risks, and prevents longer term anatomical and electrophysiological remodeling that otherwise favor AF recurrence/persistence. Yet we are not good at recognizing the development of AF in monitored hospitalized patients - prior work at our institution showed a median 1.5 day delay from automated algorithmic detection of AF to clinical response. The purpose of this work was to investigate delays in recognition and treatment of AF in floor patients, and their consequences. We undertook a retrospective review of 225 consecutively admitted patients from 12/25/20–3/29/21 who had automated algorithmic detection of AF episodes lasting at least 5 minutes. The algorithm employs both time and non-linear domain characteristics of the time series data to ensure highly sensitive and specific detection of AF. Time from alert to clinical identification, prespecified interventions and complications (Table 1), and prior history of AF were identified in each patient’s chart. Patient age, race, sex and Area Deprivation Index were also recorded. The median duration of all AF episodes was 780 seconds. 94/225 patients had at least one AF episode that was completely clinically undetected. 78/225 patients had AF that was clinically detected prior to the first automated algorithmic alert (these patients were in units where cardiorespiratory monitoring was not subject to the AF detection algorithm, e.g. the emergency dept). 53/225 patients had initial clinical identification of AF after first automated detection, and we focused on these. Median delay from AF onset to clinical identification and intervention was 89.5 minutes. Median AF episode duration in this patient group was longer, 3960s, and most were males (70%), mean age 70.5 yrs (range 40–92). Critically, patients who experienced a delay from automated AF detection to medical intervention of >60 minutes were substantially more likely to suffer an AF-related complication (8 AF-related complications vs only 1 AF-related complication in patients who experienced a sub-60 minute delay to intervention, p=0.06). Complications included transition to an increased level of care, heart failure, and myocardial infarction. In summary, we present evidence that even in patients undergoing continuous cardiorespiratory monitoring, there are considerable and medically significant delays in the detection and management of incident AF. We are in the process of implementing an alerting system to immediately notify treating teams and clinicians of automatically detected significant AF episodes to investigate whether earlier clinical recognition leads to earlier intervention, and better outcomes.

Conflict of Interest
None

SAFETY AND OUTCOMES OF VERY HIGH-POWER SHORT-DURATION ABLATION USING 90W FOR PULMONARY VEIN ISOLATION IN PATIENTS WITH ATRIAL FIBRILLATION: A REAL WORLD OBSERVATION STUDY

Introduction Pulmonary vein isolation (PVI) ablation is the established gold standard therapy for patients with symptomatic drug refractory atrial fibrillation (AF). PVI ablation is commonly carried out with either radiofrequency (RF) thermal energy or cryoenergy. Recent advancements in RF ablation, have led to the development of the novel contact force-sensing temperature-controlled very high-power short-duration (vHPSD) RF ablation. This setting delivers 90W for up to 4 seconds with a constant irrigation flow rate of 8 ml/min. The aim of this study was to compare procedural outcomes and safety with conventional radiofrequency ablation. MethodA single centre observational study was conducted with patients who underwent first time PVI ablation from 2019 to 2021. The cohort was divided into: 1) vHPSD ablation – QMODE + via QDOT MICRO TM catheter, 2) QMODE via QDOT MICRO TM catheter and 3) Conventional power-controlled RF (PCRF) ablation via THERMOCOOL SMARRTOUCH® SF (STSF) catheter. The QMODE+ vHPSD ablation group was prospectively recruited while the QMODE and PCRF group were retrospectively collected. Primary outcomes were procedural success, PVI duration, ablation duration and incidence of perioperative adverse events. Secondary outcomes were intraprocedural morphine and midazolam requirement. Results A total of 145 patients were included in the study with 70, 30 and 45 patients in the QMODE+ (vHPSD),
Abstracts

PROARRHYTHMIC EFFECTS OF FLECAINIDE

Introduction Flecainide has an established role in the treatment of common arrhythmias. The primary mechanism of action is blockade of the cardiac sodium channel, which manifests on the surface electrocardiogram (ECG) as prolongation of the PR interval and an increase in the QRS duration. The European Society of Cardiology recommends an ECG is performed within 14 days of starting therapy to screen patients for markers of proarrhythmic side effects. Our aim was to investigate the frequency of flecainide-induced arrhythmias and the role of ECG screening in contemporary practice.

