Conclusion: COVID-19 patients that develop new onset AF, without a history of AF, are associated with increased risk for developing future clinical AF. Further studies and more intensive monitoring are needed to determine the exact burden and risk of stroke and anticoagulation implication in this patient population.

POSTER PO-626:
Featured Posters: Clinical EP and Digital Health
Pod 13
Friday, April 29, 2022
12:30 PM - 2:30 PM

PO-626-01
PERFORMANCE AND ACCURACY OF A SMART WATCH SINGLE-LEAD ECG: A PILOT STUDY
David Harmon MD; Jennifer Dugan CRC; Rickey Carter PhD; Anthony Kashou MD; Zachi Izhak Attia MSEE, PhD and Paul A. Friedman MD, FHRS

Background: Prior studies have evaluated the accuracy of the Apple Watch single-lead ECG for the detection of atrial fibrillation and other arrhythmias. However, no study to date has analyzed the watch ECG morphology and interval characteristics compared to a standard 12 lead ECG, important in assessing patients on medications and for physician overreads.

Objective: To analyze the characteristics of single-lead ECGs collected by an Apple Watch compared to a clinical 12 lead ECG.

Methods: All Mayo Clinic ECG lab patients >18 years old were invited to participate in this study. Participants underwent a 30 second single-lead ECG recording with an Apple Watch Series 5 in a seated position within 5 minutes of 12 lead ECG (12 L). The collected ECGs were manually interpreted by physician staff. PR, QRS, QT intervals were manually calculated, and RR interval determined by computer.

Results: Seventy-four patients were included with 12 L ECG and Apple Watch ECG pairs. The cohort was 44.6% female (N=33) and 91.6% white (N=68) with an average age of 59.2 years. Six patients were in atrial fibrillation at the time of recording and six patients had pacemakers. The average differences between 12 L ECG and Apple Watch ECG intervals (in milliseconds) were: PR 23.3 ±18; QRS 18.9 ±16.3; QT 22 ±17.3; RR 70 ±70. Bland-Altman plots were created to visualize interval variation (Figure 1). Eleven patients were excluded from PR interval analysis due to absence (i.e. atrial fibrillation) or other conditions (significant AV block, sinus bradycardia, low P wave amplitude in L1). Apple Watch ECG result interpretation was complicated by 1) aberrant inflection points before and after QRS complexes, 2) attenuation of P waves, 3) disappearance of pacemaker spikes and 4) significant artifact from a patient’s tremor (Figure 2).

Conclusion: Apple Watch ECG processing can result in peri-QRS artifact, P-wave amplitude reduction, and pacemaker spike elimination. The Apple Watch single-lead ECG appeared most reliable in determining the QT interval and least reliable in determining the PR interval (Figure 1). The findings suggest that different processing or access to raw, unfiltered Apple Watch data may facilitate expert over-reads of these signals when applied for medical use.

PO-626-02
COMPARATIVE ARRYTHMIA PATTERNS OF IBRUTINIB AND NON-IBRUTINIB TYROSINE KINASE INHIBITORS
Chen Wei MD, MBA; Muhammad Fazal MD, MS; Ridhima Kapoor; Paul Cheng MD, PhD; Albert Joseph Rogers MBA, MD; Alexander Perino MD; Sanjiv M. Narayan MD, PhD, FHRS; June-Wha Rhee MD and Tina Baykaner MD, MPH

Background: Ibrutinib, a Bruton’s tyrosine kinase inhibitor (TKI), used primarily for hematologic malignancies, has been associated with increased incidence of atrial fibrillation (AF), but there is limited data on its association with ventricular arrhythmias (VAs). How the arrhythmia patterns of patients on ibrutinib compare to those on non-ibrutinib TKIs is unknown.

Objective: We sought to comprehensively analyze atrial and ventricular arrhythmia burden in patients on ibrutinib vs non-ibrutinib TKIs. We hypothesized that long term event monitors could reveal a high burden of both in patients on TKIs.

Methods: A single center retrospective analysis was conducted to identify consecutive patients who had 14 day event monitors while on TKI therapy.

