



## Artery Research

ISSN (Online): 1876-4401

ISSN (Print): 1872-9312

Journal Home Page: <https://www.atlantis-press.com/journals/artres>

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### **5.2: CENTRAL PULSE PRESSURE: A POSSIBLE ROBUST MARKER OF THE CARDIAC HEMODYNAMIC LOAD**

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**To cite this article:** M. Odaira, H. Tomiyama, A. Yamashina (2013) 5.2: CENTRAL PULSE PRESSURE: A POSSIBLE ROBUST MARKER OF THE CARDIAC HEMODYNAMIC LOAD, Artery Research 7:3\_4, 168–168, DOI: <https://doi.org/10.1016/j.artres.2013.10.025>

**To link to this article:** <https://doi.org/10.1016/j.artres.2013.10.025>

Published online: 14 December 2019

**Conclusion.** A higher level of PAEE is associated with lower levels of aortic stiffness indicating that the beneficial effects of PA on CVD are partially mediated by aortic stiffness.

#### 4.5 FREQUENCY RESPONSE OF BLOOD PRESSURE CUFFS BASED ON STEP RESPONSE AND FORCED SINUSOIDAL HARMONIC EXCITATION

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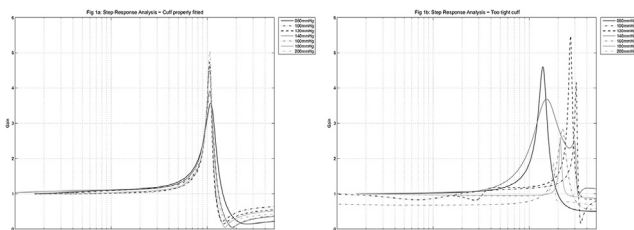
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**Background.** Pulse wave analysis (PWA) using cuff based methods emerged within the last years. Compared to traditional sensors used in PWA like catheters and piezo gauges very little is known on the frequency response of cuff based systems according to standard measurement and systems theory. Therefore the aim of this work is the investigation of the capability of blood pressure cuffs to register the dynamical behavior of the arterial pulse appropriately.

**Methods.** To evaluate the dynamic behavior of cuff based sensor chains we performed both tests on step response (similar to "pop test") as well as forced sinusoidal harmonic excitation by the means of a fully automated and standardized custom testing bench. The stepwise variation of cuff pressures and/or bladder volumes and cuff sizes was intended to account for various anatomical situations faced in clinical practice.

**Results.** The resonant frequencies of the evaluated systems are in the range of 110 Hz (Fig. 1a). Similar results have been obtained for both step response method and harmonic excitation. This behavior did not vary over a pressure range from 80 to 200 mmHg. Nevertheless we observed that pressure-volume relation had significant influence on the cuffs frequency response. In particular too tightly fitted cuffs lead to chaotic results (Fig. 1b). Too loosely fitted cuffs causes a loss in resolution.

**Conclusions.** Based on our actual data blood pressure cuffs provide acceptable capabilities to cope with the dynamics of the arterial pulse for PWA use. This is only valid for properly fitted and inflated cuffs.



### Oral Session 5

#### Invited Lecture and Free Communication Oral Presentations

In association with the Pulse of Asia

#### 5.1 VULNERABLE CAROTID PLAQUES ARE ASSOCIATED WITH THE DEVELOPMENT OF EARLY RESTENOSIS AFTER CAROTID ENDARTERECTOMY

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**Background.** Carotid endarterectomy (CEA) is widely recognised as an effective surgical method in both symptomatic and asymptomatic patients with significant internal carotid artery stenosis. Although the association between vulnerable carotid plaques and stroke is well established, little is known about their role in development of myointimal hyperplasia after carotid surgery. In the current study we aimed to establish correlation between plaque morphology (as observed by ultrasonic scan) and the degree of myointimal hyperplasia after CEA.

**Methods.** A total of 567 patients with a median age of  $65.0 \pm 5.6$  years who underwent CEA were examined using duplex ultrasound scanning prior to surgery and 12 months after. The morphology of plaques in terms of their echogenicity was graded as echolucent, predominantly echolucent, predominantly echogenic, echogenic, or calcified. The plaque surface was

categorized as smooth, irregular, or ulcerated. Chi-square test and multivariate logistic regression were used for statistical analysis.

