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Neural correlates of neurogenic stuttering following stroke

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Introduction

- Neurogenic stuttering
  - acquired speech disorder
  - following neurological disease
    - stroke
    - TBI
    - neurodegenerative diseases (Parkinson's Disease, …)
    - other etiologies (encephalitis, epilepsy, …)
  - characterized by repetition, prolongation or blocking on sounds or syllables in a manner that interrupts the normal rhythm and flow of speech (Duffy, 2005)
  - discussion concerning secondary behaviors, emotions and attitudes associated with the stuttering (Helm-Estabrooks et al, 1999; Theys et al, 2008)

Introduction

- Focus on **stroke** patients with neurogenic stuttering
  - some characteristics seem etiology-specific (Jokel et al, 2007)
  - largest etiological subgroup (Theys et al, 2008)
  - lesion sites often less diffuse compared to other etiologies
- Lesion sites reported in case studies
  - 4 lobes, both hemispheres
  - corpus callosum, cerebellum, brainstem

Introduction

- Current study: one-year prospective study
  - incidence/prevalence
  - speech, language, hearing and cognitive functioning (Theys et al, 2011)
  - brain MRI or CT-scans

AIM: Identify lesion-symptom correlates in group of stroke patients with neurogenic stuttering

Methods

- *N* = 37
- Right-handed
- Referred by speech-language pathologist/ self-identified with dysfluent speech following stroke
- Tested for:
  - stuttering
  - aphasia
  - apraxia of speech
  - dysarthria
  - cognitive problems
  - hearing problems

Methods

- Diagnosis of stuttering?
  - >3% stuttering-like dysfluencies (SLD)
    - sound repetitions
    - syllable repetitions
    - monosyllabic word repetitions
    - prolongations
    - blocks
  - during conversation, monologue or reading
  - based on %SLD subjects attributed to
    - control group (*N* = 17)
    - neurogenic stuttering group (*N* = 20)

(Conture, 1990; Guitar, 1998)

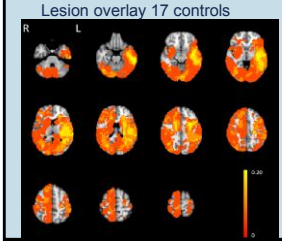
Methods

	Neurogenic stuttering (*N*=20)	Controls (*N*=17)
Median stuttering frequency, % (*IQR*)	p ≤ .001	
- conversation	4.5 (7.9)	1.5 (1.4)
- monologue	4.4 (4.2)	1.0 (1.8)
- reading of text	3.0 (3.6)	0.5 (1.5)
Co-occurring disorders, *N* (%)		
- aphasia	14 (70%)	10 of 15 (67%)
- anomia	13 (65%)	9 (53%)
- dysarthria	9 (45%)	4 (24%)
- apraxia of speech	5 (25%)	2 (12%)
- cognitive problems	6 (30%)	4 of 15 (27%)
Median age (*IQR*)	72 y. (15 y.)	69 y. (20y.)
Male-female ratio	2:1 (7 females)	2:1 (6 females)
Median number of lesioned voxels	3831 (9154)	11139 (17382)

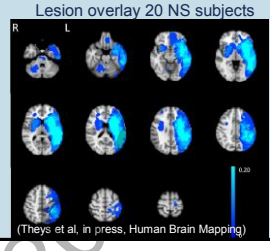
Methods

- MRI (*N*=29) or CT-scan (*N*=8)
- lesions drawn manually on FLAIR image or MNI-template in MRIcroN
- normalized to MNI-template & binarized in SPM5

Lesion overlay 17 controls



Lesion overlay 20 NS subjects



Methods

- Lesion-symptom mapping with vBLSM (voxel-based Bayesian Lesion Symptom Mapping)
- vBLSM calculates probability (*Pr*) in a certain voxel that the lesion proportion in the group with neurogenic stuttering (η_A) is larger than the proportion of lesions in the control group (η_B)

$$d(\delta)=Pr((\eta_A-\eta_B)\geq\delta)$$
with $\delta=0$ and $d(0) \geq 0.95$

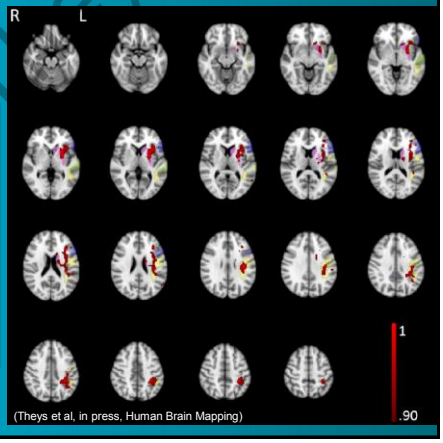
(Chen & Herskovits, 2010)

Results

- vBLSM analysis shows 9 areas in the left hemisphere with a probability of >.95 of larger lesion proportions in the neurogenic stuttering group compared to the control group
  1. GM left inferior frontal gyrus
  2. WM left inferior frontal sulcus
  3. WM left subcentral sulcus, supramarginal gyrus & angular gyrus
  4. GM left superior temporal sulcus, WM left superior temporal gyrus
  5. GM left inferior temporal sulcus
  6. GM left intraparietal sulcus
  7. left putamen
  8. left nucleus caudatus
  9. left internal capsule

(Theys et al, in press, Human Brain Mapping)

- red-black: probability map with d(0) ≥ 0.90
- pink: striatum
- blue: inferior frontal gyrus
- green: superior temporal gyrus
- yellow: superior longitudinal fasciculus



Discussion

- Areas of grey and white matter in left hemisphere differentiating between subjects with neurogenic stuttering and control patients without stuttering
  - ⇒ largely overlapping with cortico-basal ganglia-cortical ***network*** comprising of inferior frontal cortex, superior temporal cortex, intraparietal cortex, basal ganglia, superior longitudinal fasciculus and internal capsule
  - ⇒ stroke-induced neurogenic stuttering can not be linked to one specific brain area
  - ⇒ onset can be related to ***lesion in different areas in network*** involving cortico-basal ganglia-cortical structures
  - ⇒ onset dependent on disintegration of processes such as articulatory planning, perceptual processing and internal timing

Discussion

- Consistent with most case studies of stroke-induced stuttering (for review see De Nil et al, 2009)
- Limited lesion coverage does not permit exclusion of other areas as important for neurogenic stuttering
- Involvement of frontal white matter, internal capsule and striatum consistent with lesion localizations reported in a retrospective study on head injury patients (Ludlow et al, 1987)
- Many of the areas associated with neurogenic stuttering in the present study have also been associated with developmental stuttering (Neumann & Euler, 2010)
  - similar neural characteristics?
- Interindividual differences in localization ~ different behavioral characteristics?

Conclusion

dysfunction in network involving
inferior frontal cortex, superior temporal cortex,
intraparietal cortex, basal ganglia
and their interconnections

↓

disintegration of neural functions
necessary for speech

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occurrence of neurogenic stuttering-like dysfluencies
following stroke

Thank you for your attention!

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