SENIOR CAPSTONE PROJECT - COMMUNICATION DISORDERS AND SCIENCES - SUNY PLATTSBURGH

THE "STUTTERING" BRAIN: ANATOMY AND ACTIVATION DIFFERENCES

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INTRODUCTION

For many years, neuroimaging has chased the "stuttering" brain in an attempt to identify the ways in which it differs from the "non-stuttering" brain in structure, function, and connectivity, and to discover the origin of stuttering. Researchers, including Alm (2004), Brown et al. (2005), and Kell et al. (2009), have sought to understand how the knowledge of brain differences and "repair" of stuttering can provide clinical insight. This capstone will explore this field of inquiry. First, pertinent structural and activation differences in people who stutter (PWS) compared to people who are normally fluent (PNF), as revealed in research, will be examined. Second, the conclusions drawn from the findings will be discussed. Third, the clinical implications revealed by the researchers will be shared.

ORIGIN OF STUTTERING?

Broca's Area Anomaly | **White Matter Deficit** | **Basal Ganglia Dysfunction**

DEFINITIONS & BACKGROUND KNOWLEDGE:

Stuttering is a developmental disorder that affects the speech fluency of 5% of preschool children and 1% of the adult population (Bloodstein, 1995). Developmental stuttering is stuttering that develops before puberty and is referred to as persistent developmental stuttering (PDS) when it continues into adulthood (Jancke, Hanggi,& Steinmetz, 2004). Stuttering is characterized by speech events that may involve whole-word repetitions, part-word repetitions, sound prolongations, and silent blockages; secondary behaviors may be associated with this condition (American Speech-Language-Hearing Association [ASHA], 1999).

CRITICAL REVIEW AND META-ANALYSIS

Alm (2004) and Brown, Ingham, Ingham, Laird, and Fox (2005) both proposed core dysfunctions in anatomy and connectivity in the "stuttering brain." Alm (2004), in his critical review of stuttering and the basal-ganglia, proposed that the basal ganglia-thalamocortical motor circuit through the putamen to the supplementary motor area is dysfunctional in PWS (Figure 1). But this dysfunction may have a variety of causes. The basal ganglia consist of a large set of interconnected subcortical nuclei that are the largest subcortical structures in the forebrain (Alm, 2004). The functions of the basal ganglia are dependent on the functions of the cerebral cortex and the white matter connections (Alm, 2004). According to Parent (1996), the main input nucleus of the basal ganglia, the striatum, receives topographical excitatory projections from almost the entire cortex, including the sensorimotor and frontal cortex (as cited in Alm, 2004). Alm (2004), proposed several conclusions in support of basal ganglia dysfunction in PWS:

1. The basal ganglia are believed to be impaired in the ability to produce accurate timing cues in PWS (Alm, 2004). Conditions that provide compensatory timing cues, such as choral reading and singing, support this claim because these conditions create fluent speech in PWS (Alm, 2004).

2. Conditions that seem to alleviate stuttering, such as novel modes of speaking, seem to be effective because these conditions de-automatize speech control; the basal ganglia is involved with automatic sequential movement (Alm, 2004).

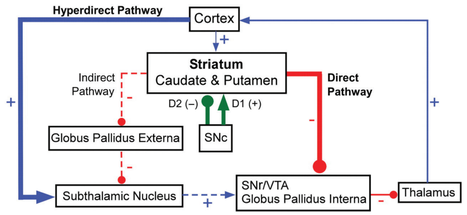

Figure 1

Alm (2004) suggested that the defective basal-ganglia circuit might be offset by a cerebellar-cortical circuit, in a by-pass route. This finding is one explanation for one of the neural signatures Brown (2005) proposes: the overactivation of the cerebellum seen in PWS.

An ALE meta-analysis examining functional neuroimaging studies of PWS and PNF identified three neural signatures of stuttering. But the role of the basal ganglia was revealed to be very small in this study (Brown et al., 2005). The three neural signatures are as follows:

- Overactivation of the right frontal operculum (the homologue for Broca's area) and anterior insula;
- Absence of activation in the auditory cortex bilaterally;
- Overactivation of the cerebellum.

