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## The “BOLD” Approach: A Multimodal Format for Understanding Communication and Learning Disorders

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Communication and learning disorders, similar to other disorders, are multiply determined and influenced by interacting and interdependent levels of genetic, physiological, social, and behavioral processes. The BOLD approach presents a multimodal format to identify, assess, treat, and guide research of these disorders. This approach takes into account the broad context of interlocking influences on communication and learning disorders, including **B**iological milieu, **O**riginal traits, **L**earned behaviors, and **D**erived behaviors (or BOLD). Although there has been an explosion of information, research, and analysis in the field of communication and learning disorders, there has been a lack of consistency in approach, presentation, and structure. The BOLD format was developed to address this lack by facilitating comparisons of ideas across disciplines and to expedite recommendations and evaluations of relevant research.

The BOLD format was developed by a physician and parent to synthesize, analyze, categorize, and interpret the vast array of emerging knowledge about communication and learning disorders. In developing this approach, its creator, Mark Rosenbloom, found it useful to (1) summarize current knowledge, (2) clarify research needs and

directions, (3) evaluate a child by clearly delineating individual behaviors and looking for underlying determinants of these behaviors, and (4) identify, implement, and evaluate appropriate interventions. The purpose of this chapter is to introduce the concepts and uses of the BOLD format.

There are numerous categorical diagnostic labels, such as attention deficit hyperactivity disorder (ADHD), autistic spectrum disorders (ASD), pervasive developmental disorder (PDD), and dyslexia, as well as specific learning disorders. Innovative investigators may use these syndromes as a window to making new observations. Over-adherence to these formal syndromes as fixed categories with expected (but not fully identified) biological pathways, however, may lead the field to overlook the significance of multiple determinants and interlocking, systematic influences within each disorder. Such limitations often result in offering unitary treatment approaches to patients labeled with one of the preceding diagnoses. As discussed in other chapters of this book, a broader, functional approach is needed to include more eclectic intervention and more treatment options, and to accommodate individual needs of children who display unique patterns of functional deficits.

In Chapter 3, “Clinical Practice for Principles of Assessment and Intervention,” it was pointed out that at the current level of knowledge and understanding, a fixed, syndrome-based approach overstates the current evidence with regard to communication and learning disorders. These disorders do not appear to have a single, fixed biological pathway. There is a tendency, however, to focus on specific behavior symptoms without sufficient attention to the full range of the underlying biological differences. In fact, the same behavior can often be caused by the opposite patterns. For example, one “autistic” child may bang his head as an attempt to drown out overwhelming sensory input. Another may engage in the same behavior in order to create sensation because he is afflicted with sensory deficits and wants to break through the extreme sensory insensitivity. Hypothetically, from a biological standpoint, the same behavior may be caused by a serotonin deficiency *or* a serotonin excess.

There is understandable pressure to go beyond existing data and oversimplify categorization of developmental and learning disorders with children who, at present, do not have identifiable fixed etiologies. The goal of the BOLD approach is to consider the multiple factors that influence communication and learning disorders with a focus on relating recent neuroscience research (see the prior two chapters) in the context of different biological levels (e.g., genetics, constitutional, and developmental patterns) with different levels of observed behavior (e.g., different levels of adaptation and maladaptation). It attempts to look relatively more microscopically at the interface between biology and experience as part of the overall Developmental-Individual differences-Relationship-based (DIR) model described in other chapters.

Research has indicated that communication and learning disorders result from complex

