Results: 3440 patients from 17 RCTs showed that Sotalol was not associated with an increased risk of all-cause mortality (RR 1.71; 95% confidence interval (CI) [0.72-4.07]; p=0.22; I² 11%) and did not increase the risk of TdP (RR 2.34; 95% CI [0.90-6.08]; p=0.08; I² 0%) (Figure 1). There was a significant reduction in recurrent AF with Sotalol (RR 0.82; 95% CI [0.76-0.90]; p<0.00001; I² 65%) but at the expense of an increase in serious adverse events leading to drug withdrawal (RR 2.12; 95% CI [1.33-3.38]; p=0.002; I² 60%). There was no difference between both groups in conversion to sinus rhythm (RR 1.23; 95% CI [0.69-2.18]; p=0.48; I² 80%), incidence of stroke (RR 0.74; 95% CI [0.18-3.09]; p=0.68; I² 0%), or MACE (RR 0.63; 95% CI [0.11-3.72]; p=0.61; I² 81%).

Conclusion: Meta-analysis of RCT data shows that Sotalol was not associated with increased risk of all-cause mortality or TdP as compared to placebo or rate control drugs. Sotalol reduced recurrent AF but at the expense of an increase in serious adverse events leading to drug withdrawal.

Table 1. Hazard ratios of DTNPV1 for ischemic stroke

<table>
<thead>
<tr>
<th></th>
<th>Non-DTNPV1 (n=1330)</th>
<th>DTNPV1 (n=459)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Definite or probable ischemic stroke</td>
<td>1286</td>
<td>50</td>
<td></td>
</tr>
<tr>
<td>Person-years</td>
<td>349,144.29</td>
<td>5,388.04</td>
<td></td>
</tr>
<tr>
<td>Incidence rate (95% CI)</td>
<td>3.68 (3.48-3.88)</td>
<td>9.28 (6.71, 11.85)</td>
<td></td>
</tr>
<tr>
<td>HR (95% CI), Model 1</td>
<td>1 (reference)</td>
<td>2.65 (2.04-3.44)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HR (95% CI), Model 2</td>
<td>1 (reference)</td>
<td>1.96 (1.50-2.56)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HR (95% CI), Model 3</td>
<td>1 (reference)</td>
<td>1.93 (1.47-2.52)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HR (95% CI), Model 4</td>
<td>1 (reference)</td>
<td>1.87 (1.44-2.45)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

* A time-varying exposure variable was used for DTNPV1 through ARIC visits 1-5
1 Per 1000 person-years
Follow-up through 2019 except for Jackson, MS participants, whose follow-up ended in 2017
Model 1 is adjusted for age, sex, and race; study center
Model 2=Model 1+additional adjustment for smoking, body mass index, systolic and diastolic blood pressure, diabetes, coronary heart disease, left ventricular hypertrophy, heart failure, use of aspirin, and use of anticoagulants
Model 3=Model 2+additional adjustment for use of other medications, including antihypertensive medications, anti-diabetic medications, and statins.
Model 4=Model 3+additional adjustment for time-dependent AF

PO-625-05

ELECTROCARDIOGRAPHIC DEEP TERMINAL NEGATIVE OF THE P WAVE IN V1 AND ISCHEMIC STROKE: THE Atherosclerosis Risk in Communities (ARIC) Study

Mingfang Li, Yuekai Ji MBBS, MS; Youmei Shen; Wendy Wang MPH; Kamakshi Lakshminarayan; Elsayed Z. Soliman MD; Minglong Chen MD, PhD, FHRS and Lin Yee Chen MD, FHRS

Background: Abnormal P-wave indices are associated with ischemic stroke independent of atrial fibrillation (AF). Deep terminal negative of the P wave in V1 (DTNPV1) is a simple ECG index that reflects underlying left atrial remodeling. However, it is unknown whether DTNPV1 is associated with ischemic stroke.

Objective: Evaluate the prospective association of DTNPV1 with ischemic stroke in the ARIC study, a community-based cohort study.

Methods: We included all participants at the baseline visit (1987-1989) and excluded those with prevalent stroke, missing covariates, and missing ECG or uninterpretable ECG due to AF. DTNPV1 was defined as the absolute depth of the terminal negative phase >100 μV (1 small box on ECG scale) in the presence of biphasic P wave in V1. The outcome was definite or probable ischemic stroke through 2019. Multivariable Cox regression with DTNPV1 as a time-dependent exposure variable was used to estimate hazard ratios (HRs) and 95% confidence intervals (CI) of DTNPV1 for incident ischemic stroke.

Results: We included 15189 participants (54 ± 6 years, 55% women, 27% Black) in this study. During a median follow-up of 27 years, 1336 cases of definite or probable ischemic stroke occurred. DTNPV1 was associated with a 2.65-fold increased risk of stroke (95% CI 2.04-3.44) after adjusting for demographics (Table 1, model 1). This association remained significant (HR 1.87, [95% CI 1.44-2.45]) after adjusting for stroke risk factors, use of aspirin and anticoagulants, and time-dependent AF.

Conclusion: DTNPV1 is significantly associated with higher risk of ischemic stroke independent of AF and stroke risk factors. Since DTNPV1 is readily discernible by the clinician at the bedside, it may be helpful to improve stroke prediction, a subject for further research.

PO-625-06

CHARACTERISTICS AND PROGNOSIS OF THE Catecholamine INDUCED QT PROLONGLONGATION SYNDROME

JEAN BAPTISTE GOURAUD MD, PhD; Jacques Mansourati MD; Nicolas Clementy MD; VINCENT PROBST; Raphael Martins MD; Frederic Sacher MD, PhD and VINCENT PROBST MD

Background: We have recently demonstrated association of unexplained sudden cardiac arrest (SCA) with inheritance of catecholamine induced QT prolongation (CIQTP). We here aim to describe incidence, characteristics and prognosis of this new syndrome in young patients with unexplained SCA or their relatives.

Methods: We reviewed the medical screening of all consecutive patients or their first-degree relatives explore from 2015 after the occurrence of a SCA before age 45. Structural heart disease or inherited arrhythmia diseases were excluded. A mental stress test was performed, as previously described, for each family members. All families with a positive mental stress were included in the study. Genetic screening was performed in at least one positive patient per family using targeted sequencing on a panel of 109 genes associated with inherited arrhythmias and cardiomyopathies.

Results: Among 456 patients screened (24 after SCA, 432 for familial screening) of 153 families, we identified 10 families (6.5%) with a catecholamine induced QT prolongation. No mutation was identified in these families. One hundred and ten patients were screened in CIQTP families. Thirty-four patients (30.9%) presented a CIQTP (mean age 42 ± 20 yo, 64.7% of women). Five (14.7%) patients presented with previous symptoms (including 4 syncope and 1 SCA). Two patients (5.9%)