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%). 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 1%).

Conclusion: Meta-analysis of RCT data shows that Sotalol was not associated with increased risk of all-cause mortality or TdP 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.

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 in V1 (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 1.20-5.44) 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.

Table 1. Hazard ratios of DTNPV1 for ischemic stroke

<table>
<thead>
<tr>
<th></th>
<th>Non-DTNPV1 (n=14,730)</th>
<th>DTNPV1*(n=459)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Definite or probable stroke (n=1336)</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>&lt;0.001</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>

DTNPV1 indicates deep terminal negative of the P wave in V1. Atherosclerosis Risk In Communities; CI, confidence interval; HR, hazard ratio.

* A time-varying exposure variable was used for DTNPV1 through ARIC visits 1-5
† 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-625-06

CHARACTERISTICS AND PROGNOSIS OF THE CATECHOLAMINE INDUCED QT PROLONGATION 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).

Objective: 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%)
were implanted with an ICD and eleven (32.3%) were treated with beta blocker therapy mainly because of QT prolongation > 500 ms after mental stress test or previous symptoms. After a 3.6 ± 1.8 years of follow up, no sudden cardiac death nor syncope occurred on beta blocker therapy except for one patient implanted with an ICD after a SCA. Under beta blocker treatment the patient was asymptomatic for 5 years. After a suddenly stop of the beta blocker treatment, the patient underwent VF. For 3 years now the patient is asymptomatic under beta blocker treatment. **Conclusion:** In our experience, CIQTP families represent 6.5% of cases of unexplained SCD and suggest systematic screening with a mental stress test for family screening after the occurrence of a SCA. Beta blocker therapy is very efficient to reduce the risk of SCA.

**PO-625-07**

**NOVEL STREAMLINED TECHNIQUE FOR LEFT ATRIAL APPENDAGE CLOSURE USING VERSACROSS LARGE ACCESS SYSTEM**  
Amin Al-Ahmad; Carola Gianni; Domenico Della Rocca; Sanghamitra Mohanty; David R. Tschopp; Rodney P. Horton and Andrea Natale

**Background:** Transseptal puncture (TSP) for left atrial (LA) access of large delivery sheaths, such as in left atrial appendage closure (LAAC), typically involves performing TSP with an 8F sheath, then “up sizing” over a wire to the device delivery sheath. This can be challenging in fibrotic or aneurysmal septa and increase the risk of air embolus.

**Objective:** Report early experience using the VersaCross Large Access (VLA) Solution (Baylis Medical) to streamline and optimize efficiency of LAAC procedures.

**Methods:** Consecutive LAAC procedures performed using the VLA system and the WATCHMAN FLX device (Boston Scientific) were retrospectively evaluated. The VLA system, consists of a 12.5F seamless sheath-dilator device, and pigtail RF wire that can be used for vascular access, TSP and WATCHMAN sheath exchange (Figure). The VersaCross pigtail RF wire (24mm) was also used to estimate the size of the left atrial appendage (LAA) and directly introduce the WATCHMAN sheath into the LAA. Contrast was injected through the WATCHMAN sheath to confirm LAA anatomy and device size. Procedural workflow efficiency was assessed in terms of the time for TSP, WATCHMAN sheath access and implant release, fluoroscopy use and procedural complications.

**Results:** A total of 12 patients underwent LAAC; 42% of patients had prior TSP for ablation. LAA morphology was 50% cauliflower and 33% chicken wing. TSP and procedure success was 100% without any intraprocedural complications. Time to TSP was 5.3 ± 3.1 mins from RF wire insertion, and 11.6 ± 3.3 min from femoral access. The 12.5F VLA device easily crossed the septum within 0.1 ± 0.3 min of TSP. Repeated passage of the seamless VLA device over the RF wire successfully dilated difficult septa (25% of cases) to confirm patency. Time to WATCHMAN release was 23.3 ± 3.4 min and overall procedure time was 26.5 ± 3.1 min. Fluoroscopy time and dose were 5.2 ± 2.3 min and 85.8 ± 42.6 mGy, respectively, with 60 ± 22.4 mL of contrast use.

**Conclusion:** The VersaCross RF system streamlined LAAC workflows, eliminating unnecessary sheath exchanges and associated risks of perforation or air embolism. The integrated 12.5F large access sheath-dilator facilitated de novo dilation of the septum for easy LA catheterization of the large bore LAAC delivery system in all patients regardless of history or septal anatomy.

**PO-625-08**

**NEW ATRIAL FIBRILLATION DURING ACUTE COVID-19 INFECTION PREDICTS DEVELOPMENT OF FUTURE CLINICAL ATRIAL FIBRILLATION**  
Justin Haloot DO; Ribesh Shrestha DO and Auroa Badin MD

**Background:** While primarily known for its impact on the respiratory system, cardiovascular complications have been well described in Coronavirus Disease-19 (COVID-19) patients. The incidence of new onset atrial fibrillation (AF) in COVID-19 hospitalized patients is reported to be as high as 12.5%. It is not clear whether this represents a reversible acute episode or if it carries a prognostic value of future clinical AF.

**Objective:** To determine the risk of developing future clinical AF in COVID-19 patients that developed new AF in the acute phase of infection.

**Methods:** We conducted a retrospective study with the global medical research network database, TriNetX. This study examined adults at least 18 years old that had COVID-19 based on ICD codes of positive SARS-CoV-2 test without previous history of AF, that developed new onset AF within the same month of the COVID-19 diagnosis. These patients were then compared to COVID-19 patients without pervious history of AF, that did not develop new onset AF. Propensity score matching was done to account for age, gender, race, ethnicity, diabetes, dyslipidemia, obesity, cardiovascular disease, pulmonary disease, neurological disease, genitourinary disease, neoplasm, cardiac medications, and cardiac procedures. We examined the risk of developing clinical AF 6 months after COVID-19.

**Results:** A total cohort size of 19,877 patients with COVID-19 that developed new onset AF in the acute phase or if it carries a prognostic value of future clinical AF were compared to 19,877 propensity matched COVID-19 patients that did not develop AF. Average age was 71.7 ± 12.9 years. 60% were male 72% were Caucasian. Approximately 60% in both cohorts had cardiovascular disease, 43% had genitourinary disease, 37% had neurological disease, 35% had dyslipidemia, 26% had diabetes, and 65% were on cardiovascular medications. We found that COVID-19 patients that developed new onset AF, without history of previous AF, were at increased risk of developing future clinical AF (OR 4.572, 95% CI 3.37 - 6.263, p < 0.001).