dynamics between genetic and environmental factors (Folstein & Rutter, 1988; Troittier, Srivastava, & Walker, 1999). The BOLD approach may prove helpful in that it addresses the factors that serve as modifiers at each of four levels. These levels include: **B**iological milieu, **O**riginal or primary traits, **L**earned or secondary behaviors (simple “coping mechanisms”) and **D**erived or tertiary behaviors. Interactions between a level and its modifiers result in changes at subsequent levels. Thus, the dynamic of environmental factors modifying the first level, biological milieu, leads to the second level of the model, original traits. Even though the second level is closely related to genetic composition, it is not purely genetic. Instead, various influences of the chemical and physical environment result in expression of particular genes. Likewise, the original traits level is modified by basic stimulus-response type interactions of the child with caregivers and others resulting in level three, learned behaviors. This level is represented by an array of “coping mechanisms,” basic in nature and rudimentary in structure. Finally, the fourth level of the model, derived behaviors, results from a dynamic interplay between a child’s learned behaviors (level three) and the response of the child’s complex social environment to his or her learned behaviors. However, unlike the third level, derived behaviors go beyond basic coping mechanisms. They are highly complex, goal directed, and require higher processing. The interconnectedness among the four levels of the model stipulates a multimodal approach for organizing a large amount of information and for assessment and treatment. What follows is a more detailed description of each level of the model.

### **LEVEL ONE: BIOLOGICAL MILIEU**

Genes, biochemistry, and fundamental structural components of the human body,

along with their environmental influences, constitute the biological milieu level. This includes the central and peripheral nervous systems and their interface with all other body systems. Although certain aspects of this level may be predictable or preventable at earlier stages of development, the structural aspects of the biological milieu are commonly thought of as essentially unchangeable in standard forms of current therapy. As knowledge expands and technology improves, modifications of biological components are becoming more possible. However, once the medical evaluation is complete for any particular child and no correctable abnormalities have been found, therapists are likely to regard biological characteristics as unmodifiable. Specific biological components that need to be addressed in connection with communication and learning disorders include, at a minimum, the basic genetic structure, central and peripheral nervous systems, neurotransmitters, the immune system, and the digestive system.

A strong genetic component of communication and learning disorders has been consistently supported by research (Folstein, Bisson, Santangelo, & Piven, 1998; MacLean et al., 1999). A 60% to 91% concordance rate for autistic spectrum disorders was found among monozygotic twin pairs, while the concordance rate among dizygotic twins was significantly lower, between 3% and 5% (Bailey et al., 1995; Gillberg, 1998; Steffenburg, 1989). In 25% of cases, autism appeared to be associated with genetic disorders, such as fragile X syndrome. Although it has been speculated that more than one gene may be involved in the etiology of the developmental disorders, the exact genetic mechanisms have not been identified. However, some advances have been made in identifying the connection between certain autism markers of brain development (three markers of the c-Harvey-

ros oncogene) and homeobox gene EN2, and between ADHD and the D<sup>2</sup> and D<sup>4</sup> dopamine receptors genes and the dopamine transporter gene (Faraone & Biederman, 1998; Trottier, Srivastava, & Walker, 1999). Investigations in this area continue and are expected to provide more important information about autism and other developmental disorders. Most researchers agree that genetic predisposition may be necessary but not sufficient to cause autism and that environmental insult plays an essential role in the development of the disorder (Folstein & Rutter, 1988; Trottier, Srivastava, & Walker, 1999). This notion will be addressed further in the discussion of biological milieu modifiers.

Research has further demonstrated an association between communication and learning disorders and certain structural abnormalities in the brain. Thus, intrinsic, neocortical dysfunction as well as structural anomalies in cerebellar hemispheres and vermian lobules, parietal lobe, and the posterior regions of the corpus callosum were found in association with autistic spectrum disorders (Minshew, Luna, & Sweeney, 1999; Saitoh & Courchesne, 1998). Structural anomalies were also detected in the corpus callosum and the midsagittal surface of dyslexic children (Robichon & Habib, 1998). Many children with autism display altered evoked-response potential and conduction time and about 50% have abnormal electroencephalograms (Trottier, Srivastava, & Walker, 1999). In addition, children with ADHD exhibited abnormalities in frontal cortex activation patterns (Baving, Laucht, & Schmidt, 1999; Faraone & Biederman, 1998; Zametkin et al., 1990). However, because virtually all of the studies in this area are correlational by nature, no definite conclusions can be made at this time about whether these abnormalities are causes or effects of the disorders.

