



Corpus callosum morphology in children who stutter

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ABSTRACT

Multiple studies have reported both functional and neuroanatomical differences between adults who stutter and their normally fluent peers. However, the reasons for these differences remain unclear although some developmental data suggest that structural brain differences may be present in school-age children who stutter. In the present study, the corpus callosum of children with persistent stuttering, children who recovered from stuttering and typically developing children between 9 and 12 years of age was compared to test if the presence of aberrant callosal morphology is implicated in this disorder. The total corpus callosum midsagittal area and area of each subsection consisting of the rostrum, anterior midbody, posterior midbody and splenium were measured using MIPAV (Medical Image Processing, Analysis, and Visualization). Voxel-based morphometry (VBM) was also used to compare white matter volume. No differences were detected in the corpus callosum area or white matter volume between children with persistent stuttering, children who recovered from stuttering and typically developing children. These results agree with dichotic listening studies that indicate children who stutter show the typical right ear advantage. Therefore, the neural reorganization across the midline shown in adults who stutter may be the result of long-term adaptations to persistent stuttering.

Learning outcomes: Educational objectives: After reading this article, the reader will be able to: (1) summarize research findings on corpus callosum development; and (2) discuss the characteristics of corpus callosum anatomy in stuttering.

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1. Introduction

The typical onset of developmental stuttering is around 3 years of age coinciding with a period of rapid speech and language development (Månsson, 2000; Reilly et al., 2009). Approximately 70% of children who stutter will recover within 2 years of onset (Månsson, 2000; Yairi & Ambrose, 2005). Children who begin stuttering at an earlier age are more likely to recover compared to those with later onsets (Buck, Lees, & Cook, 2002; Yairi & Ambrose, 2005), with boys more likely to have persistent stuttering (Craig, Hancock, Tran, Craig, & Peters, 2002; Van Borsel, Moeyaert, Rosseel, Van Loo, & Van Renterghem, 2006; Yairi & Ambrose, 1999). The performance of children with persistent stuttering also differs from children who recovered from stuttering across several dimensions including temperament, sensory and motor (Howell, Davis, & Williams,

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2008). Understanding the physiological factors that differentiate persistence and recovery remains a primary challenge for stuttering research as this could provide insight into the cause(s) of the disorder and facilitate clinical interventions that increase recovery rates.

Recovery and persistency seem to be mediated genetically to some extent as children with documented recovery or persistency are likely to have parents or siblings who respectively recovered from or persisted in stuttering (Ambrose, Cox, & Yairi, 1997). Neurological explanations are also thought to be centrally involved in persistency and recovery as stuttering is expressed as subtle alterations in the structure and function of speech-language relevant brain regions (Cykowski et al., 2008; Cykowski, Fox, Ingham, Ingham, & Robin, 2010; De Nil, Kroll, & Houle, 2001; De Nil, Kroll, Lafaille, & Houle, 2003; De Nil, Kroll, Kapur, & Houle, 2000; Foundas, Bollich, Corey, Hurley, & Keilman, 2001; Kell et al., 2009; Lu et al., 2010; Sommer, Koch, Paulus, Weiller, & Büchel, 2002; Watkins, Smith, Davis, & Howell, 2008).

We recently reported that the overall area of the corpus callosum (CC) and anterior portion of the CC was larger in adults who stutter (AWS) compared to normally fluent adults (Choo et al., 2011). Generally, a larger CC is associated with right hemisphere dominance or reduced hemispheric asymmetry for speech (Dorion et al., 2000; Gootjes et al., 2006; O'Kusky et al., 1988), which is consistent with neurological reports of increased right hemisphere activation or a lack of left hemisphere dominance in stuttering (De Nil et al., 2001; Fox et al., 1996; Neumann et al., 2003, 2005). The anterior portions of the CC including the rostrum, genu, and anterior body connect the prefrontal, premotor and supplementary motor areas. A larger anterior midbody may indicate greater inter-hemispheric communication between the left and right hemisphere motor cortices involved in speech production, which may be related to the rightward shift in motor activity reported in certain functional imaging studies of stuttering. The anterior callosa has also been implicated in auditory processing. A larger anterior CC has been associated with decreased right ear performance in right-handed individuals. One interpretation is that decreased performance results from greater competition for resources from the left ear or inhibition from the right hemisphere (Clarke, Lufkin, & Zaidel, 1993; Westerhausen & Hugdahl, 2008). The posterior regions of the CC including the isthmus and splenium which connect the parietal and temporal cortical regions are also involved in speech and language processing (Hofer & Frahm, 2006). A smaller posterior CC is thought to be linked to greater hemispheric lateralization. Patients with multiple sclerosis showed an increased right ear advantage in verbal dichotic listening tasks with progressive posterior CC loss affecting the isthmus and splenium (Gadea et al., 2009).

