Here's How to Treat Childhood Apraxia of Speech

Third Edition

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Foreword

The clinical practice and especially the science associated with childhood apraxia of speech (CAS) are relatively new compared to many other communicative disorders. In the last 20 years, there has been an explosion of work in this area, in terms of both basic science and clinical assessment tools and treatment strategies. This third edition of *Here's How to Treat Childhood Apraxia of Speech* adds to that literature in a very practical way. It is not always easy to bridge the gap between research and the application of that research to clinical practice. The authors are excellent clinicians who have brought that broad experience to this book. They reviewed a great deal of the science and evidence base in a way that practicing clinicians will appreciate and be able to use. Further, the authors recognize that every child brings individual challenges with respect to severity, personality, previous experience with therapy, comorbidities, and parent involvement, and they provide ways to approach these challenges.

The 13 chapters of this edition cover a wide range of topics. Chapter 1 discusses the nature of CAS, the neurological implications, genetic variants associated with the disorder, the core characteristics, and many associated disorders that may occur with CAS. Chapter 2 focuses on assessment strategies leading to differential diagnosis of CAS. Here the authors provide a framework for assessment in general and strategies for evaluation that covers cognition and language as well as speech. They provide some direction for assessing children for whom English is not their first language and give specific strategies for different ages, encouraging clinicians to use clinical thinking when devising assessment protocols for individual children. There are many tables and worksheets that clinicians may find helpful for assessing children with CAS, as well as many children with other types of speech sound disorders. Chapter 3 focuses on the fundamentals of treatment, with special attention to the principles of motor learning, the application of which is essential in the treatment of CAS. In addition to providing a broad description of several important principles to incorporate into treatment, the authors provide many practical tasks to allow for the repetitive practice often required in treating CAS. Further, they describe different activities that may apply to a variety of children and contexts.

The authors discuss both external and internal evidence to inform treatment decisions in Chapter 4. They describe the different published treatment methods and the evidence that has been shown for their efficacy. Chapter 5 focuses on treatment strategies for vowel accuracy and natural prosody, both which are common areas of difficulty for children with CAS. This book, however, goes beyond treatment specific to CAS. For example, Chapter 6 focuses on activities for minimally verbal children including children at risk for CAS or any child who may need very early speech-language intervention. Parents and caregivers will appreciate tables related to developmental milestones as well as activities that support early language and speech development.

While Chapter 7 addresses early literacy in CAS, the treatment strategies suggested would likely be appropriate for many children with speech sound disorders who are having trouble learning to read. The authors provide a thorough discussion of the types of phonological awareness and early literacy challenges as well as suggestions for working with these challenges in the context of speech sensorimotor planning treatment. Likewise, clinicians will appreciate Chapter 8 in which strategies for supporting the needs of older children are discussed. These strategies addressing residual articulation errors, persistent phonological patterns, cluster reduction, and reduced comprehensibility will be extremely helpful for older children with CAS as well as children with other speech sound disorders. While the authors discussed many ways to facilitate expressive language development in younger children in Chapter 6, this chapter provides information in treating ongoing language issues. Strategies for improving vocabulary, grammar, conversational skills, and use of language in social interactions will be widely appreciated by clinicians.

Two important additional chapters appear in this edition: treating children with concomitant disorders and considerations of treatment via telepractice. Many children with CAS present with a large variety of co-occurring disorders. Expressive language impairment and phonological disorders are very common, and practical suggestions for addressing these linguistic deficits are presented throughout the book. Described in Chapter 9 are treatment strategies for those children who also exhibit dysarthria, attention deficit hyperactivity disorder, autism, and developmental coordination disorder. Also discussed is how to improve social interaction skills for children with social-emotional difficulties and/or anxiety around communication. Parents as well as clinicians will appreciate the practical suggestions in this chapter. Given the COVID-19 pandemic, more treatment has been and will likely continue to be offered via telepractice. In Chapter 10, the authors give recommendations for telepractice for speech and language in general as well as specific suggestions for children with CAS. This is a timely and important issue.

