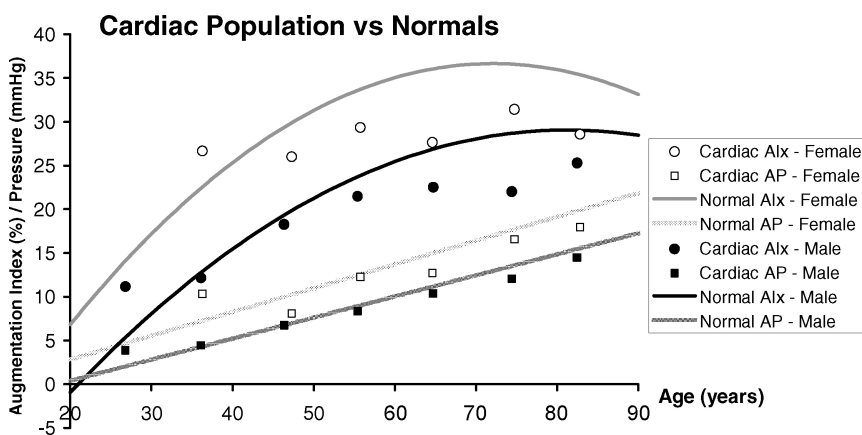


the proportion of central aortic pulse pressure that is attributed to the reflected pulse wave. We hypothesise that in a cohort of patients, who have one or more cardiac risk factors, and suspected coronary artery disease; arterial stiffness will be elevated compared to a normal population.

**Methods and results:** 910 patients presenting for coronary angiography at Westmead Hospital, Sydney, were recruited into the Australian Heart Eye Study (AHES). Brachial blood pressure was recorded (HEM 907, Omron). Radial artery waveforms were measured with a tonometer and pulse wave analysis was used to derive central blood pressures, AP and AIx (SphygmoCor, AtCor Medical). The average values for each decile of age were compared against normal reference ranges derived from 4001 healthy, normotensive individuals (McEniery et al., JACC 2005). The figure shows that while AP is consistent with normal ranges, the AIx is lower than that of normals, for both genders.



**Conclusions:** AIx and AP are not significantly increased in a cardiac population, contrary to expectations.

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This abstract has been withdrawn.

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### Calibration of Carotid Central Pressures: A Flaw in Brachial Applanation Tonometry?

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Central pressure, based on carotid and brachial applanation tonometry (AT) does not predict outcomes. This may be due to inaccuracy of brachial and carotid AT. No validation has been published.

To test its (in)accuracy, we measured radial, brachial and carotid pressure pulse waveforms by AT in 100 subjects. Carotid systolic (SP) and pulse (PP) pressures were esti-

mated by two techniques. First, the Pressure Equivalence (PE) technique calibrated brachial waves with brachial cuff pressure values, then the carotid wave was calibrated by assuming identical mean and diastolic pressures, with carotid SP extrapolated. Second, SphygmoCor<sup>®</sup> technique applied the Transfer Function (TF) to the radial waveform, calibrated to brachial cuff, to generate carotid SP and PP.

Amplification was significant between carotid and brachial (8.0 mm Hg,  $p < 0.0001$ ) with TF, but not with PE (1.5 mm Hg,  $p = NS$ ). PE gave considerable amplification between brachial to radial (8.4 mm Hg), which was not present with the TF. Form Factors (FF = (mean pressure - diastolic pressure)/PP) for carotid and brachial waves were similar (40.2 c.f. 39.1%;  $p = NS$ ), but different to the radial (34.5%;  $p < .0001$ ). Amplification as brachial PP ÷ carotid PP using PE was insignificant, but with TF was positive (18%,  $p < 0.0001$ ).

The PE method for recording PP amplification in the upper limb is inaccurate. A major problem is use of AT in carotid and brachial arteries, which cannot reliably be applanated. Findings explain inability of the PE method to predict cardiovascular outcomes, and superiority of TF. "Die Methode ist Alles" (Carl Ludwig 1852).

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### Correlation Between Radial Artery- and Peripheral Arterial Tonometry Derived Augmentation Index in Patients with Atrial Fibrillation

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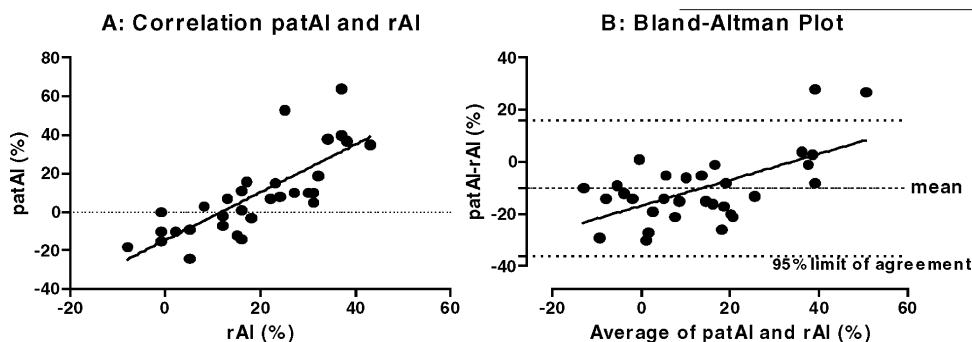
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**Introduction:** Augmentation index (AI) measures the contribution that wave reflection makes to the arterial pressure waveform. AI is considered a surrogate marker for the stiffness of the arterial system; however its utility in atrial fibrillation (AF) is unknown. The AI is routinely recorded from the radial artery (RAI). AI can also be calcu-

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  $59 \pm 12$ ) 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 ( $19 \pm 13\%$ ) was significantly ( $p < 0.005$ ) higher than patAI ( $9 \pm 21\%$ ) 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 ( $17.42 \pm 2.35$ ;  $30.75 \pm 5.31$ ;  $93.58 \pm 15.28$  respectively) in comparison to healthy control animals ( $13.19 \pm 0.65$ ;  $20.38 \pm 1.75$ ;  $54.00 \pm 4.66$  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