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Procedural learning difficulties: reuniting the developmental disorders?

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During the past 30 years, research into developmental disorders has fragmented, emphasizing differences rather than commonalities. We propose that reunification might be achieved by using a 'neural-systems' approach. Deficits in dyslexia are attributed to an intact declarative learning system combined with an impaired procedural learning system – a network that includes prefrontal language systems and basal ganglia, parietal and cerebellar structures. A typology is provided for other prevalent learning disabilities; this framework focuses on different learning skills in the understanding of learning disabilities and emphasizes the diagnostic significance of 'secondary' symptoms. This approach highlights the need for development of 'neurocognitive' tests to probe the function of components of each neural system and improve strategies for explanation, diagnosis and support of developmental disorders.

Learning and learning disabilities

Thirty years ago, the major explanatory concept in learning disabilities (also known as 'developmental disorders') was 'minimal brain dysfunction' [1,2] or 'soft neurological signs' [3]. Research was subsequently fractionated into a series of, largely, independent analyses of individual developmental disorders, of which the most prevalent are dyslexia, attention-deficit/hyperactivity disorder (ADHD), specific language impairment (SLI), developmental coordination disorder (DCD), autism and generalized learning difficulties (GLD). A key target for each disorder [4] is differentiation between 'core' symptoms (specific and central to the disorder) and 'secondary' symptoms (nonessential and, perhaps, shared with other disorders). For each disorder, despite considerable progress, there are frustrating difficulties in identifying the core symptoms, underlying cause(s) and appropriate diagnostic methods.

One of the difficulties in developing diagnostic methods is that there are considerable overlaps between disorders, with an apparent 'comorbidity' between most disorders [5–8]. Comorbidity rates for ADHD and dyslexia range from 11% to 40% [9,10]; in addition, at least 50% of dyslexic children show early motor problems [11–13]. Canadian and Swedish studies [14,15] conclude that comorbidity between (pairs of) learning disabilities is normal, rather than an exception.

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The cognitive neuroscience of learning has recently followed a parallel course, with recognition that there are separable, intertwined types, time courses and neural circuits for learning, rather than a single, monolithic learning process [16–18]. We propose that these new conceptualizations of learning abilities and networks offer a perspective that might reunite the learning disabilities, providing novel, fruitful opportunities to combine neuroscience, cognition and education.

Causal explanations of dyslexia

Research into dyslexia provides the background to the analyses. Developmental dyslexia is defined as unexpected difficulties in learning to read in children of average or above-average intelligence. A breakthrough in dyslexia research occurred in the 1980s, with the demonstration that dyslexic children lag behind their peers in phonological skills, such as sensitivity to rhyme [19,20]. This discovery provided an explanation of the behaviour symptoms (poor reading) in terms of the cognitive-level concept of weak phonology, showing the value of distinguishing the three levels of theoretical explanation: behavioural, cognitive and biological explanations [21]. In general, it is now accepted that phonological deficits in dyslexia tend to co-occur with other cognitive-level deficits, including reduced speed of processing [22].

Cognitive-level explanations suggest diagnostic tests and can inspire remediation strategies, but they cannot determine biological-level explanations, because a range of brain mechanisms could lead to impaired speed, phonology and reading. Subsequent explanations considered biological-level reasons; an influential approach is the magnocellular-deficit explanation [23–25], which attributes the difficulties in dyslexia to the magnocellular visual or auditory sensory processing system.

The understanding of brain processes has been transformed by functional imaging; a major outcome is the discovery [26–28] that the cerebellum has a crucial role in linguistic and cognitive skills, in addition to its well-established role in motor skills and coordination. The cerebellar-deficit framework for dyslexia was outlined in [29]. This provided an explanation of the cognitive-level problems in phonology, working memory and speed, and, therefore, provided a brain-level explanation of the cognitive-level theories.

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Subsequent research on the functional role of the cerebellum has provided support for the cerebellar-deficit framework [30], with developments in cognitive neuroscience confirming two-way connectivity between the cerebellum and the frontal cortex [31,32] and strongly supporting the role of the cerebellum in language-related tasks [33,34] and learning complex cognitive–motor skills, such as tool use [35,36]. Furthermore, imaging research has confirmed that the cerebellum is, indeed, active during reading [37,38]. Most directly, studies have established further evidence of functional and anatomical abnormalities in the cerebellum in dyslexia [39–42]. For independent reviews, see [43–45].

