Some problems exist in the way in which children with language disorders are classified, diagnosed, and treated. To help resolve these problems, clinicians need to function as clinical scientists in the diagnostic and therapeutic process. The classification, diagnosis, and treatment of children with language disorders should reflect hypotheses about the different levels of cause-effect relationships that exist among the structures, processes, and behaviors involved in language. Specifically, hypotheses about the primary and secondary deficits that characterize language disorders need to be made. That is, symptoms that reflect the underlying deficit must be differentiated from symptoms that reflect secondary and compensatory deficits. Clinical examples are presented to illustrate the way in which classification, diagnosis, and treatment of language-disordered children would proceed using these notions.

The purpose of this paper is multifaceted. On one level, it is an attempt to point out some of the problems that exist in the way we classify and diagnose children with language disorders and to offer some suggestions to rectify some of these problems. Accurate classification and diagnosis are important because appropriate and effective intervention often follows directly from classification and diagnosis. On a second level, this paper attempts to discourage practitioners from using assessment procedures as an end in themselves. It is much easier to observe and analyze behavior, language or otherwise, than it is to determine causal bases for behavior. Finally, on a third level, this paper represents a challenge to professionals in our field to be as scientific in the delivery of services as we are in our research and theorizing. Diagnosis and treatment of individuals with speech, language, and hearing problems require the same problem-solving skills that research does—formulating, testing, confirming, disconfirming, and reformulating hypotheses.

Why Clinical Practice is not Scientific

Despite attempts in recent years to make the classification, diagnosis, and treatment of children with language disorders more scientific, these attempts have been more successful in theory than in practice (Aram & Nation, 1975; Aram & Nation, 1982; McLean & Snyder-McLean, 1978; Nation & Aram, 1977; Rapin &

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Allen, 1980; Wolfus, Moscovitch, & Kinsbourne, 1980). There are several reasons for this. First, many clinicians do not understand what it means to be scientific in delivery of services. It is generally thought that “science” is needed only in research. Some, no doubt, view science as being rigid and inflexible where, in fact, the opposite is closer to the truth. By gathering clinical data and formulating and testing hypotheses based on these data, the clinician ensures that the clinical process remains flexible and attuned to the client’s changing needs. In this way diagnosis becomes an ongoing process rather than a one-time occurrence at the initial evaluation.

Second, to accommodate large case loads, school clinicians are often required to develop and implement procedures to expedite assessment, diagnosis, and treatment. Unfortunately, expedience and cost-effective service are thought to be inconsistent with scientifically oriented clinical procedures. It is a common misconception that such procedures need be particularly time-consuming. In fact, they can often be less time-consuming than those that are not scientifically oriented.

Third, many clinicians have limited knowledge of the biologic, cognitive, psychologic, social, and environmental forces involved in normal language development. Without this knowledge base, one cannot understand the way in which these forces might be causally related to particular deficits in language performance. Several recent books on child language disorders (Aram & Nation, 1982; Hubbell, 1981; McLean & Snyder-McLean, 1978) contain excellent frameworks and discussions of the way in which constitutional (i.e., biologic, cognitive, psychologic, and social) and environmental forces develop and interact in language-disordered children. It is only with an understanding of the development and interaction of these forces that clinicians can produce scientifically-based diagnostic hypotheses to guide assessment and intervention procedures.

Finally, many practitioners in our field have been trained to classify, diagnose, and treat language disorders according to a single orientation: (a) an etiological (medical) orientation, (b) a descriptive-linguistic orientation, or (c) a processing orientation. The use of a single orientation often has had the unfortunate consequence of focusing clinical attention to a certain set of facts and behaviors to the exclusion of other important facts and behaviors. This point will become clearer as these three orientations are described below. A more lengthy discussion can be found in Aram and Nation (1982, Ch. 2).

