Neural Network Model: Applications and Implications

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Introduction: Why Apraxia

There are many ways that can inspire in writing a book. One way, we suppose, is that the book results from a long-standing desire to explore a particular concept or idea. Another is that the book represented a continuation of a theme that was being developed over a period of time and across several books. In the current instance, both ways could be considered accurate. This book, about apraxia, is the continuation of a series of works on how the idea of the neural network organization of the way the human brain processes information can impact what we understand about the disorders practitioners confront when something goes wrong. This book is the third in our series on human neural network characteristics of the brain and disorders thereof. In that vein, we will raise themes we have been developing over the series of works that include two additional works the preceded the current series. Among these themes is the idea that the current diagnostic nosology, the Diagnostic and Statistical Manual of Mental Disorders and the behavioral grouping model upon which it is based, is inadequate to the task and probably injurious to the development of advancement of the true nature of most disorders of mental health. Another theme is the ongoing pathologizing of behaviors and groups of behaviors that may, in fact represent, normal, if atypical, variations of human behavior. Some examples of this phenomenon include the recently added to the DSM 5, namely, social engagement disorder and caffeine withdrawal disorders. Obviously, behavior in the extreme in these areas can be problematic but where the line gets drawn in practice will be an interesting process to observe.

One of the most striking things that stood out for us in preparing the material for this book is how little things have changed regarding the understanding of apraxia over the last 30 years. For the most part, apraxia remains described as the inability to make purposeful movement of one part of the body or another. The various types of apraxic conditions are essentially names of the part of the body or function that is impacted. The major theme for us, of course, is that once you understand how the neural network model explains the information processing of the brain and once you understand what can go wrong with those networks that form the basis of information processing, the basic understanding of disorders related to this system must change. The change in understanding must accompany a change in awareness of

what it means for the system to be disordered. This inevitably leads to a change in understanding that nature and characteristics of the disorders themselves. What we described for disorders of mental health and for difficulties in motivation will also be very true for apraxia. We believe that it is long past time for a new understanding of this very large group of disorders. We hope that this new understanding leads to the formulation of newer and more neurologically accurate diagnostic categories and the development of more efficient treatment approaches.

While the motivation described above is sort of accurate, that isn't really what happened. What happened was that good friends of ours had a grandchild with delayed speech development and asked us for an opinion regarding etiology, potential treatment, and long-term prognosis. The youngster was about 18 months old and speech development was not occurring in the anticipated manner. Both of us have extensive experience in the assessment of children in the 0–3 age range, so this request was well within our capability. After a thorough assessment and developmental history, it was fairly clear that a diagnosis of developmental apraxia was appropriate and the parents and grandparents were informed. Referral was made to the local early intervention program to begin treatment, consisting of intensive speech and language therapy. The family was able to supplement the treatment provided through the program, so the child received treatment several days a week from providers who had experience in the area. So far, so good.

We have known this family for many years and had permission from the parents to discuss the case with the grandparents, who were very involved and worried about what the future may hold. As might be expected, one grandparent asked one of us and the other asked the other of us. Then one night we all went out to dinner and were chatting and trying to be supportive when it became obvious that the grandparents did not receive exactly the same information regarding impact and prognosis. One of us had described developmental apraxia as an issue of motor dysregulation emphasizing the development of speech with a good prognosis, albeit after a long and intensive program of treatment. The other of us, using neural network modeling, had described a more far-reaching disorder, impacting many areas of development and provided a more circumspect prognosis.

As you might imagine, there was a rather long discussion after we got home, and we realized that what was true for many things in the world of mental health was true for developmental apraxia. There wasn't agreement on what it was other than a description of behavior, and there were many different opinions about etiology, comorbidity, and outcome. We began to wonder if what was true about developmental apraxia was true for the many other acquired apraxias as well. What we have learned in line with the answers to those questions led to the creation of this book. To preview what we have found, we would tell you that developmental apraxia is a very different phenomena from the apraxia acquired during adulthood and perhaps should be in a separate category by itself. We would also tell you, what husbands have learned since the beginning of time, that perhaps it is better to listen to the wife and not argue in the first place.

