A framework for stuttering and cluttering?
Speech segment initiation and the dual premotor model
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Can stuttering be understood?

Research on stuttering
- Still no consensus about what type of disorder stuttering is (motor, linguistic, emotional, social?)
- Still no consensus about which parts of the brain are crucial for stuttering

Why discuss the nature of stuttering?
- To facilitate treatment:
  - understanding possibilities and limitations
  - understanding the mechanisms of treatment
  - to provide relevant information to parents and persons who stutter
- The self-perception of the person who stutters
- Curiosity, the desire to understand...

Goal of this presentation
- To propose an explanatory framework for stuttering and cluttering
  - Based on the basal ganglia theory of stuttering
  - Focusing initiation of speech segments
  - Expansion to “the dual premotor theory of stuttering and cluttering”
  - Stuttering and cluttering are suggested to be the two opposite disturbances of the initiation of speech motor segments, with insufficient vs. premature initiation.

Disclaimer...
- Theories will always be simplifications in relation to reality
The brain and the basal ganglia

The cerebral cortex

The organization of the cortex

Below the cortex: the basal ganglia

The basal ganglia: automatization of cortex functions

Basal ganglia anatomy: the striatum

- caudate nucleus, aspects of cognition, language
- putamen, motor functions, incl. speech
- ventral striatum, motivation, emotions, impulses, “limbic”
The basal ganglia, anatomy

![Diagram of the basal ganglia, showing SMA (supplementary motor area), Striatum: putamen (motoric) and Substantia nigra, dopamine to striatum.]

Typical traits of basal ganglia motor disorders (Victor & Ropper, 2001)

- Typical motor symptoms
  - Motor initiation problems
  - Involuntary movements
  - Dysregulated muscular tension (dystonia)
  - Often co-contraction of antagonistic muscles
- The emotional state tend to influence the motor symptoms:
  - "nervous tension" making symptoms worse
  - relaxation improving symptoms

The basal ganglia theory of stuttering

- First account by Stier, Germany, ~1920, after study of epidemic encephalitis.
- Followed by Seeman, Prague, ~1934
- Some modern accounts:
  - Rosenberger (1980), when discussing improvement of stuttering with dopamine receptor blockers.
  - Caruso (1991)
  - Wu, Maguire, Riley, et al. (1995, 1997) PET imaging
  - Molt (1999), theoretical discussion
  - Alm (2004), theoretical discussion

Brain lesions causing stuttering with adult onset?

- Reports of stuttering after lesions to almost all part of the brain.
- Ludlow et al. (1987): the most common loci of gray matter lesions was in the basal ganglia (the striatum + the globus pallidus).
- Other frequent loci are related to the basal ganglia motor circuit:
  - the VL nucleus of the thalamus
  - the supplementary motor area (SMA)

Impaired sequence learning skills in stuttering groups

- Smits-Bandstra & De Nil (2007):
  - Review of 4 studies of "sequence skill learning and the transition to automaticity", hand and oral.
  - "support the hypothesis that dysfunction in cortico-striato-thalamo-cortical connections may be one etiological component in the development and maintenance of stuttering"
Aspects of cluttering
(Adapted from St. Louis, Raphael, Myers, & Bakker, 2003)

- Central aspect:
  - speech rate is abnormally fast, irregular, or both
- Often additional symptoms, like:
  - disorganized language and conversation
  - poor handwriting
  - distractibility, hyperactivity
- Typically temporary improvement when paying attention to speech

Neurology of cluttering

- Often genetic influence
- Seeman (1970):
  - cluttering is a result of disturbances of the basal ganglia motor system, causing dysregulation of cerebral cortex regions
- Lebrun (1996): Cluttering after brain damage:
  - primarily or exclusively after disturbances of the basal ganglia system
  - the basal ganglia + frontal cortex targets

Neurology of stuttering and cluttering

- Indications that both stuttering and cluttering is related to disturbances of the basal ganglia, resulting in dysregulated frontal cortex.
- Cluttering often occur with stuttering: likely that both symptoms are related to the same brain systems.

Timing of speech movements

- Charles Van Riper (1982):
  - The core of stuttering is a disruption of the motor sequence of the word, as a result of “disturbed timing”.
- Deecher et al. (1985):
  - speaking is a sequential task and needs exact timing, ...
  - the delivery of the “go-signal” for every element.
Core features of stuttering (Alm, 2005)

- Difficulties to move forward in the speech sequence, when the person knows what to say.
- Essence: speech is a *sequential motor behavior* (like playing the piano).

