The inverse link between AF and cholesterol level that distinguishes statin users from non-users has not been thoroughly evaluated. **Objective:** We investigated the TC - AF and LDL-C - AF relationships in statin users and non-users, respectively. **Methods:** From the Korean National Health Insurance Service database, we included 9,778,014 adults who underwent a health examination in 2009 and had prior AF history. The levels of TC and LDL-C at the health exam were categorized in quartile (Q) and decile (D) values of the total study population. The study population was grouped into statin users and non-users, and TC - AF and LDL-C - AF relationships were evaluated. **Results:** 867,336 (8.9%) were on statin use among the total population. Statin users showed higher TC level (208.4 ± 55.6 vs. 194.1 ± 39.5 mg/dL, p < 0.001) and LDL-C level (123.0 ± 102.2 vs. 121.3 ± 226.3, p = 0.001) compared to non-users. The inverse associations of TC - AF and LDL-C - AF were observed; the higher levels of TC and LDL-C were associated with the lower risk of AF. The hazard ratios (HR) and 95% confidence intervals (CI) were 0.797 (0.786 - 0.809) for the highest quartile of TC (Q4, TC ≥ 218) and 0.832 (0.82 - 0.843) for the highest quartile of LDL-C (Q4, LDL-C ≥ 135) when adjusted by age, sex, lifestyle behaviors, comorbidities, and low-income status. Statin users exhibited higher AF incidence rate than non-statin users, but the association in statin users generally tracked that seen among non-statin users demonstrating similar HR in Q4 of TC [0.812 (0.790 - 0.835) for statin users and 0.812 (0.798 - 0.826) for non-statin users] and LDL-C [0.842 (0.819 - 0.865) for statin users and 0.849 (0.835 - 0.863) for non-statin users]. **Conclusion:** The paradoxical relationship between lipid levels (TC and LDL-C) and incident AF remained consistent both in statin users and non-users. Further research is required to investigate an underlying mechanism in the cholesterol paradox of AF not disturbed by the pleiotropic effect of statin.

PO-627-04

**THREE YEAR COST SAVINGS OF AN ACUTE ATRIAL FIBRILLATION PATHWAY**

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**Background:** Atrial fibrillation (AF) is the most common cardiac arrhythmia and the treatment places a gross burden on the US healthcare system, estimated at $6.65 billion annually with 44% driven by direct inpatient cost and 29% indirect inpatient cost. Outpatient pathways offering timely access to quality care can have a substantial impact on reducing cost of AF care and reduce dependence upon emergency department (ED) care. **Objective:** We sought to quantify cost savings through a novel AF outpatient treatment protocol. **Methods:** The Atrial Fibrillation Clinic at OhioHealth’s Riverside Methodist Hospital was initiated in September 2018 with a focus on identifying low risk AF patients and expediting access for evaluation and treatment. We reviewed the total number of ED presentations, hospitalizations, and visits in our AF pathway for acute AF over a 3-year period. The hospitalization length of stay, time from referral to appointment, and outcomes of those visits were established. Acute visits in our clinic were defined as a potentially saved ED evaluation. **Results:** Over 36 months, 2,386 patients presented with atrial fibrillation to the ED resulting in 2,074 inpatient or observation stays with a mean length of stay of 4 days. Over that same period using the acute AF pathway, 1,991 patients were referred to the AF Clinic for evaluation with 169 directly from the ED and 1,822 from outpatient care sites which avoided the use of the ED altogether. We estimate $5,000 in variable cost savings per case from the reduction in use of emergency services and of potential hospitalization resulting in $9.9 million in cost savings over 3 years. The mean time to AF Clinic visit from acute referral was 1.7 business days. Same day procedures were offered to indicated patients which included 540 cardioversions, 107 TEEs, 13 atrial flutter ablations, and 60 CCTAs. **Conclusion:** Access to timely evaluation and treatment of AF using a dedicated pathway can significantly reduce the cost of care by avoiding ED visits and potential hospitalization. Opportunities for widespread patient and clinician education on how and where to seek care for acute episodes of AF should be explored.