Criticism of the cerebellar-deficit framework

Despite these successes, the framework is controversial, and the criticism represents generic challenges for the field of learning disabilities.

The cerebellum is too broad (underspecificity)

The cerebellum is a large structure, containing more than half of the neurons in the brain. For cerebellar deficit to be a viable explanation, it is crucial to specify which key cerebellar region(s) are affected.

The cerebellum is too narrow (overspecificity)

This criticism was memorably proposed as the 'innocent bystander' hypothesis [46], namely that the cerebellum receives flawed information from another brain system and hence is merely an 'innocent bystander' rather than the component responsible for the disorder – the long-standing issue of distinguishing between the cause and the correlate. This problem is exacerbated by the difficulty in devising techniques that specifically isolate cerebellar function because of its extraordinary connectivity and plasticity.

The cerebellar deficit is not a core feature of dyslexia (primacy)

This criticism has three forms: cerebellar deficit is not a necessary criterion for dyslexia (i.e. at least one-third of dyslexic children show no overt motor problems), cerebellar deficit is not a sufficient condition (i.e. many nondyslexic children show cerebellar deficit) and cerebellar deficit is only weakly correlated to the core, literacy-related symptoms [47].

These important issues challenge any developmental explanation. Consider the phonological deficit. It might also be considered too broad, that is abnormalities in a range of brain structures could lead to phonological problems. It might be considered too narrow, that is phonological deficit is only one of a range of symptoms. Phonological deficit is not a sufficient condition, that is almost all poor readers (whether dyslexic or not) show phonological deficits [4]. By contrast, a phonological deficit does seem to be necessary and is highly correlated to initial reading problems (although this might reflect the teaching strategies).

In summary, neuroscientific discoveries have confirmed that the cerebellum is centrally involved in language- and reading-related activities, thereby supporting the cerebellar-deficit hypothesis of dyslexia. However, the support for this hypothesis is weakened because the cerebellum is a large structure, with many functions, and it functions in association with other brain regions to orchestrate skill learning and execution.

Procedural motor-skill learning

Dyslexic children have difficulties in acquiring certain skills but not others. If the difference between the skills could be characterized, this would surely suggest an explanation. A longstanding cognitive-level hypothesis [48] states that dyslexic children have difficulties in learning skills to the extent that they become automatic (i.e. no longer needing explicit attentive control). The hypothesis was based on the classic three-stage analysis of skill learning [49]: an initial declarative stage (in which what to do is learnt), an intermediate procedural stage (in which how to do it is worked out) and a final autonomous stage (in which the skill becomes fluent and automatic). This framework explained why there could be problems associated with motor skills, in addition to phonology, working memory and processing speed (and, of course, reading), but provided no underlying biological-level explanation.

This automaticity-deficit framework was considerably refined following a multisession, response-blending study [50], which revealed that, if required to combine two simple reactions into a choice reaction, the dyslexic group were much more adversely affected initially and then had a much slower rate of proceduralization (speed increase). These data are directly consistent with the known role of the cerebellum in skill automatization but could also reflect a within-circuit communication problem rather than a problem in the cerebellum *per se*.

Subsequent developments [16,51] on the cognitive neuroscience of learning distinguish five phases of acquisition: fast learning (minutes), slow learning (hours), consolidation (overnight), automatization (hundreds of trials) and retention (weeks). Imaging studies [52] have revealed the involvement of cortical regions comprising the supplementary motor area, premotor area and primary motor area, in addition to subcortical regions, including the striatal regions and cerebellum. Doyon and colleagues [52] distinguished two systems: the cortico-striatal system, which involves loops from the motor cortex to the basal ganglia to thalamic nuclei and back to the motor cortex; and the cortico-cerebellar system, which involves loops from the motor cortex to the cerebellum to thalamic nuclei and back to the motor cortex. The cortex, basal ganglia and cerebellum are all involved in the initial fast- and slow-learning phases, highlighting the difficulty in isolating specific contributions. If the skill has been well learned, however, its neural representation is thought to be distributed in a network of structures involving only one of these two circuits. The cortico-striatal system is considered to be crucial for motor-sequence acquisition, whereas the cortico-cerebellar system compensates for environmental changes (motor adaptation). In theoretical learning terms, the basal ganglia uniquely provide opportunities for reward-based learning through the dopaminergic system and the cerebellum uniquely provides opportunities for 'supervised learning' through the error signal from the inferior olive [53]. The procedural learning circuits,

therefore, augment the learning capabilities of the cortex using these specialist abilities.