NEURAL NETWORKS & STUTTERING

Researchers, such as Lu et al. (2009), examined large neural networks implicated in stuttering and found the following activation differences between PWS and PNF:

- Greater activation in the right frontal operculum and anterior insula in PWS (Brown et al., 2005; Lu et al., 2009);
- weaker activation in the left anterior part of the superior temporal gyrus (Lu et al., 2009; Brown et al., 2005; De Nil et al., 2008);
- underactivation of the left anterior temporal cortex involved in auditory feedback (Lu et al., 2009);
- greater activation in the left posterior temporal cortex — involved in phonological code retrieval, (Lu et al., 2009);
- activation in the right cerebellum in PWS, but not in PNF — the cerebellum refines movement (Lu et al., 2009; Brown et al., 2005).

Unlike Brown et al. (2005), Lu et al. (2009) found activation instead of overactivation, but this still demonstrates a difference between PWS and PNF, albeit one that is more similar than different.

- The loop of the angular gyrus-middle frontal gyrus-left precentral gyrus-right anterior middle temporal gyrus was found to be disconnected in PWS (Lu et al., 2009);
- Additional connections were found between the right inferior frontal gyrus and left inferior frontal gyrus/right angular gyrus (Lu et al., 2009).

De Nil et al. (2008) examined neural activation of PWS and PNF and found the following:

- Less activation in PWS compared to PNF during natural speech:
  - Left superior temporal gyrus
- Increased activation in PWS compared to PNF during simulated stuttering:
  - Right inferior frontal gyrus

Lu et al. (2009) conclude that the disconnection listed above reflect dysfunction in what the authors refer to as the self-monitoring system. This system model integrates the cerebellum, angular gyrus, and frontal motor areas. Lu et al. (2009) also conclude that the overall results of their research support the hypothesis of Brown et al. (2005) that the underactivity of the auditory cortex is caused by repeated delivery of the perceptual prediction. Furthermore, De Nil et al. (2008) also found that PNF had peak activation in the left superior temporal gyrus, which was less activated in PWS. This lends further support to the hypothesis that this area is involved with dysfunction in the self-monitoring system of PWS (Lu et al., 2009). According to Hickok, Buchsbaum, Humphries, and Muftuler (2003), activation in the superior temporal gyrus (STG) during overt speech represents self-generated feedback and it also changes the auditory input into speech gestures (as cited in De Nil et al., 2008). Furthermore, De Nil et al. (2008) believe this finding is related to the structural anomalies that have been found in previous research and difficulty with auditory processing believed to be present in PWS.

Several structural anomalies have been found in PWS. Foundas et al. (2001) found that PWS often have extra gyri in Broca's area, as well as the sensorimotor cortex in the area related to the articulatory organs. Increased size and symmetry of the planum temporale (PT) in PWS was a significant finding in the research of Foundas et al. (2001). Although the gyrus findings also stand out, no single feature seemed to distinguished PWS from PNF; instead widely distributed neural networks differed (Foundas et al., 2001), which is what Lu et al.(2009) also found to be true.

MOTOR PLANNING AND EXECUTION IN PWS COMPARED TO PNF

Chunming, Chen, and Ning (2010) examined atypical planning and execution processes in PWS under different conditions during a picture-naming task in order to identify the planning and execution processes that PWS use. The researchers found the following activation differences:

- Atypical neural activations for the planning of speech were revealed in the bilateral inferior frontal gyrus and right putamen (Chunming et al., 2010).
- Atypical neural activations for the execution of speech were revealed in the right cerebellum, right insula, left premotor area (PMA), and angular gyrus (Chunming et al., 2010).

The structural equation modeling (SEM) results revealed two parallel neural circuits involved in atypical planning and execution in stuttering: the basal ganglia-IFG/PMA circuit and the cerebellum-PMA circuit (Chunming et al., 2010). The researchers also found that the angular gyrus seemed to be involved in the interface between atypical planning and execution processes in PWS (Chunming et al., 2010).