There were many questions to be answered and many ideas to consider. Some of these were:

In the instance of an acquired apraxia, Where a lesion to a network component was the clear culprit, was the site of the lesion important?
Given the fact the apraxia is a complex behavioral dysfunction, usually resulting from the disruption of a network consisting of interrelated components, it is logical to assume that the downstream impact would be partially dependent on where in the system the messaging was disrupted. While all cases of oral motor apraxia would have the same core central behavior, might there be subtypes with different comorbidities dependent on the lesion site? It seemed to us that there might be.

Specifically, as we shall see, lesions to the brain which impair the ability to carry out simple oral gestures on imitation include the frontal and central opercula, the anterior insula, and a small area of the first temporal convolution of the left hemisphere. All these areas contribute to the various components of a rather complex oral motor movement related to speech. The disruption of speech is evident in all the affected individuals but it is not all that happens. A lesion to the frontal opercula produces comorbidities of disrupted function different to those that were produced by a lesion to the anterior insula. Depending on the subnetworks involved, it seemed likely that there would be numerous subtypes of oral motor apraxia, each with different complexities and with different impact and prognosis.

If one of the issues with developmental apraxia is the absence of an identifiable lesion, and no observable muscle weakness, what else might happen to produce the outcome?
What exactly caused developmental apraxia and was it just one thing, which we considered unlikely, or was it a collection of things? What other issues affect the ability of a neural network to carry information? What are the factors that impact the development of a neural network in the first place? Is the prognosis for a network that never develops at all different from one with only a focal point of damage in an otherwise fully developed and functional system? For example, human speech surely is one of the most complex tasks that we humans do and likely involves numerous networks in its creation, development and expression. Disruption to any one of those networks might produce the ultimate apraxic condition. The ongoing question is whether the failure to develop one component of this system would be more or less disruptive to language functioning as compared to damage to the same component after the system had been fully functional.

Would a disrupted developmental unfolding of skills imply more complex and far-reaching comorbidities that a localized lesion in an adult brain?
This question has been around for some time and we are not sure that the argument about which is better or worse has been settled. We probably won't settle it either, but we intend to take a look at it.

How does resiliency and plasticity factor into the equation for developmental apraxia and acquired apraxia?
Related to the question above, how do resilience and plasticity impact recovery of function (or development of function)? Are there some areas of the brain that have the ability to recover or adapt at a better rate than others?

Would the idea of cognitive reserve be an important consideration in adult acquired apraxia?

Is damage that occurs later in life more impactful because of all the previous life course incidents that have transpired? What is the impact of a lesion in an already compromised brain and are some brains more capable of recovering than others? What factors contribute to the ability to recover?

We will explore these issues and more. We realized that apraxia would be an ideal way of showing the power of a neural networking model to contribute to the understanding of a complex disorder. Given our backgrounds, and the circumstances surrounding the genesis of the book, it would be fair to say that we will have a heavy emphasis on developmental apraxia of speech. This is probably fair, as it seems to be the most complex of the issues to discuss. We will discuss in detail the other, significant numbers of apraxic conditions. You will be excused if you think our biases are showing, because they are. So, come along and find out why the wife was right and basing our response on a neural networking model as explained to the grandparents was the right way to go.

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Chapter 1
Apraxia, Dyspraxia, and Motor Coordination Disorders: Definitions and Confounds

Providing the reader with a cogent and agreed-upon definition of apraxia would seem to be a valuable way to start any discussion of apraxia. We have discovered however, as is the case for other diagnoses, that goal is elusive when discussing apraxia and particularly so when addressing developmental coordination disorder or childhood/developmental apraxia of speech. This is not simply a matter of "Let's agree to disagree." The implications are significant. For those readers familiar with our other work, they will be aware of our concerns with the state of diagnostic specificity and sensitivity. The implications reach the worlds of clinical psychological practice, medical practice, neuropsychology, and neurology. Specifically, if we do not agree about what we are defining, how are we defining, treating, and researching it?

