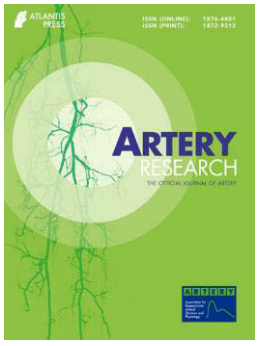


Artery Research

ISSN (Online): 1876-4401 **ISSN (Print): 1872-9312**
Journal Home Page: https://www.atlantis-press.com/journals/artres

NOVEL RESEARCH FUNDING OPPORTUNITIES

Warwick Anderson

To cite this article: Warwick Anderson (2018) NOVEL RESEARCH FUNDING OPPORTUNITIES, Artery Research 24:C, 64–65, DOI: https://doi.org/10.1016/j.artres.2018.10.012

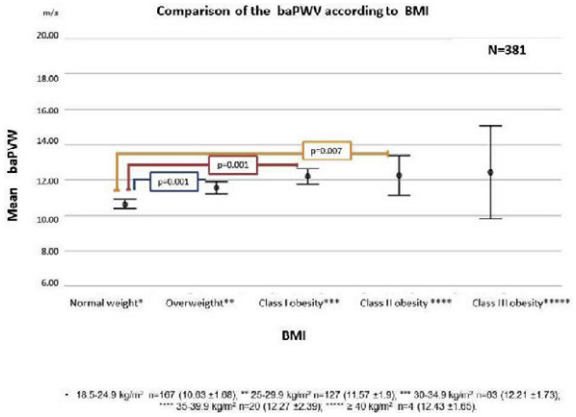
To link to this article: https://doi.org/10.1016/j.artres.2018.10.012

Published online: 7 December 2019

ASSOCIATION OF PULSE WAVE VELOCITY AND BODY MASS INDEX IN HEALTHY MEXICAN POPULATION

Daniela Avila Novoa, M. Cárdenas, E. G. Cardona, D. Cardona, F. Grover, C. Ramos, J. Trujillo
Laboratory of Vascular Mechanics of the Institute of Experimental and Clinical Therapeutics, Physiology Department, University Center Of Health Sciences, Universidad De Guadalajara, USA

Rational: Cardiovascular diseases represent the main cause of morbidity and mortality throughout the world (1). Arterial stiffness has shown to be an important predictor of cardiovascular events, and pulse wave velocity (PWV) is a marker of subclinical organ damage which can be measured by different methods, one of which is by means of the brachial ankle pulse wave velocity (baPWV) (2—3). On the other hand, obesity affects a large proportion of the population and is classified according to the body mass index (BMI). Increased BMI is associated with hypertension and increased mortality (4).
Objective: Analyse the correlation between baPWV and BMI in healthy subjects.
Methodology: An analytical cross-sectional study was carried out in healthy age 18—70 year old subjects, who attended the INTEC, (192 women, 189 men). BMI was calculated with the formula Weight (kg) /Height $(m)^2$, baPWV was measured with the VP1000 plus model BP-203RPE III. Correlations were determined with Spearman's Rho, differences between groups were determined using Anova with post hoc test.
Results: A population of 381 subjects was analyzed, a significant correlation was found between baPWV and BMI ($r = 0.332$, $p = 0.001$). Dividing patients according to the degree of BMI a significant difference was found in the baPWV between normal weight-overweight groups (10.63 ± 1.68, 11.57 ± 1.9 ($p = 0.001$), normal weight —class I obesity (10.63 ± 1.68, 12.21 ± 1.73 ($p = 0.001$) and normal weight —class II (10.63 ± 1.68; 12.27 ± 2.39 ($p = 0.007$).
Conclusion: A direct correlation between baPWV and the body mass index was found. The greater increase of the baPWV was seen between the groups of normal weight and overweight / Class I obesity/ Class II obesity. Subjects with overweight and grade I obesity represent a group with a significant increase in arterial stiffness which should receive special attention in order to decrease the development of cardiovascular disease.