PO-628

**CARDIAC RESYNCHRONISATION THERAPY ACUTELY ALTERS METABOLIC SUBSTRATE UPTAKE, CORRELATING WITH IMPROVEMENTS IN CARDIAC SYSTOLIC FUNCTION**

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**Background:** The failing heart is thought to be metabolically inflexible, and oxygen limited, shifting from free fatty acid (FFA) towards glucose oxidation. CRT acutely improves cardiac hemodynamics in patients with severe heart failure and a LBBB, however whether CRT alters metabolic substrate usage is unknown. **Objective:** To acutely assess cardiac work, efficiency, and metabolic substrate uptake in response to CRT. **Methods:** Participants with non-ischaemic cardiomyopathy were started on an insulin/dextrose infusion prior to CRT implant. During implant, measurements of cardiac work (using a pressure-volume loop catheter), coronary flow (using a doppler guide wire) and paired arterio-venous blood samples (from the left main stem and coronary sinus) were obtained with and without CRT at rest and during stress, pacing at 65% of predicted
maximum heart rate. All measurements were repeated on a FFA infusion.

Results: Eleven participants were recruited (6 male, median age 63 [IQR 60-74]). Measures of cardiac systolic performance (cardiac work and dP/dtmax) were significantly improved by CRT at rest and stress on both infusions, without an increase in myocardial oxygen demand, resulting in improvement in cardiac efficiency (insulin/dextrose at rest: 7.9%, p = 0.02; stress: 67%, p = 0.03; FFA at rest: 31%, p = 0.02; stress: 57%, p = 0.09). On insulin/dextrose, CRT at rest increased cardiac FFA uptake (14 [4.9-41.6] vs 1.8 [-6-15.4] μmol/min, p = 0.02), which positively correlated with improvement in LVEF (R = 0.86, p = 0.02). When FFA uptake was already increased during stress, CRT increased lactate uptake (31.9 [19.3-57.5] vs 23.5 [-2.7-31.2] μmol/min, p = 0.02). When FFA uptake was maximised on a FFA infusion, CRT increased ketone uptake both at rest and during stress, which positively correlated with improvement in cardiac work (R = 0.55, p = 0.04).

Conclusion: CRT improves cardiac efficiency and reverses the metabolic phenotype of heart failure towards a more physiological picture of FFA uptake. The increase in FFA uptake correlates with improved systolic function. When FFA uptake is already maximised, CRT increases lactate or ketone uptake. Metabolic flexibility is therefore retained.

PO-628-02

PERSONALIZED PACING (MYPACE): A NEW PARADIGM FOR PATIENTS WITH HEART FAILURE WITH PRESERVED EJECTION FRACTION

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Background: Pacemaker patients with preclinical or overt heart failure with preserved ejection fraction (HFpEF) may benefit from a backup heart rate (HR) that is higher than the standard 60 beats per minute (bpm) setting.

Objective: Assess the effects of a personalized backup HR (myPACE group, based on a HR algorithm) compared with 60bpm (control group).

Methods: In this double blind randomized controlled study, pacemaker patients with HFpEF and either atrial pacing alone or conduction system or biventricular pacing were assigned to the myPACE or control group for 1 year. The primary outcome was changes in Minnesota Living with Heart Failure Questionnaire (MLHOFQ) scores (range 0-125, lower is better). Secondary outcomes were changes in N-terminal brain natriuretic peptide (NTproBNP), pacemaker-detected activity levels, pacemaker-detected atrial fibrillation (AF) burden, and adverse clinical events (composite of death, stroke, AF, and HF events).

Results: The myPACE (n=50; mean ± standard deviation age 76 ± 12 years, 47% female) and control groups (n=57, mean age 75 ± 9 years, 48% female) were similar at baseline. The mean HR in the myPACE group was 77 ± 50pm. Compared with controls, MLHOFQ scores improved in the myPACE group over 1 year as shown in the Table (p=0.001). Similarly, NTproBNP levels and patient activity levels improved in the myPACE group (both p<0.02). The myPACE group had 6 adverse events in 6 patients and the control group had 19 adverse events in 14 patients.