Before we begin our discussion of apraxia and its various forms, we wish to highlight that we include developmental coordination disorder (DCD) in our discussions and by extension, what is now a subcategory of DCD as listed in the DSM 5 (APA, 2013), childhood apraxia of speech (CAS). We do this because of the extensive contribution of the motor networks that these disorders share. We do this to demonstrate how disruption of motor networks is an integral part of what are currently considered discrete disorders but may in fact represent disruption of a unified motor system with pervasive implications. We do this because if we can understand the brain through its development, we should be better able to understand the implications of a disruption somewhere along the neural network pathways. Therefore, we start at the beginning, that is, the childhood development of motor-based disorders. The field has begun to understand this position as it has, in the DSM 5, grouped DCD as a motor disorder. In addition, CAS is a further subcategory of DCD; both are under the category of motor disorders falling under the larger umbrella of neurodevelopmental disorders.

Although somewhat less so for an *acquired* apraxia, the definition of what constitutes a given apraxia is often elusive and poorly defined. This may be even more

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so with regard to the etiology. Perhaps a better way to explain the problem is that there are multiple problems. With regard to specificity, the definitions of apraxia are largely behaviorally descriptive, but sometimes too confined. And sometimes they are too ambiguous secondary to a phenotype. Often there is phenotypical overlap across disorders. In addition, some disorders are based upon the magnitude of effect (dyspraxia), sometimes.

As you will see, there is a multitude of apraxia, all ostensibly delineated by the primary area of impact. Traditionally the diagnosis and treatment of apraxia focused on a highly behaviorally segregated symptom. This is clearly understandable as from a historical perspective it reflects the early observations of those noting and then studying these phenomena which became known as apraxia. Early contributions were foundational; however, they also had a cortical and somewhat modular bias. It is understood and appreciated that from a current perspective it allows clinicians to speak in short hand to each other about what is observable, impacted, and where to begin intervention. But from a neural network model-based perspective, there is a different reality: there is a multitude of potential areas of impact and impacted processes, often treated as dissociable entities, complicated further by questions about etiology being trauma based, genetic, or developmental. Hence, if we consider this component, then does a diagnostic label, lacking in specificity, and often sensitivity, *really tell us with what we are dealing*, and how to best intervene?

Further confusing the picture is how some of these identified apraxia or dyspraxia associate with other disorders. This greatly muddles the designs and/or outcomes of research. For example, it often results in confounds in population samples which can produce "dubious" findings. An interesting, and we believe important, observation of the current authors is that research papers often include a section on "limitations of the study." We believe that sometimes what is described as the study's limitations, rather than being confounds and limitations, are actually a reflection of the diversity of the disorder, its phenotypical presentations, and manifestations across subjects rather than a limitation of the study itself. This point is elaborated upon as we go through diagnostic research across chapters.

In summary, while the current state of definition might lead one to assume that the impact is highly specific, in order to diagnose or treat a person impacted by an apraxia, we must understand that it is not an isolated clinical finding. Humans are much more sophisticated and the systems much more complicated. Neural network modeling would have us understand that the impact of the network damage has implications far in excess of the most obvious symptomology.

History and Current State of the Study of Apraxia

This is an area that has been under study for a long time. Looking at the history, we can see that the definition of apraxia has evolved. Based upon observed behavior and conjecture about underlying neurological functioning, the presumed mechanisms initially were really quite different than that of our current understanding.

Assessment of this area began as early as the 1860s with a description of apraxia, although not labeled as such, provided by John Hughlings Jackson (Pearce, 2009). In 1871, Steinthal coined the term apraxia from the Greek term apraxia, meaning inaction. It was assumed that the reason a person may fail to correctly perform purposeful movements was that they were unable to recognize the object/tool associated with the desired movement (Pearce, 2009). In 1905, Arnold Pick introduced the concept that the movement failure in apraxia was assumed to result from motor "agnosia," or motor manifestations of asymbolia.