Initiation of movements

- Distinction in motor control research:
  - *Self-initiated vs externally cued movements*
- Impairment of self-initiated but not externally cued movements after lesions to:
  - the putamen (basal ganglia) (Nixon & Passingham, 1998)
  - the SMA (Thaler et al., 1995)
- the basal ganglia-SMA-system is important for self-initiated movements.

Many stuttering children poorer at keeping a rhythm

- Anne Smith (2007):
  - 17 stuttering children, 4 to 5 years.
  - 13 fluent controls.
- Clapping hands, fixed rhythm:
  - Following a beat for 15 claps.
  - Continue with 30 unpaced claps.
  - *About 60% of the stuttering children showed higher unpaced clapping variability than any of the controls.*

Signals and noise

- Signals in the brain

Signals, noise, and signal-to-noise ratio (contrast)

- Signal:
- Noise:
- Signal + noise:
The quality of all signal transmission depends on the signal-to-noise ratio.

In the brain:
- Depends on transmitter substances, like dopamine and GABA.
- Lesions may reduce the signal and increase the noise.
- Noise may come from epileptiform activity.
- Motivation, focused attention, and motor practice may strengthen the signal-to-noise ratio.

In automatized movement sequences the basal ganglia give a pulse to the SMA, marking the end of a segment in a motor sequence → assumed to release the next segment.

Similar mechanism assumed to be involved in the initiation of speech segments.

Possible problems:
- Impaired signal: inability to move forward in speech → stuttering (repetitions, prolongations, blocks)
- Increased noise → premature signal: release of the next motor segment before the previous one was finished → cluttering (shortened or omitted segments, dysprosody)
- Mixed dysregulation:
  → "stuttering-cluttering"

Stuttering and cluttering are suggested to be the two opposite disturbances of the initiation of speech motor segments, with insufficient vs. premature initiation.

Brain imaging:
- Brain imaging of dopa uptake
  - by Wu, Maguire, Riley and coworkers, 1997
  - about 3 times higher uptake of dopa in the basal ganglia of 3 stuttering persons, compared with controls.
  - very interesting, but not confirmed
Drug effects

- Drugs affecting the dopamine system have been reported to make stuttering better or worse in some cases. (However, often with side effects.)
- Subgroups?

Developmental effects of neural systems. Relation to onset age and recovery?

  - Mean age of stuttering onset: between 2.5 and 3 years.
  - About 60-70% seems to have recovered 2 years later.
- Any neurodevelopmental changes that follows this time pattern?
  - Yes, the number of dopamine receptors in the striatum.

Neurotransmitters and receptors

- Chemical regulation of neurons by transmitters, released in the synapse.
- The transmitters activate receptors

The dopamine receptor

- At least 9 different types of dopamine receptors.
- Type D1 and D2 dominant in the basal ganglia.
- Different effects of different receptors.

The effects of the number of D2-receptors

- Schematic model:
  - High number of D2: weak general inhibition of the cortex, "high noise", increased risk for involuntary actions.
  - Low D1/D2 ratio → bad signal-to-noise ratio.

Developmental effects?

- Figure: postmortem data from general population (Alm, 2004, based on data from Seeman et al., 1987).
  - Pattern:
    - Peak in number of D2 around age 2.
    - Peak in number of D1 around age 3.
    - Early peak also found in rats.
  - Risk period: From D2 peak to D1 peak → low D1/D2-ratio → bad signal-to-noise ratio, about 2-3 years of age.
  - Tendency for lower D1/D2 ratio in boys (+) compared with girls (*) (but small number of cases)
Effects of a high number of D2-receptors?

- Reduced inhibition of the frontal cerebral cortex.
- Possible positive effects:
  - Seems to facilitate some types of learning.
  - Positive correlation between number of D2 receptors and cognitive performance, in normal population (Volkow et al., 1998; Bäckman et al., 2000).
- Increased risk for stuttering?

