

European ECSF Symposium on Fluency Disorders

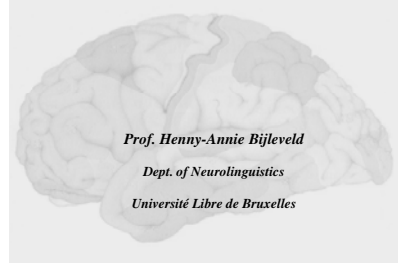


Antwerp
April 23-24, 2010

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Prof. H.A. Bijleveld

Cortical and subcortical Implications in Stuttering Relation to Emotion and Learning



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Structure Why subcortical Research in Stuttering?

- A. Why this research
- B. Research on physiologic Aspects in Stuttering
- C. Research on Subcortical Implication in speech stuttering
- D. Research on Basal Ganglia implication in Movement Disorders
- E. Research on Dopamine and BG in Stuttering
- F. Research on Limbic System and Stuttering
- G. Relation between BG, LS and cortical structures in Anxiety, Learning and Stuttering
- H. Conclusions
- I. Future Research

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A. Why subcortical brain research in stuttering?

Stuttering research outside - inside

1. research on localization and cerebral dominance
2. research on brain activation with MRI and PET scan to explore the different brain structures – cortical and subcortical –
3. fMRI research on Limbic System and BG to explore the relationship between emotions and movement disorders

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A. Why subcortical brain research in stuttering ?(2)

4. fMRI research on the role of neurotransmitters to explore the subtle dynamic interactions of cortical and subcortical activations
5. fMRI research on the impact of hormones to explore the interaction of complex brain mechanisms that can give rise to involuntary movements (certain moments & circumstances) and that can count for the dynamics of stuttering

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B. Some clinical observations Stuttering is a dynamic disorder

- Stuttering is a disturbance of speech rhythm : Columbat 1830 =>therapy based on rhythmic speech.
- Stuttering is a disturbance of coordination of speech : Schmalz 1848 =>therapy based on reduced speech rate, smooth breathing, soft onset of speech
- Stuttering is a disturbance of respiratory muscles and articulatory organs : Gutzmann 1898 =>therapy based on respiration and articulation

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B.1. Research on the physiologic basis of Stuttering

- Stuttering is a disturbance of cerebral dominance (Stier 1911, Orton, Travis 1930, Webster and Jones 1960) => Therapy based on de-automatization
- Stuttering is a disturbance of the auditory feedback (Goldiamond, Perkins, Webster, Gregory, Mysak 1960 -80) => therapy based on DAF, modification of speech, metronomous speech, syllable speech, chorus speaking, fluency shaping, role playing (cfr. Foundas 2003)

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Research on the physiologic basis of Stuttering (2)

- Stuttering is due to a disturbance of motor timing and motor control Van Riper 1982, Kent 1984, Caruso 1991, Ludlow 2003) Therapy based on slow speech
- Stuttering is a disruption of fluent speech, Ingham and Andrews 1973 => therapy based on operant conditioning, systematic reinforcement of fluent speech
- Stuttering is a disturbance, due to an abnormal activation of the LH speech areas, auditory cortex, with implication of the cerebellum, Wu, Fox, Ingham, Salmelin, Watkins (1990-2008)

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Research on the physiologic basis of stuttering (3)

- Stuttering is due to an atypical Planum Temporale anatomy and related to disturbed/ altered auditory feedback in stuttering (Foundas 2003) => Therapy based on DAF and AAF (de-automatization)
- Stuttering is associated with disturbances/ deficiencies of left grey matter and reduced white matter in LH (Kell, Chang 2008).

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Stuttering as a Disturbance of physiologic Processes

- Stuttering as the result of a disturbance of hemispheric dominance, auditory feedback, coordination or timing, can't explain the dynamic character of stuttering nor the underlying mechanisms causing the disturbances.
- Every brain is different. What is normal/standard brain functioning? What is the role of brain plasticity?
- Need for further research
- ++ RH activity sign of disturbance or of adaptation?