Hugo Liepmann presented his first paper on the topic of apraxia in 1900 (Gonzales-Rothi & Heilman, 1996; Pearce, 2009). The paper described deficits of motor function in a 48-year-old German imperial councilor whose initial presentation appeared to be dementia. The patient had difficulty with initiation and copying of gestures, although spontaneous movements, such as using utensils while eating, were normal. Liepmann suggested a disconnect of the visual, auditory, and somatosensory areas from the area of motor cortex, a disconnection of the hemispheres. He predicted cortical lesions which were confirmed upon autopsy and presented in a report in 1907. Liepmann's work led to the conclusions that apraxia was a defect dependent on lesions in the left hemisphere which contained the memory of skilled movements. He believed that plan of movement is stored in the dominant left parietal lobe. In order to execute a skilled movement, the space time plan had to be retrieved, and via cortical connections in the left sensorimotorium (precentral and postcentral gyri), the information was passed to the left primary motor areas and then transfer was made through the corpus collosum in order to activate the right motor cortex. He also concluded that lesions of the corpus callosum interrupted the process of movement located in the left hemisphere from the motor area located in the right hemisphere resulting in ideomotor apraxia of the left arm and hand. Liepmann created categories which included the distinguishing of ideomotor apraxia wherein the difficulty is with determining the nature of a single movement, from limb-kinetic apraxia wherein the movements are awkward and slowed, and ideational apraxia wherein the sequence of movements is disrupted. Liepmann's work was foundational, if not completely correct, in correlating cortical anatomy with behavior. Geschwind who largely supported Liepmann's ideas, believed the "analyses of the mechanisms underlying disturbances in motor performances (Geschwind, 1965) to be of greater importance than the listing of types of apraxia." He went on to create his own "disconnexion theory" for limb apraxia embracing Liepmann's work and Wernicke's foundational disconnection theory model for language.

Despite advances, Pearce (2009) points out that "Recent functional imaging studies correlated with neuropsychological deficits have not clarified the fundamental nature of the many different patterns of apraxia in relation to its varied anatomical lesions." He also posits that "brain diseases that damage the multiple parallel parieto-frontal circuits devoted to specific sensorimotor transformations cause different praxis deficits depending on the context in which the movement is preformed and the cognitive demands of the action."

Current Definitions

There are currently several definitions of apraxia in use. These definitions revolve around a couple of central themes. One medical definition of apraxia covers two aspects: (1) apraxia is a disorder of the brain and nervous system and (2) a disorder wherein a person is unable to perform tasks or movements when asked, even though the request or command is understood. They are willing to perform the task and the muscles needed to perform the task work properly (National Library of Medicine, 2022). However, people with apraxia cannot execute them. This definition goes on to provide more detail. Apraxia is caused by damage to the brain. When apraxia develops in a person who was previously able to perform the tasks or abilities, it is called acquired apraxia. The most common causes of acquired apraxia are:

Brain tumor
Conditions that cause gradual worsening of the brain and nervous system (neurodegenerative illness)
Dementia
Stroke
Traumatic brain injury
Hydrocephalus

There are similar definitions encompassing more or less specificity. For instance, "Apraxia which is a neurological disorder characterized by loss of the ability to execute or carry out skilled movements and gestures, despite having the desire and the physical ability to perform them." The term "dyspraxia" is used if the presentation is considered mild. "Apraxia results from dysfunction of the cerebral hemispheres of the brain, especially the parietal lobe, and can arise from many diseases or damage to the brain" (National Institute of Neurologic Disorders and Strokes, 2022). This casts a wider etiologic net by using the term dysfunction, which can refer to severity and/or etiology. This definition goes on to talk about difficulty coordinating complex muscle movements to produce an action. Other definitions concentrate only on the particular outcome behavior that cannot be performed.

Some other definitions are simpler and often leave out the idea of intentionality such as the following: "Apraxia is the loss or impairment of the ability to execute complex coordinated movements without muscular or sensory impairment" (Merriam-Webster, 2022). This definition leaves out etiology all together and focuses on phenotype. A similar definitional orientation is provided by the National Organization for Rare Diseases (2022): "Apraxia is a neurological disorder characterized by the inability to perform learned (familiar) movements on command, even though the command is understood and there is a willingness to perform the movement." Both the desire to move and the cognitive comprehension of the directive are present, but the person simply cannot execute the act.

