

## **The dual premotor model of stuttering and cluttering: a framework**

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Stuttering and cluttering are disorders of speech fluency, which may occur separately or together. An explanatory model is proposed, the dual premotor model of stuttering and cluttering. A tenet is that speech is a sequential motor behavior, in which each segment (e.g. syllable) requires a "go-signal" for its initiation. For well-learned sequences the basal ganglia seems to have a key role, providing the supplementary motor area (SMA) with timing information, for example indicating that the previous segment has been executed and it is time to release the next segment. The dual premotor model of Goldberg and Passingham states that the brain has two parallel systems for initiation of movements: the medial system (the basal ganglia and the SMA) and the lateral system (the lateral premotor cortex and the cerebellum). The medial system is assumed to be active in spontaneous speech, while the lateral systems tend to be dominant in non-automatic modes of speech, for example using increased attention to auditory or sensory perception. It is here proposed that both stuttering and cluttering are related to disturbances of the initiation of speech segments, by the medial premotor system. In this way stuttering and cluttering can be regarded as opposite symptoms of the same function, with impaired versus premature initiation of the speech segments. This model implies that impairments of the medial system may be temporarily by-passed by shifting the dominance for speech timing to the lateral system, during certain speech-modes.

### **Introduction**

Stuttering and cluttering are two disorders of speech fluency, which may occur separately or mixed together. No generally accepted theoretical model exists for either of these disorders, and the neurological mechanisms are still poorly understood. There is a range of observations and research findings about stuttering and cluttering which needs to be encompassed by an explanatory framework, linking the scattered pieces together. For example, one of the main aspects of stuttering is the various conditions that tend to temporarily alleviate the fluency problems: the rhythm effect, unison reading, singing, imitation of a foreign accent, altered auditory feedback, etc. The often remarkable effect of these conditions is likely to provide important clues regarding the basic nature of stuttering. Also the symptoms of cluttering tend to vary; in many cases the symptoms temporarily improve or disappear if attention is given to speech.

An explanatory framework is here proposed, *the dual premotor model of stuttering and cluttering*. The model is primarily based on the theoretical reasoning in Alm (2004), regarding the role of the *basal ganglia* in stuttering, and the further analyses in Alm (2005). In this later work the role of the basal ganglia was placed within the wider framework of the *dual premotor systems hypothesis* by Goldberg (1985, 1990) and Passingham (1987). In Alm (2007) this model was expanded to include both stuttering and cluttering.

### **The basal ganglia circuits**

The basal ganglia are subcortical gray matter structures (see Fig. 1) with a strong influence on the frontal lobe cortex, modulating inhibition and excitation of the cortex. The circuits through the basal ganglia seem to form relatively independent "channels", targeting different frontal cortex areas. These circuits may be divided into 3 main parts, based on functions: (a) sensorimotor (to motor cortex areas), (b) associative (to the prefrontal cortex), and (c) limbic circuits (to the anterior cingulate cortex and the medial prefrontal cortex). The exact functions

of these basal ganglia circuits are still not well understood, but a main function seems to be automatization of the corresponding frontal cortex functions, pertaining to the execution of motor sequences, attention, and motivational impulses, but probably also to higher functions such as cognition and language. The basal ganglia circuits receive input from most parts of the cerebral cortex, and also from the limbic system, placing the basal ganglia in a key position to influence behavior and cognition.

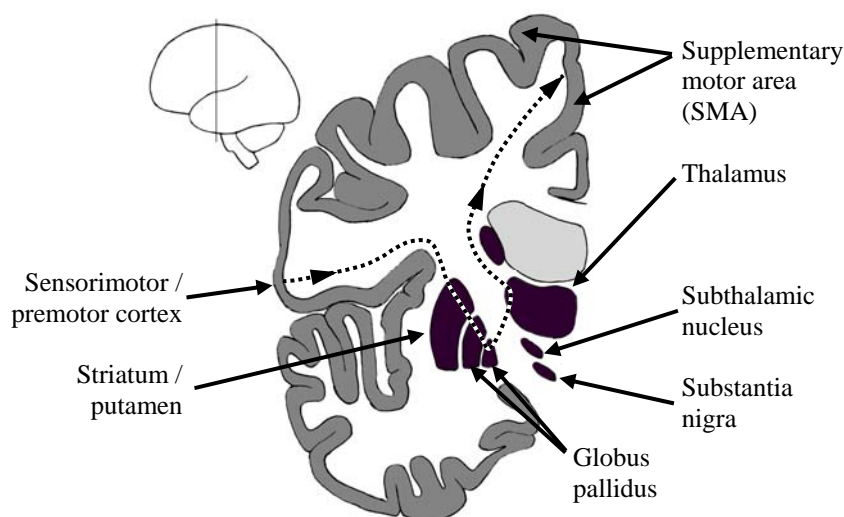
A functional principle of the basal ganglia seems to be to provide an appropriate pattern of inhibition and activation of various frontal cortex areas. The normal baseline is a diffuse inhibition of cortical impulses, combined with focal activation of selected behaviors. This balance between inhibition and excitation may be affected by a wide range of factors, such as the dopamine system, the transmitter substance GABA, and lesions or other disturbances of various parts of the basal ganglia circuits.