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C. Subcortical implication in Speech

Research on thalamic implication in speech

Stereotactic stimulation of the left thalamus => perseverations and stutterlike speech, repetitions and blocks
Schaltenbrand (1965, 1975)
Ojemann and Ward (1971)

=> difficulty in initiating speech, pitch changes, impaired rhythm
Bell (1968)

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Stereotactic stimulation of the left frontal thalamus in acquired Stuttering

to reduce epileptic attacks
=> relieve of epilepsy and stuttering
Andy and Bhatnagar (1991)
« chronic pain and stuttering may be implicated ... in reticular networks extending from the brainstem to the thalamus, and acquired stuttering may be recruited as one component of a larger syndrome complex »

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C. 1. Basal Ganglia

BG are a subcortical complex system of excitatory and inhibitory projections from the brainstem to the cortex, organized to facilitate voluntary movements

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Basal Ganglia (2)

- *Connections with the Limbic System via the amygdale and hippocampus
Dysfunction=> impaired voluntary movements and thus*
- *involved in learning, anxiety, stress*

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Basal Ganglia (3)

The thalamus could be considered as the central relay-station in the basal ganglia through the connections with Striatum- Cortex- GPi-SNr-STn

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C.2. Research on Basal Ganglia and involuntary movements is not new

- *Seeman 1934 postulated a theory of stuttering implicating the basal ganglia through the striato-pallidal pathway*
- *1995 Wu et al. Involvement of Putamen in speech motor control and Stuttering*
- *2003 Mink, 2005 Alm: BG inhibit competing movements interfering with the desired motor sequence.*

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C.2. Research on Basal Ganglia and involuntary movements/ speech

*SMA may be involved in self-initiated, well learned complex movements and timing
SMA may play an important role in speech and provides internal timing cues to facilitate the initiation of movements of a well learned motor sequence (Cunnington et al. 1996)*

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C.3. Basal ganglia lesions in acquired stuttering

*Research findings:
BG lesions in Acquired Stuttering are linked to:*

Disturbances in timing sequences, in timing cues for initiating next motor segments in speech, finger movements:

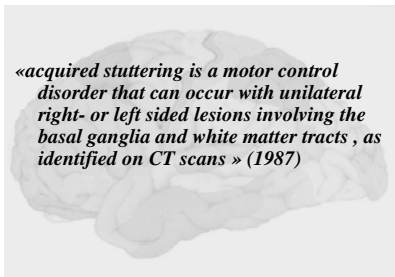
*Marshall and Neuburger (1987)
Meyers et al. (1990)
Wallesch (1990)
Ludlov et al. (1987)
Cipolotti (1988)
Bijleveld (2000)*

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Conclusions of Ludlov

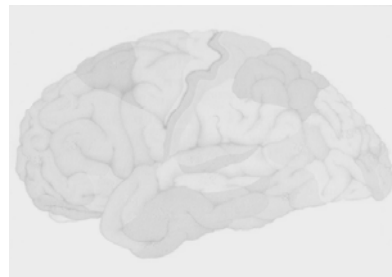
«acquired stuttering is a motor control disorder that can occur with unilateral right- or left sided lesions involving the basal ganglia and white matter tracts , as identified on CT scans » (1987)



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C.4. Studies on Basal Ganglia and developmental Stuttering



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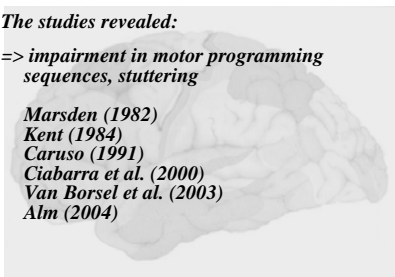
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Basal Ganglia Dysfunction and Stuttering

The studies revealed:

=> impairment in motor programming sequences, stuttering

*Marsden (1982)
Kent (1984)
Caruso (1991)
Ciabarra et al. (2000)
Van Borsel et al. (2003)
Alm (2004)*