Chapters 11 and 12 focus on how to address changing needs of the child over time and how to address that in therapy as well as writing meaningful goals and collecting data. While this certainly pertains to treating CAS, the issues discussed will be helpful to clinicians working with a variety of children with speech and language disorders. Importantly, the authors also provide helpful information regarding home practice and working with parents.

The breadth of this book is large and will therefore be of interest to a variety of audiences. Students and clinicians will find many of the practical strategies and worksheets provided to be extremely helpful in their clinical practice. Parents will find it useful for understanding the nature of their child's speech problem and feel empowered by the authors' emphasis on including parents in the management of their child's speech problems.

Margaret Fish and Amy Skinder-Meredith are experienced, excellent clinicians. They have spent many years specializing in the assessment and treatment of children with severe speech sound disorders. They have a great deal of experience in treating CAS, providing courses and workshops on the management of CAS for both students and practicing clinicians, and volunteering many hours to support families of children with CAS. This rich experience is evident throughout this book. Students and clinicians will appreciate the many intervention ideas based on best practice. The authors' desire to bring the literature to clinical application is evident in this book. I know them to have a passion for helping children with speech and language deficits, especially CAS, and it shows. They are enthusiastic in their endeavors to educate students and clinicians in pediatric motor speech disorders. This book continues that

work. The scope of the strategies presented for assessment and treatment is wide ranging, clearly written, and clinically applicable. Management of CAS is complicated, challenging, and rewarding. This book provides guidance and support that will be appreciated by many.

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CHAPTER

1

Understanding Childhood Apraxia of Speech

In 2007, the American Speech-Language-Hearing Association's (ASHA) technical report on childhood apraxia of speech (CAS) provided a definition of this complex neurological pediatric motor speech disorder that impacts both articulatory accuracy and prosody. The report provided three core characteristics observed in CAS that help the clinician differentiate it from other speech sound disorders and a list of frequently co-occurring challenges. The quest to fully understand CAS, however, keeps researchers and speech-language pathologists (SLPs) in a constant state of investigation and learning. The purpose of this chapter is to provide an overview of the journey of our understanding of CAS—from the initial discovery in the 1950s to what we currently know based on behavioral, acoustic, kinematic, genetic, and brain imaging research.

What Is CAS?

CAS falls under the umbrella of speech sound disorders (SSDs) as shown in Figure 1–1. SSDs can be due to motor-based articulation challenges, structural differences, sensory deficits, or linguistically based phonological issues. A child with an SSD could have a combination of factors making speech production difficult. For example, a child could have speech impacted by hearing loss and cleft lip and palate, in addition to CAS.

History of CAS

The history of apraxia of speech in children is an interesting one. It was first noted by Morley, Court, and Miller in 1954 in the United Kingdom. In 1950, these authors proposed a taxonomy to classify childhood speech disorders. They used the following five broad categories: deafness, defective articulation or dysarthria, retarded development of language or