It is interesting to interpret five dyslexia paradigms within this framework. A motor-sequence learning paradigm [54] established that the dyslexic participants failed to activate their cerebellum, either when first learning a task (fast-learning stage) or after they had practised the task to automaticity. By contrast, striatal activation was within the normal range for both automatic and new learning. These results indicate that the dyslexic participants were not using their cortico-cerebellar system at any stage during the learning process.

The 'response-blending' paradigm [50], discussed above, also indicates problems at the fast-learning stage, the automaticity stage and all intervening stages. This suggests problems within either the cortico-striatal system or the cortico-cerebellar system, or both. The eye-blink conditioning paradigm provides a relatively primitive learning form, with a cerebellar and/or striatal [55,56] substrate. Significant problems for dyslexic participants have been established [57,58].

In a fourth paradigm [59], a group of dyslexic participants and a group with DCD were compared with controls on their ability to adapt to the visual-field displacement caused by prisms – a cerebellar task [60]. The dyslexic group showed significantly slower adaptation than controls, with 10 out of 14 dyslexic participants showing an individual impairment. Even stronger deficits were reported in the DCD group, with all 14 participants with DCD showing individual impairments.

Fifth, studies using the implicit learning paradigm in dyslexia have found clear deficits in the serial reaction-time task [61] (specific differences in activation were reported in the supplementary motor area, parietal regions and cerebellum) and mirror-drawing task [62] (also considered to be mediated through the procedural learning circuits), whereas superior performance was obtained in an implicit spatial-learning task (considered to be mediated through medial temporal-lobe structures) [63].

The declarative and procedural memory systems

Because dyslexia has been considered primarily as a language-based disorder, many theorists have discounted insights from motor-skill learning. However, Ullman [18] applied the procedural and declarative categorization to language skills, proposing that the 'declarative memory system' underlies the 'mental lexicon', subserving the acquisition, representation and use not only of the knowledge of facts and events, but also words. The declarative memory system depends centrally on the medial temporal-lobe structures involved in the encoding, consolidation and retrieval of new memories: the hippocampal region, entorhinal cortex, perirhinal cortex, parahippocampal cortex and ventro-lateral prefrontal cortex.

The 'procedural memory system' underpins the 'mental grammar' – the learning of new rule-based procedures that govern the regularities of language – in addition to the learning of new skills and control of established sensorimotor and cognitive habits. This system comprises the basal ganglia, frontal cortex (in particular, Broca's area and premotor regions), parietal cortex, superior temporal cortex and cerebellum. The system has clear commonalities with the cortico-striatal and cortico-cerebellar motor-learning systems; however, the difference is that the language-based system interacts with the language-based regions of the frontal lobe, whereas the motor-skill system interacts with the primary motor cortex. Both systems include premotor regions.

Here, the combined framework is referred to as the 'procedural learning system', highlighting both the role in language and motor skills and the role in skill acquisition, in addition to skill execution. The system is assumed to include cortico-striatal and cortico-cerebellar loops for both motor- and language-related cortical regions.

Reclassifying learning disabilities

So far, this article has highlighted a range of stages and types of learning with associated neural circuits that normally function synergistically in acquiring complex skills. Paradoxically, this tangle of different learning abilities might be crucial in dispelling the confusion that currently surrounds learning disabilities.

Figure 1 shows a typology derived from these distinctions. Procedural difficulties are subdivided into four subtypes, depending on whether the primary problems are derived from the cortico-striatal or cortico-cerebellar system and, furthermore, whether the cortical problems are derived primarily from the language-related regions of the cortex or the primary motor cortex. It should be emphasized that this is by no means a clear-cut taxonomy and in practice, for the reasons discussed below, there is a considerable overlap in behavioural symptoms between the subtypes.

In Figure 1, starting with the declarative learning system, a weakness in declarative learning is assumed to lead to generally poor performance in tasks that require explicit knowledge and executive function and, consequently, it is associated with poor performance on standard psychometric tests of intellectual function – that is, children with generalized learning difficulty (GLD).

