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Practitioner Review: Developmental Language Disorders: A Clinical Update

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Non-specialists can identify three types of developmental language disorder. (1) mixed receptive/expressive disorders, which impair phonology, syntax, and semantics. Children who understand nothing are nonverbal, in others speech is sparse, nonfluent, poorly intelligible, and agrammatic; (2) expressive disorders with adequate comprehension affect phonologic production predominantly. Children with verbal dyspraxia, the most severe variant, may also be nonverbal but comprehend well; (3) higher order processing disorders affect semantics, pragmatics, and discourse. Semantics and pragmatics are invariably affected in preschool autistic children in whom isolated expressive deficits do not occur. Etiology of developmental language disorders is predominantly genetic. Structural brain lesions detectable by neuroimaging are exceptional. Severe receptive deficits require a sleep EEG to detect subclinical epilepsy. Early educational intervention is both critical and efficacious.

Keywords: Language acquisition, developmental language disorder, autism, epileptic aphasia, childhood aphasia, subtyping, diagnosis

Child language acquisition and its disorders are active and broad fields of research, previously summarized by Bishop in this Journal (Bishop, 1992). This review considers the often confusing field of the developmental language disorders from the clinical perspective of a child neurologist. It must be selective and focus on those aspects most relevant for updating clinicians' understanding. The review starts by outlining how children acquire their first language. It then considers clinical subtyping of the developmental language disorders (DLD), that is, inadequate language acquisition in children without known structural brain lesion, hearing loss, significant general learning disabilities, or severe social deprivation. The review contrasts children with specific language impairment (SLI or pure DLD), that is, children with an isolated deficit for language and no other associated developmental deficit, as opposed to children with DLD who have autistic features. This topic is considered because clinically the initial complaint of the parents of preschool children on the autistic spectrum is almost always inadequate language, rather than some other feature of autism such as impaired sociability or impoverished play. The paper then contrasts DLD with acquired aphasias in children, including the confusing syndrome of acquired epileptic aphasia, so-called Landau–Kleffner syndrome. It points out that there has been a major shift in our understanding of the etiology of DLD and of other developmental disorders and learning disabilities, away from the vague concept of "minimal brain damage or disability" often imputed on slender or nonexistent evidence to a perinatal insult to the immature brain, toward genetics as a major etiologic agent. Finally it touches upon appropriate investigation and management issues.

In order to assist readers unfamiliar with the field of language disorders, Table 1 provides definitions of some of the terms used in this review.

Acquisition of the Components of Language

Typically, human infants acquire the ability to speak and understand speech over the first three years of life, seemingly effortlessly and without the need for systematic instruction even though they are exposed to less than consistent exemplars of their mother tongue. This ability is specific to that age and stage of brain maturation and dwindles across childhood, to remain a markedly attenuated and imperfect aptitude in adulthood.

Longitudinal videos of infants and children, together with their parents' inventories of words and expressions produced and understood by children learning a variety of languages, indicate that there are both universals in language acquisition and differences among children. These differences depend in part on the particular languages being acquired and in part on infants' styles for accomplishing this cognitively monumental task (Bates, Thal & Janowsky, 1992). In one year infants go

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Table 1
Definitions and Abbreviations

Dysarthria	impaired speech articulation because of a motor deficit of the oromotor musculature (e.g., bulbar or pseudobulbar palsy, athetosis, cerebellar deficit)
Grammar	that part of the study of language that deals with forms and structure of words (*morphology*) and with their customary arrangement in phrases and sentences (*syntax*) (Webster)
Lexicon	stock of words, repository of word meanings in the brain
Paraphasia	substitution of a speech sound for the targeted one (*phonologic or literal paraphasia*) or of a word for the targeted one (*semantic paraphasia*)
Phonology	study of the speech sounds of a language
Pragmatics	relation between signs or linguistic expressions and their users (Webster). In the present context refers to the communicative use of verbal and nonverbal language
Semantics	the meaning of words, sentences, or communication
Abbreviations	
AD	autistic disorder, defined according to DSM IV criteria
DLD	developmental language disorder
DSM	Developmental and Statistical Manual of Mental Disorders of the American Psychiatric Association
ICD	International Classification of Diseases of the World Health Organization
PDD	pervasive developmental disorder, i.e., umbrella term used by DSM and ICD to refer to conditions on the autistic spectrum
PDD-NOS	PDD-not otherwise specified, i.e. children with some autistic features who do not fulfil criteria for AD or any of the other specified disorders on the autistic spectrum
SLI	specific language impairment, i.e., DLD without associated deficits
VAA	verbal auditory agnosia, i.e., inability to decode phonology, which results in the most severe of the DLDs

from cooing vowel sounds to producing repetitive consonant/vowel syllables such as "mama", "gaga", to producing meaningful, though imperfectly produced single words. These accomplishments indicate that the infant has learned to segment the stream of sound into communicatively meaningful chunks and to associate them with particular environmental stimuli. In the second year, words are acquired first rather slowly, one by one, then vocabulary takes off. When children have acquired some few dozen words they start to produce two word utterances which are the root of the grammar which will enable them to understand and produce meaningful sentences. In the third year, vocabulary grows from dozens to hundreds of words and syntactic knowledge from the production of two word utterances to sentences. Thereafter language acquisition involves the comprehension and production of ever more complex sentences such as passive and embedded grammatic constructions, vocabulary numbers in the thousands of words, and by school age children start to master written language.