None of these definitions cover the case of developmental apraxia or childhood apraxia of speech (CAS). Here, it is possible that a child has not yet developed speech and it is the development of speech that is being impeded. In addition, as we

write this, the cause of most developmental apraxia conditions is unknown. For CAS, definitions like this example are common: "Childhood apraxia of speech (CAS) is an uncommon speech disorder in which a child has difficulty making accurate movements when speaking. In CAS, the brain struggles to develop plans for speech movement. With this disorder, the speech muscles aren't weak, but they don't perform normally because the brain has difficulty directing or coordinating the movements" (Mayo Clinic, 2022). This definition, while alluding to brain-based etiology, largely emphasizes the coordination of the *muscle* movement on speech.

Is childhood apraxia of speech a developmental disorder we should be defining based upon the expressed phonology? According to the American Speech-Language-Hearing Association apraxia may be developmental as in childhood apraxia of speech, or the result of a medical or traumatic event, which is then an acquired apraxia (ASHA, 2021; Diehl, 2022). With a concern that people not confuse developmental as implying a possibility of "outgrowing" the disorder, and stressing the need for intervention services, ASHA states "Neither disability is something a person will outgrow, or get over, but with careful instruction and hard work, it is both a hope and belief that people with either disability can learn to work through their challenges" (ASHA, 2021; Diehl, 2022).

Perhaps the most used definition of apraxia in the neuropsychological and medical literature was developed by Rothi and Heilman (1997). This definition defines apraxia "as a neurological disorder of skilled movement that is not explained by deficits of elemental motor or sensory system." In other words, apraxia is considered as being independent from other stroke comorbidity symptoms such as hemiplegia (loss of proprioception and motor control over limb on one side) or visual deficits such as hemianopia or neglect (Bieńkiewicz et al., 2014). Thus, to some extent, apraxia is a diagnosis of exclusion; you use it when you are not exactly sure what the specific etiology is.

Developmental coordination disorder (DCD) is another diagnostic category with much confusion about presentation and etiology. According to the American Psychiatric Association (APA, 2013), this disorder includes impairment in the development of motor coordination, wherein "the acquisition and execution of coordinated motor skills is substantially below that expected" with "difficulties manifested as clumsiness, as well as slowness and inaccuracy of performance of motor skills." This disorder is also referred to in the manual as dyspraxia. Additionally, these motor difficulties are not related to a medical condition or disease such as cerebral palsy. The disorder is listed in the DSM 5 under motor disorders, under the larger category of neurodevelopmental disorders. The World Health Organization (WHO, 1992) largely concurs with this overall definition and indicates that a child with DCD must score two standard deviations below the mean accompanied by academic and daily living impact. They note that there should not be neurological disorder. Of course, the exclusion of a neurological disorder as a prerequisite for being a neurodevelopmental disorder gives one pause.

Further complicating the picture, we should note that the terms apraxia and dyspraxia are not technically synonymous. Nor are they utilized consistently across the medical field and area of speech and language. Specifically,

1. In general, dyspraxia refers to the partial loss of the ability to coordinate and perform skilled, purposeful movements and gestures with normal accuracy. Apraxia is the term that is used to describe the complete loss of this ability. The terms are, however, often used interchangeably. In addition, dysfunction of a movement related to gross or fine motor movements, in isolation or a sequence, is referred to in the literature as apraxia.
2. In contrast, ASHA utilizes the term *dyspraxia* to define a developmental disability affecting gross and fine motor skills. ASHA utilizes the term *apraxia* to describe a speech disability affecting verbal planning. In summary then, according to ASHA "while dyspraxia is a broader term used to describe muscle planning developmental disabilities, apraxia is used to describe muscle planning needed especially for speech." Therefore, two professionals, a speech pathologist and a physical therapist, working together on an interdisciplinary team, would not agree as to the terminology for a child manifesting difficulty with, for example, independent ambulation or self-care behaviors.
3. And to highlight additional sources of confusion, the term *dyspraxia* is utilized in parts of Europe (Kirby, 2007) to describe children with what is generally known as developmental coordination disorder. In a recent "white paper" on guidelines for international practice (Blank, 2019), the consensus was for using the term DCD in countries which follow the DSM 5. In countries which adhere to the ICD, they recommend the term specific developmental disorder of motor function.

At this point the reader must be wondering how to follow if we are we talking about a motor problem or a speech problem, dyspraxia or apraxia? Rightly so. The current authors utilize the larger medically accepted definition of apraxia as it relates to gross and fine motor expression. As it relates to speech, the term childhood apraxia of speech will be utilized.

