

The King's Speech:

A Psychiatric Perspective on Stuttering

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Learning Objectives

1. **Review diagnosis, epidemiology, and pathophysiology**
2. **Explore psychiatric comorbidity and functional impact**
3. **Understand treatment: then and now**
4. **Apply concepts to a formulation of Bertie**
5. **Consider what *The King's Speech* gets right and what it leaves out**

Medication may reduce the volume of the struggle – but the goal is a richer life, not just fewer stutters.

Overview

- What is stuttering?
- Epidemiology
- Pathophysiology: a multifactorial neurodevelopmental disorder
- Why can anxiety make stuttering worse without causing it?
- Psychiatric comorbidity and functional burden
- Treatment: then and now
- Bertie: a psychiatric formulation
- What *The King's Speech* gets right – and what it leaves out



A Remarkable Story - Real People

Colin Firth as King George VI
(The King's Speech, 2010)



King George VI
(1895-1952)



The only thing worse than stuttering is not being heard at all.

What is stuttering?

A neurodevelopmental fluency disorder involving disruptions in the normal flow of speech.

- Repetitions (e.g., “b-b-better”)
- Prolongations (e.g., “sssspeech”)
- Blocks (silent struggle)
- Often associated with physical tension and avoidance behaviors
- Variable across situations
- Often worsens with pressure
- Not caused by low intelligence, weakness, or poor parenting

“I…I…I…”

Epidemiology



1% of children
(ages 2-5)



75-80%
Recover spontaneously



1% of adults
(~70 million worldwide) continue to stutter



Present in all cultures,
languages, and socioeconomic groups



More common in males (3-4:1)

Pathophysiology of Developmental Stuttering

A Multifactorial Neurodevelopmental Disorder

Prefrontal Cortex

- Involved in executive function, planning, and cognitive control of speech
- Altered activation and connectivity in people who stutter (PWS)

Left Inferior Frontal Gyrus (Broca's Area)

- Critical for speech planning and language processing
- Atypical activation and reduced coordination with motor regions in PWS

Motor Cortex

- Controls the motor execution of speech
- Altered activity and timing in PWS

Somatosensory Cortex

- Processes sensory feedback from speech movements
- Atypical sensory-motor integration in PWS

