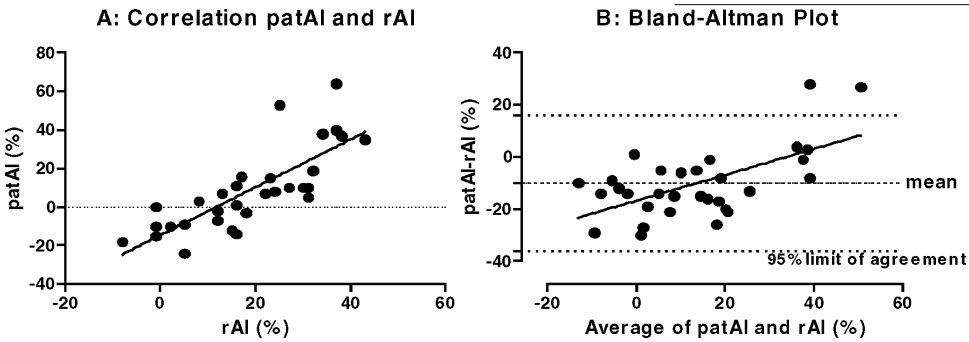
lated from the fingertips by peripheral arterial tonometry (patAI). Therefore we sought to determine whether AI calculated from patAI provides similar information to that of rAI in patients with AF.

Methods: Thirty-five consecutive patients with paroxysmal AF (age 59 ± 12) were examined during sinus rhythm. For each subject, rAI and patAI were recorded using radial applanation tonometry (SphygmoCor) and using peripheral arterial tonometry (EndoPat2000).

Results: Overall, rAI (19 ± 13%) was significantly ($p < 0.005$) higher than patAI (9 ± 21%) but both indices were highly correlated to each other. The *R* value was 0.79 ($p < 0.0001$) and the *R*-squared value was 0.62 (Fig. A). Bland–Altman plot of the difference between the two techniques (patAI-rAI values) versus their mean demonstrates that patAI under-estimates augmentation index (Fig. B). The bias calculated over the range of averaged concentrations was −10%, however, it is not constant over this range.



Conclusion: AI can be measured by radial artery tonometry and peripheral arterial tonometry. There is a good correlation between the AI calculated from both techniques; the lack of uniform bias between the values suggests that the two techniques are not interchangeable as estimates of arterial stiffness in patients with AF.

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Does Resveratrol Prevent Maladaptive Electrophysiological and Vascular Alterations in L-NAME Induced Hypertensive Rats?

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Resveratrol has demonstrated various possible cardioprotective mechanisms, which may prove to be beneficial in the treatment and prevention of various complications seen in conditions such as diabetes and hypertension. Such disease states are widely understood to result in various biochemical alterations including an increase in oxidative stress and inflammation and a decrease in the bioavailability of the potent vasodilator nitric oxide. The aim of this study was to investigate the potential protective effects of resveratrol in preventing maladaptive vascular and cardiovascular alterations in a rodent model of induced hypertension. Animals commenced treatment at eight weeks of age for a total of eight weeks (L-NAME (400 mg/L) administered in the drinking water supplied and 2 mg/kg/day resveratrol via oral gavage). Vascular organ bath studies were carried out on thoracic aorta rings and mesenteric vessels. Electrophysiological studies were carried out on the left ventricular papillary muscle and various action potential parameters examined. L-NAME induced hypertensive animals displayed a marked increase in action potential durations at 20, 50 and 90% repolarisation (17.42 ± 2.35; 30.75 ± 5.31; 93.58 ± 15.28 respectively) in comparison to healthy control animals (13.19 ± 0.65; 20.38 ± 1.75; 54.00 ± 4.66 respectively). This prolongation was not significantly prevented in resveratrol treated L-NAME animals. Vascular tissues from L-NAME animals also demonstrated decreased contractile responses to noradrenaline. These responses were significantly improved in resveratrol treated L-NAME animals. As expected, L-NAME treated animals displayed a reduced relaxation response to acetylcholine and sodium nitroprusside. There was no significant improvement in relaxation in resveratrol treated L-NAME animals.

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Dynamic Synchrotron Imaging of Diabetic Rat Coronary Microcirculation In Vivo

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In diabetes, long term micro- and macro-vascular damage often underlies the functional decline in a number of organs. Using synchrotron imaging we are now able

to detect small vessel calibres (~40 μm vs. 200 μm using a conventional X-ray device) and quantify regional differences in resistance vessels of interest, even under conditions of high heart rate (>500 bpm).

