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Toward a Narrower, More Pragmatic View of Developmental Dyspraxia

Kyle J. Steinman, MD, MAS^a, Stewart H. Mostofsky, MD^{b,c}, and Martha B. Denckla, MD^{b,c}

Stewart H. Mostofsky: mostofsky@kennedykrieger.org; Martha B. Denckla: denckla@kennedykrieger.org

^a Division of Child Neurology, Department of Neurology, University of California – San Francisco, San Francisco, CA

^b Kennedy Krieger Institute, Baltimore, MD

^c Departments of Neurology and Psychiatry, Johns Hopkins School of Medicine, Baltimore, MD

Abstract

Apraxia traditionally refers to impaired ability to carry out skilled movements in the absence of fundamental sensorimotor, language, or general cognitive impairment sufficient to preclude them. The child neurology literature includes a much broader and varied usage of the term *developmental dyspraxia*. It has been used to describe a wide range of motor symptoms, including clumsiness and general coordination difficulties, in various developmental disorders (including autistic spectrum disorders, developmental language disorders, and perinatal stroke). We argue for the need to restrict use of the term *developmental dyspraxia* to describe impaired performance of skilled gestures, recognizing that, unlike acquired adult-onset apraxia, coexisting sensory and motor problems may also be present.

Keywords

dyspraxia; ideomotor apraxia; limb-kinetic apraxia; motor skills; developmental coordination disorder; autistic disorder; developmental language disorders

Defining Apraxia

The term *apraxia*, as used in the (adult) neurologic literature, refers to an acquired disorder of higher-order motor function, resulting in impaired ability to carry out learned skilled movements in the absence of any fundamental sensorimotor impairment sufficient to preclude skilled movement.¹ In 1900, Liepmann² (as referenced in 3) described three types of limb apraxia: (1) *limb-kinetic apraxia*, (2) *ideomotor apraxia*, and (3) *ideational apraxia* (Table 1). These three forms have continued to serve as the basis for discussion of apraxia in the neurologic literature through the last century and into the current one. Most investigators in the fields of neurology and neuropsychology agree that identification of (acquired) apraxia requires that deficits in motor skills must not be explainable by elementary motor or sensory deficits, language comprehension disorder, or general cognitive impairment. Stemming from the traditional behavioral neurology terminology of Liepmann, Geschwind, and Heilman,^{2,4,5} *apraxia* is a problem that exists at the interface between motor control and cognition in which

Address Correspondence to: Kyle Steinman, MD, MAS, University of California, San Francisco, Division of Child Neurology, 350 Parnassus Ave, Suite 609, San Francisco, CA 94117, Telephone: (415) 353-4337, Fax: (415) 353-2400, steinman@neuropeds.ucsf.edu.

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the action knowledge (or *praxicon* in Heilman's terminology⁶) is lacking or cannot be accessed in order to be executed. It is this notion of *praxis*, as dependent upon a specific cognitive or neuropsychological process, that is intended by the exclusion criteria used in the traditional neurological/neuropsychological definition.

Of the three forms of limb apraxia described by Liepmann (seen in Table 1), the first – *limb-kinetic apraxia* – sits closest to the elemental motor disorder side of the motor-cognitive interface. Liepmann himself considered it the “most motor” of the apraxias, and it is, in fact, argued by some to be a motor disorder rather than a true apraxia.⁷ It is generally described as difficulty with making precise, smooth, and fast coordinated movements of independent fingers, which Heilman summarizes as “deftness.”⁸ The patient with *ideomotor apraxia* makes errors in production of gestures, seen most when asked by verbal command to pantomime transitive actions (i.e. pretending to use a tool), sometimes showing improvements when imitating another person's pantomimes, and least impaired when demonstrating the use of an actual tool. Finally, though there has been confusion about the meaning of *ideational apraxia*, most now use this term to refer to difficulty with carrying out the proper sequence of complex actions, resulting in impaired object use. Because of the excessively complicated nature of defining *ideational apraxia*, it will be set aside for the purposes of this review, which will focus on the most well-described apraxia in the neurology literature – *ideomotor apraxia* – followed by a brief discussion of *limb-kinetic apraxia* as it relates to child neurology.

Heilman and others have described several other types of limb apraxia, including *conceptual*, *conduction*, and *dissociation apraxia*, explanations of which are also beyond the scope of this review. Other authors have used the term *apraxia* to describe a wide variety of neurologic signs and symptoms, including difficulties with eye movements (*ocular apraxia*), speech (*verbal apraxia/apraxia of speech*), dressing (*dressing apraxia*), visuomotor abilities (*constructional apraxia*), and ambulation (*gait apraxia*). Using *apraxia* to describe this wide range of disabilities is controversial, and some leading experts argue strongly that these should not appropriately be considered within the rubric of apraxia (Heilman K, personal communication, 2008). This review will be limited to the use of *apraxia/dyspraxia* in reference to problems of limb movement (i.e. parallel to “limb apraxia” in the adult neurological literature).

