Method Between August 2011 and June 2014, 235 patients underwent MVP, of which 71 (30.2%) had a MI procedure performed through a 5–6 cm right anterior minithoracotomy. We adapted the Composite Physical Function questionnaire to retrospectively assess post-operative quality of life in 3 domains (scores were converted into a 0–100 scale) — Recovery Time (higher score indicated longer recovery time); Pain (higher score indicated increased pain) and Treatment Satisfaction (higher score indicated improved satisfaction). The scores were risk-adjusted using pre-operative characteristics (age, gender, urgency, Logistic EuroSCORE). Data are reported as median (interquartile range).

Results The response rate was 70.6% (n = 166) of which 51 (30.7%) underwent MI repair. Comparing St to MI, 14.8% vs 35.3% resumed 'normal activities' within 6 weeks of their operation (p = 0.003). The risk-adjusted Recovery Time domain results for St vs MI were 54.4 (44.4,69.6) vs. 44.9 (26.7, 58.0), p < 0.001; likewise, the risk-adjusted Pain domain results were 31.8 (16.9, 50.0) vs. 21.9 (12.4, 32.8), p = 0.03; and the risk-adjusted Treatment Satisfaction domain results were 98.1 (83.9, 99.9) vs. 98.3 (88.9, 100.0), p = 0.25. Comparing St to MI, 53.0% vs. 78.4% reported that they were 'Very Satisfied' with the appearance of their scar (p = 0.002).

Conclusion Minimally invasive mitral valve repair speeds recovery, reduces pain and improves cosmesis compared to a sternotomy approach. Both approaches score highly on patient satisfaction.

157 PREVALENCE OF AORTIC VALVE ABNORMALITIES IN THE ELDERLY: A SCREENING STUDY USING HAND-HELD ULTRASOUND SCANNI

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Background Degenerative aortic stenosis (AS) is increasingly recognised and treated with valve replacement in the elderly. The advent of percutaneous aortic valve intervention (TAVI) has opened opportunities for treatment in patients who would have been turned down for surgery just a few years ago, with a commensurate increase in health care-associated costs. However, there is a relative lack of data regarding the prevalence of AS in this very group of older patients, which can make planning and delivery of a TAVI service difficult.

Aim of the study

We set out to investigate the prevalence of aortic valve abnormalities by performing echocardiography with a hand-held scanner in elderly patients attending a large GP surgery for non-cardiac reasons. We hypothesised that a strategy of echo screening for AV abnormalities in this population would have a high yield in detecting clinically relevant pathology.

Methods One hundred consecutive patients aged >75years old who had no history of a heart murmur, myocardial infarction or heart failure had a detailed echocardiographic exam using a hand-held scanner without spectral Doppler capabilities. Cardiac risk factors were documented. We classified aortic valves as normal, aortic sclerosis (thickened leaflets without restriction) and aortic stenosis (thickened, restricted leaflets). Qualitative LV function assessment and colour Doppler assessment of aortic and mitral regurgitation were also obtained.

Results The mean age was 78.3 (8.7) years. The quality of the images was good in 74 studies, moderate in 20 and poor in 6. We found 48 normal valves, 28 cases of aortic sclerosis and 24 of aortic stenosis (of which 4 moderate and 1 severe). Aortic regurgitation was present in 35 cases (30 mild, 5 moderate), and LVEF was normal in 92 subjects, mildly impaired in 6 and moderately impaired in 2. There were 2 cases of severe MR and 4 of moderate MR, all organic. Neither cardiac risk factors nor mitral annular calcification were associated with aortic sclerosis or stenosis.

Conclusions Our preliminary data suggest that a strategy of echocardiographic screening in patients >75years of age identifies a significant number of abnormalities that require formal cardiology follow-up or work up for intervention, including a 5% prevalence of moderate or severe AS, and 6% prevalence of moderate or severe MR.

