Drugs for Speech: Could Pharmacological Treatment of Speech Disorders Displace Speech Therapy?

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Introduction

We can all picture speech therapy. A smiling therapist sits with their patient, likely a young boy, showing him pictures of colors and trying to get him to say the word "blue." He struggles with the introductory "buh" sound, and the therapist repeats it to him, exaggerating their facial movements in an effort to help him do the same. In this scenario, the child likely struggles with stuttering, either repeating the "buh" sound or being unable to produce it at all.

This depiction of speech therapy is common and demonstrates a large truth: adolescent boys are the largest demographic affected by stuttering (Howell, 2007). However, speech therapy serves a much broader purpose—people from all ethnicities, all age groups, and all gender identities have speech disorders and seek therapy as a result. In the United States alone, children who experience speech problems receive speech therapy at rates of around 66–67% (HHS, 2015). Additionally, stuttering is far from the only speech disorder that is treated in speech therapy. Speech disorders are complex and range from aphasia—which impairs language expression and comprehension—to dysphonia—which causes pitch and volume issues—to apraxia of speech, which disrupts lip, tongue, and jaw movements.

Speech disorders can arise for a myriad of reasons. Genetics, such as the genes GNPTAB and GNPTG, have been found to play a large role in the etiology, or cause, of stuttering (Frigerio-Domingues & Drayna, 2017). Twin studies demonstrate a higher correlation in stuttering frequency among monozygotic (identical) twins when compared to fraternal twins (Dworzynski et al., 2007). Independent of genetics, neurodegenerative conditions such as Parkinson's disease and Alzheimer's, as well as traumatic brain injuries, are associated with speech impairment by damaging the brain areas responsible for speech

processing and production. Physical conditions, such as impaired vocal cords or nerve damage, can also impact speech (Penn Medicine, 2022).

Beyond affecting different demographics and resulting from various etiologies, speech disorders can range significantly in both severity and impact. It is no secret that impaired communication abilities can have serious consequences for those afflicted by them, including but not limited to mental health issues, impaired social relationships, and missed job opportunities (Wilmot et al., 2024). Studies have found negative correlations between speech-language disorder severity and college attendance as well as evidence that adults with speech-language disorders are less likely to participate in the workforce. When they do, they are more likely to work in jobs involving unskilled manual labor and are more likely to face termination than their fluent coworkers. Furthermore, school-age children who stutter are up to six times more likely to suffer from social anxiety disorder and are around seven times more likely to have generalized anxiety disorder than their fluent classmates (Foster et al., 2023). While stuttering does not encompass all speech disorders, its impacts can be reasonably assumed to extend to children with other impairments.

Because there is so much variance between disorders, one question arises: how can speech therapy effectively treat all of them? What distinctions exist between therapeutic approaches to various disorders, and, if some aren't effective, where else can we look for therapeutic impact?

The Current State of Speech Therapy

As a current practice, speech therapy is multifaceted. Speech therapy sessions are tailored to the needs of each patient, yet standard practice uses many of the same techniques. These practices most often include exercises for breathing and swallowing, recall exercises for those struggling with word identification, and sound formation exercises (Santos-Longhurst, 2019). Speech therapy sessions are typically held frequently for about 30–60 minutes each, and many patients who have persistent disorders remain in therapy for years at a time (NLM, 2022).

Speech therapy has helped millions deal with the physical and social difficulties that result from speech disorders. Patients have learned to better produce coherent sentences, overcome inabilities to articulate certain sounds, redevelop speaking ability after strokes, and more. However, the average cost of a speech therapy session in the United States is between $100 and $250 per hour, and not everyone may be willing or able to pay this cost and/or sacrifice their time (Geller, 2024). Whether a working dad or a college student with classes, a potential beneficiary of speech therapy may not always be able to access it.

Furthermore, speech therapy is not a permanent solution for some patients. Without continued treatment, relapse into old and less fluent ways of speaking is possible, especially for patients who invest extra time and effort into a new form of communication (Tichenor & Yaruss, 2020). These methods often emphasize breathing and word-forming techniques that are unnatural to the speaker (Lowe et al., 2021). Without motivation and practice after one's therapy ends, regression—whether it be large or small—is relatively common. In extreme cases, when a patient's "new" way of speaking is too difficult to maintain and expends too much mental energy, full relapse can occur.