Now, consider the procedural learning system. Ullman [18] argues that SLI might best be considered as an impairment of procedural memory. The author notes that whereas lexical knowledge is relatively spared in SLI, there is a strong association with impaired grammar, morphology, phonology and motor skill, in addition to abnormalities of the brain structures underlying procedural memory. SLI is diagnosed primarily by difficulties in speech output, that is production rather than adaptation, and, consequently, the major locus of difficulty is classified as language–cortico-striatal.

Early phonological difficulties in dyslexia reflect an impaired awareness of implicit phonological rules, such as rhyme, and suggest a language–procedural-learning focus for the phonological-deficit hypothesis. Because of the difficulties in adaptation (cerebellum), the primary deficit in dyslexia is attributed to the language–cortico-cerebellar circuits. A key discriminator between SLI and dyslexia would, therefore, be difficulties in the cortico-striatal circuits for SLI, which is why SLI manifests earlier than dyslexia. Children with SLI who have impaired language–cortico-cerebellar function (in addition to

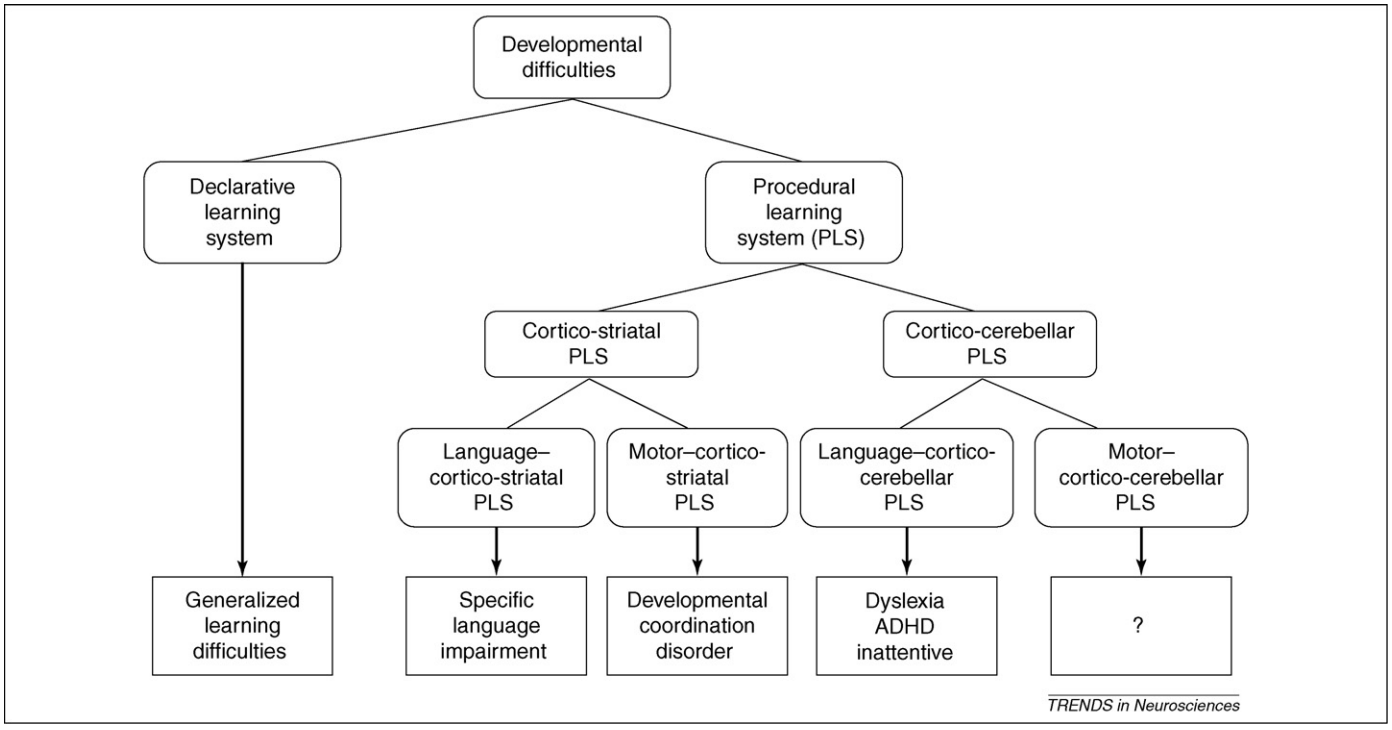

Figure 1. A neural system typography for learning difficulties. The figure provides a typology for learning disabilities derived from the neural-systems approach outlined in the text. In particular, the top-level distinction is between declarative (fact-based, explicit) and procedural skill learning. The procedural skills are split into language-related and motor skills, with each category further subdivided into cortico-striatal and cortico-cerebellar components. Proposals are made for the primary difficulties in a range of learning disabilities, in terms of one or other branch of this typology. It should be stressed that a particular child with DCD, for example, might have impairments in more than one branch, as discussed in the text. The existence of within-disorder subtypes is handled naturally by this approach, in addition to the existence of between-disorder commonalities. The questionmark in the bottom right-hand corner indicates that it is not clear whether any specific disorder has this branch as a primary route. The high incidence of difficulties in prism adaptation in DCD [59] suggests that both motor–cortico-striatal and motor–cortico-cerebellar branches might be affected in this disorder. It is important to note that a complete match between the traditional diagnostic categories and the neural-systems classification shown in Figure 1 is not expected, because the traditional classification is derived from behavioural, rather than neural, symptoms.

language–cortico-striatal impairment) go on to suffer from dyslexia too, whereas those who do not might be spared literacy problems. However, it would be premature to rule out language–cortico-striatal involvement in dyslexia, because of the evidence of abnormal activation of the putamen, in addition to the cerebellum, in implicit learning [61].