Effects of a high number of D2-receptors?
(cont’d)

- Could this explain the precocious language development reported in many children with early onset of stuttering? (Watkins, Yairi, & Ambrose, 1999)
- "Reduced inhibition" fits with some reports of temperamental characteristics in stuttering preschool children, e.g. Embrechts et al., 2000:
  - heightened levels of gross motor activity and impulsivity.
  - lowered attentional focusing.
  - (somewhat lower scores for shyness, fear and sadness, though not statistically significant)

Expanding the basal ganglia model (Alm, 2005, 2006):

Dual premotor systems

- Goldberg (1985, 1990) and Passingham (1987) independently proposed a distinction between a medial and a lateral premotor system:
  - Medial premotor system:
    - The basal ganglia
    - SMA
  - Lateral premotor system:
    - Lateral premotor cortex
    - Cerebellum

Proposed functional differences, movements in general (from Goldberg, 1985)

- The medial system
  - automatized motor sequences
  - mainly without sensory input, self-initiated
  - emotional expressions (e.g., smiles)

- The lateral system
  - movements performed with attention
  - movements in relation to sensory input, externally cued
  - non-emotional expressions

Suggested dominance in speech timing
(Alm 2005, mainly based on Goldberg, 1985)

- The medial system
  - propositional speech, reflecting emotions and thoughts
  - spontaneous speech, without conscious attention to the speaking process
  - speech linked to external stimuli, like metronome-paced speech or chorus reading
  - speech that is consciously controlled, e.g., imitation of a foreign accent, role playing, or reduced speech rate.
  - singing and rhythmic speech (reading poems, etc.)
Dual motors in a "hybrid-car"

- Petrol engine, useful for long distance
- Electric motor, useful for shorter distances
- Normal spontaneous speech
- novel speech patterns
- speech techniques
- singing
- choral reading,
speech with DAF/FAF

The dual premotor model

- Emphasizes that the motor functions of the basal ganglia are dependent of the complete circuit, the medial premotor system:

- Cortical input, (from motor cortex, Brocas area, etc)
- Supplementary Motor Area (SMA)
- Thalamus (VL nucleus)
- Basal ganglia

Anomalies outside the basal ganglia?

- Several studies with magnetic resonance imaging (MRI) since 2001 have reported structural differences:
  - Especially in the left speech (pre)motor region.
  - Kate Watkins, Smith, Davis, and Howell, 2006:

Anomalies outside the basal ganglia? (cont’d)

  - Subjects:
    - 8 right-handed boys age 9 to 12.
    - 7 persistent stuttering
    - 7 recovered from stuttering age 2 to 3 years.
    - 7 fluent controls.
  - Results:
    - Both recovered and persistent children showed reduced gray matter volume in:
      - Left inferior frontal gyrus (~Broca).
      - Both hemispheres temporal regions (posterior speech regions).
    - Recovered "worse" than the persistent (!)
    - Persistent stuttering: Reduced white matter integrity in the left hemisphere speech system.

Proposed possible interpretation (Alm, 2004)

- Input from the left cortical speech motor regions is essential for basal ganglia speech motor functions.
- Impaired signals from the cortex would leave the basal ganglia out of control
  → stronger influence of other input, e.g. limbic / emotional.
Complex system...

- Is stuttering a result of an unstable speech motor system involving both cortical/basal ganglia and left/right dimensions?
- Plotnik et al. (2005):
  - Indications that difficulties to initiate walking in Parkinson's Disease is the result of asynchronous left/right output from the basal ganglia.
  - Left/right basal ganglia asynchrony involved in stuttering?

Various methods for speech fluency

- Fluency shaping:
  - prolonged vowels, reduced speech rate
  - gentle onset
  - abdominal breathing
- McGuire:
  - monitored breathing, not abdominal
  - “Tricks”:
    - Russian “thumb-movement-method”
    - move thumb slowly when speaking
    - Swedish “foot-movement-method”☺
    - move foot slowly when speaking

“Distraction”? Or shift of brain system?
Speech with attention to body part linked to the movements, or attention to specific aspect of speech → the lateral system

Summary

- Stuttering and cluttering are suggested to be the two opposite disturbances of the initiation of speech motor segments, with insufficient vs. premature initiation.
- The disturbance is proposed to be localized to the medial premotor system
  - Circuit: cortical input → basal ganglia → thalamus → frontal cortex (especially SMA)
- The dysfunction may be temporarily bypassed by shift from the medial to the lateral system

Implications of the dual model for speech treatment of stuttering?

Very brief...

Summary, speech segment initiation and the dual premotor model of stuttering and cluttering

References
References (1)


References (2)


References (3)


References (4)