Assessing Apraxia

The traditional neurologic identification of apraxia requires exclusion of elementary motor or sensory impairments, as well as language or general cognitive disability that would explain deficits in carrying out skilled learned movements. These exclusionary requirements pose a surmountable difficulty in the assessment of apraxia. Mild elementary sensory or motor impairments may be imperceptible by basic neurologic examination yet affect the execution of skilled learned movements; even if recognized on basic neurologic examination, whether such impairments are severe enough to cause the degree of disability seen is highly subjective. Because of these challenges, traditional assessment of apraxia incorporates the use of various (sensory) stimuli to elicit skilled movements (Table 2), including spoken language (gesture-to-command), visual input (gesture-to-imitation and tool use), and tactile information (tool use). Showing that the movement can be performed better under certain circumstances than others proves that basic motor and sensory deficits are not extreme enough to preclude it. Beyond this, assessment through different modalities provides different cognitive demands and different amounts and types of sensory input, allowing specification of the different situations in which an apraxic individual can and cannot execute a movement.

Using this approach, the various apraxias are distinguished in adults (1) by the types of errors made by the individual and (2) by the means used to elicit these errors.¹ The ideomotor type is tested by asking the patient to perform symbolic/representational actions of both transitive

and intransitive types (see Table 3 for examples). The transitive type is an illustration of tool use, the predominant category for testing of praxis.

Intransitive symbolic actions represent a subcategory that is also often assessed and compared with performance of transitive actions. Some investigators have also included nonsymbolic/nonrepresentational actions, both transitive and intransitive, in their assessments. These tasks illustrate the individual's ability to imitate novel (as opposed to learned or skilled) gestures. Such novel, nonrepresentational gestures were, however, not part of the classic conceptualization of praxis. It is worth noting that the original terminology and conceptualization of *apraxia* preceded important current-day concepts related to motor skill (procedural) learning^{9,10} and the mirror neuron system.¹¹ The latter is thought to be a key component of imitative ability. Imitation of novel, nonrepresentational actions is likely best described as being based on mirror neuron system intactness rather than in terms of praxis (which has always been more closely tied to *learned* motor skills used in tool use and other meaningful movements).¹²⁻¹⁴ It is likely, however, that the two processes, both of which depend on linking perceptual (particularly visual) and motor representations of movement, are interrelated. If imitation is repeated, then procedural learning is taking place, at the endpoint of which is an established motor skill, with stored perceptual and motor representations that can be assessed on praxis examination.

Apraxia in the Neurodevelopmental Context

Applying the concept of *apraxia* in the neurodevelopmental milieu of children has proven problematic, in large part due to problems with terminology. As with many neuropsychological terms, the prefix *a-* is often replaced by *dys-* when applied in the developmental context (with the intention of distinguishing an *acquired* from a *developmental* disorder, though the original meanings of these prefixes – “lack of” vs. “abnormal” – more properly refer to severity). Thus, in order to describe children with abnormal movements of a presumed congenital (rather than acquired) cause, the term *developmental dyspraxia* has been used. Such children are often referred to generally as “clumsy.” However, “clumsy” is no longer acceptable as synonymous with “dyspraxic” because it is too vague and often is used in a way that encompasses an awkwardness of movement through the environment or basic skills like running where learning is not necessarily required. In fact, the intended meaning of *developmental dyspraxia* varies tremendously in the literature, often depending on the professional orientation of the writer (e.g. neurologist, educator, psychologist, or occupational therapist),¹⁵ as does the specificity with which it is defined. Even within the child neurology literature, use of the term is highly variable, leading to a great deal of confusion.

We propose that *developmental dyspraxia* should be used to describe a neurologic sign (with “clumsiness” as one possible associated symptom), not as a disorder unto itself. Furthermore, it should be restricted to situations in which it can be shown that impaired execution of skilled movements or gestures is out of proportion to, and not wholly explained by, basic motor impairment or perceptuomotor (e.g. visuomotor or somatosensorimotor) impairment. In neurodevelopmental disorders, comorbid conditions are more the rule than the exception. As such, we can expect that many children with *developmental dyspraxia* may also have elementary sensory or motor impairments, which makes the issue of exclusionary criteria a more difficult one (compared to that in acquired apraxia where the individual was presumed to be neurologically normal prior to some event). In the case of neurodevelopment, it is certainly possible that acquisition of skilled learned movement would be hindered by sensorimotor impairment. Given this situation, it may be difficult to determine whether developmental dyspraxia is present in the setting of sensory or motor impairment. Using the appropriate assessments, however, can optimize this possibility. Typically, this requires showing (as in adult apraxia assessments) that the gesture can be performed better under certain conditions

(such as with use of the actual tool) than under others (such as when asked to pantomime to command). (For an example, see discussion of dyspraxia in autism below.)

