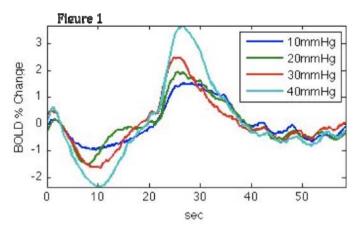
Effects of the ValSalva Maneuver and Hypercapnia on the BOLD signal

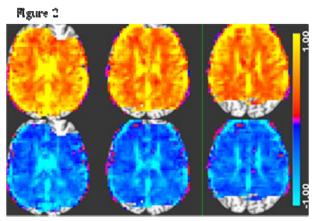
D. A. Handwerker¹, P. Wu^{1,2}, R. M. Harper³, and P. A. Bandettini^{1,4}
¹Section on Functional Imaging Methods, National Institute of Mental Health, Bethesda, MD, United States, ²Neuroscience, University of California, Los Angeles, Los Angeles, CA, United States, ³Neurobiology, David Geffen School of Medicine, UCLA, Los Angeles, CA, United States, ⁴Functional MRI Facility, National Institute of Mental Health, Bethesda, MD, United States

Introduction: Hypercapnia is frequently used to create global changes in cerebral blood flow, volume, and oxygenation that can be measured with fMRI. Several BOLD signal calibration methods use hypercapnia (1-4). The most controlled method for inducing hypercapnia requires altering the CO₂ concentration of inhaled air, but breath-holding based methods are also used. A few studies have directly compared breath-holding to CO₂ breathing (5). While breath-holding is easier to implement, a potential confound is that hypercapnia may induce changes other than just flow/oxygenation alterations (6). Thomason et al suggest that breath holding may also include a Valsalva maneuver. Valsalva-related changes in thoracic chest pressure can also alter cerebral blood flow. We examine this hypothesis by having volunteers perform Valsalva maneuvers at different chest pressures while in an MRI.

Methods: Data were collected from 9 volunteers on a GE 3T HDx scanner with a 16-element receive-only brain array coil under an IRB approved protocol. Single-shot EPI using an ASSET factor=2 were collected. (TR=400ms, TE=30ms, flip angle=30, FOV=24cm, 64X64 grid, 8 oblique slices where 4mm with 0.5mm gap). Volunteers alternated 39s of paced breathing with 20s of breath hold. They breathed through a mouth tube that was connected to an air pressure transducer. and could continuously monitor air pressure levels in the tube. During breath-hold periods for each trial, they were told to exhale to maintain a constant pressure of 10, 20, 30, or 40mmHg, or to just hold their breath after a final inhalation or exhalation. When volunteers increased chest pressure, they would be performing a Valsalva maneuver. They also performed breath-hold durations at 5, 10, 15, 20, and 25s with a 30mmHg pressure. Trial-averaged responses to each pressure level were created for each voxel.

Results: Performing a Valsalva maneuver at different thoracic chest pressures produced different BOLD signal time courses, even when the duration of the breath hold and the pattern of visual stimulation was held constant. Figure 1 shows BOLD time courses from voxels near the calcarine sulcus, averaged across 6 volunteers. Both the initial undershoot and following peak scale with chest pressure. A similar undershoot and overshoot was observed for all duration breath holds, including 5s. Figure 2 shows a mean map of the slopes of the parametric variation in undershoot (bottom row) and peak magnitudes (top row) across the 4 pressure values from 9 subjects' brains normalized to Talairach space. A clear relationship emerged between Valsalva pressure and magnitude of the rapid decrease as well as the slower increase in signal. The largest changes appeared in CSF and gray matter.





Conclusions: We show a parametric relationship between chest cavity pressure and the magnitude of the rapid decrease and slower increase in signal. Because the precise contrast mechanisms behind these changes remain unclear, these effects may be a confound in calibration studies, or may be a novel and rapid way to induce calibration-useful global BOLD signal changes. Future work characterizing the effects of selective nulling of CSF, gray matter, and blood components during the Valsalva maneuver is ongoing to separate these effects.

References:

- R. D. Hoge et al., Proc Natl Acad Sci U S A 96, 9403 (Aug 3, 1999). 1.
- E. R. Cohen et al., Neuroimage 23, 613 (Oct, 2004). 2.
- P. A. Bandettini, E. C. Wong, NMR Biomed 10, 197 (Jun-Aug, 1997). 3.
- D. A. Handwerker, A. Gazzaley, B. A. Inglis, M. D'Esposito, Hum Brain Mapp 28, 846 (Sep. 2007). 4.
- 5. A. Kastrup, G. Kruger, T. Neumann-Haefelin, M. E. Moseley, Magn Reson Imaging 19, 13 (Jan, 2001).
- 6. M. E. Thomason, G. H. Glover, *Neuroimage* 39, 206 (Jan 1, 2008).