Finally, we would point out that the definitional distinction between apraxia and dyspraxia brings up some interesting issues for neural network modeling. At first glance, one might assume that because the networks involved in both types of problems are shared, the result should be the same functional deficit, identical as it is only the degree of disrupted function that is at issue. However, that is not the case because these networks are complex and disruptions can happen in a number of locations, thereby altering the final functional behavior and related comorbidities. To be clear, effect can be in magnitude and/or reach the area of impact.

Definitional Definitions and Confounds in Apraxia Research

While there are some commonalities between current definitions, there are also some striking differences. For example, consider this definition: "Apraxia designates the impaired ability to perform a gesture, in spite of preserved motor, somatosensory, and coordination functions in the limb engaged in the action" (De Renzi &

Faglioni, 1999). This definition represents a good example of a highly "confined" definition alluded to previously. It specifies motor-based movement issues for the research study currently referred to within a particular limb. There are even problems noted within this limited view as the authors state that "a unitary account of apraxia and aphasia runs into the same difficulty that undermines the theory of a common source for praxis skills and handedness, namely the occurrence of dissociations in either direction." This statement highlights difficulties in partitioning the symptomology.

Zadikoff and Lang (2005) also take issue with the definition of apraxia noting "The definition of apraxia specifies that the disturbance of performed skilled movements cannot be explained by the more elemental motor disorders typical of patients with movement disorders." These authors go on to point out that "the term 'apraxia' has also been applied to other motor disturbances, such as 'gait apraxia' and 'apraxia of eyelid opening', are perhaps misnomers, demonstrating the lack of a coherent nomenclature in this field" (pg. 1480). We concur with their concern.

The authors go on to highlight a common theme when considering apraxia in that it has a disease or injury emphasis. They note that diseases that cause the combination of apraxia and a primary movement disorder most often involve corticobasal degeneration. Corticobasal degeneration is characterized by various apraxia and particularly affects ideomotor and limb-kinetic apraxia (both of which are described further on in this chapter) as well as buccofacial and oculomotor apraxia (also described in this chapter). They point out that corticobasal syndrome may be caused by a variety of central nervous system disorders such as Alzheimer's disease, dementias, and supranuclear palsy. Of importance, supranuclear palsy and Parkinson's disease can result in differing or varying degrees of the expression of apraxia, particularly in those with more severe cognitive dysfunction. Is this then better described as dyspraxia? Germane to their position on definition and identification is that similar presentations of apraxia can involve a variety of cerebral cortical sites as well as basal ganglia structures. This implies that they consider apraxia and its movement components to possibly be dissociable and highlight the confounding that occurs in labeling something an apraxia when the etiology can be multidetermined or wherein various disorders can produce similar symptomology.

Cassidy (2016) also notes the confusion as a result of how apraxia is defined noting that it is "an inability to perform a motor task that cannot be adequately explained by motor weakness, sensory loss or a lack of understanding." This criterion has led to a plethora of motor disorders being described as forms of apraxia, despite many of these failing to capture the essence of what apraxia really is: a disorder of motor cognition. To rectify this situation, greater specificity is proposed leading to a definition of apraxia that reflects an impairment of the storage and transformation of motor representations in the brain, either through degradation of the semantic knowledge of gestures and tool use or through the inability to translate the neural representations of higher-level goals accurately into lower-level patterns of muscle activation and inhibition (Cassidy, 2016).

The above only represents the confused state of affairs as regards limb and other muscle movement disorders. It becomes even more confusing when developmental disorders are included.