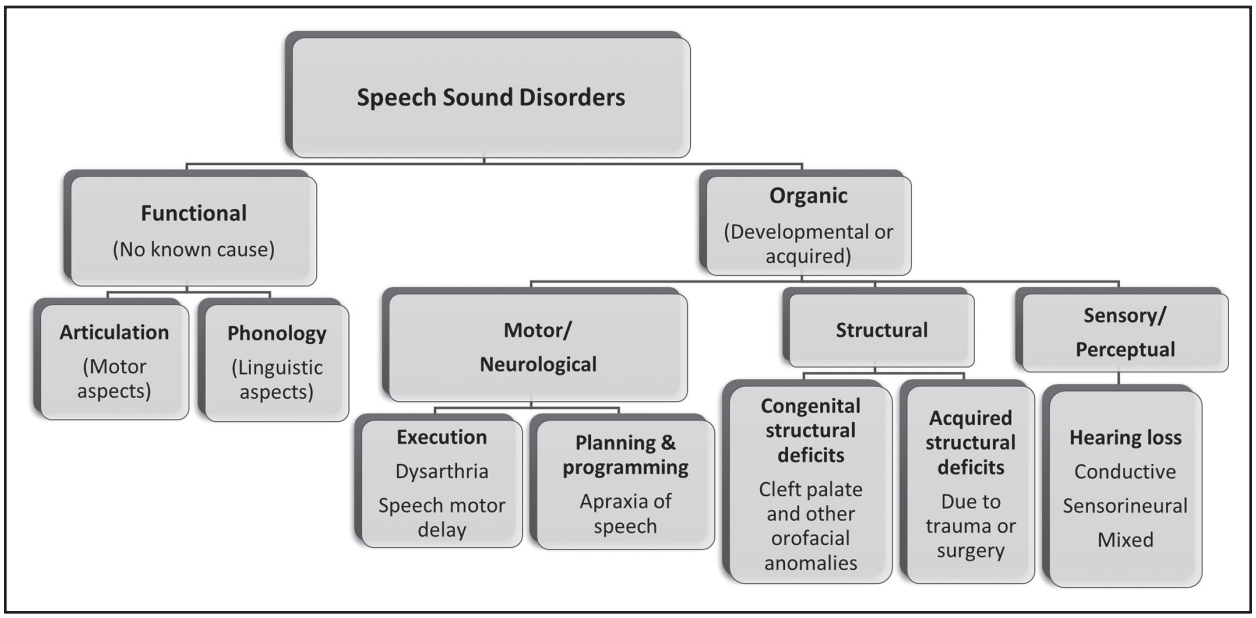

Figure 1–1. Speech Sound Disorders Classification System. (Modified from *Speech sound disorders: Articulation and phonology* [Practice portal] by American Speech-Language-Hearing Association, n.d., https://www.asha.org/Practice-Portal/Clinical-Topics/Articulation-and-Phonology/. Reproduced with permission from ASHA.)

dysphasia, dyslalia, and stammering. When they further examined the children with dysarthria in their 1954 paper, they noted that there was a subgroup of children whose lips, tongue, and palate appeared normal on voluntary movements but clumsy and awkward when they attempted the more complex and rapid movements required of speech. They labeled the disorder, "dyspraxic dysarthria" or "articulatory dyspraxia." In the United States, researchers Rosenbek and Wertz (1972) were bringing attention to a set of speech characteristics observed in children with SSDs that, in many ways, resembled the apraxia of speech affecting adults following stroke or brain injury. These children appeared to have the strength and structural integrity of the speech mechanism to be capable of speaking clearly; however, they demonstrated significant difficulty in speech production, including difficulty with phoneme sequencing, inconsistent errors, groping for sounds, difficulty imitating oral movements, difficulty imitating sounds and words, and atypical stress and intonation patterns. These children did not respond as expected to traditional types of speech therapy, often making very slow progress. Given the similarities of the nature of the speech difficulties of these children to adults with acquired apraxia, researchers began to label these children as apraxic or dyspraxic. Over the years, terms such as developmental apraxia of speech, childhood verbal apraxia, developmental verbal apraxia, developmental verbal dyspraxia, and CAS have been used to describe these types of speech characteristics in children. The term *childhood apraxia of speech* (CAS) was recommended by ASHA in 2007 as the classification term for children who demonstrate certain types of speech characteristics (e.g., inconsistent articulation errors, disordered prosody, and poor coarticulation). These characteristics are detailed in the following section. CAS is estimated to occur in approximately one to two children per thousand (Shriberg et al., 1997a), with a higher prevalence occurring in children with certain syndromes or types of genetic deviations.

Defining CAS

To provide greater clarity for SLPs working with children with CAS, ASHA formed a committee (Ad Hoc Committee on Apraxia of Speech in Children) to review the available scientific research related to CAS. The committee also described current trends in professional management of CAS and made recommendations related to assessment, treatment, and future research. In its report (ASHA, 2007a), the committee proposed the following definition of CAS:

> *Childhood apraxia of speech (CAS)* is a neurological childhood (pediatric) speech sound disorder in which the precision and consistency of movements underlying speech are impaired in the absence of neuromuscular deficits (e.g., abnormal reflexes, abnormal tone). CAS may occur as a result of known neurological impairment, in association with complex neurobehavioral disorders of known or unknown origin, or as an idiopathic neurogenic speech sound disorder. The core impairment in planning and/or programming spatiotemporal parameters of movement sequences results in errors in speech sound production and prosody. (ASHA Position Statement, 2007, para. 3)

There is a lot of information packed in this definition, so let us break it down:

> *CAS is a **neurological** childhood speech sound disorder in which the **precision and consistency of movements underlying speech** are impaired in the **absence of neuromuscular deficits**.*

What Do We Know About the Neurological Component?