Positron emission tomography (PET) studies have focused on examining brain

function in addition to the simple brain structures. These studies demonstrated abnormalities in language and auditory functioning, such as reversed hemispheric dominance during verbal auditory stimulation, reduced activation of auditory cortex during acoustic stimulation, and reduced cerebellar activation during nonverbal auditory perception (Muller et al., 1999). Hypometabolic and neuronal migration anomalies were also detected in other areas of the brain (Schifter et al., 1994). In addition, individuals with ADHD exhibited working memory patterns that were different from those displayed by controls (Schwitzer et al., 2000). Neurochemical studies have investigated the role of neurotransmitters in communication and learning disorders. It appears that levels of glutamate, dopamine, serotonin, epinephrine, norepinephrine, and beta-endorphins are altered in autism, ADHD, and other developmental disorders (Carlson, 1998; Chugani et al., 1999; Leboyer et al., 1999; Vallone, Picetti, & Borrelli, 2000). In addition, alternative hypotheses implicate an overactive brain opioid system and changes in oxytocin neurotransmission (Trottier, Srivastava, & Walker, 1999).

The biological milieu is continuously influenced by physical environmental factors. For example, an association between autism and virus serology and brain autoantibody suggested that a virus-induced autoimmune response may play a causal role in autism (Singh, Lin, & Yang, 1998; Connolly et al., 1999). This hypothesis was further supported by an increased rate of autoimmune disorders found in relatives of children with autism (Comi, Zimmerman, Frye, Law, & Peeden, 1999). A number of research studies have focused on other relationships between various environmental events and communication and learning disorders (Barton & Volkmar, 1998; Bolte, 1998; Carlson, 1998; Kobayashi & Murata, 1998; Patzold, Richdale, & Tonge, 1998).

Factors that appear to be associated with and to influence these disorders are referred to in the BOLD format as modifiers. At the biological milieu level (level one), these include pregnancy variables, delivery, neonatal complications, diet, pollution, illness, medical interventions, and other insults as yet unidentified.

By examining obstetrical records, researchers of a number of studies have investigated various associations between pregnancy, delivery, neonatal experiences, and developmental disorders (including autistic spectrum disorders and ADHD) (Bolton et al., 1997; Lord, Mulloy, Wendelboe, & Schopler, 1991; Milberger, Biederman, Faraone, Guite, & Tsuang, 1997). In spite of some controversy, most researchers agree that the following events were significantly higher among individuals with communication and learning disorders in comparison to the general population: rates of prenatal, perinatal, and neonatal complications and insults (e.g., bleeding, smoking, illicit drug use, family problems, or viral infections during pregnancy), birth complications, use of anesthetics during delivery, low birth weight, and seizures at birth (Knobloch & Pasamanick, 1975; Milberger et al., 1997; Ticher, Ring, Barak, Elizur, & Weizman, 1996; Torrey, Hersh, & McCabe, 1975). Furthermore, an association was found between pregnancy and delivery complications and the development of tardive and withdrawal dyskinesia in children with autism who were treated with haloperidol (Armenteros, Adams, Campbell, & Eisenberg, 1995). Further research is needed to explore relationships between communication and learning disorders and such teratogenic factors as malnutrition, stress, prescribed and illicit drugs, various diseases, and radiation exposure. Despite the significant excess of total obstetric complications observed in connection with developmental disorders, no single event or combination of adversities could reasonably

account for any large number of cases of a particular disorder (Deykin & MacMahon, 1980).

Diet may also modify, in various ways, the biological milieu in terms of developmental disorders. For example, some researchers suggest that abnormal levels of glutamate in children with autism may be dietary by origin, and dietary interventions have been demonstrated to be beneficial for children with ADHD (Boris & Mandel, 1994; Moreno-Fuenmayor, 1996). Ecological conditions, such as air, water, and ground pollution, have been considered in association with the etiology of autism (Sanua, 1986). Medical illnesses such as epileptic seizures and viral diseases, especially when experienced in early childhood, have also been found to be associated with developmental disorders (Kobayashi & Murata, 1998).