Drug-Induced Stuttering: What We Know and Implications

When speech therapy fails to have lasting effects or is unfavorable to someone's socioeconomic circumstances, attention can shift to the possibility of medication. While there are very few known drugs to alleviate speech disorders, and there are no FDA-approved medications specifically for stuttering, clinical data has provided insight into potential neurological targets (Maguire et al., 2020). Observations of induced speech disorders have been recorded from clinical trials and standard medications regularly given to patients. This question then remains: what are these medications targeting, and could a common factor between them be applied to the therapeutic treatment of speech disorders?

Several pharmaceuticals have resulted in side effects involving stuttering, particularly antidepressants, antiepileptics, and antipsychotics, all of which are involved in altering neuronal signals (Nikvarz & Sabouri, 2022). Neurons fire action potentials, or electrical signals that travel down the length of the axon after receiving information, and these medications impact action potential frequency, which can influence the strength of a neurological signal.

Interestingly, neurological pathway interference has not been observed to impact stuttering in all patients

identically, with some drugs causing different effects in different patients (Brady, 2020). Risperidone, an antipsychotic that balances dopamine and serotonin levels, has been observed in multiple cases to both cause and alleviate stuttering presentation (Atay et al., 2014). Clozapine, another serotonin and dopamine balancing antipsychotic used for schizophrenia, has displayed induced stuttering, even influencing severity according to medication dosage (Jaguga, 2021). Haloperidol, a dopamine antagonist antipsychotic used in similar schizophrenic cases, has displayed fluency-improving effects for many years (Murray et al., 1977; Andrews & Dozsa, 1977). With these conflicting impacts on stuttering displayed in a snapshot of the many medications shown to impact speech fluency, it is difficult to progress toward a unified pharmaceutical treatment. However, seemingly conflicting stuttering presentations can still provide great insight into speech disorder etiology and potential treatment targets.

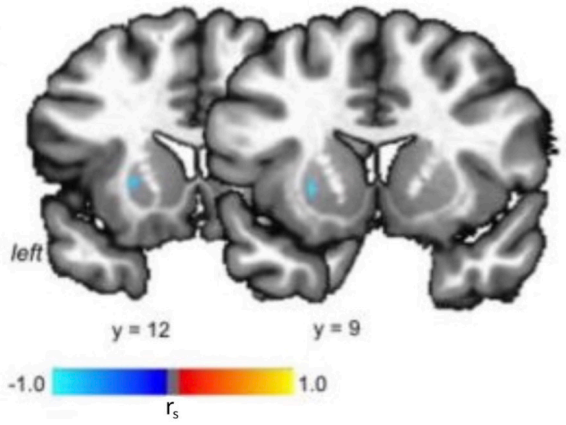

Figure 1. Speech induced brain activation and dopamine coupling is shown. This occurs in the ventromedial portion of the left hemisphere, providing evidence for a linkage between dopamine expression levels and human speech. The color bar depicts Spearman's correlation coefficients (rs), which measure direction and strength of variable association. These range from -1 to 1. The light blue color shown indicates a strong association between dopamine expression and brain activity (Simonyan et al., 2013).

Dopaminergic Pharmaceuticals: Impact and Function

One overwhelmingly common factor within the current therapeutics shown to impact speech ability is dopamine (Maguire et al., 2020). Drugs that have displayed treatment impacts in stuttering, Tourette's syndrome, and generalized fluency issues include vesicular monoamine transporter 2 (VMAT2) inhibitors—which deplete dopamine—and serotonin-dopamine antagonists (SDAs) (Makhoul & Jankovic, 2023.; Lavid, n.d.).

