

Five Subtypes of Developmental Phonological Disorders

Lawrence D. Shriberg, PhD

*Department of Communicative Disorders and
The Waisman Center on Mental Retardation and Human Development
University of Wisconsin—Madison
Madison, WI*

Evidence for subgroups among developmental phonological disorders based on etiological origins, as opposed to the alternative linguistic view of childhood phonological disorders as a learning problem of unknown origin, is presented. A three-parameter framework that arranges relevant individual differences (mechanism, cognitive-linguistic, and psychosocial) is proposed for study and classification of the phonological disorders. The disorders are studied in relation to developmental correlates and speech-sound normalization. This permits study of these variables in relation to severity and forms of expression and interaction. Subgroups of "speech delay," each of which reflects phonological consequences, are suggested. Residual articulation errors are classified based on causal origins, possibly influenced by the developmental context in which precise articulation is an acquired skill and differentiated from genetic origins posited for speech delays and those related to specific hearing, motor-speech, and psychosocial origins. Exploration of causal origins provides advantages for both assessment and intervention as well as prevention.

Keywords: Phonological disorders; subtypes; causal origins; genetic and environmental factors

A developmental phonological disorder is a clinically notable difference in the development of articulate speech that cannot be explained by significant deficits in a child's speech-hearing mechanism, cognitive-linguistic function, or psychosocial processes (Shriberg, 1980). For several years, my colleagues and I at the University of Wisconsin—Madison have been interested in the possibility of documenting distinct causal origins for developmental phonological disorders. Emerging evidence supports the likelihood of five subtypes, with causal origins for each subtype suggesting both genetic and environmental factors.

The goals of this article are (1) to present a conceptual alternative to the monolithic view of a childhood phonological disorder as a learning problem of unknown origin (Berntal and Bankson, 1993; Milisen, 1954; Powers, 1971; Van Riper, 1939; Winitz, 1977; Winitz and Darley, 1980), and (2) to illustrate the types of evi-

dence that support the probability of five etio- logically distinct subtypes. Forthcoming reports will provide specific evidence for the five sub- groups and detailed descriptions of speech and prosody-voice findings.

A CLASSIFICATION FRAMEWORK FOR DEVELOPMENTAL PHONOLOGICAL DISORDERS

A Three-parameter Framework

Figure 1 is a three-parameter framework that arranges the relevant individual differences in developmental phonological disorders for study and classification. Factors on each axis of the frame- work are first described. Then, the framework is used to profile associations and dissociations within and among five putative subtypes.

