endothelial function was seen when combination therapy was compared to statin therapy alone.
Conflict of Interest None

133 A FOLLOW UP OF LIPID PROFILE ASSESSMENT IN PATIENTS WITH ST ELEVATION MYOCARDIAL INFARCTION TREATED WITH PERCUTANEOUS CORONARY INTERVENTION (PCI) - ARE WE MISSING PATIENTS WITH FAMILIAL HYPERCHOLESTEROLAEMIA?

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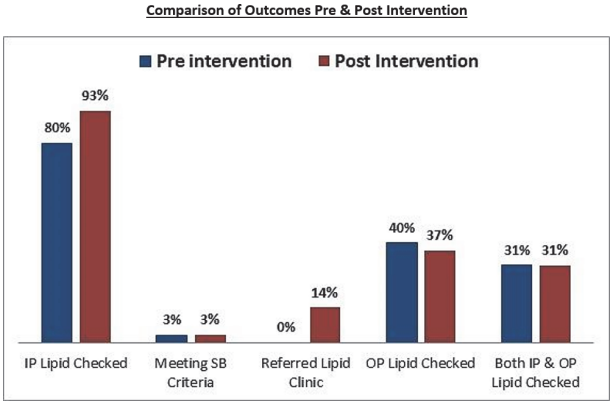
Background Familial hypercholesterolaemia (FH) is an important and under-diagnosed cause of premature cardiovascular disease. It is recommended that all patients with suspected myocardial infarction should have their lipid profile measured on admission.

The advantages of statins have been indisputably demonstrated in secondary prevention and the greatest benefit has been established with early intensive therapy following acute coronary syndrome.
Aims and Methods The main objective was to improve assessment of lipid profile in the acute setting in all patients admitted with ST-elevation myocardial infarction (STEMI). Through this, there was capacity to aid in recognition and diagnosis of familial hypercholesterolaemia (FH).

A retrospective assessment of all patients referred via the primary percutaneous intervention (PCI) pathway to the Trent Cardiac Centre between April 2016 and April 2017. We evaluated whether a random in-patient lipid profile was obtained, if patients met biochemical Simon Broome criteria for FH (total cholesterol >7.5 mmol/L +/- low-density lipoprotein cholesterol >4.9 mmol/L); were they were referred to a Lipid Clinic; was a statin prescribed and followed up was also investigated. We then implemented several changes throughout the cardiology department. These included the development of an electronic blood request order set including lipid profile for primary PCI patients. We also designed a new clerking proforma reminding clinicians to check the lipid profile. Information posters were distributed throughout the department and educational departmental sessions were held. A re-assessment was then performed of all patients between April 2018 and April 2019.



Abstract 133 Figure 1

Results The original data showed that from the 383 patients referred, 52 did not meet inclusion criteria. Of the remaining 331 patients, 67 (20%) patients did not have lipid profile checked as an in-patient. Of the 264 patients who had a lipid profile checked, 8 (3%) met biochemical Simon Broome criteria and 0 were referred to lipid clinic.

Following the intervention, the re-audit showed that from the included 284 patients, 20 (7%) patients did not have a lipid profile checked as an in-patient. Seven (3%) of the 264 patients who had an in-patient lipid profile met biochemical Simon Broome criteria; 1 (14%) was referred to lipid clinic.
Conclusion and Interpretation These results demonstrate an improvement in assessment of lipid profile in patients admitted with STEMI from 80% to 93%. There was also advancement in referral of patients to specialist lipid clinic from 0% to 14%.

It is estimated that less than 10% of the FH population in the UK have been diagnosed. Greater efforts must be made to not only perform lipid profiles in patients admitted with STEMI, but also to ensure that these results are checked and appropriate clinical decisions are made. These simple changes could potentially have hugely beneficial clinical and financial implications.
Conflict of Interest None

134 UTILITY OF 24 HOUR AMBULATORY BLOOD PRESSURE MONITORING (ABPM) IN PATIENTS WITH ORTHOSTATIC HYPOTENSION (OH) SEEN AT A SYNCOPE CLINIC

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Background Orthostatic hypotension (OH) is a disabling condition resulting from a sustained reduction in blood pressure (>20 systolic or 10 diastolic) within 3 minutes of standing. It is a common cause of syncope and is a marker of increased risk of mortality and of cardiovascular disease. OH is often secondary to medication. Patients with concurrent hypertension experiencing syncope present a complex management dilemma where a balance must be established between symptom burden and risk of cardiovascular disease.