**Classification and Diagnostic Orientations**

The use of etiological typologies for classification and diagnosis of children with language disorders grew out of the early work of McGinnis (1963), Morley (1957), and Mykelbust (1954). Guiding much of the work with language-disordered children in the 1950s and early 1960s were categories such as deafness, emotional disturbance, mental retardation, aphasia, apraxia, alexia, and agraphia. In the process of attempting to identify the child’s more global problem, however, little effort was made to obtain specific information about the child’s language performance.
The descriptive-linguistic orientation involves identifying children according to the language deficit they demonstrate (e.g., syntactic, semantic, phonologic, pragmatic). The use of linguistic descriptions to identify and classify children's language disorders was spurred by the revolution in linguistic theory emanating from Chomsky's (1957, 1965) works. The popularity of this orientation, however, was also a function of the overemphasis that had previously been placed on etiological categorizations. The past 20 years have seen the development of increasingly more precise procedures to describe children's language (Crystal, 1979; Ingram, 1981; Kamhi & Johnston, 1982; Lee, 1974; Miller, 1981). Unfortunately, during this period, specific descriptions of language abilities have become such a central part of the diagnostic process that, at times, attention to important causal factors of the language disorder has become lost in these descriptions. The descriptive-linguistic orientation thus has often had a markedly behavioral look, in that only language behavior is considered in evaluating the language disorder. This is particularly ironic in light of the antibehaviorist linguistic theory (Chomsky, 1965) from which it derived. Although those who ascribe to this orientation (e.g., Bloom & Lahey, 1978) usually classify disordered children according to major etiological categories, treatment is often based solely on the behavioral characteristics of the language disorder. In this way, a syntactic delay would be treated the same way in a retarded child as it would in a child with a developmental language disorder.

Processing orientations provided a means to bridge the gap between linguistic descriptions and etiological categorizations of children with language disorders. Generally speaking, processing orientations characterize language disorders in terms of disruptions or breakdowns that occur in the processes involved in language comprehension and production. One popular view that attempted to bridge the gap between the etiological and linguistic orientations was that children with language disorders suffered from an auditory processing deficit (e.g., Eisenson, 1972). However, since then, serious questions about the role auditory processing deficits play in language disorders have been raised (Rees, 1973, 1981; Tallal & Piercy, 1978). Recent attempts to provide processing explanations of child language disorders have been more comprehensive and integrative in their attempts to delineate how constitutional and environmental forces interact to cause language deficits (Aram & Nation, 1982; Hubbell, 1981; McLean & Snyder-McLean, 1978; Muma, 1978; Nation & Aram, 1977). Aram and Nation (1982), for example, provide a multistage process model which attempts to relate constitutional (intrinsic) and environmental (extrinsic) forces to language performance by paying particular attention to the cause-effect relationships that exist among these components.

Despite these recent attempts to link constitutional and environmental deficits with deficits in language performance, in practice, classification and diagnosis of children are still predominantly based on observable phenomena. To help resolve this problem, clinicians need to make a distinction between symptoms that reflect the underlying or primary deficit and symptoms that reflect secondary deficits that are caused by the individual's reaction to and/or compensation for the primary deficit. In other words, clinical hypotheses should be made about the different levels of cause-effect relationships that comprise the child language disorder.
Children with language disorders should be classified according to the hypothesized primary deficit. In this way, the diagnostic label would reflect an hypothesis about a causal relationship that exists between deficient constitutional and environmental forces and language performance. In the remainder of this paper, these notions will be explained further and clinical examples illustrating the way in which classification and diagnosis might proceed will be presented.

Distinguishing Between Primary and Secondary Deficits

The distinction between primary and secondary deficits has its roots in the multidimensional view of adult aphasia espoused by Luria (1958). A focal lesion to a particular part of the brain can be thought of as producing a primary breakdown of a single component of the cognitive system which supports a specific language function. The primary breakdown in turn leads to a variety of secondary deficits because of the highly interactive nature of the various language functions. As Davis (1983) notes in his discussion of these points, a primary disturbance upsets the sometimes delicate balance among these intimately related systems. For example, damage to Wernicke's area might produce a breakdown to auditory language processing, the primary deficit. This primary deficit leads to secondary symptoms such as repetition failures, lack of awareness of jargon, reading deficits and, possibly, the jargon itself (Davis, 1983). Importantly, with secondary deficits there is not necessarily an impairment to the structures and processes underlying the particular behavior in question.