158 NT-PRO BNP AND SURVIVAL IN AORTIC STENOSIS

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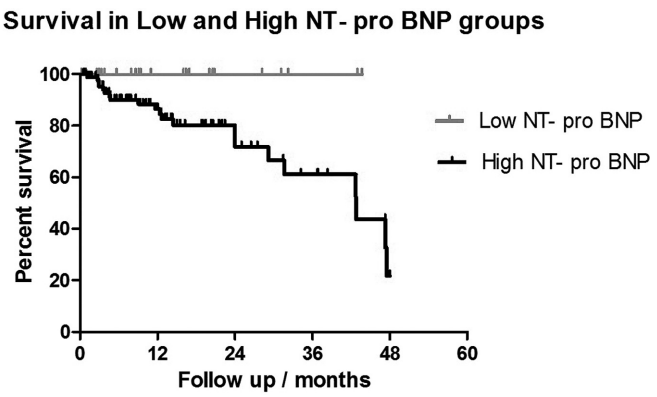
Introduction Elevated NT-pro BNP is associated with worse outcome in patients with cardiomyopathy.

Hypothesis We hypothesized that a mild elevation in NT-pro BNP may be associated with a worse outcome in patients with Aortic Stenosis (AS).

Methods Between 2011 and 2015, consecutive consenting patients with moderate (1.0–1.5 cm^2) or severe (<1 cm^2) AS considered for aortic valve intervention (either for severe AS or moderate AS with coronary disease) who had a cardiovascular magnetic resonance (CMR) scan undertaken for anatomy and function and also had blood stored for biomarker analysis were included. NT-pro BNP was measured using one-step immunoassay sandwich method with a final fluorescent detection (ELFA). Survival was obtained from hospital notes, electronic records and the National Strategic Office.

Results 112 patients (76 ± 10 years, 78 male) were included. The group was dichotomised according to NT-pro BNP value, into normal NT-pro BNP value group (values from 0–300 pg/ml, 23 patients) and high group (>300 pg/ml, 89 patients). End point was all cause mortality. At a median of 12 months follow-up, 21 patients had died, all from the high NT-pro BNP group. The high NT-pro BNP group had significantly



Abstract 158 Figure 1 Survival in low and high NT - Pro BNP groups

worse prognosis hazard ratio=3.6 (95% CI=1.24–10.44, p = 0.00184). Although the NT-pro BNP correlated with LVEF its association with outcome was independent of this.

Conclusions In a prospective mixed cohort of patients with AS undergoing investigations for consideration of aortic valve intervention, even a mild elevation in NT-pro BNP is associated with worse short term outcome. This can be taken into consideration regarding timing of intervention especially in the asymptomatic population.

Basic Science

159 A CRUCIAL ROLE OF NOX2-DERIVED REACTIVE OXYGEN SPECIES IN AGEING-ASSOCIATED METABOLIC DISORDERS AND BRAIN OXIDATIVE DAMAGE

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Ageing has been recognised to be a major risk factor for the development of cardiovascular and neurodegenerative diseases and growing evidence suggests a role for oxidative stress. A Nox2-containing NADPH oxidase has been reported to be a major source of reactive oxygen species (ROS) generation in the vascular system and in the brain. However, the role of Nox2 enzyme in ageing-related metabolic disorders and vascular neurodegeneration remains unclear. In this study, we used age-matched wild-type (WT) and Nox2-deficient ($Nox2^{-/-}$) mice on a C57BL/6 background at young (3–4 month) and ageing (20–24 month) to investigate the role of Nox2 in ageing-related oxidative stress, metabolic disorders and cerebral vascular dysfunction. There was an ageing-related increase in blood pressure in WT mice (126 mmHg for young and 148 mmHg for ageing) ($P < 0.05$); however the blood pressure was well maintained without significant change in $Nox2^{-/-}$ ageing mice. Compared to young WT mice, WT ageing mice had significantly high levels of fasting serum insulin and this was accompanied with delayed clearance of glucose ($P < 0.05$) indicating insulin resistance. In contrast, there was no indication of insulin resistance for $Nox2^{-/-}$ ageing mice. We then examined ageing-related brain oxidative stress. Compared to WT young mice, there were significant increases (2.7 ± 0.7 folds) in the levels of ROS production by WT ageing brain tissue homogenates as detected by lucigenin-chemiluminescence and DHE fluorescence. Increased ROS production in WT ageing brain was accompanied by a significant increase (1.8 ± 0.3 folds) in the Nox2 expression detected mainly in the microglial cells (labelled by Iba-1) and decreases in brain capillaries (labelled by CD31) (2.4 ± 0.8 folds) and neurons (labelled by Neuronal Nuclei) (2.9 ± 0.5 folds) (all $P < 0.05$). Knockout Nox2 abolished ageing-associated increases in brain ROS production and reduced significantly the ageing-related pathophysiological changes in the brain. In conclusion, Nox2-derived oxidative stress plays an important role in ageing-associated metabolic disorders and vascular neurodegeneration. Nox2-containg NADPH oxidase represents a valuable therapeutic target for oxidative stress-related diseases in ageing.

