increases in cardiovascular events when large populations are exposed to acute mental stress for example by earthquakes and publicised national sports events. In contrast, even in patients with known coronary artery disease and exertional angina, exercise is safe and beneficial. These differences may reflect underlying pathophysiology with vascular dysregulation limiting myocardial blood flow during mental stress.

Methods Simultaneous intracoronary pressure and flow velocity data were acquired in a target artery from 15 patients with significant coronary artery disease (FFR >0.8 and or stenosis >70%) and 11 controls following exposure to mental stress during cardiac catheterisation. Oral nitrate preparations, calcium channel antagonists and beta-blockers were stopped 24–48 h in advance. All data were acquired at rest and at peak mental stress. Mental stress involved a 6-minute mental stress test consisting of mental arithmetic and the Stroop test. Coronary flow average peak velocity (APV), microvascular resistance (MVR) and buckberg index (BI; a surrogate of subendocardial ischaemia) were calculated. Wave intensity analysis also differentiated waves that accelerate and decelerate coronary flow

Results Mental stress was associated with an increase in systolic blood pressure (SBP, 28.43 mmHg; p = 0<0001), diastolic blood pressure (DBP, 14.47 mmHg; p = 0<0001), and heart rate (HR, 13.63 bpm; p = 0<0001). Rate pressure product (RPP) a marker of myocardial oxygen demand increased by 4429 (p = 0<0001). In patients with coronary disease this increase in demand was not met by an increase in coronary flow but instead by a paradoxical increase in microvascular resistance (206.1; p = 0.0096), resulting in subendocardial ischaemia as reflected by a fall in the BI (-0.23 (p < 0.0001). In contrast, an increase in coronary flow was observed in response to mental stress in the control group (5.25; p = 0.003). This increase in coronary flow was a result of an increase in the backward expansion wave and forward compression wave reflecting an increase in sympathetic activity and myocardial contractility.

Conclusions Exposure to mental stress is associated with an increase in myocardial work and oxygen demand that is met by an increase in coronary flow in patients with unobstructed coronaries. Paradoxical microvascular dysfunction in response to mental stress does occur and this abnormal endothelial response appears to correlate with the extent of atherosclerosis in the vessel. This likely plays a key role in the mechanism of mental stress induced myocardial ischaemia and provides an exciting target for future therapies.