In this review, we intend to describe the ways in which the term *developmental dyspraxia* has been used in the child neurology literature, and present a rationale for restricting the term to a more focused usage.

The Normal Development of Praxis

To achieve a better understanding of developmental dyspraxia, expanding our knowledge about the normal development of praxis is required. Kaplan studied the development of representational gestural ability, identifying a developmental progression through stages of gestural maturity with increasing age (summarized with examples in Table 4).¹⁶ These stages (discussed by Morris¹⁷ and by Denckla & Roeltgen¹⁸) begin with manipulation of the object upon which the tool is to act without any tool depiction. With increasing age, children begin to include the tool in pantomimed action, but use a body part to represent the tool (analogous to body-part-as-tool errors in apraxic adults). Next, the child represents the tool but does so with “holding errors,” including inaccurate representation of the size or shape of the tool. The child eventually reaches the final level of accurate pantomime of transitive representational gestures. Kaplan found a developmental progression from 4- to 12-years-old, such that object manipulation was present in a portion of 4-year-olds, but was quite rare by 8. Likewise, use of body-part-as-tool was seen commonly in 4-year-olds' responses, but in less than 4% of responses by 12-years-old.¹⁶ This developmental maturation of gestural abilities underlines the need for age-matched comparisons in all studies of abnormal praxis. Further, it demonstrates the need for error analysis to determine whether errors patterns are similar to those at a younger age (suggesting “delay”) versus unique patterns specific to those with developmental dyspraxia (suggesting “deviance”).

More recent studies of the normal development of praxis have incorporated variable definitions and operationalization of praxis. Njiokiktjien *et al.*¹⁹ examined healthy children to develop a screening instrument for the development of “ideomotor praxis representation.” They defined *ideomotor praxis* as “the ideation, organization, and execution of single acts (e.g. striking a match) that cannot be divided into smaller parts of conceptualized action.” Notably, they distinguish praxis not only from motor tone and power, but also fluency and precision, in line with the traditional neurologic view of (ideomotor) *apraxia* (though fluency and precision overlap with the concept of *limb-kinetic apraxia*, as discussed later). They separated motor tasks into expressive (e.g. waving, pointing) and conventional (e.g. military salute, police traffic signs) symbolic gestures (used for communication) versus pantomimed and actual use of objects, a distinction which mimics others' division into intransitive versus transitive representational gestures. In contrast to Njiokiktjien *et al.*,¹⁹ others^{20,21} have included both “representational” and “nonrepresentational movements” in their investigation of the normal development of praxis. Though O'Hare *et al.*²¹ emphasized the importance of tool use, gesture elaboration, and motor sequencing in identifying developmental dyspraxia, their assertion that *developmental coordination disorder* is “the consensus term for this developmental disorder” is representative of the “dual meanings” of the term *developmental dyspraxia*, which adds to the confusion about just what studies of normal praxis development aim to characterize.

Developmental Dyspraxia: A Disorder to be Diagnosed?

Is *developmental dyspraxia* a primary medical or behavioral disorder? We assert, emphatically, that the answer is “no”. *Developmental dyspraxia* does not currently have status as a diagnosable disorder in either of the key diagnostic manuals of mental and behavioral disorders – the Diagnostic and Statistical Manual of Mental Disorders (DSM)-IV-Text Revision²² or the International Classification of Diseases, 9th edition, Clinical Modification²³. (The latter does

place *developmental dyspraxia* as an inclusionary category under its diagnostic label of *Specific developmental disorder of motor function*, but provides no definition nor means of diagnosing it.) Rather than as a diagnostic label, *developmental dyspraxia* should be used to refer to a specific neurologic sign of impaired execution of skilled learned movements. Unfortunately, this is far from universally true in the literature, leading to a significant amount of confusion about the meaning of the term and intent of some authors.

The Clumsy Child

In the early child neurology literature examining developmental motor problems not attributable to weakness, various authors²⁴⁻³⁵ used *developmental apraxia and agnosia* or *dyspraxia-dysgnosia syndrome* to refer, often synonymously, to “clumsiness.” In their investigations and discussions of “the clumsy child,” Gubbay, Lesný, and Iloeje include some elements that correspond with our view of *dyspraxia*, though all three also used the term exceedingly broadly. Gubbay developed a standardized motor performance battery^{24,25} that he and colleagues^{24,26,27} used to identify children with “clumsiness without any obvious neurologic cause,” to whom they applied the term *developmental apraxia and agnosia*. The battery included tasks – such as timed rolling of a ball underfoot around obstacles, rapid hand-clapping between throwing and catching a ball, timed bead threading, and skipping – that are of uncertain value in identifying dyspraxia given the multiple neurologic abilities required by each. Though dyspraxia (as traditionally defined) may make these tasks more difficult, the battery does not rule out abnormalities of fundamental neural systems – including proprioceptive deficiencies, incoordination due to abnormalities of timing and force control (considered the domains of the cerebellum and basal ganglia, respectively³⁶), visuospatial dysfunction, adventitious movements, and equilibrium problems – as the cause for slow or awkward motor performance. The non-specificity of this battery (and terminology) is attested to in an early study of Gubbay *et al.*,²⁶ in which “ideomotor apraxia” was identified in only about one-third of children with “severe clumsiness” identified by the battery.

