embolism and migraine. We aimed to report real-world experience and outcomes for all consecutive patients that had PFO closure in our hospital between March 2009 and October 2019.

Methods We retrospectively analysed baseline clinical characteristics, indications for PFO closure, procedural characteristics and long-term clinical follow-up using our dedicated hospital database and Northern

Ireland Electronic Care Record.

Results PFO closure was performed in 133 patients between March 2009 and October 2019. 59 (44%) of cases were performed between 2009-2016 with 74 (56%) cases performed between 2017-2019, coinciding with the publication of supporting randomized control trials. The mean patient age was 43 ±15 years and 69 (52%) patients were female. 16 (12.1%) of patients had a history of systemic hypertension, 4 (3%) diabetes mellitus and 35 (26%) had a smoking history. Only one patient had a thrombophilia diagnosis.

Cerebrovascular events including ischaemic stroke and TIA's were the leading indication for PFO closure in 123 (92.5%) cases. Systemic embolism, platypnea-orthodeoxia syndrome and decompressive illness were the indications in 4 (3%), 2 (1.5%) and 1(0.75%) case(s), respectively. 'Other' indications made up the remaining 3 patients.

The majority of procedures were performed under general anaesthetic (GA) in 129 (97%) cases. All cases were performed using trans-oesophageal echocardiography guidance. The mean procedure time was 38 ± 23minutes and the mean size of percutaneous device used was 25mm. Gore (52%) and Amplatzer (35%) septal occluders were the most commonly used devices.

There were no procedural deaths. Cardiac tamponade, major vascular injury, pulmonary embolism and/or device embolism did not occur in any patient. Only one patient had a new arrhythmia (atrial fibrillation (AF)) during the periprocedural period. The median length of stay was 1day.

Antithrombotic data at discharge was available for 129 (97%) patients. The main antithrombotic strategy adopted was dual antiplatelets in 112 (87%) cases, single antiplatelet in 10 (8%) cases and oral anticoagulation +/- a single antiplatelet made up the remainder of cases, respectively. No patients were readmitted to hospital for bleeding events on interrogation of NIECR.

The median follow-up duration after PFO closure was 31 months (range 2-1439months). 3 patients suffered a recurrent neurological event during follow-up, giving an event rate of 0.6/100 patient-years (PY). Infective endocarditis was not observed for any patients. 5 (3.8%) patients had a diagnosis of new AF or atrial flutter during follow-up, all of which occurred within three months of the procedure.

3 patients (2.3%) died during follow-up (median age 56 years (20-75years)) but all of these deaths were non-cardiac in nature.

Conclusions PFO closure was performed safely in our hospital with a very low rate of procedural complications. New arrhythmias and cerebrovascular events occurred in a low proportion of the population. Our real-world outcomes in combination with the previously published major randomized control trials supports the continued application of device-led PFO closure in patients with cryptogenic ischaemic stroke, TIA and/or systemic embolism.

Conflict of Interest none

18 DEVELOPING A WIRE-INJURY MODEL OF CALCIFIC AORTIC STENOSIS

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Calcific aortic stenosis (CAS) is the most common valve disease in the Western world and has no effective pharmaceutical treatment options. Stenosis can be caused by a combination of mechanical injury, inflammation, fibrosis and calcification, which eventually leads to left ventricular hypertrophy and heart failure. Males are at greater risk of developing aortic calcification and androgens are a risk factor in this condition. Elucidating the mechanisms underlying male predisposition to aortic stenosis is hampered by the lack of appropriate animal models; particularly valve-injury models which develop stenosis and calcification. This study describes introduction of a murine model for investigation of CAS in male and female mice. Damage was induced in the aortic valve of adult, male and female C57BL/6J mice by inserting a guidewire into the left ventricle under ultrasound guidance and rubbing the valve by rotating the guidewire twenty times. Pilot investigations demonstrated low mortality and weight loss (less than 15% of pre-surgery weight) but no significant changes in aortic or cardiac function (measured by ultrasound) following surgery. HE staining demonstrated variable thickening of valve cusps (30-140 μM). Cusps displayed fibrosis and stained positive for inflammatory cells (Mac2). No calcification (as determined by alizarin red staining) was observed. These results suggest that wire injury is producing mild damage and non-calcific remodelling in the aortic valve, indicating that greater damage is required to produce haemodynamic changes and aortic stenosis with calcification. Successful development of this model will provide a valuable tool for clarifying the mechanisms that predispose males to CAS.

Conflict of Interest none

Acute Coronary Syndromes & Interventional Cardiology

19 THE MECHANISM OF SUPPLY-DEMAND IMBALANCE AND CLINICAL OUTCOMES IN PATIENTS WITH TYPE 2 MYOCARDIAL INFARCTION

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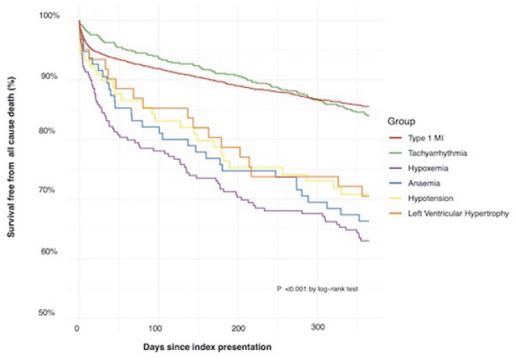
Background Type 2 myocardial infarction is common and associated with substantial risk of adverse clinical outcomes, worse than type 1 myocardial infarction, with as few as 30% of patients still alive at five years. However, this broad diagnostic term encompasses multiple mechanisms of supply-demand imbalance, which may be associated with different risks of adverse outcomes.