Differences in language learning style emerge in the second year when some normal children proceed by systematic and slowly perfected additions to their word stock, whereas others produce from very early on long largely unintelligible strings of expressive but incompletely analyzed speech-like sounds (Bates & Marchman, 1988). Differences attributable to the particular language being learned affect, for example, how early verbs, as opposed to nouns, appear in children's vocabularies. Morphologic markers for gender or case are produced at an earlier age in highly inflected languages in which word order is relatively flexible, contrasted to their later appearance in languages, such as English, where it is largely word order that indicates the grammatic role of words in sentences (Bates & MacWhinney, 1981; Leonard, Sabbadini, Leonard & Volterra, 1987).

Studies over the past 12 years have shown unequivocally that children are born with innate auditory capacities for discriminating among the variety of auditory contrasts exploited by the multitude of human languages. These contrasts vary greatly across languages and studies suggest that neonates are capable of making a larger number of *phonetic* discriminations than toddlers who, while they have perfected temporal and tonal discriminations relevant to the language(s) to which they are exposed, gradually lose the ability to make discriminations irrelevant to this (or these) languages (Aslin, Pisoni, Hennessy & Perey, 1981; Kuhl, William, Lacerda, Stevens & Lindblom, 1992). This gradual loss of phonetic perceptual ability, which spans several years, may explain why very young children are capable of speaking each of several languages like monoglots, whereas older children and adult second-language-learners retain foreign accents life-long, even in a second language that may have become their better language. The honing of auditory perception and discrimination with repeated exposure, and the loss of the ability to discriminate phonetic contrasts which the environment does not provide because they are not phonemic (i.e. do not carry linguistic meaning) in the language spoken may well be an example of the strengthening of synapses in "educated" circuits and the pruning of "uneducated" unused ones, in other words it may be an example of the way in which environmental experience "sculpts" the brain (Bates, Thal & Janowsky, 1992).

Equally early to appear as the ability to analyze phonology are the skills requisite for conversational use of language, i.e. *pragmatics*. Just as infants are innately attracted to the sounds of speech, they are innately

attracted to the human face (Trevarthen, 1983). Parents eagerly await their infant's first responsive smile, but they may be unaware that, at about that same age, infants are able to imitate facial expression (Meltzoff & Moore, 1977). Looking at the caretaker's face and cooing responsively soon follows. Responsive cooing signals that the infant is learning such conversational skills as orienting toward a speaker, listening, turn-taking, and waiting for the caretaker to pause after making a sound before taking over the role of communicator (Trevarthen, 1983).

The development of pragmatic skill is intimately tied to affect and sociability. In order to be able to respond to their caretakers' social advances, infants need to be comfortable, alert and attentive. Attachment to the caretaker is rapid but requires feedback for its maintenance (Ainsworth, 1973). It may take several days for young infants to regain full responsiveness to their mother if they have been separated from her for a week. Children brought up in isolation fail to thrive, are depressed, and do not smile (Spitz, 1945). Language learning, which depends on the repeated input over many months of the often simplified speech ("baby talk") caretakers address to their infants, requires that infants attend to the caretaker and be socially rewarded for their own social overtures and early attempts at imitating speech sounds. The importance of joint attention for language learning is exemplified by the markedly delayed language acquisition of most autistic children in whom joint attention and pragmatic skills are invariably severely deficient (Mundy, Sigman & Kasari, 1990).

A great deal of work has been expended on understanding how infants make the connection between auditory signals they have learned to segment and discriminate (i.e. words) and, first, the objects or people they stand for and, soon thereafter, the actions and attributes to which the words refer (Bates, 1979; Vihman & McCune, 1994). This ability takes place at the interface between cognitive and semantic development. It appears that infants must have stored in their memory a representation of an object or action before the word can acquire meaning independently of the particular context or episode in which it occurred. In addition to *semantics* at the word level, semantics at the sentence or discourse level is also partly linguistic — depending as it does on lexical and syntactic knowledge — and partly cognitive. Decoding sentences calls for the ability to store an entire utterance in immediate memory in order to analyze its meaning on-line (Marslen-Wilson & Warren, 1994), evaluate it in the context of what has gone before and of current priorities, and decide whether it calls for a response, be it verbal or nonverbal. Short-term multimodal memory, in which a new input is analyzed in the light of items stored in long term memory, has been shown to depend on activity in the lateral prefrontal cortex (Della Sala & Logie, 1993), whereas the assigning of weights or affective value to new information in the context of present organismic priorities calls for inputs from the amygdala (Gloor, 1978; Bachevalier, 1994).

Grammar refers to the rules for organizing words into well-formed flexible sentences that maximize precision and minimize ambiguity in information transfer. At about 18 months, almost concurrently with the burst in word acquisition, infants start producing combinations of two meaningfully related words (Bates, Thal & Janowsky, 1992). This heralds the acquisition of the complex rules of grammar which some linguists, like Chomsky (1975), look upon as the fundamental characteristic of human languages. By three years infants are capable of producing complex sentences, and by four they have acquired all of the major rules of their language.

Speech output lags behind decoding abilities (Anisfeld, 1984). The production of an intelligible word requires the discrimination of the relevant speech sounds and elaboration of the highly practiced and perfected motor programs required for their execution under auditory feedback control. Complete mastery of the production of all the phonemes of a language in all contexts takes years and may not be achieved until school age. Vowels, and consonants produced on the lips like "m", "p", "d" appear earlier than sibilant sounds like "s", "f", "th", and blends like "str". Some contrasts, like "b" and "v", or "r", "l", and "w", are difficult for many children. Isolated consonant errors that do not greatly jeopardize intelligibility should be considered immaturities rather than true language disorders, although, according to Monnin and Huntington (1974) they do reflect perceptual deficits for that specific phoneme.