Iloeje³⁷ defined *developmental apraxia* in keeping with the traditional definition, but he too used the term interchangeably with “clumsy”^{37,38} in children identified with Gubbay's battery²⁵ despite evidence of dysdiadochokinesia (inability to perform rapidly alternating movements with the hands in a smooth and regular manner, considered an element of cerebellar ataxia) as an alternative cause for clumsiness in a substantial proportion of them.

Lesný^{29,30} pointed out the confusion that results from “lumping together” of clumsy children with the indiscriminate use of the term *dyspraxia*. In a study of hyperactive children and low-birthweight children,²⁹ he described three causes for clumsiness: (1) “minor cerebellar disturbance,” (2) deficits in “praxia and gnosis,” and the less common (3) “slight dyskinetic disorder.” Lesný's work took a step in the right direction by pointing out the need to distinguish causes for clumsiness, only one of which is a problem with praxis. However, because he only assessed imitation of novel, nonrepresentational gestures (not learned, tool-oriented motor skills), inferences about dyspraxia are limited as errors could also be the result of visuo-perceptual difficulties or other causes (e.g. a dysfunctional mirror neuron system).

Developmental Coordination Disorder

Since the 1980s, *developmental dyspraxia-dysgnosia* has gone out of favor, with a variety of terms taking its place. For children with developmental motor difficulties, attempts to characterize “clumsiness,” identify etiologies, and determine best treatments have come from many different angles, and the professional orientation of the investigator/practitioner largely influences the terminology used to describe the “clumsy child.”¹⁵ Clumsy children are nowadays often diagnosed with *developmental coordination disorder*, a term first included in the DSM-III-Revision³⁹ in 1987. The current DSM-IV-Text Revision²² applies *developmental*

coordination disorder to children whose motor coordination is less than expected for chronologic age and intelligence, is not due to an identifiable medical disorder, and interferes with daily activities or academic achievement. Though this definition includes children with developmentally-based difficulties with skilled movements, it notably lacks the exclusionary assumptions that are key to the traditional neurologic definition of *apraxia*, identifying it as a disorder at the interface of motor control and cognition. Additionally, it (presumably intentionally) makes no claims about a neurological or phenomenological basis for “poor coordination,” serving instead as a catch-all to ascribe a name to the “disorder” of developmentally clumsy children. In contrast, by neurologic tradition, *developmental dyspraxia* refers specifically to clinician-observed problems with execution of skilled motor movements in the face of intact fundamental motor and perceptuomotor function.

Confusion with terminology persists, however, as some authors now use *developmental dyspraxia* interchangeably with *developmental coordination disorder* to describe subjects with developmental motor dysfunction. O'Brien *et al.*,⁴⁰ for example, examined visual processing in children with “developmental dyspraxia or developmental coordination disorder,” though the means of diagnosis and motor deficits found are not described. Of note, using the traditional exclusionary criterion for *apraxia* of motor skills out of proportion to perceptuomotor difficulties, we would argue that such a study specifically seeks an alternate cause for developmental coordination disorder other than dyspraxia. This paper therefore exemplifies further confusion in the use of the terminology: the study seeks an underlying sensory processing abnormality that, if found, would preclude the use of the term (*dyspraxia*) by which the subjects are described!

Though the synonymous use of *developmental dyspraxia* and *developmental coordination disorder* is highly prevalent in the child neurology literature, some have adhered to the traditional neurologic view of praxis when studying clumsy children. Dewey⁴¹, for example, compared normal children and those with “developmental motor problems” with a praxis assessment that evaluated types of errors made when performing transitive and intransitive limb gestures to verbal command and to imitation (in keeping with the assessments used in the adult apraxia literature). Unlike the battery developed by Gubbay,^{24,25} these tasks are aimed specifically at identifying deficits in gestural performance. Furthermore, Dewey's analysis provides the important information of type of error, not just presence or absence of error, in gestural performance.

Developmental Dyspraxia in the Setting of Neurodevelopmental Disorders

Even when using the term *developmental dyspraxia* in its appropriately restricted use as a neurologic sign, authors in the child neurology literature have overextended the term. It has been used to refer to individuals with abnormal scores on normed composite tests of motor behavior (such as the Movement Assessment Battery for Children⁴²) and even in the setting of peripheral nervous system injury.⁴³⁻⁴⁵ For the remainder of this article, we will discuss *dyspraxia* as it has been defined traditionally, and show how it has been examined in various neurodevelopmental disorders.