Although some children with CAS may have a history of clear neurological involvement, such as an intrauterine stroke, infections that attack the brain, epilepsy, or trauma, the majority do not. However, this does not rule out a neurological component. Brain imaging and genetic studies over the past two decades have revealed more insight into the neurological differences of children with CAS. This has not been an easy or straightforward discovery. Unlike adults with acquired apraxia of speech, children with CAS do not typically present with a focal brain lesion in the left hemisphere. The *heterogeneity* of the disorder also caused challenges when using neuroimaging studies that looked only at the brain morphology. Studies prior to the completion of the human genome project (1990–2003) and availability of more sophisticated neuroimaging tools, like functional magnetic resonance imaging (fMRIs) and *diffusion tensor imaging*, limited researchers in their ability to better understand the genetics and neural correlates that correspond with motor planning and sequencing for speech and the other characteristics frequently associated with CAS, such as language and literacy impairments. Advancements in neuroimaging and genetic testing methods have put us on an exciting trajectory of learning more about these children we care so deeply about.

No story illustrates the evolution of genetic and neurological discoveries better than the account of the KE family. In the late 1980s, the KE family caught the attention of a special educator in Brentford, England. The teacher, Ms. Auger, noticed she had several members of the same family that presented with motor speech and language disorders. Assuming these traits were heritable, she contacted Jane Hurst, a geneticist and researcher (Fowler, 2017). When examining three generations of the KE family, they discovered that the grandmother, four of her five children, and 11 of her 23 grandchildren had CAS in addition to other challenges,

showing this to be an *autosomal dominant monogenic trait* (Hurst et al., 1990). With the advancements made in genetics (e.g., *semiautomatic genotyping*), Fisher and colleagues (1998) were able to localize the gene responsible, *FOXP2* (*SPCH1*) on the chromosomal band 7q31. To investigate the neural correlates, the team did brain imaging on affected and unaffected family members (Vargha-Khadem et al., 1998). In the areas of speech, language, and orofacial praxis development, all the affected family members were significantly more impaired than the unaffected members. By analyzing the results produced by positron-emission tomography (PET) activation scans during a word repetition task and magnetic resonance imaging (MRI) scans, the authors found that affected family members had functional and structural abnormalities. PET scan results revealed reduced activity in the *left supplementary motor area* (SMA), the *left cingulate cortex*, and the *left preSMA/cingulate cortex*. Overactive regions included the head and tail of the *left caudate nucleus*, the *left premotor cortex with a ventral extension into Broca area*, and the *left ventral prefrontal cortex*. Consistent with these findings, the MRI scans of affected members revealed significantly more gray matter in the *lentiform nucleus* (*putamen* and *globus pallidus*) and *angular gyrus* bilaterally and less gray matter in the *preSMA/cingulate cortex Broca area*, and the *caudate nucleus* bilaterally than the unaffected members. Authors speculated that the bilateral reduction in the volume of the *caudate nucleus* may explain the orofacial dyspraxia and verbal apraxia. A follow-up study using fMRIs during a nonsense word repetition task confirmed and added to these findings. Results showed that the affected members had significantly reduced activity in the *premotor*, *supplementary*, and *primary motor cortices*, as well as in the *cerebellum* and *basal ganglia* (Liégeois et al., 2011). See Figure 1–2 for the neural correlates. These findings led to more research on the *FOXP2* gene, yet it was found that this gene only accounted for a small number of cases of CAS. As brain imaging and genetic analysis have become more sophisticated, more neurological and genetic links to CAS are being discovered.