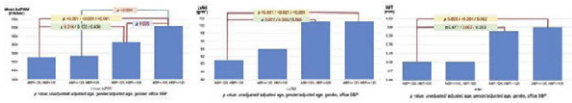


WHICH IS MORE CORRELATED WITH HYPERTENSIVE ORGAN DAMAGE, SLEEP BLOOD PRESSURE ASSESSED BY SELF-MEASURED AT HOME OR AMBULATORY BLOOD PRESSURE MONITORING?: THE JAPAN MORNING SURGE-HOME BLOOD PRESSURE (J-HOP) STUDY

Sirisawat Wanthong [1,2], Tomoyuki Kabutoya [1], Satoshi Hoshide [1], Kazuomi Kario [1]
[1]*Division of Cardiovascular Medicine, Department of Medicine, Jichi Medical University School of Medicine, Tochigi, Japan*
[2]*Department of Medicine, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand*

Objective: To assess the associations with hypertensive target organ damage (TOD) of sleep SBP assessed by self-measured home blood pressure monitoring (HBPM) and ambulatory blood pressure monitoring (ABPM).

Methods: Data of 1008 participants in the J-HOP study who measured sleep BP using both HBPM, three times during sleep (2 AM, 3 AM and 4 AM) and ABPM during sleep were analyzed. Study participants were classified into 4 groups according to sleep SBP values: group 1, HBPM <120 mmHg, ABPM<120 mmHg; group 2, HBPM <120 mmHg, ABPM≥120 mmHg; group 3, HBPM ≥120 mmHg, ABPM<120 mmHg and group 4, HBPM ≥120 mmHg, ABPM≥120 mmHg. Hypertensive TOD as indicated by brachial ankle pulse wave velocity (baPWV), left ventricular mass index (LVMI) and carotid intima media thickness (IMT), assessed in 946, 876 and 317 participants respectively, were compared among the 4 groups.
Results: Mean age was 63 ± 11 years. The percentage of male participants was 49.9. Thirty-four, 10, 20 and 36 percent of the participants were classified into groups 1, 2, 3 and 4. For groups 1,2, 3 and 4, respectively, baPWV were 1529 ± 293, 1536 ± 265, 1616 ± 255 and 1710 ± 322 cm/sec, LVMI were 91 ± 23, 94 ± 22, 101 ± 26 and 101 ± 28 g/m^2, and IMT were 0.73 ± 0.14, 0.72 ± 0.18, 0.79 ± 0.15 and 0.80 ± 0.19 mm. After age, gender and office SBP were adjusted, the baPWV, LVMI and IMT of groups 3 and 4 were significant higher than of group 1, In multivariate analyses, sleep SBP assessed by HBPM was an independent predictor of baPWV, LVMI and IMT, but that assessed by ABPM was an independent predictor of only baPWV.
Conclusions: Sleep SBP measured by HBPM was more closely associated with baPWV, LVMI and IMT than sleep SBP measured by ABPM.



Special guest lecture

MOLECULAR AND CELLULAR CHANGES IN ARTERIAL FUNCTION OVER THE LIFE COURSE — FROM ACCELERATED AGEING TO CALCIFICATION

Catherine M. Shanahan
King's College London, UK

Vascular stiffening and calcification are hallmarks of ageing and these pathologies are accelerated in patients with diabetes and renal failure. Emerging evidence has defined a role for nuclear lamina defects and the DNA damage response in driving vascular calcification. The mechanisms responsible are linked to the onset of cellular senescence and the activation of the proinflammatory senescence associated secretory phenotype (SASP) in vascular smooth muscle cells (VSMCs). In response to the SASP VSMCs can undergo phenotypic modulation and upregulate expression of proteins normally confined to cells of the osteo/chondrogenic lineage. Understanding the signaling pathways that drive this response is essential for defining novel therapeutic pathways to tackle age-associated vascular pathologies. We have been investigating the role of a number of signaling pathways involved in the DNA damage response as key drivers of calcification and VSMC osteo/chondrogenic differentiation including both the ataxia telangiectasia mutated (ATM) and poly(ADP) ribose (PARP) pathways. In addition it is clear that epigenetic changes, induced by nuclear lamina defects, precede the onset of calcification in both the aged vasculature and in patients with renal failure.

