Quantification of carotid artery blood flow before and after the acetazolamide challenge

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Introduction: The brain’s arterial input plays an important role in parenchymal perfusion, autoregulation and function. Cerebrovascular reserve may be a crucial physiological mechanism which provides increased arterial flux when necessary. It is believed that this can be assessed by a vasodilator stress-test. The objective of this work was to quantify blood flow within the internal carotid artery (ICA) in a normal, healthy, adult cohort, before and after the administration of a known potent pharmacological vasodilator, acetazolamide (a carbonic anhydrase inhibitor).

Methods: The study cohort consisted 14 subjects (7 male and 7 female) without any history of vascular disease (mean age±stdev = 26±6yrs). Imaging was performed at 3T (Achieva 3.0T, Philips Medical Systems, Best, Holland). Flow assessment was performed on each subject before and 30min after injection of 1000mg acetazolamide (Diamox sodium perenteral; Wyeth Laboratories, Maidenhead, UK) which was infused intravenously over 10min, followed by a saline flush. A standard, SENSE-enabled, 8-channel array head coil was used for quantitative flow assessment (Q-Flow, Philips) which was based on a single-slice, multi-phase, fast-field echo sequence encoding flow parallel to the slice-encode direction, approximately 3cm distal to the carotid bifurcation [fig 1a]. The phase contrast cine sequence (TE = 3.7ms; TR = 7.7ms; field echo single shot interval = 16ms; flip angle = 10°; in-plane acquisition resolution = 1.72mm x 1.60mm, interpolated to 0.86mm x 0.86mm; slice thickness = 5mm) sampled k-space in a continuous, linear ordered, phase-encoded scheme. Centrally acquired vector ECG was used to retrospectively gate 40 time points over the cardiac cycle. A constant velocity encoding factor (120cm/s) was used for all subjects. Errors bought about by second-order residual background phase offsets were minimised by the inclusion of low-pass filtering and concomitant gradient correction techniques. In addition to magnitude (anatomical) field echo images, subtraction between the 2 datasets yielded phase images plus calculated flow maps. Quantitative flow-encoded information was to be extracted via region of interest (ROI) analysis [Fig 1b]. Statistical tests were performed using paired t-tests.

Results: Group mean blood velocity significantly increased following acetazolamide (V_{PRE}=16.8 ± 2.1cm/s; V_{POST}=20.5 ± 3.7cm/s; P<0.005). Group mean blood flux (velocity x area) also highly significantly increased following acetazolamide (φ_{PRE}=5.16 ± 0.79ml/s; φ_{POST}=7.61 ± 1.18ml/s; P<0.001) [Fig 2a]. Mean heart rate did not vary between pre- and post- acetazolamide (HR_{PRE}=59 ± 9 beats/min; HR_{POST}=60 ± 9 beats/min; P=0.582), however, the ROI size (encompassing all forward flow observed on the phase-contrast images) was greater (Area_{PRE}=42 ± 7 pixels; Area_{POST}=51 ± 9cm/s; P<0.005). No gender differences were observed (P>0.05). The group mean, subject-normalised Volumetric Flow Rate (VFR) waveforms are depicted in Figure 3.

Discussion: This study demonstrates marked changes in ICA blood velocity and flux over the cardiac cycle due to administration of a potent vasodilator (acetazolamide) in a group of normal young adults. Interestingly, a greater level of statistical significance was observed in increased blood flux than in increased blood velocity. This is presumably due to localised increase in macrovascular lumen diameter and is not just a result of increased cardiac output. Indeed, the mean post-acetazolamide ROI was significantly larger and no significant elevation in heart rate was observed following acetazolamide infusion, suggesting that macro- as well as the micro-intracranial vasculature responds to the acetazolamide challenge.