Chilosi and colleagues (2015) examined 32 Italian children with idiopathic CAS for genetic and brain morphology differences. Although 70% of the children with CAS had parents or relatives with language disorders, dyslexia, or both, only six of the children had a known genetic alteration, where three of the genetic abnormalities were inherited from a parent and one was a *de novo genetic alteration*. De novo refers to a new mutation, one not inherited from either parent. Interestingly, the parents who passed on the gene did not present with a history of CAS and appeared phenotypically normal with none of the gene alterations involved the *FOXP2* gene. Findings from conventional MRI images of the 32 participants did not show significant visible neuroanatomical abnormalities and suggested CAS may be due to microstructural alterations in the brain. A follow-up study using *diffusion tensor imaging* (DTI) (Fiori et al., 2016) found differences in the structural connectivity of speech and language networks. Using this approach, researchers were able to determine three *intrahemispheric* and *interhemispheric subnetworks* that had reduced connectivity when compared to controls. When one thinks about reduced connectivity of a neural pathway, it helps to conceptualize these connections as ranging from slow and inefficient, like a hard-to-find path that has you bushwhacking through the forest, to a superhighway that allows you to move quickly. When a child has CAS, they may lack some of the superhighways that provide rapid connections for effortless acquisition of speech, language, and literacy skills. See Table 1–1 for a summary of the results of Fiori and colleagues (2016).

From a theoretical basis, connectivity issues discovered by Fiori and colleagues (2016) correlate with deficits in the areas presented in Table 1–2.