For DCD, the primary difficulty is motor output, with any language difficulties secondary; therefore, the main locus of difficulty in DCD is classified as motor–cortico-striatal. Interestingly, implicit sequence learning seems to be normal in children with DCD [64], perhaps reflecting normal function of the language–procedural circuit. Children with DCD who also suffer language difficulties (sometimes referred to as 'verbal dyspraxia') might also be classified as having language–cortico-striatal difficulties.

ADHD has frequently been attributed to abnormalities in the frontal–striatal-cerebellar circuit [65,66]. There are three subtypes of ADHD: inattentive, hyperactive and combined. Interestingly, Diamond [67] argues that patients with the inattentive subtype of ADHD suffer from abnormal frontal–parietal circuitry, whereas patients with the hyperactive subtype of ADHD suffer from frontal–striatal abnormalities. Because of the high comorbidity of the inattentive subtype of ADHD with dyslexia, it is reasonable to classify the main locus of difficulty for this subtype as language–cortico-cerebellar. Although there are not normally associated language problems, the primary problem for patients with the hyperactive subtype of ADHD is executive function; therefore, perhaps a third, executive–cortico-striatal, locus of difficulty should be introduced.

Commonalities between learning disabilities

Ramus [68] addresses the following key issue: if the learning disabilities are so distinct, why are there so many commonalities between them? Citing Galaburda's extensive neuroanatomical research [69], the author proposes that ectopias attributable to abnormal cell migration during gestation could lead to widespread difficulties, possibly including motor problems. However, Ramus stresses that the hypothesized language-area abnormalities must be the core cause of dyslexia, whereas any motor problems are secondary – that is, associated rather than causal.

This analysis reflects an ambiguity in the use of the term 'causal' in research into learning disabilities. When dyslexia researchers talk about a causal explanation, they typically focus only on reading problems, whereas a causal explanation for other learning disabilities normally refers to the range of symptoms shown.

The neural-networks framework leads to a perspective that welcomes the existence of secondary symptoms. Because of the need for cooperation between all five subsystems in the fast- and slow-learning stages, problems will occur across the range of motor and language skills, even with four 'innocent bystanders'. Initial learning can only be as strong as the weakest link in the five systems.

Comorbidities will inevitably be normal rather than an exception, but the relative strength and profile of the difficulties will provide important clues regarding the system responsible.

Comorbidity of motor and phonological problems suggests that both language and motor components of the cortico-cerebellar procedural learning system are affected, whereas intact motor skill suggests that only the language-related components are affected. Consequently, motor difficulties are a key diagnostic indicator, enabling a researcher to explore the breadth of problems in neural systems and, in due course, informing the choice of remedial strategy. A distinctive advantage of the neural-systems framework is that, rather than being unwanted noncore symptoms, these secondary symptoms are diagnostic indicators and thus at least as important as the 'core' symptoms, as discussed below.