Of course, there are biological milieu modifiers that are intentional, such as surgery or medications directed toward symptom reduction of the communication and learning disorder. Studies suggest that more than 50% of patients are treated with one or more medications and that this treatment results in some symptom relief for many of them (Aman, Van Bourgondien, Wolford, & Sarphare, 1995). In addition, reduction in autistic symptoms was reported in patients who underwent epilepsy-related brain surgery (Gillberg, Uvebrant, Carlsson, Hedstrom & Silfvenius, 1996). It has also been suggested that some of the medical interventions not targeted at developmental disorders, such as antibiotics or vaccinations, may be affecting the biological milieu. Indeed, research indicates that a significant percentage of individuals with autism have a history of extensive antibiotic use. Inasmuch as oral antibiotics disrupt protective intestinal microbiota and create a favorable environment for colonization by opportunistic pathogens, it is feasible that some of these pathogens (e.g., *Clostridium tetani*) may

result in production of neurotoxins that disrupt neurotransmission (Bolte, 1998). However, the literature remains controversial in reference to the hypothesized link between vaccines (particularly MMR) and developmental disorders (Boyles & Key, 1998; Duclos & Ward, 1998). Additional biological milieu modifiers are likely to be identified as research in this area continues.

As previously noted, biochemical interventions (medications) can be significant modifiers of level one, biological milieu, and can affect level two, original traits, as well as subsequent levels. Ideally, assessment and treatment of the communication and learning disorder should begin at level one in order to impact effectively upon original traits and address the “core” problem, especially if treatment involves biochemical interventions. Unfortunately, medications are often utilized without assessment at this primary level. Instead, they are utilized in response to symptoms at later levels. For example, Ritalin is often utilized to “control” problems at the later behavioral levels. This can result in other expressions of the primary problem, other problematic behaviors, or a compromising of a child’s ability to learn appropriate behavior without the use of medication.

## **LEVEL TWO: ORIGINAL/PRIMARY TRAITS**

Original or primary traits are the functional, operating, core determinants of the individual’s behavior. They are the biologically based individual differences in terms of the child’s motor, sensory, cognitive, and affective patterns. As noted previously, these traits are not purely genetic; they emerge as a result of interactions between one’s genetic makeup and the immediate chemical and physical environment that promotes expression of some genes while hindering that of others.

Whether original traits are modifiable depends on a child's current developmental stage in the biological life cycle. The earlier an intervention occurs, the more likely it is to result in successful modification of original traits. While evaluating and treating communication and learning disorders, a number of original traits can be considered in terms of the child's strengths or weaknesses in respective areas. These traits include but are not limited to visual and auditory acuity and processing, sensory modulation, motor planning/sequencing and kinesthetic processing, affective processing, cognitive functioning, and memory processing.

Research literature highlights a number of differences in original traits that distinguish individuals with communication and learning disorders from the general population. Thus, abnormalities in visual acuity and processing were consistently detected among children with autism (Scharre & Creedon, 1992). A nonnegligible number of children with autism were also found to have peripheral hearing impairments (Kiln, 1993). For children with learning disabilities, hearing loss compounded a significant part of their communication and educational problems (Welsh, Welsh, & Healy, 1996). Stereotypical behaviors, specifically behavioral rigidities, often displayed by children with developmental disorders were found to be associated with tactile defensiveness (Baranek, Foster, & Berkson, 1997). Some anomalies in sensory processing and sensorimotor functioning were detected among children with autism as early as at 9-12 months of age (Baranek, 1999). Multichanneled sensory processing and remarkably detailed memory for past events were reported in high-functioning individuals with autism (Cesaroni & Garber, 1991). Children with autism showed reduced expression of positive affect while interacting with their family members (Joseph & Tager-Flusberg, 1997). Finally, children with

autism did not appear impaired on metamemory tasks but rarely made spontaneous use of memory strategies (Farrant, Boucher, & Blades, 1999).