The importance of the CC for inter-hemispheric connections in support of language and cognition comes from different perspectives (see Gazzaniga, 2000; Pujol et al., 2006). Disruptions in language performance may be directly correlated with deficits in coordination of language pathways mediated by the CC (Paul, 2011). For example, children with developmental language disorder characterized by impairments in language production and/or comprehension (Bishop, 1992) have been reported to show disproportionate CC size relative to brain volume. Although the absolute size of the CC was similar between children with developmental language delay and typically developing children, the relative size of the CC to brain volume was disproportionately smaller in children with developmental language delay, and consequently, may result in greater constraints on inter-hemispheric communication (Herbert et al., 2003, 2005). In addition to developmental language delay, atypical CC development has also been associated with other developmental disorders including dyslexia, attention-deficit hyperactive disorder and autism (see Paul, 2011).

In this study, we compared the midsagittal area and white matter volume of the CC in school-age children with persistent stuttering, children who recovered and typically developing children to determine whether an enlarged CC is present in childhood stuttering. If the anterior CC is larger or has a greater volume in children who stutter, it could conceivably be associated with other aberrant early developmental processes that result in the unusual right hemisphere anatomy or function in persistent stuttering. Alternatively, enlargement of the CC could emerge as part of a neuroplastic response to prolonged stuttering, thus deviations from a typical pattern of CC development may not be present in younger children. In that case, an enlarged CC would be expressed in AWS along with the unusual brain findings reported in previous studies including atypical structural symmetry in the auditory cortex (Foundas et al., 2001; Jäncke, Hänggi, & Steinmetz, 2004).

Differences in cortical gray matter (GM) volume and white matter (WM) integrity have been found previously in a comparison of children with persistent stuttering, children who recovered from stuttering and typically developing children using a combination of voxel-based morphometry (VBM) and fractional anisotropy (FA) analyses using diffusion tensor imaging (DTI) (Chang, Erickson, Ambrose, Hasegawa-Johnson, & Ludlow, 2008). Children who recovered from stuttering and children with persistent stuttering featured reduced GM volume in the left inferior frontal gyrus (IFG) and superior temporal gyrus. The left IFG has also been implicated in AWS. In terms of WM integrity, Kell et al. (2009) reported increased FA values in the left inferior frontal region (and forceps minor of the CC) of adults with persistent stuttering. In terms of GM, there was reduced volume in the left IFG in both adults with persistent stuttering and adults who recovered from stuttering. Interestingly, greater stuttering severity was associated with lower GM volume than less severe stuttering. Additionally, other studies have reported reduced functional connectivity between the IFG and left motor regions, and reduced FA in the left (and right posterior) IFG in stuttering (Chang et al., 2011; Lu et al., 2009; Watkins et al., 2008). In contrast to these reports of left IFG anomalies, other investigations have reported increased or no GM differences in the left IFG in AWS compared to normally fluent adults (Beal et al., 2007; Jäncke et al., 2004; Lu et al., 2010). There is clear controversy over the status of the left IFG in stuttering that calls for more studies, particularly developmental studies that identify early neurological changes that could mark clinically relevant factors in stuttering. In addition to the IFG, the left arcuate fasciculus, which links Broca's and Wernicke's regions is also associated with reduced WM integrity in both children with persistent stuttering and children who recovered from stuttering (Chang et al., 2008). Although differences in the CC were not investigated by Chang et al.

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