**Results.** Internal carotid artery restenosis due to intimal hyperplasia  $\geq 50\%$  were detected in 67 patients (11,82%). The incidence of carotid restenosis was significantly higher in patients with ulcerated carotid plaques ( $P < 0.05$ ). There was no difference between rates of restenosis in patients who had plaques with smooth or irregular surface. Predominantly echolucent carotid plaque appeared to be an independent predictor of carotid restenosis in 12 months after CEA ( $P < 0.05$ ).

**Conclusion.** The results of our study suggest that vulnerable carotid plaques can lead to myointimal hyperplasia after CEA and can be considered as an independent predictor of early restenosis after carotid surgery.

#### 5.2 CENTRAL PULSE PRESSURE: A POSSIBLE ROBUST MARKER OF THE CARDIAC HEMODYNAMIC LOAD

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**Background.** No prospective study has examined whether markers of the central hemodynamics, e.g., central pulse pressure, might be a more robust marker of change in the cardiac hemodynamic load, estimated by the change during the study period of the serum levels of N-terminal fragment B-type natriuretic peptide (NT-proBNP), as compared to markers of the arterial stiffness or brachial blood pressure variables in subjects with preserved cardiac function.

**Methods.** The brachial-ankle pulse wave velocity, radial augmentation index (rAI), second peak of the radial pressure waveform, systolic and pulse pressure of the second peak of the radial pressure waveform (SP2 and PP2) and serum NT-proBNP level were measured at the start (first examination) and at the end (second examination) of this 3-year study in middle-aged healthy Japanese men ( $n = 1851$ ).

**Results.** A stepwise multivariate linear regression analysis demonstrated that only PP2, among the parameters related to arterial stiffness and central hemodynamics and also brachial blood pressure variables, was significantly associated with the serum NT-pro BNP levels in the subjects. Furthermore, only the changes of the PP2 during the study period, among the parameters related to arterial stiffness and central hemodynamics, were significantly correlated with those of the serum NT-pro BNP levels during the study period ( $\beta = 0.131, p < 0.001$ ).

**Conclusion.** Central pulse pressure, as reflected by PP2, may be a robust marker of the cardiac hemodynamic load and reflect changes in the cardiac hemodynamic load even in persons with preserved cardiac function.

**Keywords.** Natriuretic peptides, Central blood pressure, Cardiac hemodynamic load

#### 5.3 INORGANIC NITRITE, CONDUIT ARTERIES & CENTRAL BLOOD PRESSURE

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**Background.** Organic nitrates (e.g. nitroglycerin) are highly selective dilators of muscular conduit arteries. By contrast, the endogenous inorganic nitrite anion ( $\text{NO}_2^-$ ) is thought to be a hypoxia-dependent dilator of small resistance arterioles, via its reduction to vasodilating nitric oxide (NO) by deoxyhaemoglobin.

**Objective.** To establish selectivity of nitrite for resistance versus conduit arteries.

**Methods and Results** A series of forearm blood flow (FABF) studies were performed in healthy volunteers. Intra-brachial sodium nitrite ( $8.7 \mu\text{mol}/\text{min}$ ) markedly increased radial artery diameter (assessed using ultrasound) by  $37.6 \pm 9.7\%$  ( $P < 0.001$ ), with  $\text{HbO}_2 \sim 99\%$ . Furthermore, nitrite ( $0.087\text{--}87 \mu\text{mol}/\text{min}$ ) displayed similar selectivity as nitroglycerin ( $0.003\text{--}1 \mu\text{g}/\text{min}$ ) for conduit arteries, compared to resistance arterioles (FABF). Intravenous administration of sodium nitrite ( $8.7 \mu\text{mol}/\text{min}$ ) dilated the contralateral radial artery by  $10.7 \pm 1.8\%$  ( $P < 0.01$ ) and lowered central systolic blood pressure (BP) by  $\sim 12\text{mmHg}$  from  $98.3 \pm 12.3$  to  $86.7 \pm 15.1\text{mmHg}$  ( $P = 0.02$ ) without any change in peripheral BP; nitrite also reduced augmentation index and pulse wave velocity. In contrast to nitrite's effects on FABF, induction of hypoxia (breathing  $12\% \text{O}_2$ ) paradoxically inhibited nitrite-induced dilatation of the radial artery to a similar extent as hyperoxia/breathing  $100\% \text{O}_2$  (both  $P < 0.001$  compared to normoxia).