The distinction between primary and secondary deficits allows us to propose a hierarchy of cause-effect relationships. Consider, for example, the child who has a primary deficit in the cognitive processes involved in formulating syntactic categories and structures. This primary deficit obviously has an adverse effect on the child's expressive syntactic abilities, but it also might have an adverse effect on speech-sound production and/or cause the child to use only familiar words and constructions in speech. Another child with the same primary deficit might decide to talk as little as possible because of embarrassment. The way in which a child reacts to and compensates for the primary language deficit determines the nature and extent of the secondary deficits. The more maladaptive or lacking these reactions and compensatory behaviors are, the more secondary deficits there will be. These differences need to be taken into consideration in planning and implementing therapy.

Given that the distinction between primary and secondary deficits is an important one, the goal of assessment should be to differentiate between symptoms that reflect the primary deficit and symptoms that reflect secondary and other higher-order deficits. Unfortunately, this is often not so easy to do with children as it is with adult aphasics because, with children, hypotheses about deficient biological and cognitive structures and processes are usually based on specific patterns of language and language-related behaviors rather than on sites of focal lesion. Regardless of the availability of corroborative neuroanatomical data, here as elsewhere in the clinical process, the clinician needs to function as a clinical scientist. The clinician begins by formulating some hypotheses about the primary
and secondary deficits based on the initial assessment procedures. These initial hypotheses provide the basis for initial classification, diagnosis, and treatment. The scientific clinical process does not stop here, however, because as additional clinical data are gathered during subsequent therapy sessions, the initial hypotheses are either confirmed or disconfirmed and modified as necessary. To illustrate, let us consider the following two examples of children with language disorders.

Clinical Examples

Adrian is a Black male, age 5:8 (years, months). He was hospitalized at age 2:0 due to poor weight gain and failure to grow. Petit mal seizures were observed in 1980, and since that time, Adrian has been taking Zarotin twice daily. Developmental milestones were reached within normal age limits. Adrian walked unassisted and fed himself with a spoon at 13 months and was toilet trained by 3 years of age. He spoke his first words at 10 months ("Momma") and was reportedly using two-word utterances by the time he was 2 years of age. He showed no history of middle ear infections or hearing loss.

Adrian's performance on a nonverbal intelligence test, Leiter (Arthur, 1952) revealed an MA level of 5:5, well within normal limits for his age. On the Stanford-Binet, however, his MA was only 4:3, presumably reflecting his deficient verbal-based skills. His visual and auditory sequential memory abilities as measured by ITPA indicated a 1-year delay.

Adrian's receptive language was evaluated by the Test for Auditory Comprehension of Language (TACL), (Carrow, 1973) the Peabody Picture Vocabulary Test (PPVT), (Dunn, 1965) and the Auditory Association and Auditory Reception subtests on Illinois Test of Psycholinguistic Abilities (ITPA) (Kirk et al., 1968). On the TACL, Adrian performed within six months of age level, indicating some facility with prepositions, quantifiers, and action verbs. In contrast, on the PPVT, his MA level of 2:7 was approximately 3 years delayed. On both the ITPA subtests, Adrian functioned about 2 years below his age level. Cultural differences seemed to play some role in Adrian's performance on the Auditory Association task, in that acceptable responses were sometimes not geared to reflect the experiences of a Black urban child. Consider some of his responses on this task: "A jail has criminals; a hospital has doctors." "Mountains are high; valleys are flown." "The neck has a collar; the waist has a 'wasty' milk."