Purpose We aimed to assess the prevalence and clinical outcomes of different mechanisms of supply-demand imbalance



Type 1 MI	4981	4576	4432	4322
Tachyarrhythmia	616	577	559	534
Hypoxemia	219	171	154	148
Anaemia	95	78	71	66
Hypotension	89	74	67	66
Left ventricular hypertrophy	61	52	48	45

Abstract 19 Figure 1

related to survival in the High-STEACS (High-Sensitivity Troponin in the Evaluation of patients with Acute Coronary Syndrome) randomised controlled trial.

Methods The High-STEACS trial was a stepped wedge cluster randomised controlled trial in ten hospitals across Scotland, including 48,282 consecutive patients with suspected acute coronary syndrome. The diagnosis was adjudicated according to the Fourth Universal Definition of Myocardial Infarction. In patients with type 2 myocardial infarction, we prospectively adjudicated the cause for supply demand imbalance. Linkage of electronic healthcare records was used to track investigation, treatments and clinical outcomes. We used the Kaplan-Meier method, the log rank test and cox regression models adjusted for age, sex, renal function and co-morbidities to evaluate the risk of future all-cause mortality between categories.

Results We identified 1,121 patients with type 2 myocardial infarction (age 74¬ ± 14, 55% female). At one year, death from any cause occurred in 23% (258/1,121) of patients. The most common reason for supply-demand imbalance was tachyarrhythmia in 55% (616/1,121), followed by hypoxaemia in 20% (219/1,121) of patients. Tachyarrhythmia was associated with reduced future risk of all-cause mortality (adjusted HR 0.69, 95%CI 0.43-1.09), similar to those with type 1 myocardial infarction. Comparatively, patients with hypoxaemia appeared at highest risk (adjusted HR 1.75, 95%CI 1.09-2.80).

Conclusion The mechanism of myocardial oxygen supply-demand imbalance is associated with future prognosis, and should be considered when risk stratifying patients with type 2 myocardial infarction.

Conflict of Interest No conflict of interest

20 INVESTIGATING SODIUM-GLUCOSE CO-TRANSPORTER 1 (SGLT1) IN MYOCARDIUM AND ITS ROLE IN HYPERGLYCEMIA ISCHAEMIA-REPERFUSION INJURY

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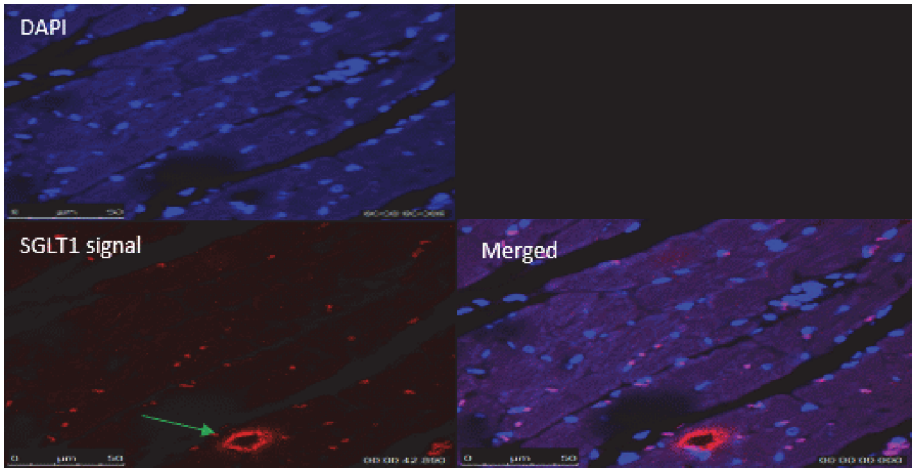
Introduction Hyperglycaemia is a common finding in diabetic and non-diabetic patients presenting with ACS, and is a powerful predictor of prognosis and mortality. The role of hyperglycaemia in ischemia-reperfusion injury (IRI) is not fully understood, and whether the Sodium Glucose co-Transporter 1 (SGLT1) plays a role in infarct augmentation, before and/or after reperfusion, remains to be elucidated. However, diabetes clinical trials have shown SGLT inhibition improves cardiovascular outcomes, yet the mechanism is not fully understood.

Purpose (1) Characterise the expression of SGLT1 in the myocardium, (2) determine the role of high glucose during IRI, (3) whether SGLT1 is involved in a glucotoxicity injury during IRI, and (4) whether inhibiting SGLT1 with an SGLT inhibitor may reduce infarct size.

Methods RT-PCR and in-situ hybridization (RNAScope) techniques were used to detect SGLT1 mRNA expression in Sprague-Dawley whole myocardium and isolated primary cardiomyocytes. An Ex-vivo Langendorff ischemia-reperfusion perfusion model was used to study the effect of high glucose (22mmol) on the myocardium at reperfusion compared to normoglycaemia (11mmol). The mixed SGLT1&2 inhibitor, Phlorizin was introduced following ischaemia, at reperfusion and its effect on infarct size measured using triphenyltetrazolium chloride (TTC) staining.



Abstract 20 Figure 1