Pulsatility Index compared to Pulsatile apparent Resistance in healthy subjects during hyperventilation and CO2-retention.

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Introduction

Introduction:
Cerebral autoregulation can be observed by changes in middle cerebral artery blood flow velocity (MCAFV) during CO2-provocations. Besides flow velocity changes, also changes in pulse morphology can be observed. We aim to improve the understanding of cerebral autoregulation by studying pulse morphology. Often the pulsatility index (PI) is used to express the changes. But PI is dependent on heart rate (HR) and PI does not express the MCAFV pulse morphology. To quantify the pulse morphology, we attempted to correct PI for arterial blood pressure (ABP), by calculating the vascular Pulsatile apparent Resistance (PaR).

Prototype of the PaR instrument

Aim

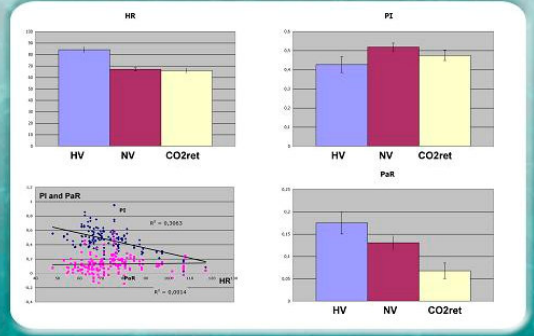
Aim:
Comparison of PI and PaR during several CO2 provocations.

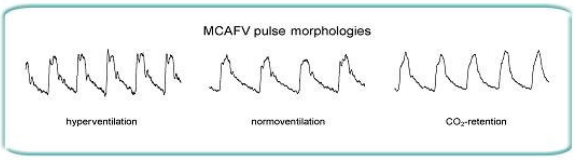
Methods

Methods:
In this study we compare values for Pulsatility Index (PI) and Pulsatile apparent Resistance (PaR) measured in a group of normal subjects. Informed consent was obtained from all subjects, all tests performed were approved by the local medical ethical committee.

In both middle cerebral arteries MCAFV was measured with a transcranial Doppler instrument (TCD). ABP was measured by means of the finger cuff method at the middle finger using a continuous blood pressure measuring device. Exhaled air CO2-concentration was measured with a capnograph and converted to end-tidal values (ETCO2). From these measurements the PI and PaR values were calculated. Measurements were performed during normoventilation (NV), hyperventilation (HV), and CO2 retention (CO2ret).

Changes observed in HR, PI and PaR during CO2 provocations





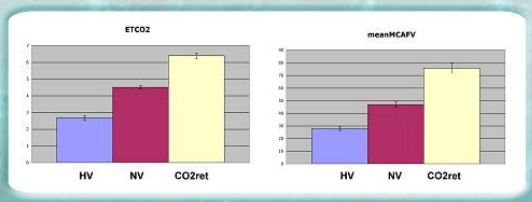
The morphology of the blood flow velocity pulses changes during CO2 reactivity testing.
PI expresses the variation of flow velocity with respect to the mean value:

$$PI = \frac{sMCAFV - dMCAFV}{mMCAFV}$$

PaR expresses the within heart beat variation of vascular resistance with respect to the mean value:

$$PaR = \frac{\frac{dABP}{dMCAFV} - \frac{sABP}{sMCAFV}}{\frac{mABP}{mMCAFV}}$$

s: systolic d: diastolic m: mean



Results

Results:
All bar graphs represent mean values of about 30 individuals (15 male, 15 female, aged 14 - 76 years), error bars indicate SEM. As was intended, the ETCO2 changed significantly during HV and CO2ret with respect to NV. As was expected, the mean MCAFV increased significantly with the increase of ETCO2. The mean MCAFV is strongly related to the ETCO2. Our subjects, performing CO2 provocations were able to vary their MCAFV with 50% with respect to normal values. Relative changes during CO2-provocations, may amount upto 50% of the values during normoventilation.

Response of the cerebral autoregulation system can be observed as a variation in of mean MCAFV, but change of the HR is an additional effect. Hyperventilation increases the heart rate (lowering PI) and decreases the mean MCAFV (elevating PI), these are opposite effects, causing a small decrease in PI. During CO2 retention mean MCAFV increases, resulting in a non-significant decrease of PI. We think that the balance between blood pressure and cerebral blood flow (i.e. cerebral autoregulation) is better monitored by including a continuous measurement of ABP. Both signals can be integrated to the new parameter PaR. Our healthy subjects showed a variation of ≥ 35% in PaR-values as response to CO2 provocations. The mean value of PaR measured was 0.13 ± 0.09 (range -0.06 - +0.45)

Conclusion

Conclusion:
Pulsatility of vascular resistance can well be expressed by PaR. In healthy subjects PaR changes in order to adapt the cerebral vascular bed to the changed CO2-exchange needed. Impaired cerebral autoregulation is indicated by low PaR values.

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A project of three companies aiming to make PaR technology commercially available has recently received the Eureka label.