ANOMALIES IN CHILDREN WHO STUTTER

Chang et al. (2008) examined the differences in the brain anatomy of children who stutter (CWS) and found the following differences in comparison to children that are normally fluent (CNF):

- CWS had reduced gray matter volume (GMV) in the following areas:
- the left inferior frontal gyrus (Broca's area)
- bilateral temporal regions (Wernicke's area on the left)
- CWS had reduced white matter integrity in the following areas:
  - Underlying area of the motor region for the face and larynx

Chang et al. (2008) also found differences between CWS and PWS:

CWS	PWS
• In CWS no differences were found in the right hemisphere. • In CWS no differences were present in right-left asymmetries.	• In PWS right hemisphere differences are present. • In PWS there are many right-left asymmetries.

Spontaneous Repair

Perhaps these areas of difference in CWS may be the origin of the "stuttering brain" (Chang et al., 2008).

Chang et al. (2008) suggest that the anatomical and functional increases in PWS that have been documented in the previous literature (e.g. Foundas et al., 2001; De Nil et al., 2008) are likely to be compensatory. It seems especially likely based on the research findings of Chang et al. (2008). It appears that the differences seen in PWS are likely to be caused by a lifetime of coping with stuttering (Chang et al., 2008). Relying on the right hemisphere speech areas over many years of persistent stuttering could result in increased GMV on the right side and reduced asymmetry (Chang et al., 2008). Kell (2009) found that unassisted recovery from stuttering in adulthood was found to be associated with activation of the left BA 47/12, adjacent to the white matter anomaly found in both CWS and PWS (Chang et al., 2008; Foundas et al., 2001) The grey cross in figure 2 represents the anatomical anomalies found in PWS (Chang et al., 2008; Kell et al., 2009).

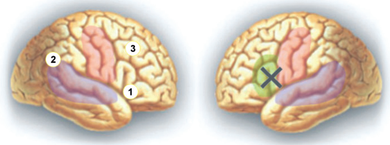

Figure 2 (Kell et al., 2009)

Fluency-shaping therapies "normalize" the overactivation of the right hemisphere, normalize basal ganglia activity, and activate the left hemisphere speech and language areas (De Nil et al., 2003; Neumann et al., 2008; Giraud et al., 2008).

Successful management of stuttering typically requires repeated therapy and maintenance in order to maintain gains in fluency; so the shift of brain activity to the left hemisphere in PWS after therapy is usually transient (Kell et al., 2009). This indicates an insufficient neural repair process (Kell et al., 2009).

Kell et al. (2009) believe identifying the area of spontaneous recovery in the left BA 47/12 region, adjacent to a white matter anomaly seen in persistent stutterers but found to be normal in recovered stutterers, demonstrates that the optimal repair follows a "very focal perianomalous plasticity" (p. 2758).

CONCLUSION

In conclusion, researchers now feel that stuttering is a neurodevelopmental disorder with the core dysfunction revolving around the left hemisphere — Broca's area and underlying white matter — anomalies (Chang et al., 2008) and that the right hemisphere overactivations, bilateral auditory underactivations, and basal ganglia dysfunction seen in PWS represent compensatory mechanisms in the brain's attempt to "normalize" the stuttering brain.

In the future, the knowledge of brain structure and activations present in CWS may allow for early diagnosis and intervention before the stuttering becomes persistent (Chang et al., 2008). Perhaps taking advantage of the plasticity of the child's brain may allow for earlier and more permanent neural changes prior to the right hemisphere compensations.

Biofeedback has been used successfully in many applications and may possibly be applied to the "stuttering" brain. Heart rate variability biofeedback appears to increase activity in the medial prefrontal cortex which inhibits the amygdala and insula, decreasing state anxiety (Prinsloo et al., 2013). If a PWS could "see" that they were activating the optimal repair region, BA 47/12, through some type of biofeedback mechanism then perhaps that could assist PWS to utilize that region during therapy and then be able to generalize it outside the clinic.

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