Sympathetic nervous system does not influence the cardiac contribution to the relationship between blood pressure and pial artery pulsation oscillations in healthy volunteers

Marcin Gruszecki[1], Pawel J. Winklewski[2], Yurii Tkachenko[3], Kamila Mazur[4], Jacek Kot[3], Wojciech Guminski[5], Krzysztof Czuszynski[4], Jerzy Wtorek[4], Andrzej F. Frydrychowski[2]

[1]Department of Radiology Informatics and Statistics, Medical University of Gdansk
[2]Institute of Human Physiology, Medical University of Gdansk
[3]National Centre for Hyperbaric Medicine, Institute of Maritime and Tropical Medicine, Medical University of Gdansk
[4]Department of Biomedical Engineering, Gdansk University of Technology
[5]Department of Computer Communications, Gdansk University of Technology

1. Introduction.

Evidence accumulates that variation in cardiac output either acutely or chronically leads to a change in cerebral blood flow [Brassard et al. 2015, Meng et al. 2015]. However, the mechanisms linking cardiac output with brain haemodynamic remains largely unrecognized. The aim of this study was to assess changes in heart generated relationship between blood pressure (BP) and subarachnoid space width (SAS) oscillations. Based on our previous experiments [Winklewski et al. 2015a, 2015b] we speculated that sympathetic nervous system (SNS) may tend to stabilize BP-SAS coupling in extreme conditions while do not affect the relationship between these signals during more physiological stimuli. Therefore, we hypothesised that both handgrip test (HGT) and cold test (CT) would not affect the cardiac contribution to the relationship between BP-SAS oscillations, regardless of the fact that the stimuli evoked by the tests are likely transmitted by different central sympathetic circuits.

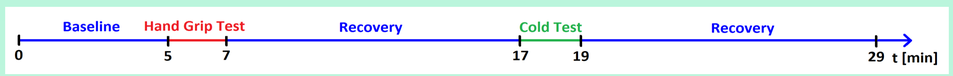
2. Experiment.

Volunteers

Experiments were performed on a group of 20 healthy volunteers (6 females; age 28.5±7.5 years; BMI = 24.2±3.6 kg/m2); none of them were smokers. None of the participants suffered from known disorders or were taking any medication, a general and neurological examination was performed before the experiment. Nicotine, coffee, tea, cocoa and methylxanthine-containing food and beverages were not permitted for 8 hours before the tests.

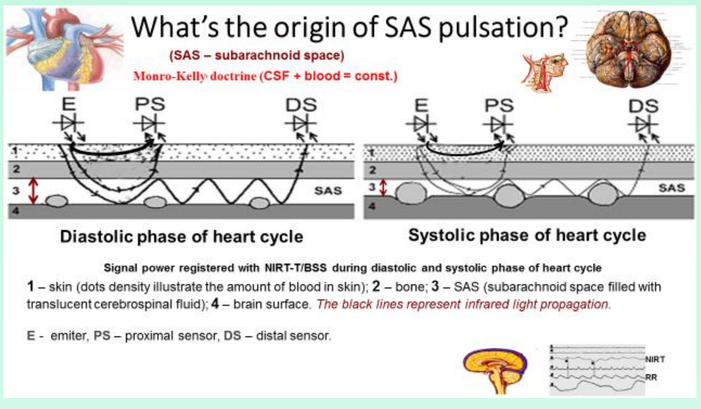
Experimental design

All tests were conducted in a comfortable quiet room with a comfortable temperature.



Measurments

Respiratory rate, minute ventilation, end-tidal $EtCO_2$ and end-tidal EtO_2 were measured using a metabolic and spirometry module of the medical monitoring system. Oxyhaemoglobin saturation (**SaO_2**) was measured continuously with a finger-clip sensor. **Cerebral blood flow velocity** (**CBFV**) was measured using Doppler ultrasound of the left internal carotid artery. **Heart rate** (**HR)** and **BP** were recorded using a finger-pulse photoplethysmograph. **SAS** was measured using a Near-Infrared Transillumination Backscattering Sounding (NIR-T/BSS).



Schematic description of mechanism underlying rapid changes in the subarachnoid space width. During heart systolic phase pial artery are filled with blood, cerebrospinal fluid (CSF) is pushed to spinal part, and the subarachnoid space width decrease. The reverse happens during diastolic heart phase (less blood in pial artery, CSF is back from spinal part and the subarachnoid space becomes wider).