Clinical Subtyping in DLD

Children with DLD present to clinicians with a variety of clinical pictures (Rapin & Allen, 1983; Bishop & Rosenbloom, 1987). This is hardly surprising considering the complexity of decoding, processing, and encoding language operations. Yet much of the experimental work on DLD rests on the assumption that SLI (DLD without associated deficits) is a unitary condition and attempts to find a single explanation for its varied manifestations (Bishop, 1992). Locke (1994) argues not only for DLD as a unitary condition but goes so far as to argue against DLD as a specific disorder; he views it as but one of the consequences of delayed brain maturation causing generalized brain dysfunction. Experimenters' unitary view of SLI may be due in part to the fact that they select children for experimental studies on the basis of discrepancy criteria between language ability and mental age, language age, or score on a standardized language test, rather than on the basis of the child's conversational use of language, and on the basis of exclusionary criteria such as lack of hearing loss, known brain lesion or diagnosed neurologic illness, mental deficiency, autistic behaviors, and social deprivation. The sample captured will depend a great deal upon the particular discrepancy criterion chosen (Aram, Morris & Hall, 1993). Also, standardized language tests tend to focus on language at the word level and provide little or no data at the sentence or conversational level and, with the rare exception of a few specialized tests, no data on pragmatics.

Clinicians, notably neurologists, identify children with DLD on the basis of positive signs of inadequate language, based on their expectation of the intelligibility,

Table 2
Rapin and Allen Clinically Defined DLD Subtypes

Mixed receptive/expressive disorders
- *Verbal auditory agnosia (VAA)*: profoundly impaired comprehension deficit because of very deficient phonologic decoding with resultant severe expressive deficit. Children are nonverbal or have very limited expression with defective phonology. More frequent in autistic than DLD children.
- *Phonologic/syntactic deficit disorder*: comprehension equal to or better than production which is impoverished, with short, often ungrammatical utterances, impaired phonology, and a limited vocabulary. The most prevalent variant of DLD. Occurs in both DLD and AD children.

Expressive disorders (comprehension normal or near normal; not seen in autistic children)
- *Verbal dyspraxia*: extremely dysfluent subtype with sparse output and very poor phonology.
- *Speech programming deficit disorder*: fluent subtype with jargon.

Higher order processing disorders
- *Lexical deficit disorder*: severe word finding deficits and comprehension difficulty for connected speech. Spontaneous language superior to language on demand. Inadequate formulation of discourse. Early on, jargon, pseudo-stuttering because of word-finding deficit, and simplified syntax, are frequent. Later, phonology and syntax unimpaired. Occurs in both DLD and AD children
- *Semantic pragmatic deficit disorder*: verbosity with comprehension deficits for connected speech. Word finding deficits and atypical word choices frequent. Phonology and syntax unimpaired. Inadequate conversational skills: speaking aloud to no one in particular, poor maintenance of topic, answering besides the point of a question. Most prevalent in verbal autistic children in whom it may be associated with conspicuous verbal and nonverbal pragmatic deficits, immediate and delayed echolalia, and verbal perseveration. Less frequent in DLD, seen in Williams syndrome and hydrocephalus.

syntax, vocabulary, comprehension, and conversational skills of children of a similar age. Thus clinicians are likely to see a broader range of language disorders than the more narrow range of children with SLI selected by experimentalists. Some clinicians may choose to validate their clinical impression by using a checklist, such as the Early Language Milestones (ELM) (Coplan, 1989) or the Clinical Linguistic and Auditory Milestone Scale (CLAMS) (Capute et al., 1986), which spell out the range of expectations for any given age. The MacArthur Communicative Development Inventories (Fenson et al., 1993), for which there are norms for American English, Mexican English, Japanese, Italian, and several other languages, when filled out at regular intervals by parents provide a reliable way to track comprehension, production and use of language in infants under the age of 30 months.

It may be appropriate to state at this point that there is no unanimity on the classification of DLD children into qualitatively distinct syndromes, even though, at a minimum, receptive and expressive syndromes have been identified by statistical analysis of standardized language and neuropsychologic test data (e.g. Aram & Nation, 1975; Wilson & Risucci, 1986; Rapin et al., 1996). Bishop (1992) points out that some early attempts at subtyping were weakened by the small number of subjects relative to the number of variables used. Whether DLD is one disorder or whether subtypes of DLD reflect dysfunction in discrete parts of the language processing systems of the immature brain is a critical issue if one's goal is to gain an understanding of the neural pathogenesis of DLD. Resolving these issues calls not only for the study of larger numbers of children selected according to well defined but less restrictive criteria and evaluated with a broad range of well standardized measures, it also calls for external validation of any empirical behavioral classification with such new biologic correlates as genetic markers for particular syndromes, morphometric and functional brain imaging, and electrophysiologic measures (Fletcher, 1985). As a first approximation, DLD can be divided clinically into three categories: receptive/expressive, expressive, and higher order processing disorders at one or more of the language-processing levels, which include phonology, syntax, semantics and pragmatics. These disorders correspond in broad terms to the receptive, expressive, and conduction aphasias of adults with acquired focal brain lesions (Rapin & Allen, 1988). We review here Rapin and Allen's (1983) attempt to subtype DLD in preschool children into these three broad clinically-defined categories. They suggest that clinicians may find it useful to distinguish subsyndromes within these three variants, based on systematic observation of the preschool children's conversational skills in an interactive play setting. Empirical determination of whether these subsyndromes differ on a severity continuum or have a categorical biologic basis remains to be determined. They are listed in Table 2.