Results: A total of 108 patients with hematologic malignancies were included (n=72 on ibrutinib and n=36 on non-ibrutinib TKI) (Figure A). The non-ibrutinib TKIs at the time of event monitoring were Ibrutinib (n=12), Dasatinib (n=12), Ruxolitinib (n=5), Nilotinib (n=3), Bosutinib (n=2), and Ponatinib (n=2). During ibrutinib therapy, the most common arrhythmias seen on 14-day event monitors were SVT (n=32, 44.4%), AF (n=32, 44%), and NSVT (n=31, 43%). In comparison, the non-ibrutinib TKI group had significantly lower rates of documented AF (n=7, 19%; p=0.01) and NSVT (n=8, 22%; p=0.03). The rates of non-AF SVT were not significantly different between the groups. There were non-statistically significant trends toward increased >1% PAC burden (18% (n=13) vs 8% (n=3)) and >1% PVC burden (22.2% (n=16) vs 11% (n=4)) in the ibrutinib group (Figure B). TKI therapy was held in 25% (n=18) of patients on ibrutinib vs.
In this large dataset of ambulatory cardiac monitors by cardiology and EP specialists (p = 0.002 and p=0.018).

**Conclusion:** In this large dataset of ambulatory cardiac monitors on patients treated with ibrutinib and non-ibrutinib TKIs, we report a higher prevalence of atrial and ventricular arrhythmias in those receiving ibrutinib vs non-ibrutinib TKIs, with a high incidence of treatment interruption due to arrhythmia burden. More research is needed to optimize strategies to diagnose, monitor, and manage TKI related arrhythmias.

### A

**Characteristics**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>All patients (n=189)</th>
<th>Patients on ibrutinib (n=72)</th>
<th>Patients on non-ibrutinib TKIs (n=56)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>74±12.3</td>
<td>76±9.9</td>
<td>70±15.5</td>
<td>0.005</td>
</tr>
<tr>
<td>Sex (%)</td>
<td>58%</td>
<td>57%</td>
<td>58%</td>
<td>0.809</td>
</tr>
<tr>
<td>Male</td>
<td>72 (66.7)</td>
<td>54 (75)</td>
<td>18 (30)</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>36 (33.3)</td>
<td>28 (25)</td>
<td>18 (30)</td>
<td></td>
</tr>
<tr>
<td>Body Mass Index (kg/m²)</td>
<td>25±4.7</td>
<td>23±4.0</td>
<td>25±8.6</td>
<td>0.049</td>
</tr>
<tr>
<td>Body volume index (cm³/kg)</td>
<td>34±13.0</td>
<td>34±13.0</td>
<td>35±12.0</td>
<td>0.677</td>
</tr>
<tr>
<td>EF (%)</td>
<td>58±8.4</td>
<td>58±10.9</td>
<td>60±6.9</td>
<td>0.210</td>
</tr>
</tbody>
</table>

**Coronary Medical Conditions (N, %)**

- **Cognitive Heart Failure:** 45 (24.7) vs 26 (36.1); p = 0.008
- **Vascular Disease:** 44 (23.6) vs 31 (43.1); p = 0.079
- **Hypertension:** 70 (37.9) vs 49 (68.1); p = 0.046
- **Hypertensive:** 68 (36.3) vs 46 (64.7); p = 0.573
- **Diabetes Mellitus:** 24 (13.1) vs 13 (19.6); p = 0.069
- **Coronary Artery Disease:** 39 (21.1) vs 28 (41.9); p = 0.018

**History of AF (prior to TKI therapy):**

- **Duration of AF (months):** 5 (4.9) vs 3.6 (2.2); p = 0.046

**Patients on anti-arrhythmic drug therapy (N, %):**

- **Patients on anti-arrhythmic drug therapy that was initiated after TKI treatment (N, %):** 4 (2.2) vs 1 (1.1); p = 0.741

**Care team involvement (N, %):**

- **General Cardiologist:** 78 (42.2) vs 50 (89.4); p = 0.062
- **Electrophysiologist:** 25 (23.1) vs 20 (27.8); p = 0.167

### B

**Arrhythmia Patterns: Ibrutinib vs Non-Ibrutinib TKIs**

- **Ventricular Fibrillation (VF):** 1 (0.5%) vs 0 (0.0%); p = 0.002
- **Sustained Ventricular Tachycardia (VT):** 2 (1.1%) vs 1 (1.2%); p = 0.002

**Figure:** Cardiac event monitor findings while on TKI

**PO-626-04**

**THE PERI-INFARCT “GRAY ZONE” OF MYOCARDIAL FIBROSIS IS A BETTER PREDICTOR OF VENTRICULAR ARRHYTHMIAS THAN DENSE CORE FIBROSION IN PATIENTS WITH PREVIOUS MYOCARDIAL INFARCTION**