A similar pattern of strengths and weaknesses was found in Adrian's expressive language. His vocabulary was quite limited as indicated by a type-token ratio of .38. Templin's normative data on type-token ratios reported in Miller (1981) indicated that TTR values of .50 occurred across a wide range of normal children. A sample of Adrian's language revealed a Developmental Sentence Score (DSS) (Lee, 1974), of 6.85, which placed him slightly below the 10th percentile for his age. A measure of grammatical marker use, Grammatical Marker Error (GME) Index (Kamhi & Johnston, 1982) was .12, indicating that Adrian rarely had difficulty correctly encoding grammatical morphemes such as noun and verb inflections, copulas, and auxiliaries. As a result, Adrian generally produced well-formed sentences. In fact, not counting 10 dialectal forms (e.g., "ain’t"), he produced only three ungrammati-
cal sentences. Like his lexical production, however, his sentence constructions lacked variety. This lack of variety and tendency to produce simple constructions contributed to his relatively low DSS score.

Semantically, Adrian expressed all the semantic relations common to the speech of young children, including agents, patients, beneficiaries, experiencers, locatives, factitives, and comitatives. His phonological development was characterized by the presence of some common early developmental phonological patterns, such as cluster reduction and unstressed syllable deletion. Speech intelligibility was generally good, and he demonstrated a full phonetic inventory. Pragmatically, he expressed a variety of communicative functions, including questions and requests for action; however, less than 5% of his utterances were initiations of a communicative act.

Given this clinical information, what can be said about Adrian’s primary and secondary deficits and the causal bases for these deficits? Let us begin with the descriptive-linguistic hypothesis that he suffers from a primary deficit in lexical semantics, with a secondary deficit in certain aspects of expressive syntax. This hypothesis is based on the fact that his vocabulary skills, both expressive and receptive, were considerably more delayed than his syntactic and phonological development.

This descriptive-linguistic hypothesis about Adrian’s primary and secondary deficits needs to be followed by hypotheses concerning the possible causal bases for these deficits. Were the lexical and syntactic deficits caused by the same or different factors? Consider first the possible causes of the lexical deficit, including (a) defective lexicalization structures and/or processes; (b) limited exposure to objects, events, and experiences in the world; and (c) a developmental history of failure to thrive and seizures.

Insofar as Adrian was able to express relatively abstract concepts such as “both,” “already,” various conjunctions, pronouns, and quantifiers, he possessed the ability to form conceptual categories and map these categories onto words. Though perhaps inefficient, the biological and cognitive structures and processes that allow for conceptualization and lexical expression to occur were intact. With regard to the second possibility, there was no reason to believe that Adrian suffered from environmental deprivation. Parent interviews suggested a home environment with diverse opportunities for language stimulation. For an initial causal hypothesis, therefore, the third alternative was chosen. That is, it was hypothesized that Adrian’s lexical deficit was caused by his early failure to thrive and seizures.

Premack (1980) has written that competence means, in part, an indigenous disposition to exercise that competence. This notion seems to have particular relevance for Adrian in that he either chose not to exercise his disposition to learn language or was prevented from exercising this disposition. Initially, perhaps Adrian was prevented from exercising his competence because of frequent illnesses. These illnesses could have kept his interactions with the world to a minimum and deprived him of the mental and physical energy needed for lexical development to proceed at a normal rate. When the illnesses abated, Adrian may have maintained his limited involvement with the world, perhaps because he had grown accustomed to this low level of interaction. Adrian’s deficit might best be described

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as a “motivational” deficit, because he either willingly or unwillingly was not using his language faculties to the extent they should be used. The lexical deficit and the potentially deficient structures and processes which underlie lexical development are thus being viewed as a secondary deficit to the primary motivational one. The motivational deficit had more effect on lexical rather than syntactic development, probably because lexical development depends more on interactions with objects and events in the world. The motivational deficit also restricted pragmatic development as evidenced by the sparsity of communicative acts initiated by Adrian. A more rigorous pragmatic or social-behavioral analysis would no doubt reveal other secondary deficits in these areas.