Dopamine-related medications that influence speech are either agonists or antagonists—meaning that they either activate or block dopamine receptors—and therefore stimulate or inhibit neuronal activation. These medications are often used in the treatment of mental health disorders such as depression and schizophrenia that are associated with dopamine dysregulation, such as the deficit of dopamine in the ventral tegmental area and the hyperactivity in the subcortical brain associated with major depressive disorder and schizophrenia respectively (Conn et al., 2020; Belujon & Grace, 2017). This coexisting impact of dopamine agonists and antagonists on these disorders and speech has led to one theory surrounding speech disorders (independent of those caused by physical injury): similar to the hyperactivity associated with these mental health conditions, speech disorders such as stuttering and aphasia may be associated with unusual activity levels in dopaminergic pathways within the brain, leading to either hyper- or hypoactivity that inhibits the proper synthesis and production of speech (Turk et al., 2021).

Many studies have demonstrated clear connections between dopamine and speech production. A recent study has provided the first concrete evidence of endogenous dopamine expression during human speech production, showing expression in the ventromedial portion of the lateral striatum in the left hemisphere, or the brain region most strongly associated with motor and reward (**Fig. 1**, Simonyan et al., 2013). The basal ganglia is the center for dopamine production under normal conditions, after which the neurotransmitter spreads to the frontal cortex, midbrain, and brainstem. Dopamine is largely accepted as crucial to motor function, as its modulation has proven very important to speech production and other types of vocalizations. Despite the large scientific acceptance of dopamine as crucial to motor control within the brain, not much is understood about the chemical basis of this impact on speech (Turk et al., 2021).

Recent studies have implicated astrocytes—star-shaped glial cells that support the nervous system—in speech regulation because of their close connection to the dopaminergic pathways in the brain and impact in involved conditions such as Parkinson's or stuttering (**Fig. 2**). Another indicative finding is that calcium levels, which dictate glial cell function, often change in unison with dopamine levels in relevant brain areas (Turk et al., 2021).