Autistic Spectrum Disorders

Dyspraxia has been described as a feature seen in autistic spectrum disorders (referred to here for simplicity as “autism”). Some authors have examined praxis in autism in ways inconsistent with the traditional approach we espouse. When studying motor symptoms in patients with autism, Ming *et al.*⁴⁶ defined *motor apraxia* as “impairment of the ability to execute skilled movements and gestures, despite having the desire and the physical ability to perform them,” a definition which is suggestive of that used traditionally, but which lacks an explicit description of the necessary exclusionary elements. Further, they used retrospective clinical

chart review of clinical office visits with a pediatric neurologist to identify *dyspraxia*. Such an approach was recognized as inappropriate by Gubbay²⁸ (in Deuel & Doar⁴⁷), who stated that dyspraxia is “not readily identified by routine neurologic history or examination,” as the necessary elements of a traditional praxis examination are highly unlikely to be included unless praxis is considered *a priori*.

Rapin and colleagues^{48,49} have looked for evidence of apraxia in children with autism, developmental language disorders, and low IQ, operationalizing *apraxia* as “the neurologists’ clinical impression rather than a formal test for apraxia.”⁴⁸ A discrepancy was found between “apraxia” identified by the neurologists’ observations of specific motor behaviors and the overall final clinical impression (including historical information) of whether the child was “apraxic”. The investigators suggest that history obtained from the parent may have influenced the overall clinical impression of *apraxia*, implying an overlap between *apraxia* and the more-general “clumsiness” in at least that measure. This attests to the inadequacy of history and/or routine neurologic examination for assessment of praxis.

Other autism investigators have used a traditional neurologic approach, defining *developmental dyspraxia* in much more specific terms. Appropriately emphasizing the nature of praxis between motor and cognitive control, Weimer *et al.* defined *apraxia* as “the general inability to carry out skilled purposeful, and coordinated motor activity,” occurring when “the motor pathways are intact, while the conceptualization of the movement is impaired.”⁵⁰ They examined the ability of children with Asperger syndrome to pantomime gestures to verbal command, to pantomime to imitation, and to use real objects appropriately. Their battery assessed both transitive and intransitive limb (ideomotor) praxis (as well as other types not discussed here), with errors categorized into types (e.g. symbolic content, body part as object, hand posture, imprecision) which corresponded to apraxia subtypes. No significant difference in performance was found on limb apraxia subtests between those with Asperger syndrome and controls (though Asperger patients had more apraxia errors on all subtests combined than controls). Specific analysis of apraxia error types revealed more errors of hand posture than control subjects.

When examining praxis in autism, two of this review’s authors (MBD and SHM) and their colleagues⁵¹⁻⁵³ have used a developmental adaptation of the traditional definition, examining performance of skilled gestures in response to verbal command and imitation and actual object use. Like Weimer *et al.*,⁵⁰ we graded praxis not only in terms of success or failure of gesture performance, but also included analysis of the types of errors made. Doing so, we showed that children with autism made more errors of praxis in response to all stimuli (to command, to imitation, and with tool use) than controls, and a greater proportion of body-part-for-tool errors than controls.⁵¹

These findings are consistent with those from another group who, similar to our approach, examined for specific error types in performance of gestures to command and with imitation.⁵⁴ Importantly, they found that children with autism were impaired in gestural performance compared not only with typically-developing peers, but also with children with development coordination disorder, attention-deficit/hyperactivity disorder, and the combination of the two.

In a follow-up study,⁵² we showed that impaired performance on the above praxis examination was still present at highly significant levels in children with autism even after accounting for basic motor skill performance (measured as speed of repetitive movements of the hands and feet from the Revised *Physical and Neurological Examination for Soft Signs*⁵⁵) with hierarchical regression modeling. This methodology and these findings speak to the classic definition of praxis as impairment out of proportion to deficits in fundamental motor abilities. Further, this study showed that praxis performance was significantly correlated with scores on

the Autism Diagnostic Observation Schedule,⁵⁶ indicating an association of apraxia with the core symptoms of the disorder.

Most recently, we have shown that children with autism show deficits in “postural knowledge” (i.e., knowledge about gestural postures), suggesting they have difficulty acquiring knowledge of the correct representations of skilled movements. As expected, postural knowledge was robustly correlated with praxis performance in children with autism. Nevertheless, hierarchical regression analyses revealed that deficits in postural knowledge, as well as basic motor skill, could not entirely account for impaired praxis performance in autism.⁵³ The findings suggest that dyspraxia in autism is, at least in part, due to problems with transforming spatio-temporal representations of movements into the motor sequences necessary to accurately perform skilled gestures. This perceptual-motor mapping critical to praxis has been linked to parietal-premotor networks, particularly in the left hemisphere.⁶ Relevant to praxis development, these circuits are necessary for the formation of internal models of action that not only guide goal-directed movements but also (through feed-forward mechanisms) form the basis by which we interpret others' actions.⁵⁷ In a recent study examining generalization of newly acquired movement patterns⁵⁸ we found that in forming internal models of action, children with autism show a bias toward reliance on proprioceptive, rather than visual, feedback. Furthermore, in the same study we found that for children with autism this bias was strongly predictive of impaired motor skill assessed on praxis examination, as well as impaired social skill. These associations may reflect a developmental process by which abnormalities in neural mechanisms underlying motor learning contribute to impaired formation of internal models of action necessary for acquiring motor (as well as social) skills.⁵⁹