Diagnosing learning disabilities

Before considering diagnosis, it is important to note that the standard diagnostic measures for most learning disabilities are at the behavioural or cognitive level and, therefore, are incapable of differentiating between functional differences in the efficiency and use of neural systems.

A further difficulty is that similar behavioural outcomes can be achieved by different neural systems. Dyslexic children can achieve at normal levels by 'consciously compensating' for their lack of automaticity [48]. Similarly, Ullman [18] proposed that the declarative and procedural memory systems form a dynamically interacting network that yields both cooperative and competitive learning and processing. This can lead to a 'winner takes all' effect, such that dysfunction or underuse of one system leads to enhanced learning in the other system.

Five methods of overcoming these potential confounds have been outlined by earlier research. First, one approach attempts to prevent conscious compensation by administering a dual task, to take up any spare declarative resources [48]. Second, 'primitive skills' can be tested – typically those that are not amenable to conscious control or for which conscious control leads to slower performance [11]. Third, a series of tasks can be administered that increase systematically in difficulty, to identify a point at which skill begins to break down [70]. Fourth, a multi-session, longitudinal-design-facilitated, independent analysis of the five phases of learning acquisition (fast, slow, consolidation, automatization and retention) discussed above [16,51]. Fifth, brain imaging can be used to assess whether the dyslexic participants were indeed using the normal brain regions [54].

In addition to these specialist research designs, a key requirement for the field is to develop 'neurocognitive' diagnostic methods to identify which parts of neural circuits function abnormally. For declarative learning, an example would be a paired-associate learning task, involving arbitrary visual stimuli that cannot be labelled. Several 'microtests' of procedural learning have already been noted, including response blending and prism adaptation. Paradigms investigating motor learning and consolidation [17] also provide the opportunity for developing 'benchmarks' for fast learning, consolidation and longer-term learning and transfer. Most importantly, it is valuable to combine the traditional measures with brain imaging data.

There is a strong genetic component to several learning disabilities, but genetic analyses have not fully realized their potential because the involvement is polygenic and multifactorial and it is difficult to map specific genes onto specific behaviours [71]. Converging use of the neural-systems framework, genetic analyses and brain imaging [72] might prove to be particularly valuable in this endeavour.

Concluding remarks: the neural-systems level

In summary, this review has noted the overlaps between the specific developmental disorders and outlined the difficulties in diagnosing the underlying cause(s) by considering developmental dyslexia. Here, alternative conceptualizations, in terms of phonological or cerebellar deficit, are by no means either mutually exclusive or necessarily equivalent. Key difficulties for each framework arise because of underspecificity, overspecificity and primacy. The neural-systems approach addresses these three central issues. It attacks the criticism of specificity by providing a methodology for starting with a broad, but ending with a narrow, focus, advocating the use of tests and designs to identify both the affected circuit(s) and the affected components within the circuit(s). This approach addresses the issue of 'innocent bystanders' by acknowledging the inevitable confounding of the different systems but again providing a methodology for identifying the responsible component(s).

Neural systems also provide an intermediary level between the brain and cognition that is particularly appropriate for analysis of learning skills and disabilities. The neural system is close enough to the cognitive processes and skills underlying performance to support investigations of behaviour and education and it is close enough to the underlying brain mechanisms to support investigations of cognitive neuroscience.

There is clearly considerable room for development within the typology of neural systems and the classification is far from exhaustive. A clear omission from the categorization is the magnocellular sensory system. There is, however, no impediment to extending the typology to include other brain systems as they are discovered. In parallel with these developments, there is a crucial need for neurocognitive tests that isolate the functioning of specific components of the circuits involved.

Regardless of the precise model emerging, the neural-systems framework highlights the fact that, contrary to current practice, secondary symptoms are of central importance in identifying which of the potential cortico-subcortical systems are affected. It is only by considering the profile of each individual's symptoms in its entirety that a properly informed diagnosis can be made that goes beneath the surface manifestations to identify and, in due course, remedy the underlying problems. This change to a deeper, but more inclusive, analysis could transform the field of learning disabilities.

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