1. *Receptive/expressive DLD syndromes*: One of the major differences between children and adults is that purely receptive deficits with preserved expression do not exist in young children because at that age language is not the overlearned skill of adults who can continue to speak without the need for feedback. Even acquired peripheral deafness in a preschool child is likely to result in quasi total loss of the ability to speak unless heroic efforts are made to maintain speech: after a year or so some children deafened at 2 or 3 years are virtually indistinguishable from children with congenital sensorineural hearing losses, even though the deafened children might already have learned to produce sentences and have had a sizeable vocabulary. Deafened preschoolers have the advantage of an awareness of the power of speech and communication which is not as well

developed in most of the congenitally deaf children of hearing parents and may be conspicuously lacking in young nonverbal autistic children.

The most severe receptive/expressive deficit syndrome is *verbal auditory agnosia (VAA) or word deafness* (Worster-Drought & Allen, 1930). VAA jeopardizes the decoding of phonology, the requisite first step for language comprehension. VAA is particularly frequent in the syndrome of acquired epileptic aphasia, to be discussed later, but also occurs as a developmental disorder which is more frequent in children on the autistic spectrum than in children with SLI (Allen & Rapin, 1992). Inability to analyze speech at the phonologic level precludes all subsequent language processing operations. As a result children with VAA may be as handicapped as deaf children and are mute or largely nonverbal. Those who improve typically have defective phonology and their comprehension and speech sound discrimination are unlikely to recover fully (Klein et al., 1995). The key, is that if the disorder is isolated, these children are able to acquire language through the visual channel. They are the children who profit the most from communication boards, sign language, or being taught language by reading, using computers or other augmentative communication devices (Allen, 1989; Allen, Mendelson & Rapin, 1989). Those who are severely autistic, especially if also learning disabled, generally have a guarded prognosis for the acquisition of speech but some can learn some sign language, often with subsequent behavioral amelioration (Bonvillian, Nelson & Rhyne, 1981).

A less severe variant of receptive/expressive deficit disorder differs from VAA by the fact that, although comprehension is impaired, it is equal to, or often considerably better than, production. These children tend to speak in short telegraphic utterances, and often missing from their productions are the morphologic markers (word inflections) and small invariant closed-class words such as articles, prepositions, conjunctions, and pronouns. Their vocabulary is meagre and they may have word finding problems. If they make paraphasic errors, these tend to be phonologic rather than semantic paraphasias. They too may use superordinate categorical words rather than the more precise target that would be appropriate.

This *receptive/expressive phonologic/syntactic deficit syndrome* is the most prevalent disorder among children with DLD. It is in this type of child that Tallal demonstrated for the first time the children's problem with processing stop consonants (Tallal & Piercy, 1974), a problem subsequently expanded to involve a variety of brief acoustic stimuli (Tallal & Piercy, 1978). Recent work has shown that the deficit for processing rapid stimuli also involves visual and somatosensory stimuli (Tallal, Miller & Fitch, 1993). The deficit is most dense in children with VAA (Frumkin & Rapin, 1980). Perhaps the mixed receptive/expressive deficit syndrome is on a severity continuum with VAA, and its syntactic, lexical, and expressive deficits are secondary to the auditory decoding deficit that forestalls normal language development; again, more work will be required to determine that this single perceptual deficit provides an adequate explanation for all the children with this most common of the DLD syndromes.

In contrast to the always mixed expressive/receptive disorders, isolated *expressive — or largely expressive — DLD syndromes* are quite common because, inasmuch as language processing is unidirectional — albeit with many feedback loops — an output disorder does not preclude comprehension and processing. Most receptive/expressive and expressive disorders affect both phonology and syntax, which makes them clinically salient because they jeopardize intelligibility, sentence structure, and, in many cases, the richness of the vocabulary as well.

Rapin and Allen (1988) divide the expressive deficit syndrome into two subsyndromes on the basis of verbal fluency. The first is a very dysfluent subsyndrome, *verbal dyspraxia*, which has been identified by many other investigators (Le Normand, 1995; Hurst, Baraitser, Auger, Graham & Norell, 1990; Ferry, Hall & Hicks, 1975; Yoss & Darley, 1974) and may even have a specific genetic etiology (Hurst, Baraitser, Auger, Graham & Norell, 1990). Children with verbal dyspraxia speak little and effortfully, they produce single words or truncated word approximations, often with very distorted or missing consonants. If their language is limited to single utterances there is no opportunity for them to produce grammatical forms. Some investigators consider verbal dyspraxia a severe speech production disorder rather than a language disorder, an unsettled argument which hinges on different models of the language reception/production system (Stackhouse, 1992). Prognosis is guarded despite active remedial training, with the most severely affected children remaining expressively handicapped as adults (Ferry, Hall & Hicks, 1975).

It is important not to confuse verbal dyspraxia with *dysarthria* which is a sensorimotor disorder of speech production, not a language disorder. Dysarthria results from a motor deficit in the control of the orofacial musculature, such as that caused by spastic and, especially, athetoid cerebral palsy, or by cerebellar disorders. Verbal dyspraxia is a language disorder presumed to be due to defective programming of verbal motor outputs, of the translation of verbal "images" into the motor commands for their execution by the orofacial musculature. Dysarthria is regularly associated with drooling, difficulty chewing and swallowing. Note, however, that a significant number of children with verbal dyspraxia, and of those with mixed receptive/expressive disorders, have some oromotor deficits, however these are not severe enough to account for their defective expressive phonology.

