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

J. M. Reeve[1], D. Selvarajah[2], N. Craig[1], P. D. Griffiths[1], S. Tesfaye[2], and I. D. Wilkinson[1]
[1]Academic Radiology, University of Sheffield, Sheffield, S Yorkshire, United Kingdom, [2]Diabetes, Royal Hallamshire Hospital, Sheffield, S Yorkshire, United Kingdom

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-chanel 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 = 10^0; 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.

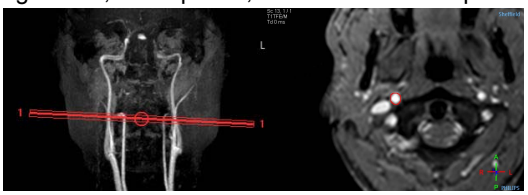

Fig 1. a) 2D Flow slice position perpendicular to the long-axis of the right ICA and (b) example ROI.

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.

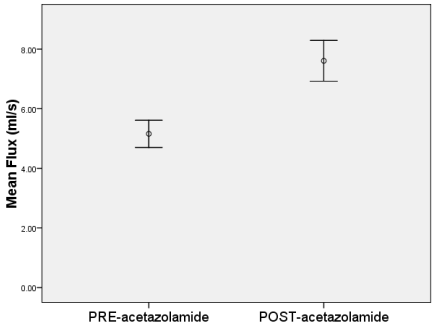

Figure 2. Group mean blood flux pre- and post- acetazolamide (paired t-test, P<0.001).

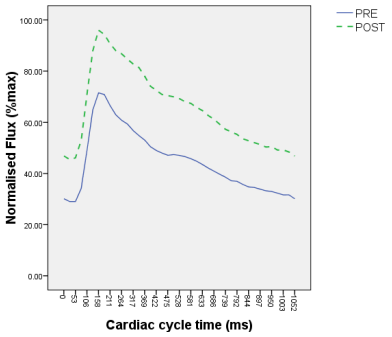

Figure 3. Effects of acetazolamide on group mean VFR waveform.

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.