### **Stuttering and the basal ganglia**

*Historical overview.* The idea that stuttering is related to disturbance of the basal ganglia function is not new. Already around 1920 the German physician Sahli made this proposal, based on observations of epidemic encephalitis. In more contemporary literature on stuttering the basal ganglia appeared in Rosenberger (1980), in the discussion of the effect of dopamine receptor blockers to relieve stuttering. Dopamine is the main regulator of basal ganglia functions. In 1987 Ludlow et al. published a detailed study of the localization of lesions causing stuttering in adults. The only gray matter structures that were significantly more frequently affected in the stuttering group were parts of the basal ganglia. Wu et al. (1995) reported reduced metabolism in a part of the left basal ganglia, and later the same group found highly increased uptake of the precursor of dopamine in a small study of three stuttering adults (Wu et al., 1997). Molt (1999) discussed possible links between stuttering and the basal ganglia, based on similarities between stuttering and Tourette syndrome, dystonia, and other neurological disorders.

*Other basal ganglia motor disorders.* A typical characteristic of various basal ganglia motor disorders is emotional influence on the symptoms, with "nervous tension" making them worse and "relaxation" bringing about an improvement (Victor and Ropper, 2001). Basal ganglia motor disorders often also involve motor initiation problems, involuntary movements, and dysregulated muscular tension.

*Drug effects.* The drugs with the strongest effect on stuttering, making it better or worse, typically affect dopamine (Alm, 2004). Dopamine has effects in many parts of the brain, but the most explicit effects seems to be in the basal ganglia, with immediate and often dramatic influence on movements, mood, and the general level of activity.



*Figure 1.* Basal ganglia nuclei and the thalamus. The basal ganglia motor circuit is marked schematically, by the dotted line, from motor cortex areas to the supplementary motor area (SMA).

### **Cluttering and the basal ganglia**

Cluttering is characterized by abnormally fast and/or irregular speech rate, often with omission of syllables and slurred articulation (St. Louis, Raphael, Myers, & Bakker, 2003). Seeman (1970) argued that cluttering is the result of disturbances of the basal ganglia motor system, causing dysregulation of cerebral cortex regions. This view was shared by Lebrun (1996), based on his claim that cluttering after brain damage appears primarily, if not exclusively, after lesions of the basal ganglia system. He suggested that this system may contain the timer that sets the pace of articulation.

Persons with cluttering have been reported to frequently display other symptoms such as disorganised language, poor handwriting, dysregulated movements, restlessness, and attention deficits (Seeman, 1970; Daly, 1993; St. Louis et al. , 2003). It is quite possible that this cluster of symptoms may be related to impaired inhibition of various frontal cortex areas as a result of dysregulated basal ganglia circuits. For example, restlessness and attention deficits are often linked to basal ganglia disturbances, and handwriting tend to be affected in basal ganglia disorders such as Parkinson's disease. Deso Weiss (1964) suggested that these various symptoms in cluttering are expressions of a "central language imbalance", but may instead be understood as expressions of a basal ganglia disinhibition syndrome.

### **Initiation and timing of speech movements**

Charles Van Riper (1982) proposed that the core of stuttering is a disruption of the motor sequence of the word, as a result of disturbed timing. This is in accord with the proposed definition of stuttering in Alm (2005), that stuttering is characterized by difficulties to move forward in the speech sequence, when the person knows what to say. A key concept in this view is that speech is a sequential motor task which needs exact timing, in order to produce the appropriate prosody. This aspect of speech was emphasized by Deecke et al. (1985): that each motor segment (e.g. syllable) needs a "go-signal" in order to be executed. As mentioned above it is likely that the basal ganglia has a central role in the execution of well-learned movement sequences, supporting the functions of the supplementary motor area (the SMA)

by providing timing cues. Experimental studies of hand movements in monkeys indicate that the basal ganglia produce a "completion signal", marking the end of a completed segment in a well-learned movement sequence. This completion signal is assumed to cue the SMA to release the next motor segment in the sequence (Brotchie et al., 1991), by providing brief activation of a part of the SMA. It is possible that similar mechanisms are important in speech.

Based on this model it is easy to imagine that the initiation of speech segments may be disturbed in different ways, as a result of dysregulated excitation and inhibition of the cortex. If the completion signal is not strong enough the next segment may not be released, possibly resulting in repetitions or prolongations of the previous segment, or halting of the sequence. Such insufficient initiation of speech segments may be a core mechanism of stuttering.<sup>1</sup> On the other hand, impaired cortical inhibition might result in premature initiation of speech segments, before receiving a completion signal for the previous part. Such premature initiation of speech segments may be a core mechanism of cluttering, resulting in shortened or omitted segments, incomplete articulation, increased speech rate or spurts, and disturbed prosody. In summary, it is proposed that stuttering and cluttering are the two opposite disturbances of the initiation of speech segments, with insufficient versus premature initiation. In some cases the symptoms of stuttering and cluttering occurs in the same individual, so called "stuttering-cluttering", with a mix of initiation difficulties and cluttered speech. It seems possible that this could result from a general dysregulation of the relevant basal ganglia circuits and their cortical targets, with a combination of impaired inhibition and insufficient activation. Another way to describe this state is in terms of *signal-to-noise ratio*, where one may have a combination of increased noised and weakened signal.