Interventional Cardiology

32 TRANSRADIAL CATHETERISATION: A CLINICAL TRANSLATIONAL MODEL OF HUMAN ARTERIAL INJURY *IN VIVO*

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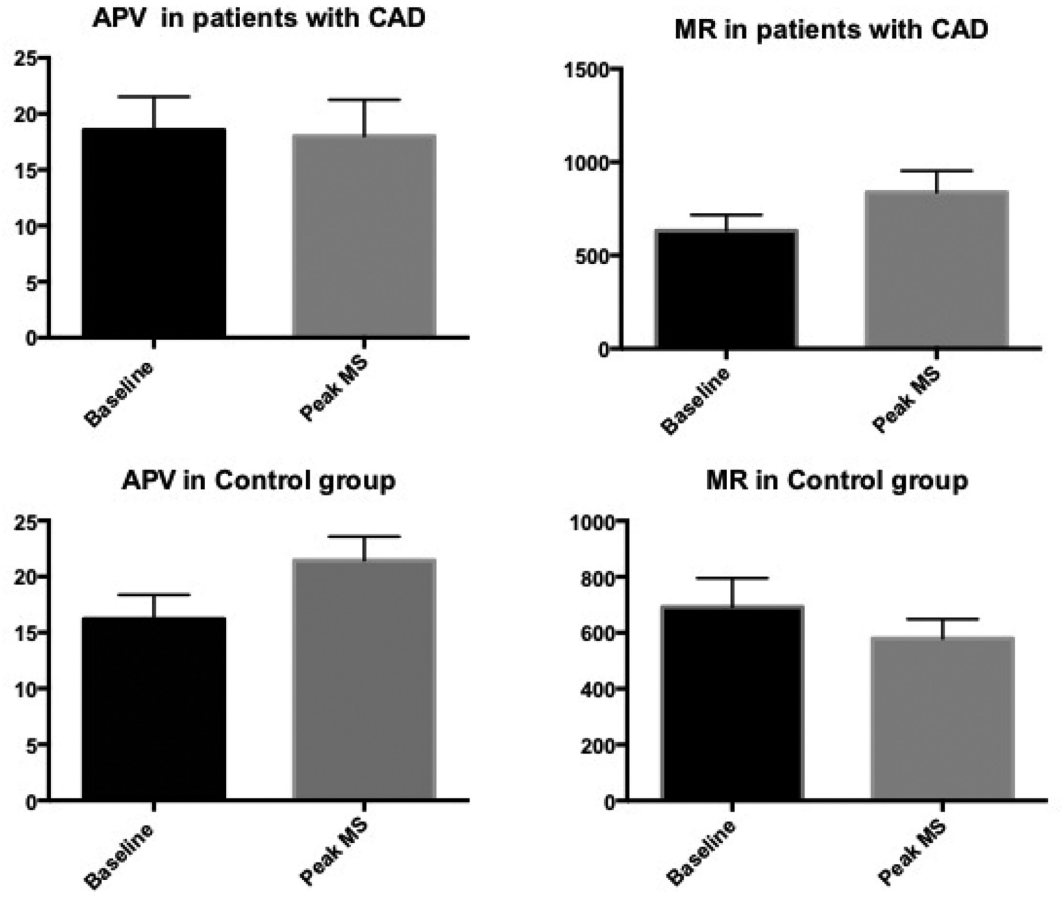

Abstract 31 Figure 1

Introduction Endothelial outgrowth cells (EOC) isolated from the circulation can be expanded in culture and have major potential as a therapy for vascular regeneration, but investigation of their role in vascular repair has been limited by the lack of a safe model of arterial injury in man. We developed a GMP-compliant culture process for EOC production and characterised the functional response to vascular injury in patients undergoing transradial cardiac catheterization.
Methods Patients with stable angina (n = 50, 63.8 ± 10.8 years) undergoing transradial cardiac catheterization had an assessment of flow-mediated dilatation (FMD) in both radial arteries prior to catheterisation and at 24 h, 1, 4 and 12 weeks. Peripheral blood was obtained for the isolation and quantification of EOC, and for the development of a GMP-complaint protocol.
Results FMD was attenuated in the catheterised artery compared to the non-catheterised artery at 24 h (4.3 ± 3.4 vs. 10.7 ± 5.6, P < 0.05) and at one week (3.1 ± 4.1 vs. 7.7 ± 5.6, P < 0.05), but had recovered by 12 weeks. EOCs were obtained in 66.67% of patients (14/21) and using a GMP-compliant protocol we were able to produce 32.1 ± 11.2 × 10^6 cells over four weeks.
Conclusions Transradial catheterization is associated with endothelial denudation and radial artery vasomotor dysfunction, and represents an accessible and reproducible model of vascular injury in man. The development of an efficient GMP-compliant culture protocol for the manufacture of EOC will permit first-in-man clinical trials to assess whether EOCs are able to home to and incorporate at sites of vascular injury in man.

33 TIMING OF SURGICAL AND PERCUTANEOUS REVASCULARISATION FOR LEFT MAIN STEM CORONARY ARTERY DISEASE IN THE WEST OF SCOTLAND