Conclusion: Among pacemaker patients with preclinical or overt HFpEF a moderately increased, personalized backup HR setting improves quality of life, NTproBNP, and activity levels compared with the standard 60bpm setting, without an excess of adverse effects.

Table. Between-group differences in the myPACE study

<table>
<thead>
<tr>
<th>Primary outcomes</th>
<th>myPACE group</th>
<th>Control group</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline scores, mean ± SD</td>
<td>58.6 ± 13.9</td>
<td>59.0 ± 14.3</td>
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<tr>
<td>1 month scores, mean ± SD</td>
<td>52.7 ± 13.6</td>
<td>53.3 ± 13.3</td>
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<tr>
<td>1 year scores, mean ± SD</td>
<td>51.4 ± 13.1</td>
<td>51.7 ± 13.2</td>
<td>0.92</td>
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</table>

<table>
<thead>
<tr>
<th>Secondary outcomes</th>
<th>myPACE group</th>
<th>Control group</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>NTproBNP (pg/ml) median (IQR)</td>
<td>12.2 (7.2-21.2)</td>
<td>12.2 (7.2-21.2)</td>
<td>0.92</td>
</tr>
<tr>
<td>NTproBNP (pg/ml) from baseline</td>
<td>1.8 (0.9-3.7)</td>
<td>1.8 (0.9-3.7)</td>
<td>0.92</td>
</tr>
</tbody>
</table>

PO-628-03

ASSOCIATION OF MINERALOCORTICOID RECEPTOR ANTAGONIST USE WITH MORTALITY AND VENTRICULAR ARRHYTHMIA RISK IN PATIENTS WITH IMPLANTABLE CARDBIOVERTER-DEFIBRILLATORS

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Background: Heart failure (HF) patients with implantable devices have improved outcomes on optimal Guideline-Directed Medical Therapy (GDMT), yet few studies have evaluated the impact of mineralocorticoid receptor antagonists (MRA) on mortality and arrhythmia risk in patients who may be unable to tolerate optimal GDMT.

Objective: To assess the impact of MRA use on the risk of all-cause mortality and ventricular tachyarrhythmia (VTA) in HF patients with an ICD or CRT-D who were not concomitantly treated with both beta blockers (BB) and ACE-inhibitors/angiotensin receptor blockers (ACE/ARB) in 4 MADIT (Multicenter Automatic Defibrillator Implantation Trial; MADIT-II, MADIT-III, MADIT-RIT, MADIT-RISK) and Lanrozaline in High-Risk Patients with ICD (RAID) clinical trials.

Methods: We included 5504 patients enrolled in 5 landmark ICD clinical trials. Patients were categorized by MRA use in addition to baseline BB and/or ACE/ARB use into subgroups of those who were prescribed both BB and ACE/ARB (2-drug GDMT) and those who were prescribed only one of the two drugs (1-drug GDMT) at enrollment. Cox proportional hazards regression models and Fine and Gray regression models, stratified by studies, were performed to assess the association between time-dependent MRA use and risks of all-cause mortality and VTA. VTA was defined as any treated or monitored sustained ventricular tachycardia (>170 bpm) or ventricular fibrillation.

Results: Among 5504 study patients, 4392 (80%) were prescribed 2-drug GDMT and 1016 (18%) were prescribed 1-drug GDMT. MRA was prescribed to 1656 (30%) patients. Multivariate analysis demonstrated that MRA use was associated with a 52% reduction in mortality risk when prescribed in patients on 1-drug GDMT (HR=0.48 [95% CI: 0.27-0.86], p=0.014). The mortality benefit of MRA therapy was attenuated among patients on 2-drug GDMT (HR=1.05 [95% CI: 0.82-1.34], p<0.7) (Figure 1). Further, MRA use was associated with a 36% reduction in VTA risk in patients on 1-drug GDMT (HR=0.64 [95% CI: 0.40-1.01], p=0.05), while among patients on 2-drug GDMT the association of MRA use with VTA risk was also attenuated (HR=1.09 [95% CI: 0.93-1.29], p=0.28) (Figure 2).

Conclusion: In HF patients with an ICD or CRT-D who are unable to tolerate 2-drug GDMT, MRA use is associated with a reduction in the risk of mortality and VTA.