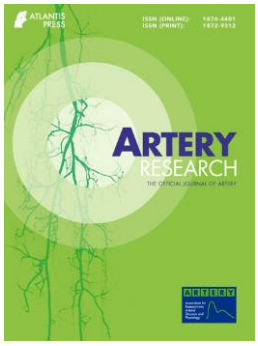

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4.6: WAVE INTENSITY ANALYSIS IN THE PULMONARY ARTERY

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BCL11B gene expression levels among those with different genotypes. In addition, rs1461587G>T and rs17773233G>T polymorphisms influence aortic stiffness measured ex vivo, confirming previous observations. Further functional studies are required to elucidate the role of this locus on aortic stiffness.

4.3
IMPACT OF AGE AND GENDER ON THE HAEMODYNAMIC DETERMINANTS OF BLOOD PRESSURE ACROSS THE ADULT AGE-SPAN

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Background: Systolic and diastolic blood pressure (BP) oscillate around the mean arterial pressure (MAP), which is determined, physiologically, by the cardiac output (CO) and peripheral vascular resistance (PVR). Although data describing the influence of age and gender on BP are widely available, few studies have examined the normal age-related changes in CO and PVR and the impact of gender on these changes, in a sufficiently large cohort of healthy individuals.
Methods: Detailed haemodynamic data including blood pressure (BP), CO and PVR were available in 5580 individuals (2518 males), aged between 18-92 years. Data were stratified according to gender and decade of age.
Results: As expected, MAP increased progressively with age in both males and females ($P<0.001$ for both). PVR was significantly higher in females at all ages and increased significantly with age in both sexes ($P<0.001$ for both). In contrast, CO was significantly higher in males at all ages and declined significantly with age in both sexes ($P<0.001$ for both). The decline in CO was due to a decline in stroke volume (SV) ($P<0.001$ for both), since there was no effect of age on heart rate. Adjusting CO and SV for body size abolished the gender-related differences but the age-related differences remained.
Conclusion: These data indicate that the physiological determinants of blood pressure vary between genders and with ageing, supporting the need for differential approaches in understanding, and treating high BP across the adult age-span.

4.4
THE EFFECT OF B-VITAMIN SUPPLEMENTATION ON ARTERIAL STIFFNESS IN ELDERLY

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Background: Hyperhomocysteinemia is an important cardiovascular risk indicator in the oldest old and is also associated with elevated arterial stiffness in this age group. Several intervention trials reported a lack of benefit of B-vitamin supplementation on cardiovascular outcomes, therefore we aimed to investigate the effect of B-vitamin supplementation on arterial stiffness and cardiovascular events in hyperhomocysteinemic elderly subjects.
Methods and Results: The B-PROOF study is a double-blind randomized-controlled trial, including 2919 elderly ($\geq$ 65 years), with hyperhomocysteinemia (12-50 μmol/l), treated with B-vitamins (500 μg vitamin B12 and 400 μg folic acid) or placebo for 2 years. In a subgroup ($n = 569$) the effect of B-vitamins on pulse wave velocity (PWV) was investigated. In the total B-PROOF population, incidents of cardiovascular and cerebrovascular events were determined via structured questionnaires and also blood pressure was measured. Compared to placebo, B-vitamins lowered serum homocysteine by 3.6 μmol/L ($p<0.001$). Analysis of covariance showed no effect of B-vitamins supplementation on PWV levels, but aortic pulse pressure was higher in the intervention than in the placebo group (49.6 mmHg vs. 47.2 mmHg; $p = 0.02$). Furthermore, a significant reduction of cerebrovascular events in females (OR 0.33 95%CI [0.15 ; 0.71]), but not in males was observed.
Conclusions: B-vitamins supplementation in hyperhomocysteinemic elderly has no effect on PWV and caused a modest increase in aortic pressure, but also a reduction in cerebrovascular events in females. Arterial stiffness is not likely to be the underlying pathway of the negative trial outcomes.

4.5
DO BACKWARD PRESSURE WAVES ARISE FROM "REFLECTIONS" OR FROM A "RESERVOIR"?

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Objective: Pressure waves in the aorta can be described as the summation of forward pressure wave generated by ventricular contraction and backward wave. Backward wave is usually regarded as being due to reflections from discontinuities in the arterial tree but could arise as a result of wave emptying backwards from "reservoir". We examined these possibilities in numerical models and with clinical data obtained during modulation of ventricular and arterial function using inotropic, vasodilator and vasopressor drugs.
Methods: Numerical models included simple Windkessel models with no wave propagation and a distributed single tube model terminated with impedance which allows wave propagation. Clinical data was obtained by carotid tonometry and Doppler sonography during modulation of cardiovascular function in healthy volunteers with dobutamine (2.5 - 7.5 μg/Kg/min), norepinephrine (12.5 - 50 ng/Kg/min), phentolamine (10 - 40 μg/min) and nitroglycerin (0.03 - 0.30 μg/min). Wave intensity analysis and arterial reservoir theory were applied to numerical models and clinical data.
Results: For numerical modelling, backward pressure over a range 0 - 50 mmHg was highly correlated with reservoir pressure both in Windkessel ($R=0.958$, $P<0.001$) and single tube models ($R=0.990$, $P<0.001$). For clinical data, there was a linear relationship between backward pressure over a range 5 - 20 mmHg and reservoir pressure ($R=0.911$, $P<0.001$) for all the subjects at rest and after inotropic/vasomotor stimulation. Augmentation pressure was neither related to the reflected pressure, nor to the reservoir pressure.
Conclusion: This study shows that the backward pressure wave may arise in large part from an arterial reservoir.

4.6
WAVE INTENSITY ANALYSIS IN THE PULMONARY ARTERY

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Introduction: Little is known about the roles of wave travel and wave reflection in the development of right ventricular dysfunction. The objective of this study is to apply wave intensity analysis (WIA) in the pulmonary artery to assess right ventriculo-arterial function and coupling in man.
Methods: Right heart catheterisation was performed using a pressure and Doppler flow sensor tipped catheter to obtain simultaneous pressure and flow velocity measurements in the pulmonary artery (PA). Recordings were made at rest as well as during a modified Valsalva manoeuvre and handgrip exercise. WIA was subsequently applied to the acquired data.
Results: 7 patients (48 ± 14 years, 5 male) undergoing cardiac catheterisation and with normal mean pulmonary arterial pressure (17 ± 3 mmHg) and without significant cardiovascular disease or lung disease were studied. In the main PA, WIA showed a forward (proximally originating) compression wave in early systole caused by right ventricular ejection and a forward expansion wave prior to closure of the pulmonary valve that decreased the arterial pressure and flow in late systole. Backward (reflecting) waves were minimal. Wave speed was 2.64 ± 1.39 m/s. The wave pattern was unchanged by respiration and handgrip exercise, however, during Valsalva manoeuvre the magnitude of the waves reduced.
Conclusion: Contrary to previous work in animals, our data show that minimal backward waves are present in the pulmonary artery indicating well matched ventriculo-arterial coupling in individuals without pulmonary artery disease. Patients with pulmonary hypertension will be assessed in the continuation of this study.

5.1
EPOXYEICOSATRIENOIC ACIDS IN THE REGULATION OF VASCULAR TONE

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Background: Epoxyeicosatrienoic acids (EETs) are released from the endothelium and regulate vascular tone as an endothelium-derived