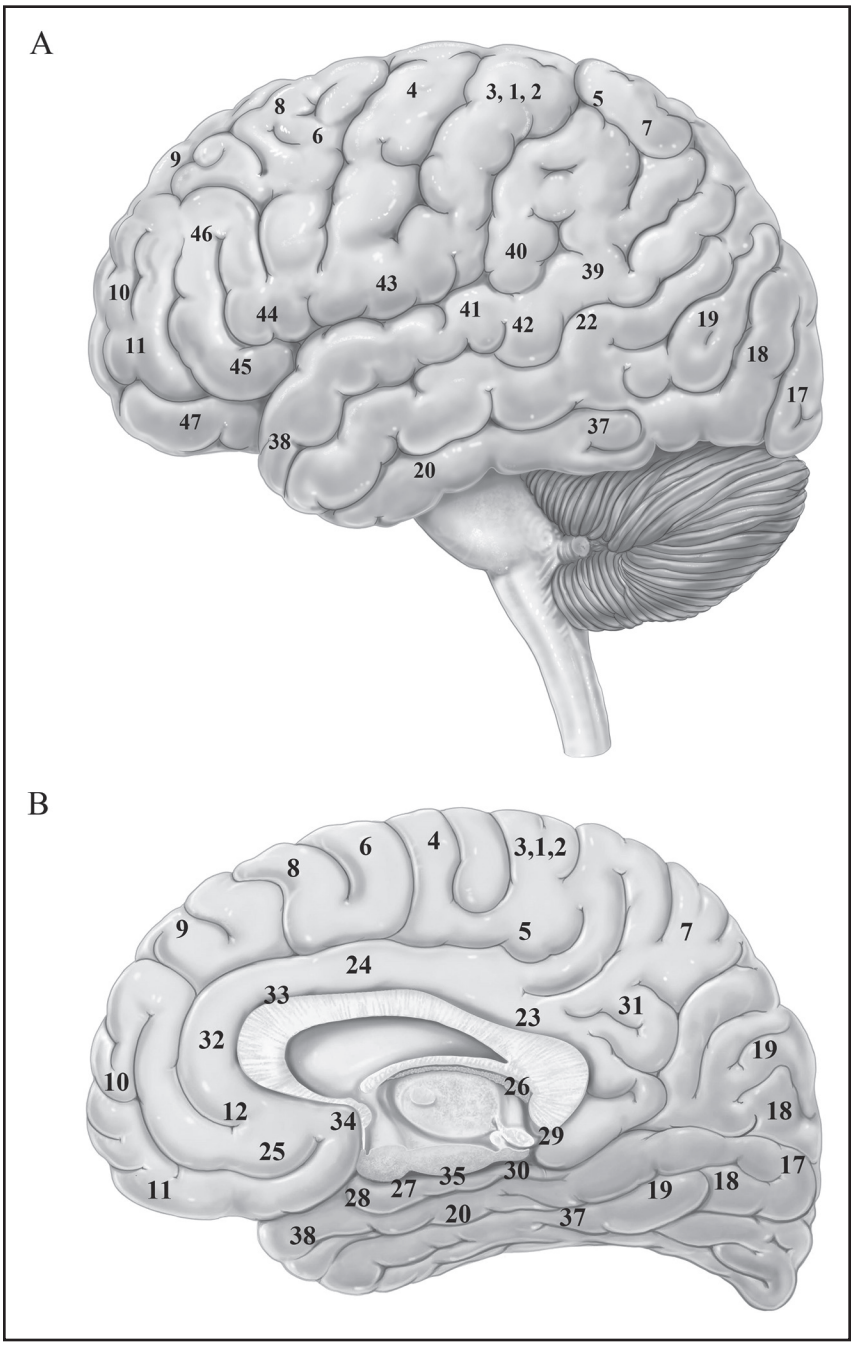

Figure 1–2. A. Brodmann areas shown on the literal surface of the left hemisphere. **B.** Brodmann areas shown on the medial surface of the right hemisphere. (From *Clinical neuroscience for communication disorders: Neuroanatomy and* neurophysiology, by M. Lehman Blake and J. K. Hoepner, 2023, p. 8, Figure 1–8. ©Plural Publishing.)

Table 1–1. Impacted Subnetworks and Structures and Corresponding Behavioral Consequences

Network	Structures Involved	Significant Correlations
Subnetwork 1	Left inferior and superior frontal gyrus, left superior and middle temporal gyrus, and left posterior–central gyrus.	Low performance on oromotor skills Low DDK rate Poor expressive grammar Poor lexical production
Subnetwork 2	Right supplementary motor area, left middle and inferior frontal gyrus, left precuneus and cuneus, right superior occipital gyrus, and right cerebellum	Low DDK rate
Subnetwork 3	Right angular gyrus, right superior temporal gyrus and right inferior occipital gyrus	No correlation found

Table 1–2. Neural Structures and Their Behavioral Correlates Impacted in Research Participants With CAS

Subnetwork	Neural Structures Involved	Proposed Functions of the Neural Structure
1	Middle and superior temporal gyri	Phonemic discrimination
1	Inferior frontal gyrus	Phonological and syntactic processing Feedback-based articulatory control
1	Temporal-frontal connectivity disruption	Mismatch between auditory feedback and oromotor control
2	Precuneus	Conceptual planning during lexical search Action initiation
2	Right supplementary motor area (SMA)	Speech planning and motor and cognitive triggering
2	Cerebellum Cerebellum + SMA	Alteration in feed-forward mechanisms of speech control Altered motor planning
3	Angular gyrus	Semantic representation

Note. Data from "Neuroanatomical Correlates of Childhood Apraxia of Speech: A Connectomic Approach," by S. Fiori, A. Guzzetta, J. Mitra, K. Pannek, R. Pasquariello, P. Cipriani, M. Tosetti, G. Cioni, S. Rose, and A. Chilosi, 2016, *NeuroImage: Clinical*, *12*, pp. 894–901. https://doi.org/10.1016/j.nicl.2016.11.003

Treatment studies have provided additional information on neurological differences of the brain in children with CAS. In 2014, Kadis and colleagues found differences in cortical thickness in the *left posterior supramarginal gyrus* when using *vertex-based thickness analysis with high-resolution MRIs*. Prior to treatment, children with idiopathic CAS had thicker left posterior supramarginal gyri than controls. Eight weeks post PROMPT (*PROMPTS for Restructuring Oral*

