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Stuttering and the basal ganglia

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Editor's note: Per Alm has a Ph.D. in neuropsychology and has been working with research on stuttering in Sweden but is currently teaching at the University of Alberta, Edmonton. We asked Per if he could write something about his research and his view on stuttering and the brain.

The variability of stuttering is often striking to the listener and mysterious for the person who stutters.

Based on personal experiences I have felt that the lack of real knowledge about the underlying mechanisms of stuttering is a problem in many ways. It makes it more difficult to treat, it leaves the field open to unfounded speculations, and it makes it difficult for persons who stutter to reach self-understanding. For this reason, I changed my vocation from engineering to research on the nature of stuttering, about 10 years ago.

When I began my Ph.D. studies, it was not easy to know what to do to advance the understanding of stuttering. So many ingenious efforts had already been made. Comparing this research to a puzzle, it felt important to try to make sense of the scattered pieces. Therefore I worked both with experimental studies and theoretical overviews of previous research findings.

In this article I will focus on the main theoretical work, regarding the possible relation between stuttering and the basal ganglia, brain structures involved in automatization (Alm, 2004). This work led to the proposal of the dual premotor systems model of stuttering (Alm, 2005). The idea that stuttering is related to the basal ganglia is not new. The first account came from the German physician Sahli, based on studies of epidemic encephalitis around 1920. Among later examples can be mentioned Caruso (1991) and Molt (1999).

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Conditions where stuttering tends to be absent provide important clues about the underlying dysfunction. One such condition is the "rhythm effect": persons who stutter usually become fluent when speaking to the pace of a metronome. They seem unable to stutter when following the beats. This distinction, to have a problem performing a motor sequence on your own, but being able to do it when receiving external "timing cues," is characteristic of dysfunction of the basal ganglia motor circuits (including related structures, such as the supplementary motor area (SMA)). Normally, the basal ganglia seem to provide "go-signals" for the segments in a motor sequence, e.g. the syllables in speech.

Without sufficiently strong go-signals the speech cannot be started or will be disrupted. The rhythm effect appears to be a key feature of stuttering, providing a strong indication that stuttering is related to some type of disturbance of the basal ganglia motor circuits.

There are several other indications for the important role of the basal ganglia in stuttering, for example: (1) Lesions that cause "acquired stuttering" often affect the basal ganglia (Ludlow et al., 1987). (2) The drugs that have shown the clearest effect on stuttering, to make it better or worse, affect the dopamine system. Dopamine is a key transmitter that regulates the function of the basal ganglia. (3) Basal ganglia motor disorders characteristically worsen during stress and "nervous tension," and improve under relaxed conditions.

In the review on stuttering and the basal ganglia (Alm, 2004), it was also argued that the typical pattern of onset of stuttering around age 2.5 to 3, with a large percentage of early recoveries, may be related to a natural phase of the development of the basal ganglia. Specifically, it has been shown that children in general have a peak in the number of dopamine receptors type D2 in the basal ganglia at this time. There are theoretical arguments for how a large number of D2 receptors may increase the risk for stuttering.

Furthermore, the drugs that have shown the best documented effect on stuttering act by blocking these D2 receptors. The number of D2 receptors has also been reported to show correlation with cognitive

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performance, which is in accord with the observation that children with early onset of stuttering often display precocious language development (Watkins, Yairi, & Ambrose, 1999).

In the thesis, On the causal mechanisms of stuttering (Alm, 2005), the basal ganglia model was developed further, based on the theoretical work on the human motor system proposed by Goldberg (1985, 1991) and others. Goldberg argued that the human brain has two parallel premotor systems, i.e. systems involved in planning and execution of movements, including speech. (However, he did not discuss stuttering.) According to this model, both systems have the ability to provide go-signals for movements, but under somewhat different conditions. The lateral system, consisting of the lateral premotor cortex and the cerebellum, is active when the movement is controlled in relation to the sensory input — like when speaking to the pace of a metronome or reading in unison. Similarly, the lateral system is dominant when speech is controlled by auditory or somatosensory feedback.

In contrast, the medial system, consisting of the basal ganglia and the SMA, operates based on automatized programs without external feedback. This system is dominant during spontaneous speech, especially if the speech is propositional, i.e. that it conveys thoughts or emotions.

The lateral system is also assumed to be active when a movement is executed with increased attention and conscious control, while the medial system dominates for automatic responses. This is claimed to be the reason why it is difficult to get a natural smile when asked by a photographer — voluntary and spontaneous smiles are created by two different systems in the brain. This distinction suggests that the lateral system is in charge when speaking in a way that is not automatic, like imitating an accent or playing a role.

This dual premotor systems model of stuttering provides a novel explanation for most of the well-known fluency inducing conditions in stuttering. Stuttering is related to a disturbance of the medial system, but when the control is shifted from the medial to

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the lateral system the problem is bypassed. As mentioned above, this could pertain to the metronome effect, unison reading, imitation of an accent, and role play. Furthermore, there are research data supporting that it is the lateral system that is dominant for go-signals during singing and rhythmic speech, conditions know to improve fluency.

An interesting question is how the effect of altered auditory feedback on stuttering can be explained, for example frequency altered feedback (FAF). There are now several brain imaging studies showing specific activation of the lateral premotor system when listening to speech sounds. Moreover, recent brain imaging data (Watkins, Davis, & Howell, 2005) have shown increased activation of the auditory cortex during speech with FAF. These findings point to increased control from the lateral system during altered feedback, so that difficulties with the medial system may be bypassed. This hypothesis is supported by reports that some speech difficulties in Parkinson's disease, which is a basal ganglia disorder, may be improved by either delayed or frequency altered auditory feedback.

An important aspect of the dual premotor model is that it emphasizes that the basal ganglia system is part of a larger medial system, including the complete loop from the cortex through the basal ganglia and the thalamus, and back to the cortex (the SMA). For example, as suggested in Alm (2004), the production of go-signals from the basal ganglia may be disturbed because of deficient input from the primary motor cortex. In this way the basal ganglia model is compatible with the recent reports of structural anomalies of the cortex and the white matter, e.g. in the sensorimotor region for the speech organs (Foundas et al., 2001; Sommer et al., 2002; Jancke et al., 2004; Watkins et al., 2005). A more detailed account of this dual premotor model of stuttering is currently under way.

A pdf-file of the thesis can be downloaded from *http://theses.lub.lu.se/postgrad/ (However, the online version does not yet include all the research papers, because of copyright questions. For missing papers contact Per.Alm@psychology.lu.se. Printed copies of the complete thesis can be ordered from the Swedish*

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