Developmental Speech and Language Disorders

Studies of children who fit within the diagnostic category of developmental speech and language disorder have yielded contradictory results on the question of increased prevalence of *developmental dyspraxia*. Hill⁶⁰ examined *developmental dyspraxia* in children with specific language impairment, comparing them with children with developmental coordination disorder; age-matched controls; and younger, motor speed-matched controls. Subjects were asked to pantomime transitive and intransitive representational gestures to verbal command and to imitation (scored as correct or incorrect, without analysis of error type). *Apraxia* for representational gestures was found in both specific language impairment and developmental coordination disorder groups compared to age-matched and speed-matched controls.

Aram & Horwitz⁶¹ examined another population of children with developmental speech/language disorder, “developmental verbal apraxia” (a term plagued by its own controversies), for problems with non-verbal praxis, including manual gestures. Asking children to pantomime the use of objects, they found a distribution of performance that did not deviate from their expectations for a normal population. Dewey and colleagues⁶² compared limb praxis in this population to children with “phonologic speech disorders” and control children with normal speech. Children performed intransitive gestures (to verbal command and to imitation) and transitive gestures (to verbal command, to imitation, and with the object). Interestingly, they found that children with abnormalities of sequential vocal motor movements (i.e. “verbal apraxia”) performed worse than language-impaired children without “verbal apraxia” and than normal controls on gestures to command and imitation, but no worse with actual object use. This parallels the difficulties seen in adults with acquired ideomotor apraxia who show significant improvement in praxis with actual tools, suggesting the possibility of a similar underlying neurological deficit.

Congenital Left Hemisphere Lesions

Left hemisphere specialization for the execution of skilled movements is recognized in the adult apraxia literature.¹ To explore the innateness of this specialization, Nass⁶³ examined children with unilateral perinatal cerebral injury resulting in hemiparetic cerebral palsy. She examined “skilled motor abilities” in children with a pantomime-to-verbal command task (“praxis”), as well as a finger sequencing task (getting at “motor dexterity” or *limb-kinetic apraxia*; see below) and a finger tapping task (which we would consider a simple, repetitive movement, and therefore not an evaluation of praxis). Neither right nor left- and right-hemisphere lesion groups exhibited dyspraxia to verbal command (except for use of body-part-as-tool, which was still considered normal-for-age in her population). Potential explanations provided by the authors included that apraxia to verbal command might not be apparent until reaching adolescence when the developmentally appropriate body-part-as-tool is no longer seen in typically developing children. Another potential explanation relates to the lack of side-of-lesion effect in language deficits after perinatal stroke, which may result in a different relationship between laterality and apraxia to verbal command than the association of aphasia and apraxia (both of left-hemispheric specialization) in adults.

Relationship to IQ

In order to better understand the relationship of dyspraxia to IQ, Deuel and Doar⁴⁷ examined *praxis* in two populations of school-age children – those from a public school with average academic performance and those referred to an outpatient neurology clinic for school problems. To assess for *dyspraxia*, they assembled a “quantitative assay” using items from the Lincoln-Oseretsky Motor Development Scale and adapted from adult apraxia batteries. Their scores included three subscales – imitation of nonsense gestures, pantomime to verbal command, and actual object use – thereby incorporating elements of the traditional approach in adults as well as imitation of nonrepresentational gestures. They identified 24 of 164 subjects with a “grand mean dyspraxia z-score” (across z-scores for each of the three subscales) further than one standard deviation below the group mean, which they empirically defined as the “dyspraxic group.” Within this group, three individuals fulfilled their criteria for *ideomotor apraxia* (having z-scores on pantomime to command and imitation of nonsense gestures more than 1 standard deviation below their z-score on the actual object use subscale). While they found a positive correlation between with full scale IQ and praxis abilities amongst their whole cohort of 164, such a correlation was not present within the dyspraxia group alone. This, they argued, supports the idea of a dissociation between praxis and IQ, at least in generally dyspraxic individuals.