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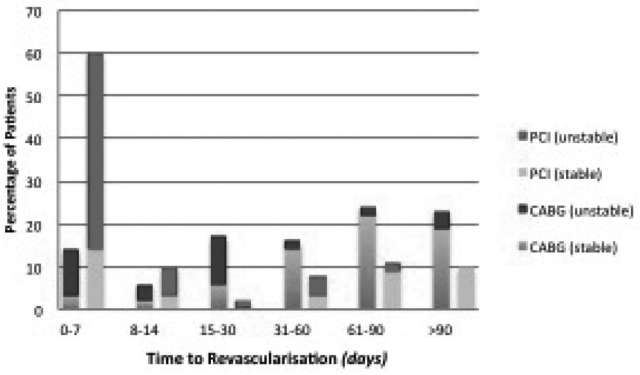
Introduction Significant (>50%) left main stem stenosis (LMS) is found in 4–6% of patients undergoing diagnostic angiography. Unprotected LMS lesions are associated with a poor prognosis and the mortality for non-revascularised disease may be as high as 37% at 3 years. In order to reduce morbidity and mortality associated with delayed treatment, European guidelines recommend that all patients with LMS disease undergo revascularisation within 2 weeks.
Aims We audited all patients in our high-volume tertiary centre that underwent revascularisation by coronary artery bypass grafting (CABG) or percutaneous coronary intervention (PCI) for significant LMS stenosis during a 6-month period. We sought to investigate timing between coronary angiography and revascularisation for stable and unstable presentations and compare our findings to the international guidelines.
Methods Patients with significant LMS disease observed during invasive coronary angiography (Feb–Aug 2014) were retrospectively identified using the coronary intervention patient-database at the West of Scotland Regional Heart and Lung Centre (Golden Jubilee National Hospital). Data were collected from angiography reports and electronic patient records. Patients who did not undergo revascularisation or who had primary PCI were excluded from the analyses. Patients were considered unstable if the indication for angiography was acute coronary syndrome, including ST-elevation myocardial infarction without primary PCI.

	All (n=165)	CABG (n=103)	PCI (n=62)
Male sex n (%)	125 (76)	83 (81)	42 (68)
Age n (%)			
20-39 years	1 (1)	1 (1)	0
40-59 years	33 (20)	27 (26)	6 (10)
60-79 years	102 (62)	63 (58)	39 (63)
≥80 years	29 (18)	12 (12)	17 (27)
Comorbidities n (%)			
Diabetes	44 (27)	24 (23)	20 (32)
Previous myocardial infarction	58 (35)	31 (30)	27 (44)
Previous stroke	18 (11)	11 (11)	7 (11)
Peripheral vascular disease	22 (13)	9 (9)	13 (21)
Angiogram indication n (%)			
Stable	93 (56)	68 (66)	25 (40)
Unstable	72 (44)	35 (34)	37 (60)
Time to revascularisation n (%)			
≤14 days			
All	63 (38)	20 (21)	43 (69)
Stable	16 (25^)	5 (25^)	11 (26^)
Unstable	47 (75^)	15 (75^)	32 (74^)
>14 days			
All	102 (62)	83 (81)	19 (31)
Stable	77 (75^)	63 (76^)	14 (74^)
Unstable	25 (25^)	20 (24^)	5 (26^)

Abstract 33 Table 1 Baseline character

Results 207 patients were found to have significant LMS disease during the audit period, 42 were excluded (not revascularised or primary PCI). A total of 165 patients underwent non-emergency revascularisation. 103/165 (62%) patients were revascularised by CABG. A larger proportion of patients in the PCI group were female, older, had additional comorbidities and were unstable at presentation compared with the CABG group (Table 1). 102/165 (62%) patients waited >14 days for revascularisation. Figure 1 shows the distribution of timing of revascularisation by CABG and PCI, highlighting the stability of patients within each group. 16/93 (17%) stable patients and 47/72 (65%) unstable patients were revascularised within 14 days, with the majority undergoing PCI (43/64, 68%). The main reason for delay >14 days for CABG was the wait for outpatient surgery (Figure 2). In the PCI group waiting >14 days, 5/19 (26%) patients were referred back for PCI after being deemed unsuitable for surgery (Figure 3).
Conclusion Huge service developments have been made in recent years to deliver timely revascularisation for patients, particularly those with unstable coronary disease. Despite this, we have found that patients with the highest risk coronary anatomy are experiencing significant delays to revascularisation, particularly by CABG. Further efforts to streamline the



Abstract 33 Figure 1 Time to revascularisation stratified by patient stability