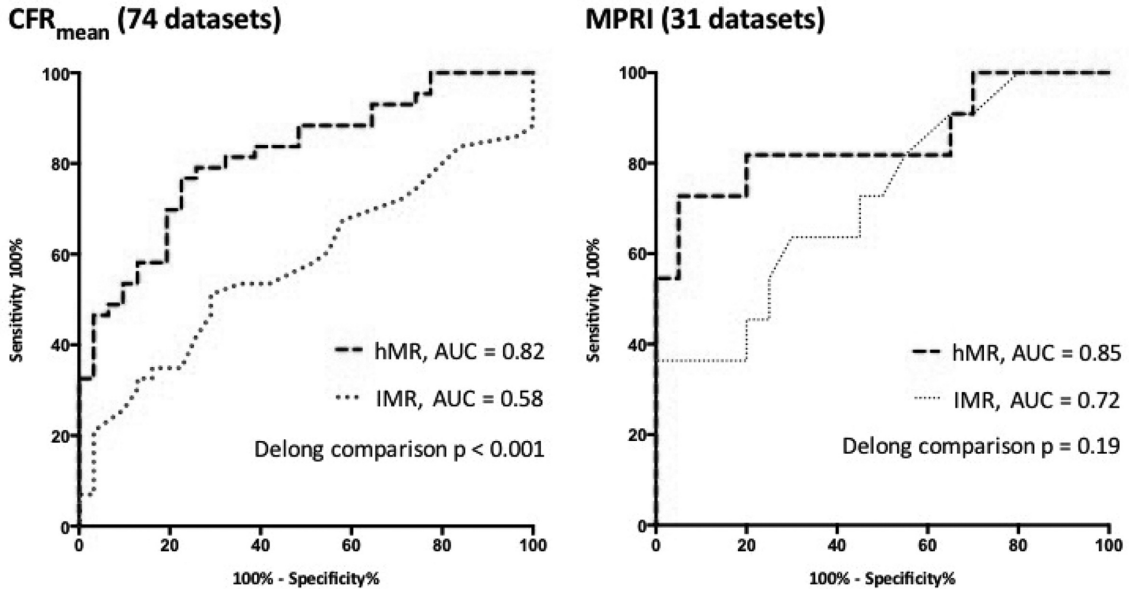

Abstract 30 Figure 1 Defining best measure of microvascular resistance

B. 47 datasets were obtained:

1. n = 10
2. n = 24
3. n = 13. (12 patients did both conditions 2 and 3, in randomized order).

MVR increased during CAI alone ($p = 0.04$), and decreased during exercise ($p < 0.0001$). Exercise with CAI was associated with less decrease in MVR (NS). The increase in CBF was similarly 34% less during exercise with CAI ($p = 0.04$) versus without ($p < 0.0001$). Coronary perfusion efficiency increased during exercise ($p < 0.05$), but CAI during exercise abolished this.

Conclusions A. Doppler-derived hMR may have superior diagnostic accuracy than IMR at predicting invasive and non-invasive measures of micro-vascular function.

B. CAI attenuates the reduction in MVR and the increases in CBF and coronary perfusion efficiency that normally occur during exercise. These suggest impedance of coronary vasodilatation and ventricular relaxation, rendering the heart more susceptible to ischaemia.