Basal Ganglia

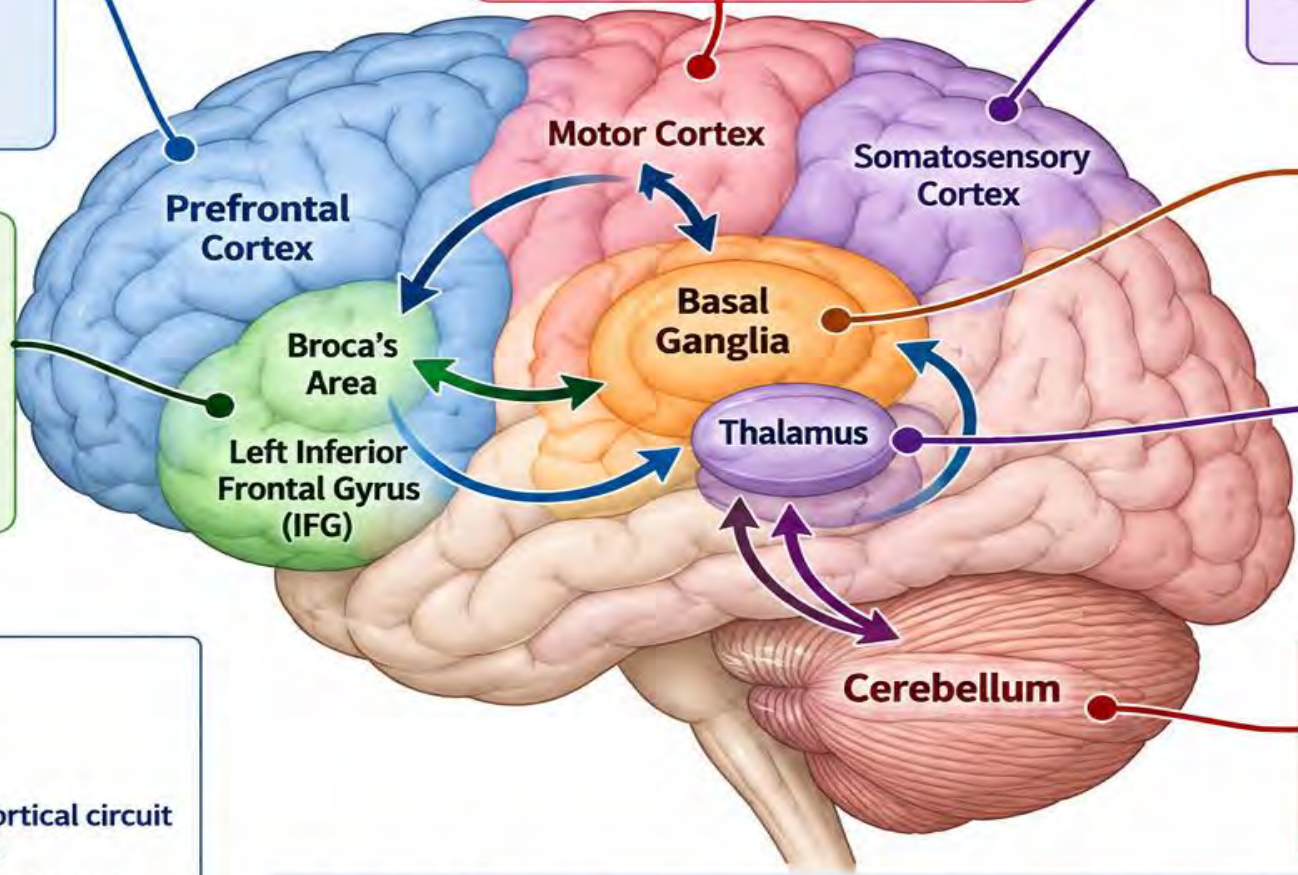
- Involved in motor learning, timing, and sequence initiation
- Structural and functional differences observed in PWS

Thalamus

- Relays information between subcortical and cortical regions
- Altered thalamo-cortical connectivity in PWS

Cerebellum

- Important for motor coordination, timing, and error monitoring
- Atypical structure and activation in PWS



Key Neural Circuits

- **Cortico-striatal circuit** (planning and initiation)
- **Basal ganglia–thalamo-cortical circuit** (motor control and timing)
- **Cerebello-thalamo-cortical circuit** (coordination and feedback)

Stuttering arises from atypical structure and function within a distributed speech network, involving cortical, subcortical, and cerebellar regions.

Why Can Anxiety Make Stuttering Worse Without Causing It?

Anxiety Amplifies	But Does Not Cause It
Increases physiological arousal (sympathetic activation)	Stuttering is present even in low-anxiety situations
Heightens self-monitoring and performance pressure	Occurs in young children before social anxiety develops
Narrows attentional focus	Persists during sleep, singing, and automatic speech
Increase muscle tension and speech effort	Not explained by psychological trauma or parenting style
Leads to avoidance and safety behaviors	Anxiety is a consequence and a perpetuating factor, not the origin

Stuttering and anxiety interact - but they are distinct.



DSM-5-TR: Childhood-Onset Fluency Disorder (Stuttering)

- Frequent disruptions in normal fluency
- Onset in early developmental period
- Causes clinically significant distress or impairment
- Not attributable to another condition
- If another condition is present, severity exceeds what would be expected

Stuttering is a disorder of fluency, not of intelligence, psychological strength, or parenting.

Differential Diagnosis

Type	Key Features	Clinical Clues
Developmental stuttering	Onset in childhood, chronic course	Most common
Neurologic stuttering	Acquired after brain injury (e.g., stroke, TBI, neurodegenerative)	Older onset, other neurologic signs
Psychogenic/ functional	Sudden onset, often – after psychological stress	Inconsistent patterns
Medication-induced	Associated with dopamine agents (e.g., antipsychotics, levodopa)	Temporal relationship to medication