3. Analysis.

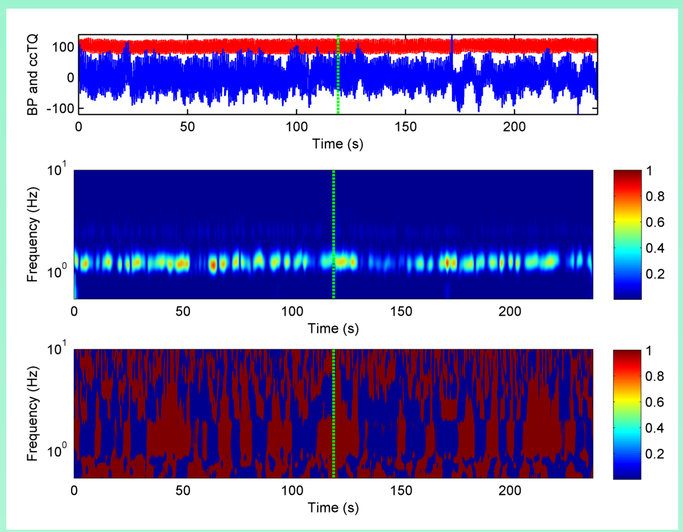
Wavelet transform analysis was used to assess the relationship between BP and SAS oscillations. Wavelet coherence (WCO) and wavelet phase coherence (WPCO) were estimated with Morlet function as a mother wavelet.

Wilcoxon signed-rank test was used to compare the changes in all measured variables.

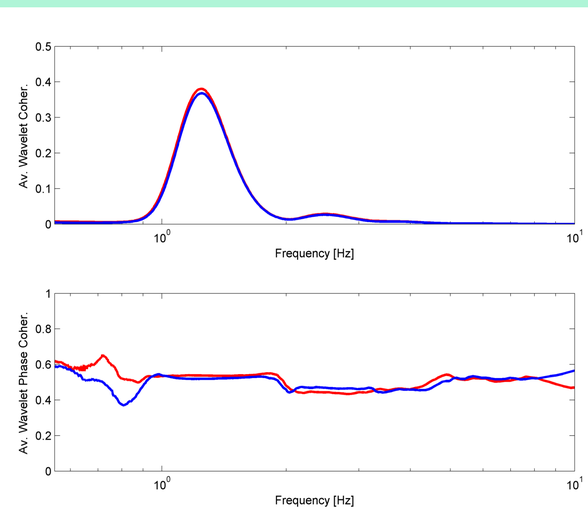
5. Results.

HGT evoked an increase in BP (+15.9%; P<0.001), HR (14.7; P<0.001), SaO_2 (+0.5; P<0.001) EtO_2 (+2.1; P<0.05), while SAS was diminished (-8.12%; P<0.001). CBFV (+2.9%; NS) and $EtCO_2$ (-0.7; NS) did not change during HGT.

CT evoked an increase in BP (+7.4%; P<0.001), SAS (+3.5%; P<0.05) and SaO_2 (+0.3%; P<0.05). HR (+2.3%; NS), CBFV (+2.0%; NS), EtO_2 (-0.7%; NS) and $EtCO_2$ (+0.9%; NS) remained unchanged.



Representative WCO (middle panel) and WPCO (lower panel) tracings of Cold Test. BP (red) and SAS (blue) signals are provided in the upper panel. The green dashed line separate baseline and cold test period.



Time average of wavelet coherance and wavelet phase coherance estimated for 120s baseline before cold test (red line) and 120s of cold test (blue line).

6. Conclusions.

There were two main findings of the study:

1) Short sympathetic activation does not affect the cardiac contribution to the relationship between BP—SAS oscillations,
2) HGT and CT display divergent effects on the width of the subarachnoid space.

We believe that combination of NIR-T/BSS with advanced signal analysis tools most likely represents a promising approach in describing the interrelations and pathways involved in heart failure, obstructive sleep apnoea and related cerebrovascular diseases. The presented results establish therefore reference for future clinical studies which are warranted. We have shown that SNS activation does not affect the cardiac and respiratory contribution to the relationship between the BP and SAS oscillations in healthy subjects. In fact, it seems that a high sympathetic drive tends to stabilise the relationship between the analysed signals.

Acknowledgments: MG is supported by the Alumni Programme founded by the Royal Society.