Muscular Phonetic Targets) treatment, eight of the nine children with CAS showed thinning of this area, suggesting increased neural plasticity of the brain with the pruning of unnecessary neurons for more efficient motor speech planning. Fiori et al. (2021) examined the behavioral and neurological impact of PROMPT compared to a language, nonspeech oral motor training (LNSOM) approach. Results indicated both groups had a favorable change in the *corticobulbar pathways* for speech motor control, with greater changes in the PROMPT group. More specifically, the use of fractional anisotropy indicated an increase in the *left dorsal* and *ventral corticobulbar tracts*. When interpreting results of studies that use fractional anisotropy, it helps to understand the basic premise. "Reduction of *fractional anisotropy* and related increase of mean diffusivity are reported to be associated with impaired connectivity, whereas processes connected with learning can determine neuroplastic effects and have been associated with fractional anisotropy increase and mean diffusivity decrease" (Fiori et al., 2021, p. 965). Although a correlational analysis between severity of motor speech control and measures of diffusivity were unable to be performed, positive changes were seen in both areas. In addition to studies giving us insight into the compromised neural networks for children with CAS, they also show how appropriate therapy can strengthen these pathways. It is exciting to know that, with appropriate therapy implementation, SLPs have the tools to change the brain.

To summarize, the neuroanatomical and physiological differences found thus far in children with CAS include the following areas: left posterior central gyrus (1, 2, 3); primary motor cortex (4); left premotor cortex (6); left precuneus (7); left cuneus (17); right superior occipital gyrus (19); left middle temporal gyrus (21); left caudate nucleus (23); left cingulate cortex (32); left and right superior temporal gyrus (41); Broca area (44, 45); and left ventral prefrontal cortex (46); in addition to the basal ganglia and cerebellum. See Figure 1–2 for a reference to the Brodmann areas and Figure 1–3 for the basal ganglia and cerebellum.

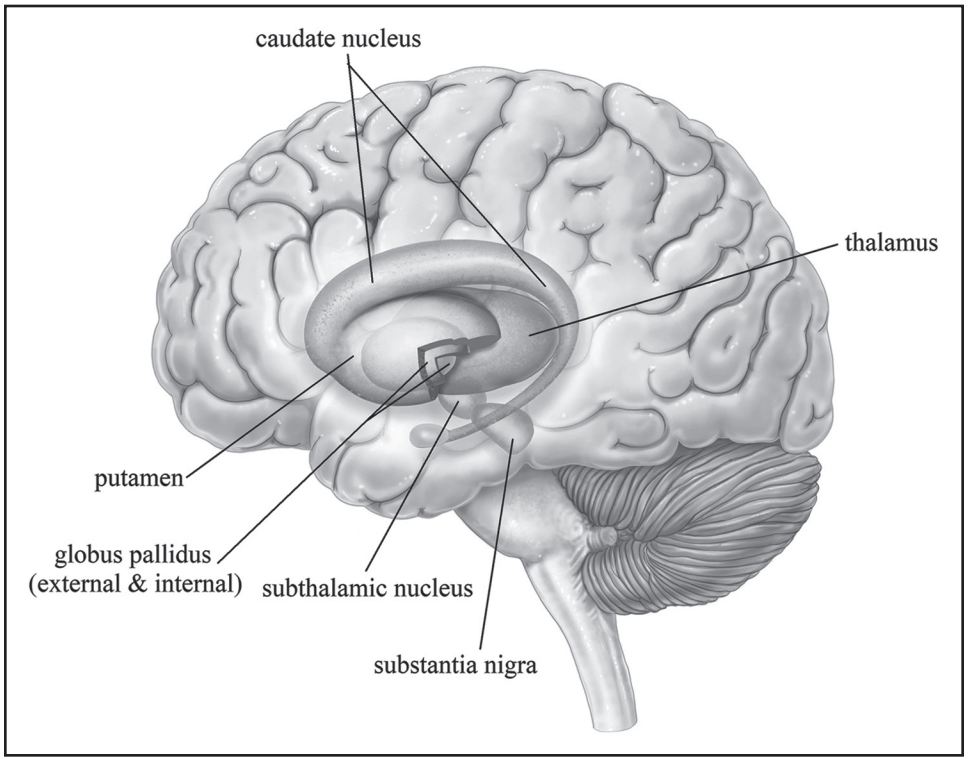

Figure 1–3. Structures of the basal ganglia, thalamus, and cerebellum from the lateral view of the left hemisphere. (From *Clinical neuroscience for communication disorders: Neuroanatomy and neurophysiology*, by M. Lehman Blake and J. K. Hoepner, 2023, p. 27, Figure 1–30. ©Plural Publishing.)