Occasional children present with a fluent variant, the *phonologic programming deficit syndrome*. These children tend to speak in long phonologically flawed, poorly intelligible utterances. Poor intelligibility makes it difficult to judge the adequacy of their syntactic skills. Prognosis is better than in verbal dyspraxia. When the children become verbal, they often have difficulty organizing coherent discourse, for example telling a story or providing an explanation, skills that also call for semantic abilities. Comprehension may be quite good but is not universally perfect, casting doubt on this DLD

Table 3
The Autistic (Pervasive Developmental Disorder or PDD) Spectrum

Autism (PDD) is a behaviorally defined disorder that refers to individuals with deficits in (1) social interaction, (2) communication and imaginative play, and (3) range of interests and activities. Although the cognitive skills of the population of individuals on the autistic spectrum is lower than that of the normal population, IQ is not a defining feature of the disorder. In general, the verbal IQ tends to be lower than the performance IQ. Autism has many different causes, a wide range of severity, and in the absence of other overt signs of brain dysfunction has strong genetic implications

DSM IV (1994)

- *Autistic Disorder (AD)*: A total of 6 or more (of 12 listed) deficits, with one in each (at least 2 in the first) of the following areas in (1) social interaction, (2) social communication, (3) restricted repetitive and stereotyped patterns of behavior, interests, and activities, with onset of deficits in at least one of these areas prior to age 3 years.
- *Rett's Disorder*: A syndrome limited to previously normal girls with decelerating head growth in infancy, early autistic behaviors, severe mental deficiency, hand wringing, clapping, licking or other stereotyped hand movements. Most are nonverbal, many develop epilepsy, episodic hyperventilation, muscular hypotonia, and scoliosis. Although indefinite survival occurs, there is an excess of early deaths.
- *Childhood Disintegrative Disorder*: Appearance of autistic behaviors following completely normal development until at least age 2 years. May be associated with loss of motor and cognitive skills and with epilepsy. Must not be attributable to a defined progressive degenerative disease of the brain.
- *Asperger's Disorder*: Socially and functionally disabling autistic behaviors in the face of adequate language and cognitive skills. May be associated with clumsiness. Cognitive verbal skills may be superior to performance skills.
- *PDD-NOS* (PDD not otherwise specified): Patients on the autistic spectrum who do not qualify for AD because they have fewer than 6 of the listed deficits or do not have them in the prescribed distribution and do not fulfil criteria for any of the other PDD categories.

ICD 10 (1993)

- *Childhood Autism*: is similar to DSM IV AD, with onset before age 3 years, although some the descriptors differ, and the usual lack of prior normal development is specified.
- *Atypical Autism*: onset after age 3 years, or criteria for number of abnormal symptoms is not met.
- *Rett's Syndrome*: corresponds to the DSM IV definition
- *Asperger's Syndrome*: corresponds to the DSM IV definition. Emphasizes frequent but not required isolated special skills.
- *Other Childhood Disintegrative Disorder*: Similar to DSM IV definition.

being fully specific to phonologic production. It is the existence of such overlaps that some investigators advance as an argument for a unitary definition of DLD.

Higher order processing disorders tend to be less salient than disorders that impoverish or distort expressive speech and are thus easily overlooked; the children may speak in full, well articulated sentences but what is amiss is semantics and, often, pragmatics. *Pragmatic disorders* are most prevalent in verbal children who are in the autistic (or Pervasive Developmental Disorder, in present parlance) spectrum and will be discussed later.

Semantic deficits at the word level are likely to be manifest as word retrieval difficulty, which may be so severe as to result in pseudo-stuttering when a child hesitates and tries repeatedly to come up with a needed word. Word-level semantic problems usually reflect an impoverished lexicon (repository of word meanings; Locke, 1994) and may be characterized by the retrieval of vague superordinate words such as "thing", or by a less specific categorical word for the sought target, e.g. "thing you sit on" for "chair". Semantic paraphasias may reflect retrieval of an incorrect token from within the same semantic field as the target, e.g. "chair" for "table". Word-level semantic problems are sometimes characterized by an atypically organized lexicon, in which case the child, who is likely to be a verbal child on the autistic spectrum, may retrieve non-prototypical exemplars of a category in a word fluency test, e.g. "penguin" rather than "robin" when attempting to come up with bird names (Dunn, Gomes & Sebastian, 1996).

Semantic deficits at the sentence level also occur, where the distinction between linguistic and cognitive deficits is particularly unclear. For example, it is difficult to know whether children who respond to a "when" or "why" question by chaining to a word in the question rather than answering the question (although they can be shown to know the answer to the question) have failed to answer correctly because they do not yet possess the linguistic device provided by that question form or because they do not understand time or causation.

Contrasts Between the Language of Children with DLD and Those on the Autistic Spectrum

Pervasive development disorder (PDD) is a behaviorally defined syndrome and not a specific "disease" with a unique etiology. It is defined in DSM IV (American Psychiatric Association, 1994) and ICD 10 (World Health Organization, 1993) as deficient development of sociability, interactive language, imaginative play, and range of interests, with frequent stereotypic repetitive activities. Both ICD 10 and DSM IV divide the PDD or autism spectrum on the basis of a defined number and distribution of items endorsed in the areas of sociability, communication and play, and repertoire of activities into Autistic Disorder (DSM IV) or Childhood Autism (ICD 10) — the classical, more severe variant, and other less severe variants such as Pervasive

Developmental Disorder-Not Otherwise Specified (PDD-NOS) in DSM IV and Atypical Autism in ICD 10, and Asperger Syndrome (Table 3). In this review, I use the term autistic to refer to the entire autistic spectrum, and Autistic Disorder to refer to classic, rigidly defined autism. Both DSM IV and ICD 10 also include Rett syndrome in the autistic spectrum, a biologically distinct very severe syndrome limited to girls (Hagberg, Aicardi, Dias & Ramos, 1983; Hagberg, 1993).

Autism and its variants are now known to be but one of the developmental disorders of brain function, with a variety of different etiologies and widely varied severity (Gillberg, 1992; Gillberg & Coleman, 1992; Rutter et al., 1984). Although intelligence is not a defining feature of the syndrome, a majority of individuals on the autistic spectrum are significantly behind in their mental development.

