

## **Neurological stuttering research:**

**Broad Findings:** PET, fMRI, and MEG studies have found over activations in primary and premotor cortex, anterior cingulate cortex, and cerebellum, as well as deactivations in auditory cortex (DeNil, Jokel, & Rochon, 2007). “Although some investigations have revealed atypical right hemisphere activation in adults with developmental stuttering, many studies have reported a general over activation of regions in both the left and right hemisphere (DeNil, 2004). In addition, several recent studies have shown that developmental stuttering is characterized not only by functional but also by structural differences, especially in the frontolateral and auditory cortex (Foundas, Bollich, Corey, Hurley, & Heilman, 2001; Foundas et al., 2003; Sommer, Koch, Paulus, Weiller, & Buchel, 2002)” (DeNil, Jokel, & Rochon, 2007, p. 330).

**Broad Findings:** Conflicting with DeNil’s statement that findings of atypical right hemisphere activation are perhaps not as absolute as some think, Bennett (2006) lists several studies confirming this phenomenon of increased right hemisphere activation during various speaking tasks (e.g., Braun et al., 1997; DeNil et al., 2000; Fox, Ingham, & Ingham, 1996; Ingham, 2001; Van Borsel, Achten, Santens, Lahorte, & Voet, 2003).

**Broad Findings:** Fogle (2008) summarizes the general themes emerging from the neuroimaging literature as follows: “(1) the neural systems in stuttered speech can be distinguished from that of normal speech; (2) areas known to be associated with motor speech and language production are found to show differences in levels of activity among individuals who stutter compared to individuals who do not stutter; (3) stuttering is not necessarily related to one structural or neural pathway; and (4) stuttering is particularly associated with hemispheric asymmetry, including increased activity in motor centers in the non-dominant (typically the right) hemisphere (DeNil, 2004; Kent; 2000; Ward; 2006)” (p. 371).

**Caveats:** Any differences reported in neuroimaging studies do not necessarily reflect a causative relationship between stuttering and abnormal neural functioning, organization, or structure. As Buchell and Somer (2004) note, brain differences between IWS and normally fluent speakers “may not be the cause of stuttering, but rather a compensatory process” (p. 161) developed through years of living with the disorder.

**Treatment Effects:** Some studies have found increased cerebellum activity right after intensive stuttering treatment (DeNil, Kroll, & Houle, 2001; DeNil, Kroll, Lafaille, & Houle, 2003; Ingham et al., 2001). This has been interpreted to signify increased attention to speech production following therapy.

- DeNil’s (1999) findings suggest that treatment may lead to reorganization of neurological processes. For instance, he found increased activation in left hemisphere cortical motor regions at post-treatment, coupled with altered activation patterns in the anterior cingulate region (possibly reflecting changes in cognitive & emotional states).
- The findings of Neumann et al. (2003) also point toward changes in neural organization post-therapy. At pre-treatment, subjects showed widespread

over activation in right hemisphere regions while reading aloud. After treatment, the activation patterns during this task were distributed bilaterally.

Kully, Langevin and Lomheim (2007) report that some clients who went through the ISTAR-Comprehensive Stuttering Program were inept at self-evaluating whether or not they accurately produced a target skill (e.g., easy onsets, light contacts). The authors claimed that this difficulty was consistent with neuroimaging research showing deactivation of regions involved in self-monitoring speech and voice in adults who stutter (Ingham, Ingham, Finn, & Fox, 2003).

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