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Are Thalamic Aphasia and Neglect Due to Cortical Hypoperfusion?

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Introduction

Although aphasia and hemispatial neglect are classically designated as ‘cortical’ deficits, language deficits or hemispatial neglect following lesions to subcortical regions have been reported in many studies (e.g., Weiller et al., 1993). However, whether or not the cause of aphasia and hemispatial neglect is due to subcortical lesion has been a matter of controversy. Previous work in the lab has shown that most cases of aphasia or hemispatial neglect due to acute subcortical infarct can be accounted for by concurrent cortical hypoperfusion (Hillis et al., 2002). It should be noted that in the Hillis et al., 2002 study, lesions were mostly restricted corona radiata and caudate. Thalamic lesions were not included.

The primary objective of this study was to determine if aphasia or neglect occurs after acute thalamic stroke without cortical hypoperfusion detected with dynamic contrast perfusion weighted imaging (PWI).

Methods

We tested a series of 1471 patients on a battery of language tests (naming, comprehension, and repetition) for left hemisphere stroke patients or hemispatial neglect (line cancellation, a gap detection test, copying a scene) and visual and tactile extinction for right hemisphere stroke patients and obtained MRI scans within 48 hours of onset of stroke. Of these patients, we identified 9 patients with isolated left thalamic infarcts and 11 patients with isolated right thalamic infarcts who had concurrent perfusion scans. Volume of infarct and hypoperfusion were measured using Perfuse or ImageJ.

Results and Discussion

There was no association between naming impairment and left cortical hypoperfusion by chi square ($X^2=0.03; p=0.86$). 4/9 patients with left thalamic lesion had naming deficits but no cortical hypoperfusion. 2/9 patients had cortical hypoperfusion but no naming deficits. One patient with cortical hypoperfusion had auditory comprehension deficits. Error rate in comprehension were not correlated with volume of infarct or volume of hypoperfusion. Only 2/11 patients with right thalamic infarcts had hemispatial neglect, and both had cortical hypoperfusion ($X^2=11; p=.001$). Deviation to the right on line bisection (indicating severity of neglect) was correlated with volume of thalamic lesion (r$ho=.61; p=.04$) and volume of cortical hypoperfusion (r=.67; p=.02).

Results suggest that left thalamic strokes can cause naming impairment (perhaps via diaschisis)
irrespective of lesion volume or cortical hypoperfusion. However, thalamic neglect may be largely due to cortical hypoperfusion. This provides further evidence that the thalamus (particularly the intralaminar nucleus and pulvinar) either directly or indirectly participates in language based on lesion studies in both animals and humans and functional imaging. To further explore the relationship between thalamus and language processing, we are investigating fMRI and DTI data in left thalamic stroke patients with and without cortical hypoperfusion.

References