Severely delayed and often highly deviant language is regularly the presenting complaint of parents of preschool children on the autistic spectrum. The one exception is the subgroup *Asperger syndrome* which is defined on the basis of the acquisition of language at the expected age, normal verbal intelligence often associated with lower nonverbal rather than verbal skills, an especially narrow range of interests, and, frequently, motor clumsiness (Frith, 1991; Wing, 1991; Szatmari, Bartolucci & Bremner, 1989). In all other preschool children on the autistic spectrum, absent or delayed and deviant language is salient.

As is the case in children with DLD, autistic children present with a variety of language disorders. Allen and Rapin (1992) compared clinically-defined language subtypes among 229 children on the autistic spectrum and 262 nonautistic children with DLD they had evaluated personally. Ninety two DLD children (35%) and none of the autistic children were judged to have expressive disorders with normal/near normal comprehension. Among the remaining 399 children, 63% of the autistic and 77% of the DLD had mixed receptive/expressive disorders (including VAA), and 37% of the autistic and 23% of the DLD had higher-order processing deficits. VAA, including nonverbal or minimally verbal children with extremely poor phonology and minimal comprehension, was much more prevalent in the autistic sample (23%) than in the DLD sample (5%) (unpublished data). In another study comparing 197 autistic children without severe learning difficulties with 215 DLD children, Tuchman, Rapin and Shinnar (1991) found that 59% of 29 children with VAA were autistic and 41% dysphasic, and among 81 children with higher-order processing deficits, 77% were autistic and 23% were dysphasic. Again, there were no autistic children among the 72 children with purely expressive disorders. Very impaired comprehension and persistent lack of expressive language are associated with poor cognitive outcome in autism.

The *higher-order processing deficits* of verbal autistic children are characterized by adequate phonology and syntax but deficits at the level of semantics and pragmatics (Tager-Flusberg, 1981; 1989). Impaired pragmatics or conversational skill is the most salient characteristic of autistic communication. *Nonverbal pragmatics* include the production and interpretation of the facial expressions, body postures, gestures, and prosody (suprasegmental acoustic aspects of speech) that play a critical role in clarifying the intent of verbal communications. For example, autistic children are likely not to look at the person they are speaking to, not to use gestures to supplement speech, to speak in a monotonous voice with a choppy robotic rhythm or in a high pitched singsong that may make affirmative sentences sound like questions, and to ignore threatening facial expressions or a raised tone of voice. A telling early sign of impaired nonverbal pragmatics is failure to point to a desired object and to look up when called by name, skills most infants achieve by their first birthday.

In addition to nonverbal pragmatic deficits, the *verbal pragmatics* of autistic children are also deficient, for example initiating communication, engaging in meaningful dialogue, using language as a tool to comment or fulfil needs, providing appropriate pauses for turn-taking, staying on topic, or supplying a conversational partner with adequate information to make the communication intelligible (Bishop & Adams, 1990; Tager-Flusberg, 1989). Verbal autistic children may engage in long monologues that have no discernible communicative intent, or ignore signs of impatience from a partner tired of listening to a topic of exclusively personal interest.

Echolalia, the repeating of what has just been heard, is normal in toddlers but may persist for many years in autistic children. Children at a loss for words, who are aware that it is their turn to speak, may use echolalia as a conversational device to fill that slot. Echolalia also provides more processing time for comprehension (Prizant & Rydell, 1984). Delayed echolalia or the reciting of scripts characterizes autistic children with good verbal memories who can be taught to modify these overlearned, overused utterances and to call upon them appropriately in various contexts.

Pragmatics refers specifically to the communicative use of language and thus the impaired pragmatics of autism are widely considered to be but another aspect of deficient sociability. This view does not provide an adequate explanation for echolalia, which probably has more to do with inadequate comprehension and lexical retrieval deficits.

If pragmatics is at the interface of language and sociability, semantics is at the interface of language and cognition (Tager-Flusberg, 1992). Semantic deficits in autism encompass the organization of the lexicon (repository of word meanings) and the retrieval of words in spontaneous speech, the comprehension of verbal utterances at the sentence or idea level, and the formulation of coherent discourse. For clinical purposes, a quick way to gauge comprehension is to ask the child open-ended questions such as why, when and how. Many autistic children who can answer concrete questions appropriately and who can be shown to know the answer to an open-ended question may answer quite beside the point of the question. Their perseverative questioning about topics to which they know the answer, their veering completely off topic in the middle of a conversation, and their use of pedantic words when a simple word would do, give their speech a strong flavor

of inappropriacy and a looseness that may be mistaken for a thought disorder or psychosis.

Rapin and Allen (1983) coined the term *semantic-pragmatic deficit syndrome* for verbose preschool children with a higher order processing disorder. Although this syndrome predominates among verbal autistic preschoolers whose pragmatic deficits are salient, it is also encountered in an occasional non-autistic DLD child and in some non-autistic hydrocephalic children. At preschool, these chatter-box children initiate more verbal interactions than normal children but do so inappropriately. They may repeatedly ask questions to which they know the answer and perseverate, often with overlearned scripts. Bishop and Adams (1989) analyzed conversations of 14 8–12-year-old children with semantic–pragmatic deficit disorder, comparing them to the conversations of 20 normal children and of 43 children with other developmental language disorders. Their interpretation was that the inappropriacy of these children's language was indicative of cognitive and social limitations and comprehension failure rather than a specific linguistic deficit.