The discussion above has focused motor aspects of speech production. However, in some cases of cluttering the main disturbance seems to occur on the language level, with disorganized sequencing of words and ideas. As mentioned above, the basal ganglia include both motor circuits and associative circuits related to higher functions of cognition and language. The basic architecture of the motor circuits and the associative circuits are very similar, but supports different functions. Little is known about the exact functions of the associative circuits, but it seems possible that they could be involved in the higher levels of speech sequencing. In other words, it may be speculated that both the motor and language aspects of cluttered speech are related to the same type of basal ganglia disinhibition, but pertaining to different frontal cortex areas.

### **The dual premotor model**

In Alm (2005) the basal ganglia model of stuttering was developed further, and the role of the basal ganglia was put within the wider framework of the *dual premotor systems hypothesis* by Goldberg (1985, 1990) and Passingham (1987). This model emphasizes that the motor functions of the basal ganglia are part of an integral system, the *medial premotor system*, which is paralleled by the *lateral premotor system*. The main components of the medial system are the basal ganglia and the SMA, while the lateral system consists of the *lateral premotor cortex* and the *cerebellum* (se Fig. 2). These two systems were proposed to dominate under different conditions: The medial system being dominant in automatized sequences performed without attention, while the lateral system typically operates in relation to sensory input, based on feedback control, and/or with voluntary conscious control.

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<sup>1</sup> This model was outlined in Alm (2004), with the key features that the core dysfunction of stuttering is impaired ability of the basal ganglia to produce timing cues (via the SMA) for the initiation of the next motor segment in speech. (The proposal did not specify the segment unit, e.g. syllables or phonemes.) A similar model has recently been put forward by Packman, Code, and Onslow (in press), stating that stuttering comprises difficulties to initiate syllables, related to a disturbance of the functions of the SMA.

Goldberg (1985) discussed the roles of these two systems in speech, but did not relate the model to stuttering.

This dual premotor model implies that the brain has two parallel systems for speech timing, and that the problems of stuttering and cluttering typically is only related to one of them, the medial system. This may provide an explanation for many of the characteristic fluency inducing conditions in stuttering: Certain conditions shift the dominance from the medial to the lateral system, thereby temporarily by-passing the problem of the medial system. This mechanism is suggested to be relevant for the effects of choral reading, metronome-paced speech, singing, altered auditory feedback, imitation of a foreign accent or role play, or when using a consciously controlled speech rate. Furthermore, this model may also provide an explanation for the typical effect of attention to speech in cluttering: the attention to speech may support a shift of speech control towards the lateral system.

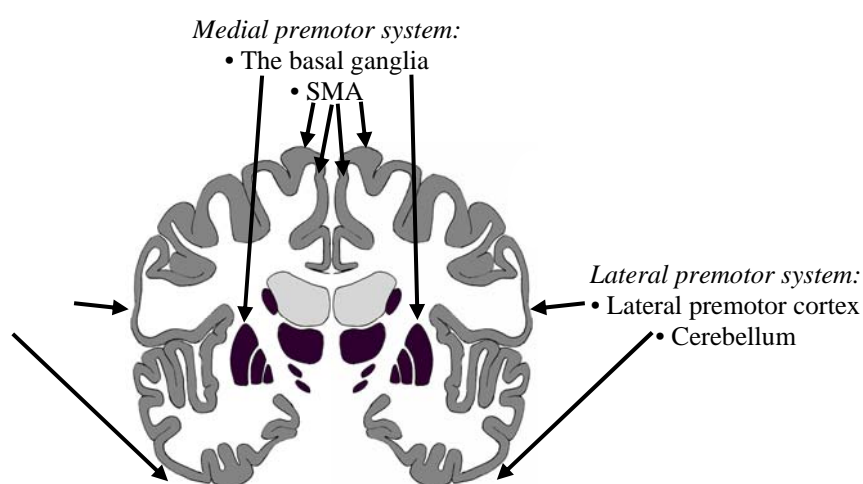


Figure 2. Dual premotor systems, based on Goldberg (1985, 1990) and Passingham 1987.

### Summary and conclusions

The fluency disorders stuttering and cluttering are proposed to be the two opposite disturbances of the initiation of speech segments (e.g. syllables) by the medial premotor system, with insufficient versus premature initiation of speech segments. The medial premotor system, according to Goldberg, consists of the complete circuits starting with the cortical input to the basal ganglia, passing through the basal ganglia and the thalamus, and finally targeting the SMA (see Fig. 1). The normal functions of the basal ganglia are dependent on the integrity of this complete circuit. This means that the disturbances of basal ganglia functions discussed in this presentation may result from disturbances in any part of the medial premotor system, for example impaired input from the cortex to the basal ganglia (see Fig. 1). Without correct input the basal ganglia may be left out of control.

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