A number of these and other anomalies in original traits can be referred directly to the biological characteristics described in the previous section. For example, neurological impairments within the brain stem, the cerebellum, the midbrain, and the frontal lobe could be associated with deficits in affective processing, sensory processing, motor planning, and cognitive flexibility (Huebner, 1992). More specifically, abnormal hippocampal system function leads to the disrupted integration of information known as canaesthesia. Abnormal amygdaloid system function disrupts affect association and results in impaired assignment of the affective significance to stimuli. Impaired oxytocin system function flattens social bonding and affiliativeness, and abnormal organization of temporal and parietal polysensory regions yields aberrant overprocessing of primary representations, leading to extended selective attention (Waterhouse, Fein, & Modahl, 1996).

Modifiers of original traits include sensory stimuli from the child's environment. The nature of the environmental stimuli will affect the child's ability to progress normally through subsequent developmental stages. A child with adequate ability to process the sounds, sights, movements, events, and circumstances impinging upon her senses from the surrounding environment will progress adequately unless the environmental stimuli are inadequate, depriving, excessive, threatening, overly harsh, erratic, unusual, or inconsistent. Developmental delay, or even a disorder, may then result. The child's ability to advance adaptively to the level of learned behaviors or derived behaviors may also be compromised inasmuch as there is diminished ability to respond appropriately to

normal patterns of learning contingencies, modeling, and interaction. A child with inadequate processing abilities may, of course, also experience developmental delay or disorder even in a “normal” environment. A normal environment may be excessive or depriving for a child with abnormal processing abilities. Modifying the environment specifically to target abnormalities in processing may result in adequate compensations and allow for normal development. Of course, the interactions between environmental stimuli that are depriving or excessively harsh and sensory abilities that are inadequate place a child at highest risk for communication and learning disorders or delays, especially at particular critical stages of development.

### **LEVEL THREE: LEARNED/SECONDARY BEHAVIORS**

Various types of learned behaviors emerge as a result of interaction between the child’s original traits and rudimentary sensory environmental stimuli with caregivers, family members, peers and therapists, the media, the computer, and other visual, auditory, gustatory, olfactory, and tactile stimuli. These learned behaviors can best be described as basic “coping mechanisms.” They are generally connected to spoken and written language (e.g., in children diagnosed with ASD, dyslexia, and specific learning disabilities), memory and attention (e.g., in children with ADHD and ASD), impulsivity (e.g., in children with ADHD, autism, and other developmental disorders), and social interactions (especially in children with ASD) (Berger & Posner, 2000; Hardan & Sahl, 1997; Jolliffe & Baron-Cohen, 1999).

All of the learned behaviors can be divided into three broad categories: executive developmental functions, adaptive coping behaviors, and maladaptive coping behaviors.

Executive functions are behaviors that serve to organize a series of events over a long period of time. They include prolongation, or a person’s ability to hold and evaluate events in working memory; separation; regulation of affect based on his ability to distinguish facts from feelings; internalization of language; and reconstitution, which involves analysis and synthesis of events. The development of these functions are often impaired or delayed in children with learning and communication disorders (Pennington & Ozonoff, 1996). This leads to a number of problems, including deficient self-regulation of mood and behavior, impaired ability to organize and plan behavior over time, inability to direct behavior toward the future, and diminished social effectiveness and adaptability (Houghton et al., 1999; Pennington & Ozonoff, 1996; Vig & Jedrysek, 1995).

Some of the research on autism has posited an association between failure to learn adaptive social behavior and autistic children’s lack of a “theory of mind” (Baron-Cohen, 1996; Happe, 1995, 1997). “Theory of mind” is a term used to describe one’s understanding of the motives, knowledge, and beliefs of others. This includes understanding of the mental states of self and others, including wanting, feeling, believing, and thinking (Bartsch & Wellman, 1995; Baron-Cohen, Leslie, & Frith, 1985). Such understanding is achieved and usually inferred by normally developed individuals through the acquisition of the ability for affective signaling between an infant and her caregiver and such related skills as eye contact and joint attention (Greenspan, 1995, 1997). It has been proposed that children with communication and learning disorders may be delayed in acquiring abilities related to the theory of mind and that they may have problems generalizing these abilities due to a deficient sense of “self” and/or the absence of healthy social contexts and inadequate community

contacts (Hadwin, Baron-Cohen, Howlin, & Hill, 1997; Sparrevojn & Howie, 1995). However, in many cases, such apparent delays or deficiencies may actually only signify insufficient means of assessment. Examples of a more appropriate assessment model will be presented later in this chapter.