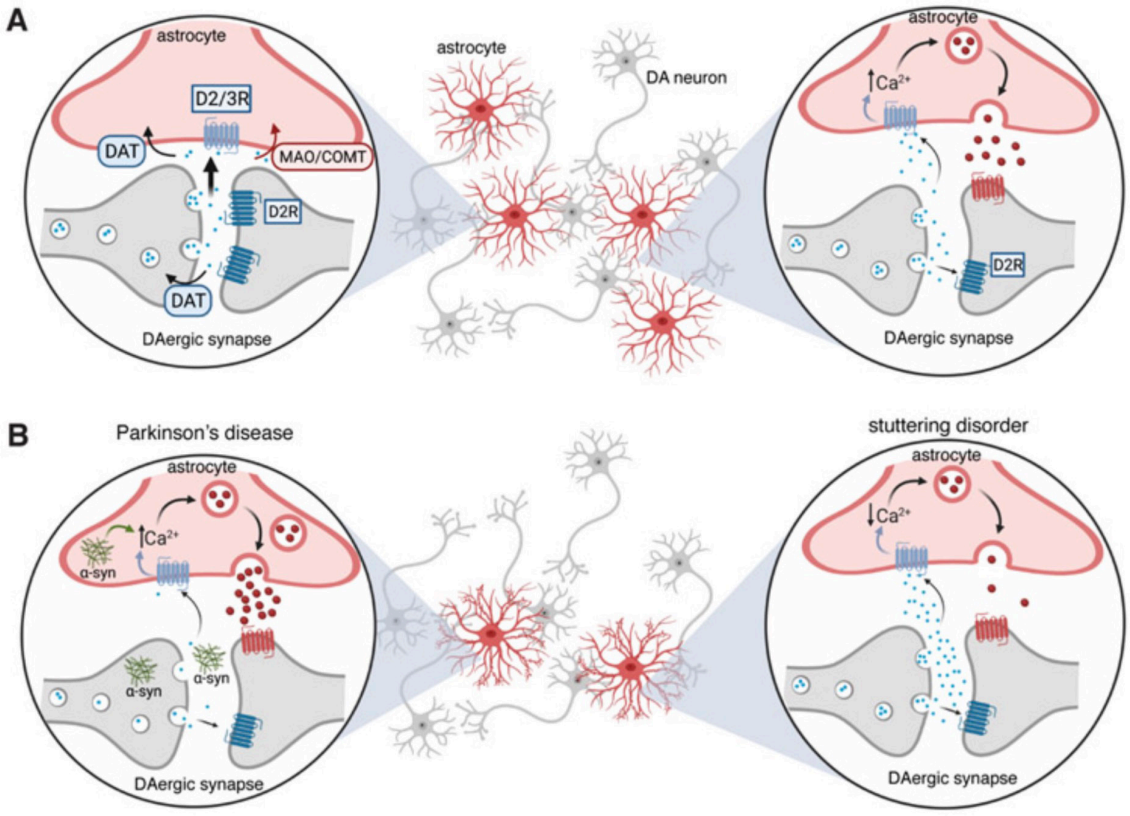

Figure 2. [A] Dopaminergic circuits involving astrocytes in the basal ganglia are depicted. In each of the four pictures, the presynaptic dopaminergic neuron (right gray) releases dopamine into the synaptic cleft that is detected by postsynaptic dopamine receptors on the postsynaptic neuron (right gray). Astrocytes (red) play a role in recycling dopamine, as after they detect dopamine their intracellular calcium levels increase, leading to the release of gliotransmitters(chemicals that influence neuronal function and communication). **[B]** Two different diseased synapses are shown. Parkinson's disease results in excessive release of gliotransmitters. In the proposed stuttering model, excessive dopamine release actually results in decreased calcium levels in the astrocytes, leading to decreased gliotransmitter release (Turk et al., 2021).

These areas include the substantia nigra pars compacta (SNc), which is a brain region comprised of dopaminergic neurons associated with motor and emotional regulation as well as speech, and the ventral tegmental area (VTA), which also mediates dopamine levels and plays a crucial role in reward processing. Lastly, the nucleus accumbens (NAc) is also implicated, which is associated with speech and emotional behavior. An apparent connection between emotionality and speech production can be observed when considering brain region functionality, which further supports the evidence surrounding dopamine, normally used to treat many emotionally driven disorders, in speech disorder treatment.

Further, dopamine has even displayed an impact on neurodegenerative speech conditions such as Parkinson's disease (PD). In PD, characteristic loss of laryngeal muscle activity is thought to derive from a loss of binding at dopamine receptors, an effect that was propagated in a study that utilized dopamine antagonists to further inhibit dopamine activity (Feng et al., 2009). This led to severe vocal inability, demonstrating an etiology stemming from dopamine-associated underactivity as opposed to the hyperactivity usually thought to cause disorders like stuttering.

Differential Drug Use: Treating the Source or the Symptoms

The success of dopaminergic pharmaceuticals as well as the apparent linkage between emotional brain centers and those responsible for speech production raises important questions: what aspect of speech disorders are these drugs targeting? Are these dopaminergic therapeutics truly targeting the speech pathways, or potentially targeting the emotional pathways to which they are connected? As it is known that anxiety and depression levels have positive correlations with most speech disorder severities, could it be that these drugs are just having a typical therapeutic effect, calming the nerves to help fluency (Bernard & Norbury, 2023; Lockett, 2021). If this is true, would it make the therapeutic

pursuit of these agents less valid? These questions can only be answered by more research. Ultimately, the purpose of therapeutic development is to offer speech disorder patients alternative pathways to obtain the therapeutic effect they are pursuing.

Dopaminergic drugs also pose safety risks, as do many commercial therapeutics if used in the wrong dosages or with unknown conflicting medical conditions. The most common side effects include dizziness, headaches, and arrhythmia (irregular heartbeat); in older patients, psychiatric disturbances such as anxiety, depression, and hallucinations can become more common. These risks may be well worth it to a patient with severe social and workplace repercussions for their speech disorder and must be evaluated on a case-by-case basis.

Conclusion: Where are we now?

Therapeutic development is no easy task, and the conflicting presentations of speech disorders in patients are another barrier. If FDA approval is not guaranteed or even feasible, will speech disorder medications be offered on a case-by-case, experimental basis? As of now, the future of therapeutic development for speech disorders remains both uncertain and largely patient-based. Patients willing to experiment with different medications for the sake of their eventual fluency will advance our records and eventually improve their social efficacy, but will this advance therapeutic development? The fundamental complexity of speech is daunting, and time will reveal whether such individual efforts can bridge the gap in our understanding.

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