**Pedro Lopes MD; Gonçalo Cunha MD; Pedro Freitas MD; Bruno Rocha MD; francisco albuquerque MD; Daniel Gomes MD; Mariana Paiva MD; Rita Amador MD; Daniel Jorge Matos MD; Gustavo Rodrigues MD; João Carmo MD; Maria Salomé Carvalho MD; Pedro Miguel Galvão Galvao Barata Pereira MD; FRANCISCO Moscoso COSTA MD; Pedro Carmo MD; Sara Guerreiro MD; Diogo M. Cavaco MD; João Abecasis MD; Francis B. Morgado MD; Miguel Mendes MD; António Ferreira MD and Pedro Adragao MD, PhD**

**Background:** Current sudden cardiac death (SCD) risk stratification relies heavily on left ventricular ejection fraction (LVEF), but markers to refine risk assessment are needed. Dense core fibrosis (DCF) and peri-infarct “gray zone” of myocardial fibrosis (GZF) on late gadolinium enhancement (LGE) cardiac magnetic resonance (CMR) have been proposed as arrhythmogenic substrates.

**Objective:** To determine whether DCF and GZF could predict the occurrence of ventricular arrhythmias in patients with previous myocardial infarction.

**Methods:** We performed a single centre retrospective study enrolling consecutive patients with previous myocardial infarction undergoing CMR before implantable cardioverter-defibrillator (ICD) implantation. Areas of LGE were subdivided into “core DCF” and “peri-infarct” GZF zones based on signal intensity (>5 SD, and 2-5 SD above the mean of reference myocardium, respectively). The primary endpoint was a composite of sudden arrhythmic death, appropriate ICD shock, ventricular fibrillation (VF), or sustained ventricular tachycardia (VT) as detected by the device.

**Results:** A total of 88 patients (median age 61 years [IQR 54-73], 84% male, median LVEF 30% [IQR 23-36%]) were included.

**PO-626-03**

**PRECLINICAL SAFETY OF ADKCNH2-G628S FOR POST-OP AF**

J. Kevin Donahue MD and David D. McManus MD, MSci, FHRS

**Background:** Post-operative atrial fibrillation (POAF) occurs in 30% of cardiac surgeries, increasing risk of stroke, MI, and death. We propose gene therapy with an adenovirus encoding the G628S mutation of KCNH2 (AdKCNH2-G628S) to prevent POAF. We have reported preclinical efficacy in pigs.

**Objective:** To determine safety and vector biodistribution of AdKCNH2-G628S atrial gene painting in rabbits.

**Methods:** We assessed 5 groups of animals (saline, poloxamer-saline, 1.5e10 virus particles (vp) atrial delivery, 1.5e12 vp atrial delivery, 1.5e12 vp whole heart delivery) with 3 sacrifice time points (7, 21 and 42 days) and 5 animals per gender per group at each time point, except the whole heart delivery group was only assessed with the 42 day sacrifice time point. Toxicity measures included general well-being, detailed clinical signs and vital sign assessment, body weights, serum chemistry, hematology, IL-6 and troponin, echocardiography, complete necropsy, tissue harvest and microscopic examination, vector presence in blood and tissues. A subset of animals had telemeters placed for continuous rhythm analysis. The study was compliant with 21CFR58 Good Laboratory Practice.

**Results:** All animals had findings consistent with open-chest surgery with no between-group differences in behavior, clinical or vital signs, labs, ECG or echocardiographic findings. Specifically, QTC did not differ between groups. Animals in all groups had sinus bradycardia and sinus arrhythmia before and after the surgical procedure. No ventricular tachyarrhythmias were observed. IL-6 and troponin-I increased in the early post-op period to a similar extent in all groups. Post-mortem eval was consistent with recent cardiac surgery and not different between groups except a slight mononuclear cell infiltrate in 4/10 low dose animals. Vector was found in the heart and surrounding organs. No systemic vector biodistribution was observed. No systemic pathology or toxicity was noted.

**Conclusion:** AdKCNH2-G628S atrial gene painting is safe and effective in preclinical study. These findings support a phase 1 clinical trial of AdKCNH2-G628S atrial gene painting to prevent POAF. Clinical trial design will be reviewed in the abstract presentation.