Comparison of Developmental Language Disorders with Acquired Aphasias in Childhood

Developmental language disorders (DLD) are almost never associated with focal structural brain lesions detectable with neuroimaging. Although the majority of children with DLD speak adequately by school age — which has given rise to the theory of a developmental lag rather than a disorder (Bishop & Edmundson, 1987; Locke, 1994), many of these children will come to attention a second time at school age when they will have difficulty learning to read or spell (Aram, Ekelman & Nation, 1984; Bishop & Adams, 1990). This chronicity of DLD is often contrasted with the transient nature of the acquired aphasias in children with focal brain lesions and with the lack of language disorder in many children with congenital hemiplegias, whether they reflect left or right hemispheric lesions.

In adults, dominant hemispheric lesions affecting eloquent cortical or subcortical areas result in reasonably predictable aphasia syndromes, even though representation of language in the individual brain varies (Damasio, 1989). Recovery from aphasia also varies but deficits often persist life-long. Unilateral congenital lesions in these same areas do not result in aphasia in children because of plasticity in the organization of the immature brain (Aram & Eisele, 1992). This is not to say that there is no price to pay for these early lesions: unilateral lesions are likely to retard babbling, development of communicative gestures, and the emergence of single words and syntax. This is true whether the lesion is on the left or the right, anterior or posterior (Bates, Thal & Janowsky, 1992). Both comprehension and production are delayed, with comprehension improving earlier than expression. It is only after the age of 18 months that congenital left-sided lesions have a demonstrably greater tendency than right-sided lesions to continue to delay expression. Surprisingly, in these toddlers posterior lesions delay expression more severely than anterior lesions, whereas localization does not correlate with comprehension. When there is a price to pay for the resilience of language development in the face of an early lateralized brain lesion it is likely to be in the realm of spatial cognition (Nass, Peterson & Koch, 1989).

Contrary to what earlier investigators reported, the variety of aphasic syndromes encountered in children with later-acquired unilateral brain lesions closely follow those of adults (van Hout, 1992). A major difference is that fluent receptive aphasias are distinctly less common in children, presumably because children who are still in the process of acquiring linguistic skills require feedback in order to program language. Another major difference is that prognosis for recovery of oral language is considerably better than in adults, albeit persistent deficits can jeopardize reading, writing, and the use of language as a tool for acquiring new information.

The major exception to the relatively favorable prognosis of acquired aphasia in young children is *acquired epileptic aphasia or Landau-Kleffner syndrome* (Landau & Kleffner, 1957). This syndrome refers to loss of language in a previously normally developing child, in the context of either clinical seizures or, in the absence of clinical seizures, an unequivocally paroxysmal EEG. A telling characteristic is that the severity and even the character of the language deficit may fluctuate over time (Deonna, 1991). Prognosis for disappearance of seizures is almost universally favorable, whereas it is unpredictable for language recovery, with a significant proportion of children remaining seriously impaired (Dugas, Gerard, Franc & Sagar, 1991; Bishop, 1985). Recovery may not take place for many months or even several years, that is, over a much longer period than in an acquired aphasia caused by a discrete lateralized lesion. In contrast to acquired aphasia, where a younger age generally affects prognosis favorably, exactly the opposite is the case in acquired epileptic aphasia (Bishop, 1985).

The main type of language deficit in acquired epileptic aphasia is VAA (Rapin, Mattis, Rowan & Golden, 1977). The EEG discharges are typically temporo-parietal, unilateral or bilateral, and may fluctuate from side to side. In some cases they predominate during sleep and even take on the characteristics of electrical status epilepticus in slow-wave sleep (Jayakar & Seshia, 1991). Functional imaging performed in a small number of children has shown perfusion deficits in posterior temporal cortex (Lou, 1992). Predominantly expressive disorders (Deonna, Roulet, Fontan & Marcoz, 1993) are far less common than receptive disorders and are presumed to involve anterior Rolandic cortex instead. Whether the epilepsy is responsible for the language loss, as proposed by Deonna (1991) among others, or whether both the epilepsy and the language loss are common manifestations of an underlying brain dysfunction remains undetermined.

Again, there is a confusing overlap among syndromes. Some children lose their language without epileptiform EEGs, some were not developing normally prior to language regression, and a larger number, mostly without evidence for epilepsy, lose not only their language but their social and, in some cases, their cognitive skills and become frankly autistic. Clearly, the term Landau–Kleffner is inappropriate for such children.

If the regression occurs between 1 and 3 years and the children have either pre-existing or worsening autistic symptomatology the term autistic regression is appropriate. If global regression occurs after the emergence of full sentences in a previously entirely normal child, disintegrative disorder is the term suggested by DSM IV (American Psychiatric Association, 1994) and ICD 10 (World Health Organization, 1993). Finally, an unknown but probably small proportion of children with autistic regression or disintegrative disorder have EEG abnormalities identical to those of children with the Landau-Kleffner syndrome (Rapin, 1995). Further work will be required to achieve a rational classification of these disorders.

Etiology

Past theories about the causes of DLD and other developmental disorders of brain function focused on acquired damage to the immature brain, in particular by prematurity and obstetrical misadventures, and on an unfavorable social environment. It is now well established that perinatal brain injury alone is seldom responsible for a specific developmental disability in the absence of other evidence of brain damage (Broman, Nichols, Shaughnessy & Kennedy, 1987; Rutter, Bailey, Bolton & Le Couteur, 1991). This is not to deny the importance of unfavorable environmental forces, such as malnutrition (Hertzig, Birch, Richardson & Tizard, 1972) and social deprivation (Blank & Allen, 1976), on cognitive development. The past decade has seen a dramatic shift in emphasis, brought about by awareness of the preponderant role of genetic factors in dyslexia (Cardon et al., 1994), some subtypes of DLD (Hurst, Baraitser, Auger, Graham & Norell, 1990; Lewis, 1992), autism (Folstein & Rutter, 1977; Smalley, 1991; Bailey et al., 1995), attention deficit disorder (Biederman et al., 1986), Tourette syndrome (Pauls & Leckman, 1986), and developmental disorders in general (Pennington, 1991). Outcome is determined by the combined effects of genetic and environmental influences. For example, the majority of even very premature infants who did not suffer severe ischemic or hemorrhagic brain damage, although seriously delayed initially, catch up provided they are exposed to a stimulating environment (Hack et al., 1991).