As far as the adaptability of these learned behaviors or coping mechanisms, it may be suggested that if all the child's biological milieu, original traits, and their modifiers were known, the so-called "maladaptive" behaviors could be understood in terms of their origin and purpose. This would make them not only predictable but also elucidate how the behaviors may be attempts at adaptation. Unfortunately, syndrome diagnoses are often made based on limited information about learned behaviors and in the absence of a broader picture of levels one and two. However, since the same maladaptive coping behaviors may originate from different combinations of biological milieus, original traits, and their modifiers, such generalized diagnoses may be misleading in reference to the choice of interventions. In order for the intervention strategies to be successful, all levels must be considered.

Modifiers of learned behaviors include the responses of other people in the child's environment. For example, abnormal behaviors tend to evoke responses from parents, relatives, caregivers, teachers, and the community, which may modify the learned behaviors toward better adjustment or toward further abnormality. These responses from others to the developing child's learned behaviors will tend to modify them therapeutically in proportion to available knowledge about the child's specific biological milieu, environmental modifiers, and other levels of the model noted in earlier sections. In turn, learned behaviors may be modified negatively due to ignorance about the dynamics of these multilayered and interconnecting levels.

This may include professional interventions that over-focus on one dynamic or ignore important interacting variables. Some inappropriately interventions may include sole use of medications to control behavior, inappropriate rigid reliance upon any one-dimensional treatment strategy, isolating a child in a locked facility, or restricting him to a special needs environment where there are no "typical" children. Of course, the responses from people in a child's environment to his learned behaviors lead, in turn, to further responses from the child that are called "derived behaviors," or level four, of the BOLD model. These behaviors will be even more disruptive if responses, including interventions, toward abnormally learned behaviors are shortsighted, narrow, or otherwise inappropriate.

#### **LEVEL FOUR: DERIVED BEHAVIORS**

Derived behaviors are represented by children's responses to the initial reactions that their original traits and learned behaviors evoke in other people and society. These are multidimensional patterns of behaviors that are far more complex than the rudimentary coping mechanisms referred to at the third level. Because they can be particularly disruptive or dysfunctional, derived behaviors represent the most obvious, and usually the most alarming, symptoms of the learning and communication disorder. This is the level at which evaluation and diagnosis frequently occur, and the level likely to be targeted by treatment interventions. Unfortunately, derived behaviors are often so far removed from the biological milieu and original traits that it becomes very difficult, if not impossible, to properly assess a child solely at this fourth level and to implement appropriate intervention strategies. Examples of derived behaviors may be easily recognized as commonly assessed symptoms of numerous syndromes

falling under the category of learning and communication disorders. These behaviors may include head-banging, rocking, spinning, continuous self-stimulation, and other stereotypical behaviors. Also, there may be aggressiveness toward others expressed in biting, kicking, head-butting, or spitting. Other problem behaviors may include idiosyncratic rituals and compulsions, inappropriate yelling out, continuous seeking out of attention by inappropriate acting out, severe temper tantrums, angry outbursts, social isolation, withdrawal, and lack of development of spoken language.

