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Hyperventilation-induced reduction of cerebral blood flow measured with pseudo-continuous arterial spin labeling (pCASL)

J. Debatisse[1,2,3], C.J. McGinnity[1,4], S.N. Yaakub[1,4], S. Jeljeli[4], J. Stirling[5], M. Koutroumanidis[5], G. Charles-Edwards[6], E. De Vita[6] and A. Hammers[1,4]

[1]School of Biomedical Engineering and Imaging Sciences, King's College London, London SE[1 7]EH, UK

[2]Univ Lyon, CarMeN Laboratory, INSERM, INRA, INSA Lyon, Université Claude Bernard Lyon [1], Lyon, France

[3]Siemens Healthcare SAS, Saint-Denis, France

[4]King's College London & Guy's and St Thomas' PET Centre, St Thomas' Hospital, London SE[1 7]EH, UK

[5]Department of Clinical Neurophysiology and Epilepsy, Guy's and St Thomas' NHS Foundation Trust, London, UK

[6]Department of Biomedical Engineering, Centre for Medical Engineering, School of Biomedical Engineering & Imaging Sciences, King's College London, King's Health Partners, St Thomas' Hospital, London, SE[1 7]EH, UK

Abstract

Objectives

The cerebral vasculature adapts cerebral perfusion to variations in ventilatory volume and composition of inspired gases. Modulation of respiration induces a vasomotor response resulting in quantitative changes in cerebral blood flow (CBF). Hyperventilation induces hypocapnia by increasing elimination of carbon dioxide, leading to a cerebral vasoconstriction, with invasive measurements reporting a ~30% decrease in CBF (Kety and Schmidt, 1946). Here we quantify the effect of hyperventilation on CBF using pseudo-continuous arterial spin labeling (pCASL).

Methods

Eleven participants (median ± interquartile range age 31 ± 16.5, three males) were studied on a 3 T Siemens Healthcare PET-MR system. 3D T1-weighted MPRAGE (1 mm^3 voxels) was used for co-registration and region-of-interest (ROI) identification using FSL-FAST (https://fsl.fmrib.ox.ac.uk/fsl/fslwiki/).

Normal breathing and 3-min hyperventilation (HV) intervals were interleaved. HV consisted of paced breathing at a rate of 30-breaths/minute, as per standard clinical electroencephalography (EEG) practice, with continuous auditory cue (i.e. “in, out, in, out…”).

Asl

was acquired using pCASL (Wu, 2007): 2-shots, 4 × 4 × 5.5 mm^3 resolution, TR = 4.5 s, TE 17.32 ms, 1.8 s labeling, 2.1 s post-labeling delay; 1 M0 image and 12, 10, and 7, 18 tag-control pairs for preHV (4 min), HV, rest (2 min) and post-HV (6 min) intervals respectively. Data was distortion corrected using FSL-topup.

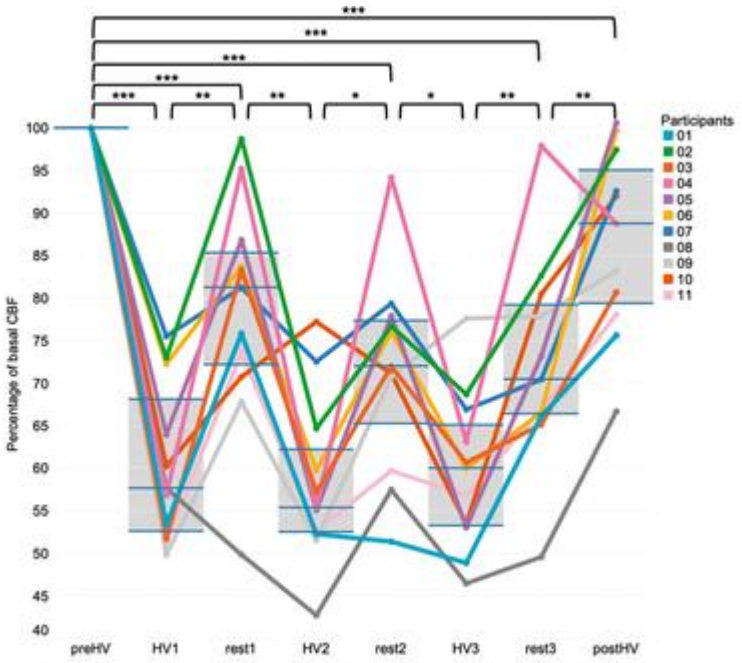
Perfusion quantification used FSL Oxford-ASL v3.9.13 (Chappell, 2009), co-registered to the structural space. GM CBF measurements were extracted and averaged across each interval (rest or HV). Paired t-test were used to assess significance of inter-interval CBF differences.

Results

Good compliance with the task was observed, with marked EEG slowing. Hyperventilation significantly lowered CBF measurements with a median decrease of 42% in GM (IQR = 16%) after HV1. After 2 min of rest, CBF did not return to its basal value and was 19% lower than baseline. The following sessions of HV induced a similar decrease compared to the basal CBF value (i.e. median ± IQR; 45 ± 10% for HV2; 40 ± 12% for HV3). A long-term effect of hyperventilation is observed on the rest sessions, with a 28% and 30% decrease in CBF in rest2 and rest3 relative to baseline, respectively. Eight minutes after the last HV phase, CBF was still 11% lower than its basal value.

Discussion

We confirm in this study the detection of hyperventilation-induced decrease of absolute CBF with pCASL MRI (Tancredi, 2013), noting marked between-subject variability. Decreases persist for several minutes after the cessation of overbreathing. Our hyperventilation – pCASL protocol with simultaneous PET-MR holds promise as a method to study the neurochemistry of seizures (Bartenstein, 1993).



Evolution of CBF changes during the task in grey matter. CBF was normalized to the pre-hyperventilation basal CBF value and expressed as percentage of basal CBF for each participant. Distribution bands represent median (middle line), first quartile (upper line) and third quartile (lower line). Paired t-test, 10 degrees of freedom, *** p<0.0001; ** p<0.001; * p<0.01

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