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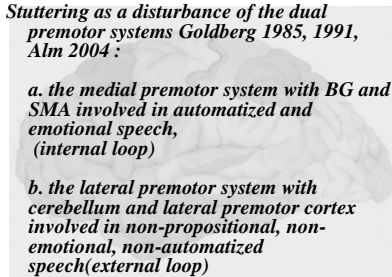
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Basal Ganglia Dysfunction and Stuttering

Stuttering as a disturbance of the dual premotor systems Goldberg 1985, 1991, Alm 2004 :

a. the medial premotor system with BG and SMA involved in automatized and emotional speech, (internal loop)

b. the lateral premotor system with cerebellum and lateral premotor cortex involved in non-propositional, non-emotional, non-automatized speech(external loop)

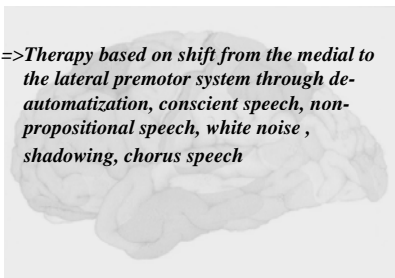


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Basal Ganglia Dysfunction and Stuttering (2)

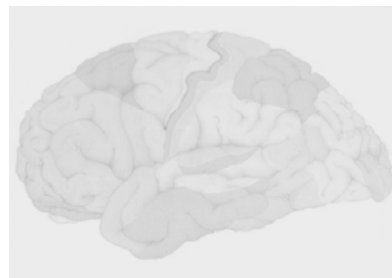
=>Therapy based on shift from the medial to the lateral premotor system through de-automatization, conscient speech, non-propositional speech, white noise , shadowing, chorus speech



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D. Basal Ganglia Dysfunction in Other Movement Disorders



D.1. Basal Ganglia in Gilles de la Tourette

TS is a developmental neurological disorder characterized by motor and vocal tics, rapid and involuntary stereotyped movements, with occasionally coprolalia and stuttering

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Ts is a developmental dopamine disorder

Groenewegen et al. (2003)

Yoshiko Nomura & Masaya Segawa (2003) stress the strong relation between basal ganglia and limbic system

Stern et al. (2000)

abnormal brain activity in anterior cingulate cortex (limbic system), SMA ... putamen and caudatum in TS

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Ts is often accompanied by OCB, hyperactivity, attention deficit

*Kurlan (1994, 1997)
Marcus and Kurlan (2001)*

« Ts is a part of a clinical spectrum that includes a range of increasing functional impairment, indicating various degrees of abnormality in basal ganglia development »

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Research Similarities between Stuttering and TS

- *Abwender et al. (1998), Molt (1999): point to involvement of limbic system and basal ganglia*
- *PET Scan: Wu et al. (1997): higher bloodflow in striatum (caudatum) during stuttering; decrease in stuttering after neuroleptic haloperidol => role of the dopamine (Maguire 2003)*

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Research Similarities between Stuttering and TS (2)

- *Marsden (1982) and Mink (1996) Basal ganglia have a control and facilitator function in motor and cognitive processes*
- *Shah et al. (2003) : stuttering may be badly diagnosed as stuttering instead of a form of TS
But: TS following RH temporal lobectomy for seizure control (Chemali et al.2003)*

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D.2. Sydenham's Chorea and Basal Ganglia

*Characterized by :
involuntary movements,
hypotonia and occasionally emotional instability*

*OCB + psychopathological vulnerability + hyperactivity in Sc
Swedo et al. (1993),
Pauls and Leckman (1986)*

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E. Research on Dopamine and Basal Ganglia

According to Mink and Thach (1993) adapted by Alm (2004), dopamine projections from Subst. Nigra pars compacta (SNc) to Striatum follow two pathways

- direct pathway with excitatory D-1 receptors: facilitation of learning behavior with + dopamine through the direct pathway (or risk of involuntary movements)
- indirect pathway with inhibitory D-2 receptors: background inhibition of behavioral activity through the indirect D-2 pathway.