Young investigator session

NOVEL RESEARCH FUNDING OPPORTUNITIES

Warwick Anderson
Human Frontier Science Program, France

It is a fortunate paradox that research motivated simply by a wish to better understand the natural world can later result in the most powerful applications imaginable. By definition, the outcome of any research is unknown until the research is finished. The outcomes are even less predictable at the frontiers of knowledge but the rewards can be commensurately greater. Internationally, there has been increased funding of more "mission-focused" and translational research during the last decade. This is to be applauded and is yielding strong dividends for the public funds invested. But frontier basic science, where the most creative and talented researchers work on the ideas and hypotheses that they believe are the most important, is a long-term investment in the future. It is still the major engine for progress in science, industry and society. Frontier basic life science research is the domain of the Human Frontier Science Program. The Program was created as

the Cold War ended and was aimed at increasing collaborative science in the basic life sciences. Now supported by 14 countries and the EU, HFSPO research grants fund basic, interdisciplinary team-based research; research whose aim is to break through the known frontiers of science and foster collaboration across continents. Our postdoctoral Fellowships scheme offer top postdoctoral biologists and non-biologists the opportunity to change fields and research new ideas in a new laboratory in a new country. Thirty years on, 27 researchers funded by HFSPO have gone on to win a Nobel Prize.

Servier organised in collaboration with servier

ARTERIAL STIFFNESS AS A RISK FACTOR FOR CEREBRAL VASCULAR LESIONS

Dariusz Gasecki
Department of Adult Neurology in Medical University of Gdansk, Poland

Increased arterial stiffness, a biomarker of vascular aging, is a recognized subclinical organ damage, and may thus serve as predictor of cardiovascular disease. The predictive value of arterial stiffness is higher in patients with higher baseline cardiovascular risk, such as in patients with hypertension. According to European Society of Hypertension, increased arterial stiffness should be recommended as a negative prognostic factor in the management of patients with hypertension.
Arterial stiffness, an important determinant of transition of pulse wave energy from the heart into the periphery, could improve our understanding of the consequences of the hemodynamic-related vascular stress, especially in low-impedance organs, including the brain.
Epidemiological studies have demonstrated arterial stiffness as a risk factor for silent cerebral lesions, stroke, and cognitive impairment. Arterial stiffness was found to be independently associated with all components of cerebral small vessel disease including silent lacunar infarcts, white matter hyperintensities, and microbleeds, although there are some methodological differences between the various surrogate markers. Arterial stiffness may be important also in recovery after ischemic stroke. Aortic stiffness was found to be an independent predictor of both short-term clinical improvement and long-term functional outcome after ischemic stroke. Furthermore, increased aortic stiffness has been shown to be linked to acute hypertensive response after ischemic stroke.
However, the vascular, physiological, and metabolic roles of arterial stiffness in cerebrovascular diseases remain unclear. Better understanding of the hemodynamic consequences of arterial stiffness on brain damage is necessary, not only to select the most appropriate therapeutic management but also to optimize prevention, which should be started early in individuals at high risk of developing cerebral vascular lesions.

Focus update

CHILDHOOD DETERMINANTS OF ARTERIAL DYSFUNCTION CENTRAL AND PERIPHERAL BLOOD PRESSURE

Renate Oberhoffer
Institute of Preventive Pediatrics, Technical University Munich, Germany

Arterial Hypertension is one of the leading causes of death and disability worldwide. Measurement of peripheral brachial blood pressure using cuff sphygmomanometry belongs to the clinical routine assessment from childhood on. Based on age and length related reference values, it is widely accepted as surrogate marker of later cardiovascular events. Peripheral arterial pressure, however, does not necessarily reflect central aortic pressure, which seems to predict cardiovascular (CV) risk even better: many vital organs like brain and kidneys are perfused with aortic pressure, and keeping the systolic pressure amplification within the arterial tree in mind, the difference between peripheral and central blood pressure shows a wide variety.
Nowadays, oscillometric devices are able to measure central aortic as easy as peripheral blood pressure, but the clinical acceptance of the method is still low. Reference values for central arterial pressure in youth are existing and could help in CV risk stratification: we defined central systolic pressure ranges from 90 $\pm$ 5.8 mm Hg to 110.5 $\pm$ 9.6 mm Hg in boys and from 91.2 $\pm$ 7.5 mm Hg to 109.1 $\pm$ 8.6 mmHg in a cohort of 1445 children and young adults. Other central blood pressure measures, however, may show different results, and factors influencing the pressure amplification and end organ damage in childhood have to be defined in large series.