Developmental Limb-Kinetic Apraxia & Dyspraxic Dysgraphia

As discussed above, since its description by Liepmann, the nature of *limb-kinetic apraxia* as an elemental motor disorder versus an apraxia has been the subject of extensive debate. This issue has direct relevance in the realm of neurodevelopment in consideration of the neurobiologic and phenomenologic basis for *developmental coordination disorder*. Tests and assessments addressing developmental coordination disorder often include finger sequencing as an element of the developmental neurological examination. The inclusion of a finger sequencing component in the Revised *Physical and Neurological Examination for Soft Signs*⁵⁵ (added to 1985 revision based on normative work originally published in 1972 and 1974) was considered until recently by this author (MBD) as simply a late-developing aspect of a general “motor coordination.” This view was changed by two quite different but ultimately convergent contributions by colleagues.

First, in terms of the transition from the classic literature, timed finger sequencing is considered by Heilman (personal communication, 2008) to exemplify *limb-kinetic apraxia* as originally

described by Liepmann.⁶⁴ Thus, although it is not clear which aspects of developmental coordination disorder are independent of and which are actually part of or overlapping with developmental dyspraxia, the overlap is most obvious, according to Heilman (personal communication, 2008), in this area of sequential control of the fingers. The other parts of the developmental coordination domain are variably overlapping with the category of dyspraxia, although within the *Physical and Neurological Examination for Soft Signs*⁵⁵ the patterned movements of hand pronation-supination and foot heel-toe “rocking” back and forth are possibly considered to be limb-kinetic praxis because they are not simple repetitive movements.⁶⁵

Secondly, Virginia Berninger and colleagues have published both strictly behavioral⁶⁶ and, more recently, fMRI⁶⁷ studies firmly linking timed finger sequencing (done as specified within the *Physical and Neurological Examination for Soft Signs*⁵⁵) to the development of handwriting skills. Since use of a writing implement to write is certainly a high-level example of “praxis,” its developmental association with finger sequencing appears to reinforce in a very pragmatic modern context the appropriateness of its status (finger sequencing) as limb-kinetic praxis.

Thus, although not originally conceptualized as *limb-kinetic dyspraxia*, the finger sequencing portion of the developmental neurological examination has acquired pragmatic predictive value.⁶⁷ In relation to handwriting, then, and dyspraxic dysgraphia, finger sequencing deserves praxic status. An inclusion of “deftness” is likely appropriate, therefore, in the evaluation children with developmental dyspraxia, though it remains to be seen whether this is equally valid via the qualitative (e.g. whether a pantomimed transitive gesture was performed “with precision”¹⁹) and quantitative methods (e.g. finger sequencing speed⁶³) used in some of the studies discussed above.

A Narrower, More Pragmatic View

As we have tried to show with the above examples, *developmental dyspraxia* and its multiple variants (*agnosia-apraxia*, *developmental apraxia*, etc.) have been used in a wide variety of contexts in the child neurology literature, ranging from a diagnosable disorder (synonymous with *developmental coordination disorder*) to a symptom (synonymous with “clumsy”) to its proper use as a neurologic sign. In some cases it is well-defined and its ascertainment is in keeping with this definition; in other cases, it is described in only vague terms, and it is ascertained in inadequately specific ways.

Problematically, the relationship between neurologic sign and brain lesion, which is relatively well established in adult acquired apraxia (frontal lobes, left inferior parietal lobe, and corpus callosum)¹ has not been identified in developmental forms of impaired gestural performance. While this is not unusual in the realm of neurodevelopmental dysfunction (cf. developmental language disorder), it adds to the already difficult task of determining how to “lump” or “split.”

We would argue that appropriate “splitting” of the group of “clumsy children” must occur based on what is known from the adult neurology literature, using a narrow, traditional neurology-based definition of *dyspraxia*. To summarize, this definition requires impairments in execution of skilled learned movements (or gestures) that are out-of-proportion to deficits in basic motor, perceptuomotor, linguistic, or general cognitive function. While recognizing that these other co-existing impairments are common in populations with neurodevelopmental disability, they should not be sufficient to explain the impairments in skilled movement execution. To demonstrate this, gestural tool-oriented performance should be assessed under multiple conditions as in adult acquired apraxia, including examination of skilled movement in response to verbal command, imitation, and actual tool use (though with developmental adaptations, including use of age-appropriate tasks). Representational or symbolic gestures

must be evaluated to enable appropriate conclusions about praxis. A complete assessment of developmental dyspraxia should include differences in transitive and intransitive gestural performance. Nonsymbolic gestures are necessarily novel, and therefore do not provide sufficient information about *skilled learned* movements to address the question of praxis in isolation, but can be included to provide supplementary information regarding the interface of praxis with imitation and learning.

Gestural abilities develop throughout childhood along an apparent developmental trajectory, though specific nature of this trajectory has only begun to be defined. Therefore, assessments for developmental dyspraxia must include age-matched controls in order to determine whether inadequate execution of skilled movements is worse than expected for age. Further, gestural performance must be examined for error types, to determine whether error patterns represent delays in the normal trajectory (e.g., body-part-as-object errors) or outright deviance from typical development and from children with other (e.g. perceptuomotor) causes for abnormal skilled movement execution. Error types looked for should include spatial (e.g. posture), temporal (e.g. timing), body-part-as-object, and content (e.g. perseveration) errors (as described by Gonzalez-Roth *et al.*68). Additionally, there is evidence that assessment of dexterity (fluency and precision of sequential finger movements) should be included, though how best to characterize this is not yet clear.