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E. Research on Dopamine and Basal Ganglia (2)

- A well balanced regulation of dopamine and D1/D2 relation is essential for the functioning of BG
- Some stutters go better with D2 blockers; others worsen. Some go better with +D-1 stimulating dopamine, others don't.
- There might be subgroups of stutters: stimulant responsive and D-2 blocker responsive (Alm 2004)

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E. Research on Dopamine and Basal Ganglia (2)

Study of Garraux et al. (2007): dopamine and learning

- Dopamine input to the Striatum (Putamen) =>faster learning of movements.
- Paradoxical findings:
 - a. faster learning corresponding to lower brain activity (cfr. Braun et al.1997)
 - b. +++ dopamine in SMA = faster learning of movement = --- dopamine the the Striatum
 - c. learning aspect: prediction of movement/place

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F. Limbic System Emotions, Learning and Stuttering

Kleist 1934 postulated the role of the limbic system- and cingulate cortex- in emotions and learning. Hypothalamus might be the centre

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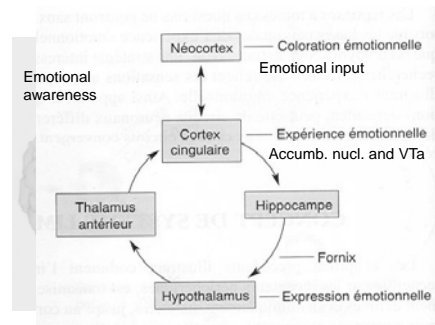
Limbic system

Papez (1937) proposed a theory for the expression of emotion in human language => circuit de Papez

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Circuit de Papez



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Other observations

- **Lamendella (1976)**
emotional language is expression of the limbic system :
« Papez gave emotion a testable physiological basis »
- **Jürgens (1982):**
lesion in A CC=>emotional utterance
- **De Nil et al. (2001,2003)**
higher activity in ACC => stuttering
(cfr. Baxter et al. 1987, 1992 , Freire Maia et al. 1999)

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G. Integrating BG , Limbic System, learning

- **Research of A.R. Damasio(2003):**
- **Distinction between emotion and sentiment**
- **link between emotions, stress and learning**
- **Functioning of LS, memory, learning and emotions depend on a delicate system of regulation of the neuromodulators: dopamine and noradrenaline and the stress hormone cortisol**

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G. Integrating BG , Limbic System, learning

- dopamine from the Snc to the Striatum=>thalamus=> => SMA for learning of movements**
- dopamine + noradrenaline from theAmygdale => hippocampus for attention and learning**
- dopamine from the Ventral Tegmental Area (Vta) : emotional aspects in learning: =>hippocampus to strenghten the learning process**
- dopamine from the Accumbens nucleus in the loop of Vta and hippocampus (motivation LS)**

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G. Integrating BG , Limbic System, learning

- excessive liberation of dopamine from VTA in excessive stress/anxiety =>inhibition of the hippocampus and the learning process**
- Liberation of stress hormone cortisol (adrenal gland) under the controle of the Hypothalamus =>hippocampus**
In case of excessive stress:+++ dopamine and +++ cortisol induce a dysfunction of the hippocampus and learning
- repetition of identical situations create identical reaction patterns**

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G. Integrating BG , Limbic System, learning and Stuttering

- **Anxiety gives rise to more dopamine and cortisol release =>temporary dysfunction of the basal ganglia
=> temporary loss of inhibitory control of movements =>stuttering**
- **Thus: psychologic and physiologic factors form a whole unit**

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H. Conclusions

- **Well learned motor sequences show decreased brain activity**
- **Stuttering is a dynamic process**
- **Role of the basal ganglia in the control of movements and learning**
- **Role of limbic system in the expression of anxiety and learning**
- **Interconnectivity in this network through**
- **the complex interactions of neurotransmitters, subcortical and cortical mechanisms**

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I. Research questions

- ***Observation in real communication situation in fMRI***
- ***Observation in children < adults***
- ***fMRI of dev. and acquired stuttering***
- ***Research on dopamine and hormones in stuttering***
- ***Research on the implication of genes***

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