Modifiers of these behaviors include various intervention programs. A number of such programs have been developed (Rogers & Lewis, 1989; Schopler, Mesibov & Hearshey, 1995; Strain & Hoyson, 1988). Some programs are highly structured and have a behavioral orientation; others are multidisciplinary and have a relational focus (Campbell, Schopler, & Hallin, 1996; Greenspan & Wieder, 1998; Lovaas, 1981, 1987; Robinson, 1997; Wieder, 1992, 1996). Such programs work on modifying derived behaviors through a variety of means, including facilitating communication, decreasing inattention and irritability, improving cognitive and social skills, and promoting generalization and maintenance of new adaptive behaviors (Bondy & Peterson, 1990; DeGangi & Greenspan, 1997; Greenspan, 1992; Greenspan & Wieder, 1997, 1998; Olley, Robbins, & Morelli-Robbins, 1993; Stokes & Osnes, 1988).

Many intervention programs have traditionally remained self-contained by including only the population of children with communication and learning disorders, with no emphasis on providing them with opportunities to socialize with children from general populations. Research seems to indicate, however, that development of many of the maladaptive derived behaviors could be prevented by placing the children in an environment that

includes adaptive peers (Bricker & Cripe, 1992; Fewell & Oelwein, 1990; Giangreco, Dennis, Coninger, Edleman, & Shattman, 1993). Such an environment may allow them to learn positive social roles and communication skills by observing and imitating their peers. Mesibov (1984) suggested that many children with communication and learning disorders exhibit social deficits due to the lack of friends and positive role models. This suggestion is confirmed by research findings that children with learning and communication disorders benefited from opportunities to interact with their more typically developed peers (Mahoney & Powell, 1992; Odom & McEvoy, 1988). Their gains were more significant than those of children in self-contained programs, and included increased social behavior as a result of being actively engaged in social interaction by their more typically developed peers. Also, children with learning and communication disorders gained enhanced development of language as well as improved cognitive, social, motor, and other age-appropriate skills (Mahoney, Robinson, & Powell, 1988; McHale, 1993; Peck, Odom, & Bricker, 1993; Roeyers, 1996; Strain & Kerr, 1981; Strain, Kerr, & Ragland, 1979; Yoder, Kaiser, & Alpert, 1991). Effective and creative curricula that ensure inclusion of both children with developmental disorders and their more typically developed peers are currently available (Jorgensen, 1997; Onosko & Jorgensen, 1997; Sizer, 1992).

### **USING THE BOLD APPROACH TO ENHANCE ASSESSMENT**

As previously noted, the BOLD format was developed as a multimodal approach to communication and learning disorders. What follows is a breakdown of how this approach may enhance one of these applications; namely, the assessment of communication

and learning disorders. Looking at the different levels contributing to a child's challenges may help in asking the additional questions that will reveal the different "contributors" to the child's behaviors.

A comprehensive, team-based approach to assessment can use the different levels as reminders of the different functions involved and the relationship among them. The biological milieu—that is, all relevant biological areas, including individual physical differences and possible modifiers of this level (e.g., birth complications, medical interventions)—is a basic level. Assessment may then proceed sequentially to original traits (functional developmental level) and corresponding modifiers (e.g., various sensory and environmental stimuli), then learned behaviors and modifiers (e.g., responses of caregivers), and, finally, derived behaviors and modifiers (e.g., therapeutic interventions at home, family, and school interactions).

Assessment of the biological milieu (level one) can rule out possible metabolic abnormalities, seizure activity, brain lesions, neurological disorders, immune disorders, and other likely influences on communication and learning disorders. The child's biochemical makeup, genetic structure, and central and peripheral nervous systems can be assessed by means of standard medical evaluations, pediatric neurological evaluations, metabolic/endocrine screenings, nutritional and genetic screenings, MRIs, PET scans, and EEGs.

Modifiers of the child's biological milieu, such as pregnancy complications, birth complications, medications, surgery, diet, and contaminants, can also be evaluated by these and other procedures. Assessments of the modifiers of the biological milieu are important because some modifiers, such as diet, medications, and pollutants, may be preventable or reversible. Moreover, such assessments may shed light on

different expressions of the same genetic structure, for example, as seen in identical twins.

Assessment of original traits (level two) should examine functional strengths and/or weaknesses in auditory reception and processing, visual-spatial perception, and processing, olfactory, gustatory, tactile, and other sensory input/processing modes, basic cognitive abilities, and memory. Specialists in pediatrics, ophthalmology, audiology, and other processing areas may rule out abnormalities in vision, hearing, perception, and sensorimotor processing. Psychological testing may rule out cognitive processing, memory deficits, and other complications.