There are definitions of apraxia that include speech difficulties. Some refer to speech, some to language. These are often regarded as a unique disorder in some ways. At one recent point, "Apraxia of speech (AOS) has emerged as the term to describe a motor speech disorder characterized by an impaired ability to coordinate the sequential, articulatory movements necessary to produce speech sounds" (Ogar et al., Apraxia of Speech An overview, 2005). Clearly, this definition requires deficiencies in the motor components of speech making the two elements nondissociable. The authors go on to point out that "confusion in the literature around AOS stems from the fact that terminology associated with this disorder has varied greatly. Also, symptoms associated with AOS often co-occur or overlap with those caused by neuromuscular deficits indicative of the dysarthrias and the linguistic errors associated with aphasia" (pg. 427). An example of the overlap is as follows: childhood dysarthria (CD) or childhood apraxia of speech (CAS) is suspected in children who, in addition to such errors, have imprecise and/or unstable spatiotemporal distortions of vowels and consonants, inappropriate prosody, and deficits in voice (Shriberg et al., 2019).Despite this, the authors insist that "AOS is, however, a distinct motor speech disorder." A careful inspection of this definition would note that there were disorders where speech was disrupted in exactly the same manner that it is with apraxia, making discriminating between the two conditions a matter of art as opposed to science. For example, they point out that "AOS is often confused with conduction aphasia, perhaps because sound level errors (substitutions, additions, transpositions or omissions) are prominent in both disorders. However, the nature of errors is *thought* to be different" (pg. 429). As with the other disorder definitions we have looked at, this definition is disease or insult based. The authors posit that while vascular lesions are the most common cause of AOS, the disorder also results from tumors and trauma.

To put a fine point on this discussion, we offer this from the online medical dictionary which, based on an article from the *American Journal of Speech-Language-Pathology* in 2003, provided the following description of the diagnostic problem: "The diagnostic criteria used to identify developmental apraxia of speech (DAS) have been at the center of controversy for decades. Despite the difficulty in determining the characteristics that differentiate DAS from other speech acquisition disorders, many children are identified with this disorder. The current report presents the criteria used by 75 speech-language pathologists to establish a diagnosis of DAS. Although 50 different characteristics were identified, 6 of these characteristics accounted for 51.5% of the responses. These characteristics included inconsistent productions, general oral-motor difficulties, groping, inability to imitate sounds, increasing difficulty with increased utterance length, and poor sequencing of sounds. These results are consistent with the general ambiguity of the diagnostic criteria of DAS and suggest that no single deficit is used among clinicians" (The Medical Dictionary, 2021).

This led to the following definition: "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 and 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, 2021).

"An understanding of developmental apraxia depends on consistent utilization of a group of symptoms for diagnosis so that data-based results can be used to generate inferences about the disorder" (Davis et al., 1998). This is a point which is also made and is relevant to the diagnosis of developmental coordination disorder. The criteria having been outlined above will not be repeated here. However, the current point, consistency in distinguishing symptoms, is clearly problematic across these two developmental disorders. In addition, as alluded to by Zadikoff and Lang (2005) in reference to limb and ideational apraxia, etc., there are problems with confounding variables including symptoms being *caused* by other disorders (etiology outside this diagnostic category) and symptoms clouded by other disorders, e.g., separating speech difficulties from other disorders, causing symptoms which may exacerbate or confound those of the "identified" neurodevelopmental disorder.

These issues of definition and explanation clearly and succinctly articulate many of the issues that motivated us to write this book. It suggests that what was called developmental apraxia, and is now referred to as childhood apraxia of speech, or developmental coordination disorder may well have been better described as developmental and, perhaps, represents a distinct and dissociable diagnostic entity.

Which brings us to what we consider a well-taken point, referred to previously by the current authors and elucidated by Geuze et al. (2015): "Clinical and research diagnostic criteria serve different purposes." Clinical issues include "decisions related to special education, treatment, remedial teaching, and reimbursement of costs related to services rendered." Research issues revolve around accurately identifying an entity, etiology, and presentation rather than a confounded construct. The current authors would argue that the latter, rather than the former, is critical for treatment. For example, the successful treatment of an attention-deficit disorder requires a greater understanding than just the involvement of attention. In fact, unless one understands the interplay of attention, motivation, reward circuitry, and attention being specific to task, one is not really possessing a therapeutic understanding of the diagnosis.

In summary, it should be apparent that there are multiple definitions of apraxia, all circulating around a central theme describing an inability to willfully move a specific muscle group. Within that wide net, there are those disorders that are clearly related to damage to the neurological integrity of the individual through either disease or insult and those disorders that are presumed to have damage, although no damage is identifiable. There is a third group which constitutes disorders for which