

previously been reported. We conclude that higher dosages of both preparations when administered together are effective and well tolerated in JHS patients with drug refractory POTS.

161 CATHETER ABLATION OF ATRIAL FIBRILLATION ON UNINTERRUPTED WARFARIN USING STANDARD AND DUTY CYCLED RADIOFREQUENCY ENERGY: SAFE AND EFFECTIVE

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Introduction Catheter ablation (CA) for atrial fibrillation (AF) is growing exponentially. Although ablation for paroxysmal AF (PAF) is associated with shorter procedure times and less extensive left atrial ablation vs persistent AF thromboembolic complications can occur in both sub-groups. Inadequate anticoagulation leads to thrombotic complications and excessive anticoagulation can lead to bleeding risks. Many centres adopt a policy of discontinuing warfarin in the immediate run-up to the procedure, covering the procedure with unfractionated heparin and “bridging” postoperative patients with low molecular weight heparins (LMWH) back onto warfarin. We wished to determine the safety of CA for AF with a therapeutic INR using both the single transseptal approach and duty cycled radiofrequency energy (RF) with non irrigated PVAC catheters and the double transseptal puncture technique using irrigated RF catheters and either CARTO or NAVX electroanatomical mapping.

Methods A retrospective analysis of 173 patients who underwent CA for AF while taking uninterrupted warfarin. Procedural target International Normalised Ratio (INR) was 2–3 with a peri-procedural target ACT of 300–350 s. In sub therapeutic INR patients weight adjusted LMWH was used post procedure with warfarin until INR was >2. Standard technique employed was large area circumferential ablation using conventional RF energy or pulmonary vein isolation using duty cycled RF energy. Data was gathered for demographics, procedural INR, total dose of unfractionated heparin, fluoroscopy time, and type of radiofrequency energy used. Endpoints were minor bleeding, major bleeding (requiring transfusion), vascular complications, pericardial tamponade and stroke/TIA within 28 days of the procedure.

Results There were 128/173 male patients, age range between 21 and 73 years (mean 57 years). 122 underwent ablation for PAF and 51 for persistent AF. Mean procedural INR was 2.4 (range 1.7–3.9). Mean unfractionated heparin dose was 6000 units (range 1000–14500). Mean fluoroscopy time for the PVAC group was 23.4 mins (range 8.3–50.1 mins). Mean fluoroscopy time for CARTO/NAVX group was 31mins (range 14.10–58.44 mins). There were no major bleeding complications. There was 1 minor bleeding complication with a groin pseudoaneurysm. There were 2 cases of pericardial tamponade (2/173%–1.2%) both managed with percutaneous pericardial drainage. There were no stroke/TIAs.

Conclusion These data demonstrate that CA for AF by both single and double transseptal technique using both standard RF and duty cycled RF while maintaining a therapeutic INR is a safe procedure. Maintaining a therapeutic INR reduces the risk of embolic events associated with “bridging” heparin without an increase in bleeding complications. This technique is convenient for patients and avoids switching between LMWH and warfarin and ensures patient safety by maintaining therapeutic anticoagulation before, during and after the procedure.

162 THE CLINICAL MANAGEMENT OF RELATIVES OF YOUNG SUDDEN ARRHYTHMIC DEATH VICTIMS; ICDs ARE RARELY INDICATED

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Introduction Following National Service Framework guidance on the management of sudden cardiac death (SCD), regional inherited cardiac conditions clinics were established to identify and treat those at increased risk of dying from an arrhythmic condition. Studies have examined the yield of different diagnostic strategies but the outcome of management in these patients has not been reported.

Methods We present data on 193 consecutive patients (108 families) referred to a regional inherited cardiac conditions clinic because of SCD/aborted cardiac arrest of a relative. The mean age on referral was 38±17 yrs and mean duration of follow-up was 15 months (range 1 day to 56 months). All individuals underwent clinical assessment by history, examination, ECG and echo. If treadmill exercise test had not previously been performed this was undertaken. Further imaging by CMR or contrast echo was performed in those with structurally abnormal hearts or with T wave inversion in V₂/V₃. Ajmaline provocative testing was performed in those with a history and/or ECG suggestive of Brugada syndrome.

Results Of the 193 patients, 43 individuals (22%) from 36 families (33%) were diagnosed with an inheritable cause of SCD and 145 patients were clinically normal (see Abstract 162 table 1). Five patients were found to have other conditions (LV non-compaction, AVNRT, skeletal myopathy, mild AS and congenital sub-aortic membrane). Of the 43 patients diagnosed with an inheritable condition, 21 had medication commenced by the clinic (β-blockers (21), ACEi/ARB(2), Spironolactone[1]). ICDs were implanted as per HRUK guidelines, resulting in 4 patients having an ICD inserted on clinic recommendation (2 HCM, 1 DCM, 1 ARVC). To date no appropriate therapies have been administered (follow-up 8–29 months) but there was 1 inappropriate shock from a fractured lead. Three individuals had β-blockade withdrawn after negative genetic testing for an established familial mutations (2 CPVT, 1 LQT), one ICD was removed and one deactivated (both negative for CPVT). Of the 145 patients thought to be clinically normal, 85 were reassured and discharged, 13 failed to return to clinic and 47 are regular reviewed as the risk of developing an inheritable condition cannot be excluded; this includes those with family histories of HCM (7), ARVC (12), DCM (9), CPVT (5), Brugada (4) and LQT(1). To date no deaths have occurred in those diagnosed with inherited causes of SCD (follow-up mean 20, 1–52 range) or those clinically normal with ongoing review (follow-up mean 22 months, 1–56 range).

Abstract 162 Table 1

Diagnosis of patient	Numbers
Clinically normal	145
LQTS	12
Brugada	2
CPVT	5
ARVC	7
DCM	7
HCM	10

Conclusion A diagnosis of an inheritable cause of SCD was obtained in 22% of individuals and 33% families with a history of SCD/aborted cardiac arrest in a relative. The number of ICDs inserted was very small (2%) and there have been no appropriate therapies in this