As noted previously, a child's ability to advance adaptively to subsequent developmental levels may be compromised, even with normal original traits, if these traits (hearing, vision, and others) are abnormally modified by an environment that is depriving, excessive, threatening, or otherwise harsh. Thus, a child may unfortunately not experience normal patterns of learning contingencies, not respond to conditioning stimuli and reinforcement, nor be able to benefit normally from social modeling. Assessment of the status of the child's environment may include psychosocial histories, immediate observations of child-caregiver interactions, and on-site evaluations at the child's home or school.

Likewise, assessment of learned behaviors (level three) may include on-site observation of learning contingencies in the child's usual environment to rule out possible inadequate social interactions, parenting styles, or deficient preschool or academic conditioning. The child's developmental level of functioning (e.g., age appropriateness) is assessed at this level and may also include formal tests, such as psychological evaluations, behavior checklists, and tests for learning disabilities. Also included may be tests to rule out abnormalities in spoken and/or written communications,

impulse control problems, attention deficits, low frustration tolerance, inability to postpone immediate gratification, deficits in mood regulation, faulty cognitive responses to stress that may influence affect (e.g., overgeneralizing or “catastrophizing”), empathy (“emotional quotient”), and social skills delays.

Assessment of modifiers of learned behaviors involves evaluations of others’ reactions or interventions to the child’s problematic learned behaviors. The responses of parents, relatives, peers, and the child’s community to his learned behaviors may modify those behaviors therapeutically or even worsen them. It will be important to assess what kinds of informal and formal interventions have been applied or are currently in place. Of course, this can be done by taking a thorough history of previous clinical interventions and *in vivo* observations of the child in his natural environment at home, in school, at clinics, and in the community. Assessment may rule out responses or interventions that are ignorant of the interacting dynamics of the various levels affecting communication and learning disorders. Interventions or responses that are not comprehensive, over-focus on only one dynamic, or are too narrow or shallow may be uncovered.

Assessment of derived behaviors (level four) involves ascertaining the child’s responses, in turn, to the interventions or reactions of others toward her learned behaviors. These responses of the child are often the alarming or stereotypical behaviors that are the hallmark of the communication and learning disorders, such as head-banging, chronic self-stimulation, rocking, spinning, aggressive outbursts, and withdrawal. Assessment of these may include matching the problem behavior to antecedent stimuli or triggers (e.g., child bangs head when intervention is isolation from peers). Also, assessment will ascertain and list the quantity or number of behaviors

and their type and severity. This may be done through direct observations, anecdotal reports from others, use of checklists, narrative descriptions, and other means.

Finally, assessment of modifiers of derived behaviors involves assessment of the interventions applied toward the most problematic behaviors. The interventions may be assessed in terms of observed effectiveness, short- or long-range effectiveness, whether they generate further problems, and whether they make use of multiple influencing variables. Assessment of modifiers of derived behaviors can identify whether interventions are unitary, multimodal, comprehensive, shortsighted, or too self-contained. The assessment may evaluate if interventions are increasing communication, attention, social interest, mood regulation, and cognitive skills, and whether interventions are making use of beneficial interactions with normal peers. At times, an intervention may over-focus on a particular behavior and undermine the more important relationship and positive feelings a child has with a caregiver, therapist, or teacher. Although the child may change specific behaviors, the results of missing the most important component of the intervention may show up in negative changes in the child’s mood, flexibility, and overall thinking and problem-solving capacities.

As new research in the field of communication and learning disorders becomes available, the BOLD format may serve as a framework to channel and organize new knowledge on the understanding between different biological levels of organization (genetic, constitutional, and developmental) with various levels of observed behavior. As a multimodal approach, it may sort comprehensive new information for the purpose of meaningful synthesis with established knowledge and prompt new directions in research and intervention. ■

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