Investigations and Interventions

All of the developmental disorders, including DLD and autism, are behaviorally defined conditions. The differential diagnosis of inadequate language acquisition includes four main considerations: (1) hearing loss; (2) DLD; (3) autistic spectrum disorder; and (4) general cognitive incompetence (mental retardation). These behavioral diagnoses are not mutually exclusive, of course. Therefore and without exception, the first step in making a diagnosis is a prompt and definitive test of hearing, which may involve, besides behavioral audiometry, a physiologic test such as brain stem auditory evoked potentials (Stappels, 1989) or cochlear emissions (Decreton, Hanssens & De Sloovere, 1991). Besides testing hearing, the differential diagnosis among these four conditions is made on the basis of the history, clinical observation — in particular of language use in imaginative play with a partner — and on the basis of deficits on formal tests administered by professionals such as psychologists, neuropsychologists, and speech/language pathologists or therapists. Making one of these behavioral diagnoses does not specify their biologic causes or etiologies, which differ among children. Attempting to define etiology requires the input from a physician who will attempt to determine whether there is evidence for a known genetic or acquired syndrome, or for diffuse or focal brain dysfunction that mandates further testing. In the absence of such evidence, routine neuroimaging of the brain, tests of body fluids for rare metabolic disorders, and chromosome studies have little place unless the test is designed to substantiate a clinical hunch regarding etiologic diagnosis. Undirected laboratory testing, besides its expense, occasionally yields false positive results that may lead to further fruitless tests. The one test, besides audiometry, that is recommended in any child with a severe language disorder, especially if there is a suspicion of language regression or fluctuations in its severity or there are autistic features, is a prolonged sleep EEG to rule out unsuspected epileptiform activity. The finding of such activity in the absence of clinical seizures does not mandate the prescription of medication. Nonetheless there is a sufficient number of reports of improvement in language, and even in autistic behaviors, following the administration of spike-suppressant antiepileptic medications, ACTH, or steroids (Echenne et al., 1992; Lerman, Lerman-Sagie & Kivity, 1991; Marescaux et al., 1990) to warrant considering pharmacologic intervention, with the full realization that its results are unpredictable.

The mainstay of management of children with DLD is education. In children with predominantly expressive disorders, Whitehurst and Fischel (1994) recommend waiting until age 4 years to provide speech and language therapy because language improves spontaneously in many of the children, unless they are otherwise handicapped, in which case these authors strongly advocate admission to a preschool for language-impaired children. In my opinion this recommendation does not apply to children with verbal dyspraxia and adequate comprehension who may become extremely frustrated by their inability to make their needs and ideas known. Many of these children will invent an idiosyncratic gesture language. Because prognosis for the development of functional speech is not always favorable in verbal dyspraxia (Ferry, Hall & Hicks, 1975), I recommend intensive education which includes the provision of communication boards or the teaching of formal sign language in a preschool where it is the vernacular. It is not the case that learning sign language will retard the development of speech, quite the contrary.

Children with severe receptive/expressive disorders, especially those with VAA or autistic behaviors, require early admission to a specialized preschool for language-impaired children (Allen, Mendelson & Rapin, 1989). This setting provides intensive training in conversational language in a naturalistic setting in addition to individualized speech and language therapy. It also

fosters the development of sociability and play skills with peers. The provision of language by eye, including gestures, sign language, communication boards and computers, and reading are even more essential for children with severe comprehension programs than for those with verbal dyspraxia. Superior programs provide parent training and support which greatly amplifies the effectiveness of the intervention.

Behavior altering medication plays a minor role in the management of preschool children with developmental disorders. The simultaneous prescription of multiple drugs is discouraged. If any medication is recommended, it must be targeted at a specific behavior problem such as attention deficit, impulsivity, uncontrollable aggression, and the like, or at seizures. Indications for antiepileptic medications have already been discussed. If attention deficits are severe, a trial of stimulants (methylphenidate, dextroamphetamine, or pemoline) may be helpful, although these medications are not useful for the deficient joint attention of children on the autistic spectrum. Pharmacologic intervention should be considered an interim measure that provides the time for behavioral intervention and retraining to gain effectiveness. Long-term administration of psychotropic medications, especially those like the phenothiazines and butyrophenones that may, in some children, produce potentially irreversible side-effects, must be strenuously avoided.

The goal of early intervention is to maximize the rate of language learning and use at the optimal language-learning period of childhood, to minimize secondary behavioral consequences of inadequate communication, and to educate parents regarding effective and ineffective therapies. Prognosis for the acquisition of serviceable speech by school age or earlier is good, except in children with VAA, verbal dyspraxia, and in persistently nonverbal autistic children. In these severely affected children, prognosis is less predictable and a proportion of them may remain significantly impaired life-long. As is true of all the learning disabilities, acquisition of a previously deficient ability does not imply recovery; it implies compensation. Consequently one needs to anticipate potential later difficulties with the acquisition of written language. In the face of adequate overall intelligence, the goal of intervention is to make sure that these circumscribed cognitive deficits do not preclude an educational opportunity commensurate with the child's aptitudes.

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