The question that remains is whether the syntactic deficit is secondary to the primary motivational deficit or secondary to the lexical deficit. It is probably secondary to both. In some respects, the syntactic deficit is secondary to the motivational deficit because the motivational deficit to some extent probably diminished Adrian’s desire to generate diverse syntactic constructions. At the same time, Adrian’s lexical deficit might lead to a disproportionate amount of cognitive energy being devoted to the lexicalization process leaving less energy and attention to be devoted to other language domains. In this way, the lexical deficit might be responsible, in part, for Adrian’s use of simple syntactic constructions and lack of syntactic diversity. The syntactic deficit, thus, would be secondary to the lexical deficit and a third-level effect of the primary motivational deficit.

Based on these hypotheses, therapy focused on Adrian’s primary motivational deficit and secondary lexical and pragmatic deficits. Improving Adrian’s desire to interact and initiate communicative acts formed the basis of our therapy approach. By showing Adrian the power of language, the fun in using language, and the diversity in language, we hoped to enhance his desire to learn about objects and events in the world. In general, Adrian was encouraged to use his intact language-learning abilities to their fullest extent. Despite no formal syntax or vocabulary training, in six months’ time Adrian’s PPVT and DSS scores improved by more than a year. Admittedly the same result might have been obtained with a different set of hypotheses (even no hypotheses) and different therapy, but language gains achieved by scientific means should occur more quickly and more frequently than language gains achieved in other ways.

Let us compare Adrian to David, a 5:0-year-old boy being raised in a middle-class environment. In contrast to Adrian, David had no significant medical history and a normal developmental history. On the Leiter, David obtained an MA of 5:7 or more than six months above his age level. His auditory and visual sequential memory skills were 1 year below age level. However, on the PPVT, and TACL, and the Auditory Association and Auditory Reception subtests of the ITPA, he performed within normal age limits. Nevertheless, despite David’s age-appropriate cognitive, conceptual, and vocabulary skills, his DSS score of 4.72 placed his expressive language abilities well below the 10th percentile for his age. His GME was .59, meaning that he made errors encoding grammatical markers more than half the time and that more than half of his utterances were not well formed. To illustrate, some of David’s utterances were: “He throw stick.” “He said, me no get stick.” “That dog go like this.” “He playing fiddle.” “He run away.” “I go look at that boats.” “I’m not
old enough go to baseball.” “He lay down cause he not go get it.” As is apparent from these utterances, David had particular trouble correctly encoding grammatical markers.

In contrast to David’s syntactic difficulties, his semantic system seemed to be intact. His sentences encompassed all the various semantic notions and generally contained more than one proposition (see Kamhi & Johnston, 1982, for a discussion of propositional complexity). David, then, in contradistinction to Adrian, is language-impaired, with a primary deficit in the structures and/or processes that underlie syntactic functioning. Although no notable secondary deficits were apparent in David’s behavior at this time, it is not difficult to envision the potentially adverse effects his language impairment might have on future academic performance and social development. These adverse effects would be secondary symptoms of the primary syntactic formulation deficit. At this point, therapy should attempt to improve David’s syntactic formulation abilities while at the same time attempting to maintain his nonmaladaptive reactions to his language impairment.

In these two clinical examples, the importance of differentiating between primary and secondary deficits should be clear as should the need for clinicians to function as clinical scientists in order to make such distinctions. By formulating and reformulating hypotheses about different levels of cause-effect relationships, it was possible to uncover important differences in Adrian and David, despite the fact that descriptively they might both be identified as syntactically disordered children. In Adrian, the syntactic problem seemed to be secondary to the primary motivational deficit as well as to the secondary lexical deficit, whereas in David, the syntactic disorder reflected a primary deficit in the structures and/or processes underlying syntactic functioning.

Conclusion

This paper has argued that clinicians need to function as clinical scientists throughout the diagnostic and therapeutic process. In addition, it was suggested that classification, diagnosis, and treatment of children with language disorders should derive from clinical hypotheses about the primary and secondary deficits that characterize these disorders. These hypotheses can then be confirmed or disconfirmed as new clinical data are obtained. In this way, the classification, diagnosis, and treatment of children with language disorders become and remain ongoing scientific clinical processes.

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