160 MODULATION OF MYOCARDIAL ISCHAEMIC INJURY BY CARBON MONOXIDE RELEASING MOLECULE-A1 IN A PORCINE MODEL OF ACUTE REPERFUSION INFARCTION

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Introduction Conventional pharmacological treatments for acute myocardial infarction (AMI), a life threatening complication of a sudden coronary occlusion, are limited by their efficiency and side effects. New therapeutic strategies are thus needed to improve outcomes. Carbon monoxide (CO) is cardio-protective at nanomolar concentrations. Carbon monoxide-releasing molecules (CORMs), capable of carrying and releasing controlled quantities of CO in cellular systems, are a promising therapeutic that overcome the limitations of CO gas. CORM-A1 is a water soluble and releases CO slowly through hydrolysis at physiological conditions. We investigated the efficacy and safety of CORM-A1 in reducing infarct size in a clinically relevant porcine model of re-perfused AMI.

Methods Male Yorkshire White pigs (25–33 kg) underwent a balloon-induced coronary occlusion at the middle segment of left anterior descending artery beyond the first diagonal branch for 60min. From 15min post-occlusion, sodium borate (control) or CORM-A1 (4.27 mM, each) were infused over a period of 60min. Left ventricular (LV) function and blood pressure were assessed by cardiac catheterization. Cardiac biomarkers, hepatic and renal functions were compared between the groups. Seven days after AMI, animals were culled and *in-situ* double staining with Evans blue and 2,3,5-triphenyltetrazolium (TTC) performed to measure infarct size. Myocardial inflammation, proliferation and apoptosis were evaluated by immunohistochemistry, immunoblotting, and TUNEL assay.

Results CORM-A1 treated pigs had a reduced infarcted area and improved LV function, but no significant change in blood pressure, compared to controls. Infarct size was 35 ± 7% of the area at risk (ischaemic area) in CORM-A1 pigs compared to 90 ± 5% in controls ($p < 0.0001$, n = 3–8/group). Myocardium from CORM-A1 treated animals had fewer TUNEL positive (30.5 ± 4.7 vs. 46.2 ± 6.6%, $p < 0.05$, n = 37), Ki67 positive (7.7 ± 2.3 vs. 29.0 ± 4.0%, $p < 0.01$, n = 3–7) and inflammation positive cells (4.3 ± 1.8 vs. 36.7 ± 7.1%, $p < 0.01$, n = 3–7) in the infarcted regions, compared to controls. CORM-A1 infused animals also had significantly reduced (2–3-fold) neovascular formation (vWF staining) in the infarcted areas compared to controls (22.0 ± 3.6 vs. 53.3 ± 8.9%, $p < 0.01$, n = 3–7). A similar pattern was seen in the ischaemic areas. These changes were associated with a down-regulation of HIF-1a expression in the myocardium of CORM-A1 treated animals.

Conclusions Our data suggest CORM-A1 as a key modulator of myocardial repair following re-perfused AMI injury. Injury is reduced in CORM-A1 treated animals by reducing inflammation, proliferation and cell death whilst maintaining healthy repair via neovascularisation. This study suggests the development of CORM-A1 as a potential new therapeutic for treatment of patients with AMI and warrants further clinical studies.