Application of these examination principles will allow for proper identification and characterization of *developmental dyspraxia*. *Dyspraxia* cannot be identified with specificity on traditional neurologic examination (including general observation of tool use alone) or by history. Also, it must be remembered that *dyspraxia* is a neurologic sign that can exist in children with other neurodevelopmental disorders (e.g. autism, language disorders) or in children who have no other signs of neurologic impairment. As such, it is not a diagnosis unto itself.

This narrowed usage of the term *developmental dyspraxia* will facilitate research advances in understanding the phenomenology, neurologic basis, and treatment of dyspraxia, as well as to guide clearer communication about clinical diagnosis and treatment. Further, our views of praxis must take into account modern knowledge and conceptualization of motor activities, including the antecedent processes on the path to praxis, including the mirror neuron system and procedural learning. As yet to be explained are the relationships between the mirror neuron system, which appears to have a large role in “one-shot”/non-repeated movement imitation; procedural learning, involved in the repetition (with associated motivation) of a new motor task; and praxis, the conceptualization and execution of learned skilled motor tasks. With more focused use of terminology, ongoing research will be able to make greater strides toward elucidating these relationships and enabling a better understanding of the role of dyspraxia in neurodevelopmental disability.

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Table 1

Three types of limb apraxia described by Liepmann

Type	Description
Limb-Kinetic Apraxia	Difficulty with precise, smooth, and fast coordinated movements of independent fingers (“deftness”). Argued by some to be a motor disorder, not apraxia.
Ideomotor Apraxia	Errors in production of skilled movements despite intact motor, sensory, language, and general cognitive function. In adult acquired apraxia, most obvious when asked to pantomime transitive actions to verbal command, better when pantomiming in imitation, and least impaired when demonstrating actual tool use.
Ideational Apraxia	Impaired object use due to difficulty with carrying out the proper sequence of actions to complete a complex, multi-step task (as now used by most, though confusion about this term persists).

Table 2

Traditional assessment of apraxia incorporates various stimuli to elicit skilled movements

Method	Eliciting Stimuli	Example
Gesture-to-Command	Spoken language	“Show me how you would hammer a nail into the wall.”
Gesture-to-Imitation	Visual input	Examiner pantomimes hammering a nail into the wall and asks the patient to imitate.
Tool Use	Visual and haptic (somatosensory) input	Examiner provides the patient a hammer and asks him to show how it is used.

Table 3

Examples of subtypes of gesture assessment in adults

Subtypes		Gestures
Symbolic / representational	Transitive	Brushing your teeth; pouring a glass of lemonade; kicking a ball
	Intransitive	Waving goodbye; beckoning to come here; saluting
Nonsymbolic / nonrepresentational [*]	Transitive	Touching your nose; touching your left thumb to your right palm
	Intransitive	Wiggling your fingers

^{*} See text for discussion of appropriateness of using nonsymbolic/nonrepresentational gestures in assessment of “praxis”.

Table 4

Kaplan's stages of normal praxis development from 4- to 12-years-old

Stages	Description	Example
Object manipulation	Manipulation of the object upon which the tool is to act without any tool depiction.	Touching or grabbing teeth without depicting toothbrush.
Body part as tool	Includes the tool in pantomimed action, but uses a body part to represent the tool.	Using index finger as toothbrush.
Holding errors	Represents the tool but with "holding errors," including inaccurate representation of tool size or shape.	"Holding" the toothbrush as if it were as thick as a tennis racket handle.
Accurate pantomime	Accurate pantomime of transitive representational gestures.	As if using a real toothbrush.

Table 5

Tips for Assessing and Describing Dyspraxia in the Neurodevelopmental Context

- Look for impaired execution of **skilled learned movements**
- **Exclude** other potential neurologic causes (e.g. basic motor deficits, perceptuomotor dysfunction, general cognitive impairment)
- Examine differences in movement execution **under various conditions** (in response to various sensory inputs) – e.g. pantomiming gestures to verbal command, pantomiming to imitation, and using real objects appropriately
- Assess execution of **representational/symbolic** gestures
- Assess **transitive & intransitive** gestures
- Analyze **error types** in skilled movement execution
- **Compare** to a group of age-matched children
- *Dyspraxia* is typically **not identifiable by history or routine neurologic examination**
- *Dyspraxia* is a **neurologic sign**, to be distinguished from a symptom (e.g. “clumsiness” – may or may not be caused by *dyspraxia*) or a disorder (e.g. *developmental coordination disorder* – may or may involve *dyspraxia*)