Methods We performed a retrospective study of all patients either initiated on flecainide or who underwent a dose up titration our outpatient electrophysiology service over a three-year period in. Alongside basic demographic data, we collected information on risk assessment prior to prescription including baseline ECG, imaging and ischaemia testing. We also studied the effectiveness of post-initiation ECG screening, in particular whether this was performed and whether flecainide was discontinued if: QRS duration increased by >25%, there was a new high grade atrioventricular block or bundle branch block, or a type 1 Brugada pattern. Finally, we looked at the frequency of all side effects attributed to flecainide after its prescription.

Results A total of 318 prescriptions were issued to 306 patients over the study period, of which 239 (75%) were new, 61 (19%) were an up titration and 18 (6%) were a rein titration of flecainide. The most commonest indication was atrial fibrillation (241/318; 76%). The majority of patients underwent some form of risk assessment prior to prescribing flecainide, including echocardiography (316/318; 99%) and an ECG within the past six months (307/318; 97%). 47 patients (15%) had an assessment for ischaemic heart disease prior to prescribing flecainide. The commonest indication was atrial fibrillation (241/318; 76%). The majority of patients underwent some form of risk assessment prior to prescribing flecainide, including echocardiography (316/318; 99%) and an ECG within the past six months (307/318; 97%). 47 patients (15%) had an assessment for ischaemic heart disease prior to prescribing flecainide.

Conclusion Significant QRS widening or a new bundle branch block was observed in 7% however discontinuation of flecainide occurred in a minority of cases. New high grade AV block and a Brugada pattern were rare (<1%). Whilst side effects were common, only 2% were potentially cardiac in nature and no serious harm was detected. Greater awareness of a performing a post-initiation ECG is needed and the markers for pro-arrhythmia, however most discontinuations of observational study design, these preliminary findings are promising with respect to periprocedural outcomes and safety of QMODE+. Longer term outcomes with respect to maintenance of sinus rhythm and symptomatic burden will be pertinent to assessing the overall efficacy of this novel technology.

Conflict of Interest None to declare

Abstract 95 Figure 1 Intraprocedural outcomes with respect to total PVI duration, energy application time and fluoroscopy duration

Abstract 95 Figure 2 Intraprocedural sedation requirement: IV morphine and midazolam

QMODE and PCRF, respectively. PVI was successfully attained in all patients. QMODE+ (vHPSD) demonstrated significantly reduced time required for PVI and total energy application in comparison to the QMODE and PCRF groups (69.4 ± 3.54 vs. 92.9 ± 4.86 vs. 93.6 ± 4.34 min, P<0.0001; 10.2 ± 0.484 vs. 33.9 ± 1.42 vs. 36.0 ± 1.56 min, P<0.0001, respectively). Intravenous morphine and midazolam requirement was lower in the QMODE+ (vHPSD) group compared to the QMODE and PCRF groups (10.6 ± 0.50 vs. 16.1 ± 0.935 vs. 15.3 ± 0.686 mg, P<0.0001; 4.04 ± 0.43 vs. 8.63 ± 1.07 vs. 8.58 ± 0.821 mg, P<0.0001). QMODE+ (vHPSD) observed a non-significant reduction in fluoroscopy time compared to QMODE and PCRF (12.9 ± 0.923 vs. 14.1 ± 2.34 vs. 16.1 ± 1.54 min, P=0.15). No adverse procedural events were observed in QMODE+ (vHPSD) while the QMODE group exhibited one cardiac tamponade and the PCRF group exhibited an embolic stoke and two pericardial effusions that did not require drainage.

Conclusion In this study, QMODE+ (vHPSD) demonstrated a comparable, if not superior safety profile to the other treatment arms. Procedural duration and energy application time was substantially reduced while a non-significant reduction was observed for fluoroscopy time for QMODE+. Furthermore, sedation requirement was reduced and thus potentially conveyed greater patient tolerability of the procedure when conducted with QMODE+. Notwithstanding the limitations of