Acute Coronary Syndromes

31 UNRAVELLING THE MECHANISMS OF MENTAL STRESS INDUCED MYOCARDIAL ISCHAEMIA

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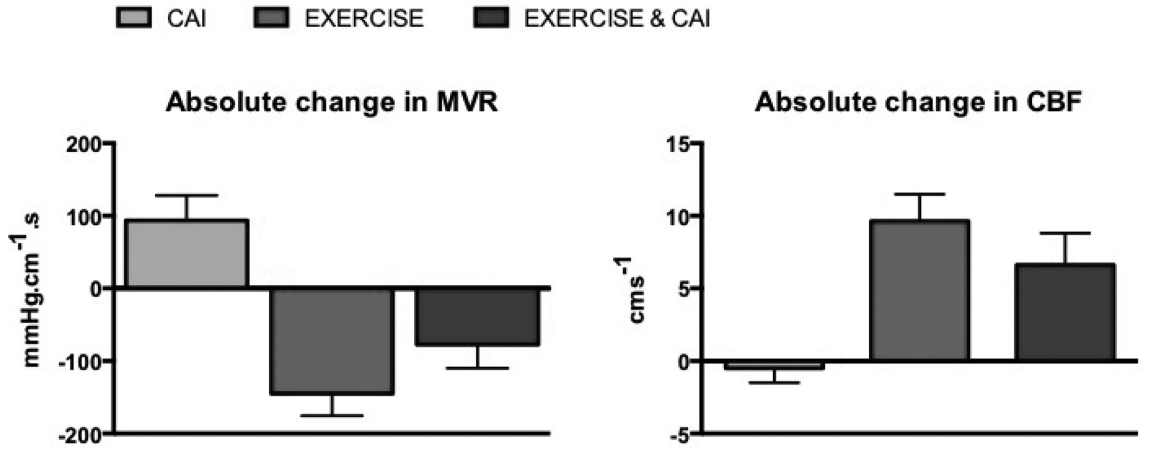
Background Mental stress triggers myocardial ischaemia at cardiac workloads that are lower than those that cause exercise-induced ischaemia in the same patient. Clinical relevance is highlighted by observational studies demonstrating marked



Abstract 30 Figure 2 Simultaneous measurement of microvascular resistance during cold air inhalation and exercise

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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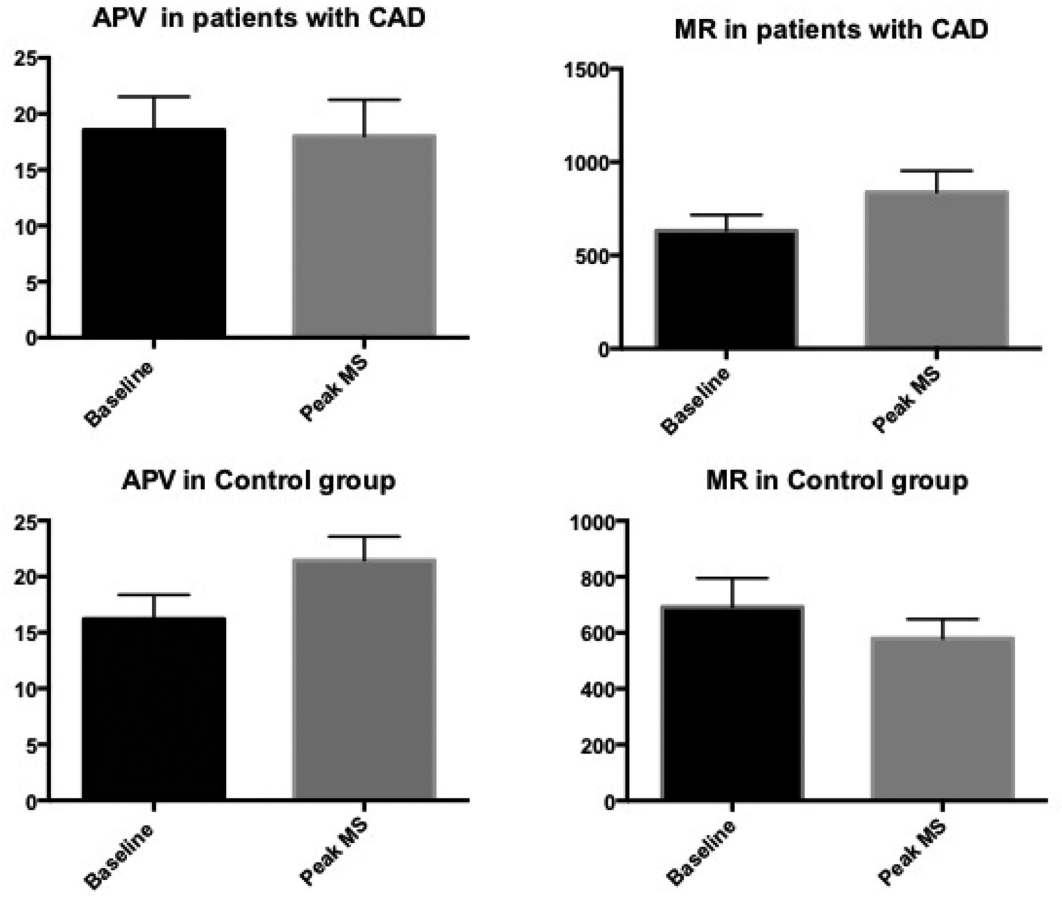

Abstract 31 Figure 1