The current European Society Cardiology (ESC) syncope guidance suggests ABPM should be used in patients with 'autonomic failure' to assess nocturnal hypertension or drug-induced hypotension. Could this be improved with further explicit criteria on which patients should be monitored and interpretation of results?
Purpose The aim of this study is to review the use of 24 hour ABPM in OH within a tertiary referral syncope clinic.
Methods A retrospective analysis was performed electronically for patients with a final diagnosis of OH seen in the syncope clinic between March 2017 and May 2019. We collected data on comorbidities, medication history, physical mobility, clinic blood pressure measurements, 24 hour ABPM results (if performed) and medication changes. Comparisons were made between patients who had ABPM and those who did not. Statistics were calculated using Fisher's Exact Test (2 tailed).
Results 119 patients had a final diagnosis of OH in the study period. 45 had ABPM, 74 did not. The ABPM group had a significantly higher proportion of diagnosed hypertension at 51.1% vs 23% (p=0.0025). A similar proportion of patients

in both groups had medication changed however the ABPM group were significantly more likely to have antihypertensive therapy added at 19.4% vs 1.8% (p=0.0053). Discussion: In a secondary care syncope clinic ABPM is more likely to be performed in patients with a history of hypertension. Despite OH often being due to medication, the need for adequate BP control is important in reducing risk of cardiovascular morbidity. Current ESC guidance targets BP for those aged 65 and over to be under 139/79 if tolerated. In symptomatic OH patients it is crucial to establish accurate blood pressure measurements in order to assess need for additional therapy. This can be provided by a 24 hour ABPM. Management of these patients must balance their symptoms with their comorbidities and target blood pressure control.

Conclusion Using 24 hour ABPM in OH patients can aid clinical decision making in the sub-group with hypertension to guide the need for alteration/ addition of antihypertensive therapy.

Conflict of Interest nil

Young Investigators Award

A THE ENDOTHELIUM AS A PARACRINE MODULATOR OF ADIPOSE FUNCTION: A ROLE FOR ENDOTHELIAL IGF-1R IN THE SETTING OF NUTRITIONAL OBESITY

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In obesity the relationship between white adipose tissue expansion and neovascularisation becomes uncoupled leading to inadequate perfusion of adipose tissue. Under these circumstances the secretory profile of adipocytes becomes unfavourable and pro-atherosclerotic.

We hypothesised that reducing endothelial insulin like growth factor 1 receptor (IGF-1R) expression affects adipose tissue remodelling as a result of communication between endothelial cells and adipocytes.

To study the effect of endothelial IGF-1R deficiency, we developed a mouse with inducible endothelial specific IGF-1R deficiency (ECIGF-1R^{KD}). In the context of diet induced obesity, ECIGF-1R^{KD} mice were more insulin sensitive and had increased energy expenditure compared to littermate controls. ECIGF-1R^{KD} mice also had favourable changes specific to the white adipose tissue, including; increased uncoupling protein-1 and vascular endothelial growth factor expression, enhanced endothelial sprouting and greater vascularisation.

The mechanisms underpinning the specific effect of endothelial specific IGF-1R deficiency on white adipose tissue were then explored in more detail. Lineage tracing experiments eliminated the possibility that ECIGF-1R^{KD} endothelial cells were directly differentiating into brown/brite adipocytes. *In vitro* treatment of primary human white adipocytes with conditioned media from isolated ECIGF-1R^{KD} endothelial cells revealed an altered secretome which caused browning of human white adipocytes in culture.

The favourable metabolic profile seen in ECIGF-1R^{KD} mice are the result of an altered endothelial secretome. The endothelial secretome, its production in the context of IGF-1R depletion, and its action on white adipose tissue provide a potential novel therapeutic strategy to combat the negative metabolic consequences of diet-induced obesity.

B OXIDIZED PKARIα PROTECTS AGAINST ISCHEMIA-REPERFUSION INJURY BY INHIBITING LYSOSOMAL-TRIGGERED CALCIUM RELEASE

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Reperfusion-induced calcium overload profoundly affects the extent of myocardial injury following ischemia, impacting long-term morbidity and mortality. Reactive oxygen species play a crucial role in shaping the amplitude and spatiotemporal patterns of the intracellular calcium signal, but the mechanism governing this interplay remain unclear. Here we show that, in vivo, myocardial ischemia and reperfusion (I/R) potently induce formation of an intermolecular-disulfide within the type I regulatory subunits of protein kinase A (PKARIα), both in mice and in humans. This conformation does not increase intrinsic PKA catalytic activity, but rather promotes AKAP-mediated subcellular compartmentalization of PKARIα to the lysosome, where it inhibits calcium release from two-pore channels and prevents global calcium release from nearby ryanodine receptors. This regulatory mechanism is shown to be crucial for limiting I/R-induced cell death, as 'redox dead' Cys17Ser PKARIα knock-in mice, which are incapable of undergoing RIα disulfide formation, display substantially larger infarct sizes with concomitant reductions in left ventricular contractile recovery, both of which are prevented by inhibition of lysosomal calcium release at the time of reperfusion. These findings reveal a hitherto unknown role for PKARIα, in its disulfide-activated state, to regulate calcium homeostasis and, in this way, potently protect the myocardium from post-ischemic injury.

C IN-VIVO GRAFTING OF LARGE ENGINEERED HEART TISSUE PATCHES FOR CARDIAC REPAIR

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Introduction Engineered heart tissue (EHT) strategies, by combining cells within a hydrogel matrix may overcome the limitations of intracoronary/myocardial cell delivery routes. EHTs regenerate heart muscle in small animal models but data