Debate

ARE CENTRAL HEMODYNAMIC PARAMETERS BETTER PROGNOSTIC MARKERS THAN PERIPHERAL BLOOD PRESSURE IN STROKE?

Alberto Avolio
Department of Biomedical Sciences, Faculty of Medicine and Health Sciences, Macquarie University, Sydney, Australia

Ernesto L. Schiffrin
Lady Davis Institute for Medical Research, Department of Medicine, Sir Mortimer B. Davis-Jewish General Hospital, McGill University, Montreal, Quebec, Canada

The complex pathophysiological mechanisms involved in stroke confound the associative and causal role of blood pressure. This is particularly relevant in monitoring changes in blood pressure with respect to treatment efficacy and stroke outcome, which is highly dependent on the both patient and stroke characteristics. However, whereas blood pressure may have a variable prognostic role in both the underlying causes of stroke as well as stroke outcome due to modifications of intracranial pressure and autoregulation of cerebral blood flow, particularly in the elderly, measures of pulsatile phenomena, such as arterial stiffness affecting wave propagation have been shown to be strong independent predictors of overall cardiovascular events. Aortic stiffness has been shown to be related to cognitive decline, cerebral small vessel disease and acute hypertensive response and outcome following ischemic stroke.
In this debate, a case will be made that measures associated with arterial stiffness (pulse wave velocity, magnitude of forward and backward waves, intensity of wave reflection), pulsatility of arterial pressure and flow and central aortic pressure are better prognostic markers than conventional brachial blood pressure for assessment of stroke outcome. Specifically, this will involve measurement of arterial stiffness gradient to assess the amount of pulsatile energy generated by central hemodynamics and that is transmitted to the peripheral cerebral tissue leading to potential microvascular damage. These measures can provide additional information beyond brachial blood pressure to enable better alignment of patient and stroke characteristics and so improve management of stroke and mitigate cerebrovascular risk.
The effects of aortic pulsatility impact on the heart, brain, kidneys and other vascular beds, particularly in the elderly with elevated blood pressure, in who pulsatility is enhanced due to large vessel stiffness and is transmitted further distally along blood vessels into smaller arteries, affecting target organs to a greater degree. The brain, like the heart and the kidney, "sees" central blood pressure more directly than peripheral, brachial, blood pressure. Thus, central blood pressure could be a more accurate and direct reflection of the pathophysiological effects of elevated blood pressure on the brain than peripheral blood pressure. Indeed, central blood pressure appears to have a closer correlation with risk of stroke or other target organ damage than peripheral blood pressure. However, peripheral blood pressure has been the subject of millions person-years studies, many of them with hard end-points such as stroke, myocardial infarction, renal failure, death, compared to a few thousand patient studies for central pressure and some measure of target organ damage. Brachial pressure is measured around the world, and definitions, thresholds and goals have been established on the basis of huge numbers of data. Beyond controversies on the accuracy and reliability of central pressure measurements, there are nowhere equivalent numbers available for central pressure or the data needed to provide evidence-based directives for treatment of elevated blood pressure based on central pressure. Thus, a small advantage in risk prediction does not justify the adoption of measurement of central pressure over a proven method that is cheaper and generalized across the world, including in low- and middle-income countries with financially constrained health care systems.

McDonald lecture

THE HAEMODYNAMIC GENESIS OF HYPERTENSION

Phil Chowienczyk
Kings College London, UK

Hypertension has classically been attributed to an increase in peripheral vascular resistance. Such an increase in peripheral vascular resistance would increase mean and diastolic blood pressure but have little influence on pulse pressure. However, hypertension in our ageing society occurs mainly as a