Cerebrovascular Reserve Impairment is associated with Recurrent Events in Patients with Carotid Artery Occlusion

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Introduction

Cerebrovascular reserve (CVR) measurements have been shown to be useful in predicting haemodynamic impairment and risk of future ischaemic events in patients with carotid artery disease[1, 2]. CVR can be assessed by using a vasodilatory stimulus such as acetazolamide or carbon dioxide (CO_2), whereby the residual vasodilatory capacity of resistance vessels is probed. CVR measurements provide reliable information about the ability of the circulation to increase blood flow in response to demand, and any preexisting vasodilation will reduce the reserve capacity. Identification of patients with carotid artery disease and a significant haemodynamic impairment is important for selection for vascular intervention such as carotid endarterectomy/ stenting or for specifically carotid artery occlusion extracranial to intracranial bypass surgery. We have recently shown good reproducibility for a non-invasive technique to map CVR capacity using hypercapnia fMRI[3], and also confirmed its effect on patients with carotid artery disease[4]. The aim of this study was to investigate the CVR of patients with carotid artery occlusion in relation to the presence of recurrent symptoms.

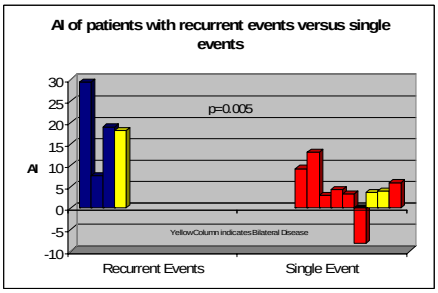
Methods

13 patients with symptomatic (2 stroke, 11 TIA) internal carotid artery occlusion were scanned using a clinical 1.5T Intera (Philips, Best, Netherlands) MR scanner. In the contralateral ICA, there was one artery with 70-99% stenosis, two with 80-95% and the rest did not have any significant disease. 9 patients had only a single symptomatic event and the remaining 4 patients had recurrent symptoms following their initial episode. Using a standard non-rebreathing anaesthetic circuit, 8 % carbon dioxide (CO_2) was administered to achieve transient episodes of hypercapnia; and the patients physiological parameters were continuously measured (blood pressure, pulse and arterial oxygen saturations). All data was analysed using FSL software, this method has been refined over the past year. High resolution MPRAGE images were acquired in all patients and segmented into grey and white matter, this process ensured that areas of severe infarction and encephalomalacia were removed from the analysis. Before the time-series analysis all the functional images were preprocessed using motion correction, high-pass filtering and smoothing. The statistical threshold for the CO_2 parametric maps was uncorrected $p<0.05$. To quantify the relative signal change for each patient the mean % signal change(%SC) following CO_2 stimulation was extracted from the grey matter of the MCA territory of each hemisphere, with the %SC being taken from the thresholded contrast maps and not normalised to CO_2. The degree of side-to-side asymmetry in the brain was calculated using the Hemispheric Asymmetry Index ($AI = 200*(CVR_{nonoccl} - CVR_{occl})/(CVR_{nonoccl} + CVR_{occl})$) for all patients.

Results

There was no significant change in the blood pressure and oxygen saturations throughout the paradigm. The mean %SC in the MCA territory distal to the occlusion [2.50% (SD 0.32)] was significantly lower than the mean %SC in the contralateral MCA territory [2.740% (SD 0.44)] (Paired T test, p=0.007). There was no significant difference between recurrent symptoms versus single symptoms GM MCA CVR. In the 4 patients with recurrent symptoms, the mean AI was 18.5 compared with 4.3 for those patients with only single events (Independent T-Test, p=0.005) (Figure 1,2).

Figure 1 – Graph of AI in patients comparing recurrent versus single events. Note – Yellow indicates bilateral carotid disease



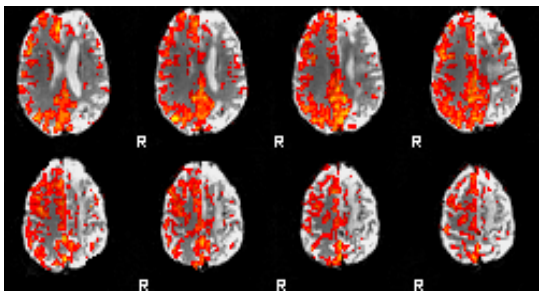


Figure 2 – CVR map from patient with left carotid artery occlusion and recurrent symptoms. Note – decreased CVR in left hemisphere.

Conclusion

Measurement of CVR capacity using CO_2 reactivity can provide important information on the haemodynamic status of patients with carotid artery occlusion. Our findings confirm previous work looking at impaired cerebrovascular reserve in patients with carotid artery occlusion[5]. In patients with recurrent symptomatic episodes we found a significantly increased asymmetry with (AI~18) ipsilateral reduced CVR. It is tempting to speculate that these recurrent events may be haemodynamically explained by reduced CVR in relation to the degree of compensatory vasodilatation during rest.

References

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