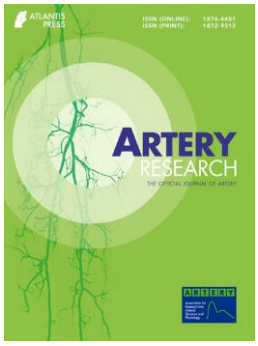

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P167: PULSE PRESSURE AMPLIFICATION AND AUGMENTATION INDEX CHANGE IN OPPOSITE MANNER WITH ARTERIAL STIFFNESS INDEPENDENTLY OF SYSTEMIC RESISTANCE

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Methods: The study was conducted on 2091 participants in the Avon Longitudinal Study of Parents and Children (ALSPAC), a prospective population-based birth cohort study, aged 17. BP measurement and echocardiography was performed and heart rate (HR), stroke volume (SV) and TPR calculated. Data are means (SD).
Results: Table 1 shows selected characteristics of the sample. Higher quintiles of systolic BP were associated with higher SV, higher HR and higher TPR. However, the proportional contribution made by SV, HR and TPR to mean arterial pressure differed little by systolic BP quintile (stroke volume (32−34%) heart rate (25−29%) and TPR (39−41%)).

Variable	Males (n = 939)	Females (n = 1152)	All (n = 2091)
y	(0.3)	(0.3)	(0.3)
kg/m^2	(3.7)	(4.2)	(4.0)
mmHg	11)/64(8)	9)/65(7)	11)/65(8)
bpm	0	0	0
L/min	1.0)	0.8)	0.9)
ml	3	1	3
mmHg ml/min	(5.9)	(6.0)	(6.0)

Conclusions: Higher blood pressure is attributable to a combination of higher cardiac output (i.e. SV×HR) and higher TPR in a population-based sample of adolescents. There is no evidence of a disproportionate contribution from CO at higher BP levels.

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HEAD-DOWN TILT BED-REST SIGNIFICANTLY INCREASES CENTRAL ARTERIAL STIFFNESS

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The vascular system is subject to continual variation in mechanical stresses, both physiological and pathological. Vascular remodeling via changes in vessel wall properties, including thickness and stiffness, is a major feature of aging and cardiovascular disease.
A more detailed understanding of the interplay between mechanical stress, aging, CVD and vascular remodeling will aid prevention of increased cardiovascular risk following long term microgravity.
This study aims at assessing vascular remodeling processes resulting from a 60-day head-down-tilt bed-rest period during the European Space Agency Study (Toulouse, France).
We hypothesize that arterial remodeling processes are modified by long term bed- rest and constitute a significant cardiovascular risk in the long term for astronauts. Applanation tonometry is used to assess carotid to femoral pulse wave velocity (PWV) and non-invasive ultrasound imaging are used to assess arterial remodelling processes at the carotid, femoral, brachial and popliteal arteries.Measurements are performed at baseline; at day 29 and 52 of bed-rest; and at day 6 and 30 of the recovery period.
The preliminary results including 10 first subjects, demonstrate a strong effect of bed- rest on arterial PWV.The average PWV at baseline equals $7{,}6 \pm 1.4$m/s and is increased to 9.0 ± 1.9m/s after 29 days, and, 9.3 ± 1.8m/s after 52 days bed-rest. This increase is significantly different between baseline, and, 29 and 52 days bed-rest ($p < 0.005$).
Increase in PWV suggests a rapid and significant stiffening of the central arteries, which on healthy subjects corresponds to an aging process wich occurs many years. Low gravity conditions as during bed-rest induce significant arterial stiffening that could be linked to long term CVD risks for either patients in bed-rest or astronauts.

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PULSE PRESSURE AMPLIFICATION AND AUGMENTATION INDEX CHANGE IN OPPOSITE MANNER WITH ARTERIAL STIFFNESS INDEPENDENTLY OF SYSTEMIC RESISTANCE

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Background: Pulse Pressure Amplification (PPA) is the increase in Pulse Pressure (PP) from proximal to distal arteries. The Augmentation Index (AIx) is the secondary increase in aortic pressure in systole relative to PP. With aging and increased arterial stiffness the PPA *de*creases while the AIx *in*creases. Since both depend on the reflection of pressure waves, the finding that PPA and AIx change in opposite ways seems surprising.
Methods: Aortic PPA, AIx and Reflection Magnitude ($RM = P_{reflected}/P_{forward}$) were determined in a multibranched model and during control and Valsalva Maneuver in the human.
Results: During the Valsalva Maneuver reflections decrease: the lower mean arterial pressure results in lower stiffness and Pulse Wave Velocity (PWV) while Systemic Vascular Resistance (SVR) is increased. The model confirms that SVR plays a minimal role in terms of reflections. Reflections result from many reflections sites in the larger arteries. The lower PWV implies shorter wave length and thus artery length/wave length increases. This increase makes the differences in travel times from the many reflection sites to the heart more different resulting in lower total reflection: RM and AIx decrease. The lower PWV, thus the shorter wave length, also implies an increase in travel time over the aorta, and larger amplification. (It has been shown that local reflections change little with changes in stiffness.)
Conclusions: Reflections are mainly determined by travel times of reflected waves of the larger arteries. Mean pressure determines arterial stiffness and the stiffness change, via PWV, results in the opposite changes in RM and PPA.

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ENDOTHELIAL REGULATION OF AWV IS IMPAIRED DURING INCREASE IN BLOOD FLOW IN ESSENTIAL HYPERTENSION

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Background: Arterial wall viscosity (AWV) depends on endothelium-derived factors in physiological conditions (1,2). Hypertension is characterized by an altered FMD during sustained flow increase due to endothelial dysfunction (3). Whether NO and EETs regulate change in AWV during increase in flow in hypertensive patients (HT) as compared with normotensive controls (NT) remains to be evaluated.
Methods: Radial artery diameter, wall thickness and arterial pressure were measured in 18 untreated essential HT and 14 frequency matched