Quantification of Venous Vessel Size in Human Brain in Response to Hypercapnia and Hyperoxia

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Introduction

Both hypercapnia and hyperoxia influence the vessel size and induce vasodilation and vasoconstriction, respectively [1-2]. Quantitative measurement of vasodilation and vasoconstriction is a technical challenge. In this study, we measure the vasodilation and vasoconstriction non-invasively using the blood oxygenation level dependent (BOLD) contrast that is induced respectively by breathing hypercapnic and hyperoxic gas to explore the changes in R_2^* and R_2 that depend on the vessel radius [3-4]. We investigate the influence of hypercapnia and hyperoxia on the venous vessel radius in human brain and characterise the difference of venous vessel radius between hypercapnia and hyperoxia.

Materials and Methods

Eight healthy volunteers (ages: 21 - 37; 5 male) were scanned following informed consent. A specially designed unidirectional breathing circuit (Intersurgical Ltd, Wokingham, UK) was used to deliver either room air or 6% CO_2 (100% O_2). The experimental paradigm consisted of two 3-minute blocks of breathing 6% CO_2 (100% O_2) interleaved with three 2-minute blocks of breathing room air. Physiological parameters (heart rate, arterial oxygen saturation, end-tidal CO_2 and O_2) were continuously monitored. On a Philips Achieva 3T MR system (Philips Medical Systems, Best, The Netherlands) a single-shot dual echo EPI sequence was used for simultaneous acquisition of GE and SE EPI images. Imaging parameters were: TR = 3.5 s, TE_{GE}/TE_{SE} = 30/90 ms, flip angle = 90°, FOV = 200x200 mm^2, matrix = 68x67, number of slices = 28, slice thickness = 3 mm, slice gap = 1 mm. A total of 206 dual-echo volumes were acquired over a period of 12 minutes. Additionally, a T_1-weighted TFE sequence was used to acquire a high-resolution structural images with TR = 8.2 ms, TE = 3.8 ms, flip angle = 8°, matrix = 240x240x160 and FOV = 240x240x160 mm^3. Images were processed and analysed using the SPM8 software package (http://www.fil.ion.ucl.ac.uk/spm) and Matlab (The Mathworks, Natick, MA, USA). GE and SE EPI volumes were spatially normalised (MNI standard space) and smoothed using an 8 mm FWHM Gaussian kernel. Parametric maps of ΔR_2^*, ΔR_2 and $q = \Delta R_2^*/\Delta R_2$ were calculated on a pixel-by-pixel basis. The calculated q values were converted into vessel radii using a corresponding calibration curve that was derived individually based on the physiological parameters of hypercapnia and hyperoxia using a previously described biophysical model [5].

Results

Figure 1 shows axial cross sections through the parametric map of mean venous vessel radius (R), averaged from 8 subjects in MNI standard space, in hypercapnia (top row) and hyperoxia (middle row). In addition, a z-score map is shown at the bottom of the figure to identify statistically the vessel size difference between hypercapnia and hyperoxia. A positive z-value means that the venous vessel radius in hypercapnia is larger than that in hyperoxia while a negative z-values means that the vessel size in hypercapnia is smaller than that in hyperoxia. A threshold is used for z-score map where $z \geq 2$ and $z \leq -2$ are shown in the colour coded z-map. A different contrast is clearly shown in the colour coded mean venous vessel size maps between hypercapnia and hyperoxia, meaning that the venous vessel size in hypercapnia is largely different from that in hyperoxia. The quantitative z-map demonstrates that most z-values are larger than 2 while only a few z-values are less than -2, illustrating that the venous vessel radius in hypercapnia is profoundly larger than that in hyperoxia. The individual mean venous vessel radii in hypercapnia and hyperoxia for grey matter (GM) and white matter (WM) are shown in Table 1. The individual and group average results show that the venous vessel radii were larger in hypercapnia than these in hyperoxia for both grey and white matter.

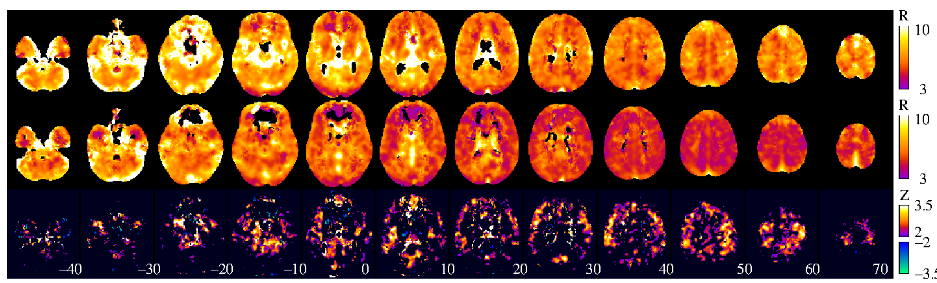

Figure 1. Parametric maps of the mean venous vessel radius (μm), averaged from 8 subjects in MNI standard space, in hypercapnia (top) and hyperoxia (middle). The vessel size difference between hypercapnia and hyperoxia is shown statistically in a z-score map (bottom).

	Hypercapnia		Hyperoxia	
Subject	R_{GM} (μm)	R_{WM} (μm)	R_{GM} (μm)	R_{WM} (μm)
1	7.6 ± 4.5	7.0 ± 3.0	5.5 ± 2.3	5.4 ± 2.0
2	7.3 ± 3.5	6.5 ± 2.0	5.4 ± 1.7	5.0 ± 1.5
3	7.7 ± 4.3	7.3 ± 2.7	5.9 ± 2.4	5.7 ± 1.9
4	7.3 ± 4.1	6.6 ± 2.4	5.6 ± 2.8	5.4 ± 2.5
5	6.8 ± 3.3	5.7 ± 1.8	5.4 ± 2.1	5.3 ± 1.7
6	7.4 ± 3.8	6.4 ± 2.3	5.7 ± 2.6	5.4 ± 2.1
7	7.5 ± 3.4	6.9 ± 2.3	5.6 ± 2.3	5.3 ± 1.5
8	7.0 ± 3.5	6.2 ± 1.9	5.6 ± 2.0	5.3 ± 1.8
Mean ± SD	**7.3 ± 0.3**	**6.6 ± 0.5**	**5.6 ± 0.2**	**5.4 ± 0.2**

Table 1. Venous vessel radius in grey matter (GM) and whiter matter (WM), obtained in hypercapnia and hyperoxia from 8 subjects (R (hypercapnia) > R (hyperoxia), p < 0.005).

Conclusion

We have shown that it is feasible to quantify the mean venous vessel size in hypercapnia and hyperoxia by breathing either 6% CO_2 or 100% O_2 that both can induce BOLD contrast which is used in the exploration of the mean venous vessel size in an MRI voxel. Our data show that the venous vessel radius is larger in hypercapnia than that in hyperoxia, indicating vasodilation and vasoconstriction effects induced respectively by hypercapnia and hyperoxia. Our results suggest that an exposure to hypercapnia incurs a very different physiological impact from an exposure to hyperoxia on regulating blood supply through the effective change of blood vessel size.

References

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