Experiments were conducted at the Japanese Synchrotron, SPring-8, using anaesthetised Sprague–Dawley rats three weeks after treatment with vehicle (control) or streptozotocin (diabetic, 65 mg/kg i.p.). The right carotid artery was cannulated and angiograms of the coronary vasculature were recorded. Using cine-radiograms we investigated endothelium-dependent and -independent vasodilatory responses in individual coronary vessels, in vivo. Change is from baseline.

Diabetic animals had elevated blood glucose concentration ($p < 0.001$) and reduced final body weight ($p < 0.001$) and mean arterial pressure ($p < 0.05$). Vessel recruitment was lower in diabetics during acetylcholine (ACh, $p < 0.05$), while nitric oxide synthase (NOS) and cyclooxygenase (COX) blockade, resulted in a strong trend towards loss of visible microvessels ($p = 0.059$). Diabetic animals displayed numerous focal stenoses during NOS/COX blockade which persisted and increased after ACh infusion. Segmental constriction was also noted in diabetic rats during NOS/COX blockade which remained following ACh infusion. Diabetes was further associated with smaller vessel calibre in the 101–200 μm vessels during NOS/COX blockade ($p < 0.05$).

Synchrotron imaging provides a novel method to investigate coronary microvascular function in disease models, in vivo, and this study indicates that the early diabetic state is associated with localised and systemic impairment in coronary endothelial function in the diabetic microvasculature.

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Effect of Adenosine on Pulmonary Vascular Resistance in Patients with Pulmonary Hypertension due to Left Heart Disease and Scleroderma

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Introduction: Functional assessment of the pulmonary vasculature is an important tool in the diagnosis of pulmonary hypertension. The effect of adenosine on pulmonary haemodynamics in left heart disease is not well understood

Methods: Right heart catheterisation was performed in eight subjects with mean PA pressure (mPAP) >25 mm Hg. In four subjects this was due to scleroderma (SCL) whilst four patients had heart failure with normal ejection fraction (HFNEF) defined by a pulmonary capillary wedge pressure (PCWP, mm Hg) >18 mm Hg and an ejection fraction >50%. Invasive haemodynamic measures were obtained before and during an adenosine infusion (140 mcg/kg/mins)

Results: Four SCL patients had a mean age of 58 ± 13 years, all were female and none were on pulmonary vasodilator therapy. Four HFNEF patients had a mean age of 62 ± 8 years, all were female and were receiving systemic antihypertensive therapy. The four patients with HFNEF all had LV end diastolic pressure >18 mm Hg (mean 21.5 ± 4.2 mm Hg). The effect of adenosine on pulmonary haemodynamics is summarised in the table: in patients with SCL, adenosine infusion resulted in a significant decrease in PVR in both groups, and an increase in CO in HFNEF patients. Despite the reduction in PVR, there was a trend to increased mPAP in HFNEF patients

Conclusions: Adenosine is an effective pulmonary vasodilator. Further study is required to elucidate whether the effect on pulmonary arterial and wedge pressures may vary with the etiology of pulmonary hypertension.

	SCL pre	SCL adenosine	HFNEF pre	HFNEF adenosine
mPAP	47.5 ± 12.9	45.3 ± 10.2	27.8 ± 5.9	30.3 ± 3.7
PCWP	15.3 ± 6.6	16.8 ± 6.6	18.2 ± 6.2	20.0 ± 5.0
CO	5.9 ± 1.7	8.1 ± 4.4	6.2 ± 1.5	10.1 ± 2.3†
PVR	5.8 ± 2.3	4.2 ± 2.3†	1.6 ± 0.6	1.1 ± 0.4*

* $p < 0.05$ for paired *t*-test comparison with pre adenosine.

† $p < 0.01$ for paired *t*-test comparison with pre adenosine.

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Gender Differences in Internal Mammary Artery Vasoconstrictor Responses

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Background: Women have poorer in-hospital outcomes following coronary artery bypass grafting compared with men although the reasons are unclear. Gender-mediated differences in the vascular reactivity of the arterial conduit may contribute to these differential outcomes. This study investigated gender-dependent differences in the vasoconstrictor responses of the internal mammary artery used for coronary artery bypass grafting.

Methods: Internal mammary artery segments were obtained from patients undergoing coronary artery bypass grafting and subjected to vascular myography. Following assessment of endothelial function, concentration-response curves were determined to noradrenaline, phenylephrine, seotonin, U46619 (thromboxane analogue) and endothelin. The EC50 values for each vasoconstrictor are summarised in the table below, expressed as mean ± SEM (*significant difference between males and females, $p < 0.05$).