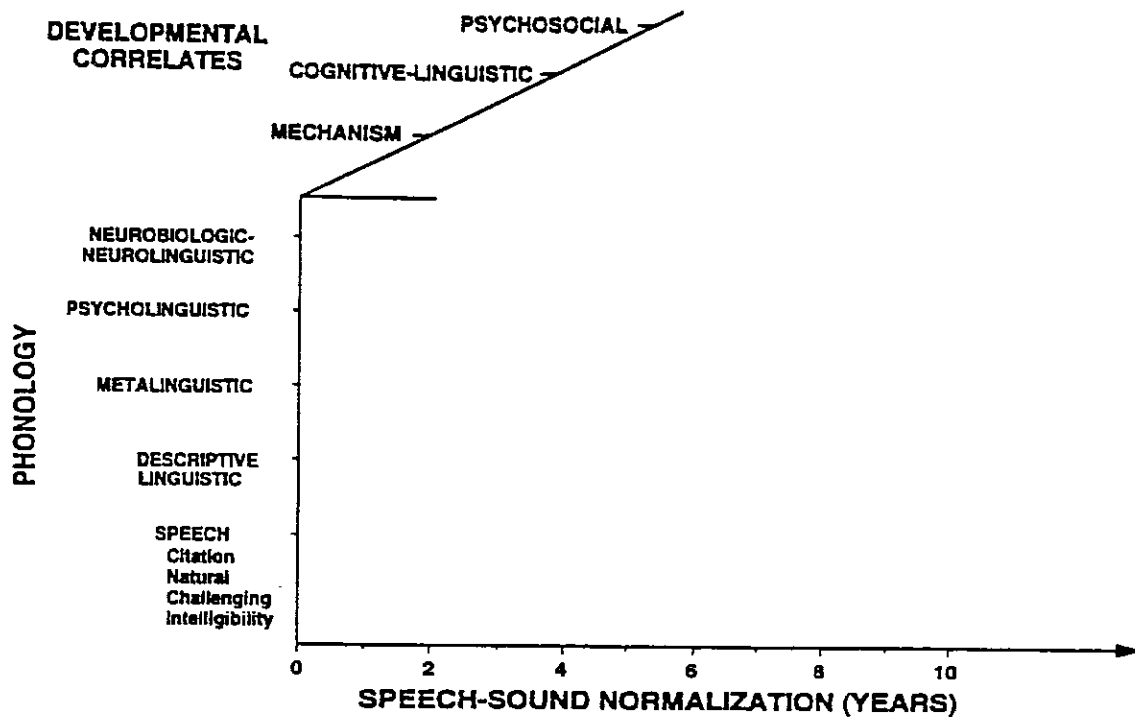


Figure 1. Three-parameter framework for etiologic classification of developmental phonological disorders.

The parameter termed *phonology* in Figure 1 divides normal and disordered phonological processing into five levels:

1. The level of phonology closest to the products of gene regulation reflects neurobiologic and neurolinguistic substrates. Phonological processing activity at this level is assessed using a variety of neuroimaging and other technologies (e.g., Plante, Swisher, and Vance, 1989, 1991).
2. At the next level of phonologically relevant behavior are psycholinguistic processes, such as those reflected in measures of temporal processing of stimuli with varying phonological attributes (e.g., Tallal, Sainburg, and Jernigan, 1991).
3. The third phonological level is metalinguistic information, such as data derived from tasks that assess a speaker's awareness of speech sounds and ability to segment words into constituent syllables (e.g., Ball, 1993; Magnusson, 1991).
4. Fourth is the descriptive linguistic level, which models the organization of phonology using analytic units that relate segmental and suprasegmental processes to one another and to other language domains. During the past two decades, many descriptive linguistic procedures used to describe a speaker's comprehension and production status have been developed specifically for children and adults with phonological disorders (cf. Bernthal and Bankson, 1993; Kohn, 1988). Each of these phonological analyses invokes alternative basic units, including those associated with structuralist phonology, generative phonology, natural phonology, and, most recently, nonlinear phonology.
5. The fifth level of phonological behavior represented in Figure 1 is manifest speech. Several types of manifest speech can be used as the corpus for one of the four levels of inferential analyses above, or may constitute an alternative phonological construct. The four types of

speech samples included in Figure 1 are (1) citation forms or decontextualized speech, such as phonological data obtained from articulation tests and word lists; (2) natural speech, such as measures of the distribution and precision of speech in conversational speech samples; (3) challenging speech, such as responses obtained from rapid syllable repetition tasks and tongue twisters; and (4) intelligibility indices and handicap scales, which are the most distal measures of phonology relative to genotype, but closest to communication in the ambient environment.

The axis in Figure 1 termed *developmental correlates* provides a parameter to account for a speaker's current status and history on all potential developmental correlates of the primary disorder. Case history data and assessment protocol results are organized into three domains, termed speech-hearing mechanism, cognitive-linguistic processing, and psychosocial function (cf. Shriberg, 1982; Shriberg and Kwiatkowski, in review; Shriberg et al., 1986). For each of these three domains, it has been useful to trichotomize a speaker's status on each of several hundred developmental variables as within the normal range, questionable, or not within the normal range.

The axis termed *speech-sound normalization* reflects the temporal or life-span parameter of a developmental phonological disorder. As developed presently, classification of disorders can include consideration of age of onset and/or age of normalization, each of which may reflect information about regulatory processes in both genetic and nongenetic domains.

To summarize, the three parameters in Figure 1 provide a means to study and classify variables associated with developmental phonological disorders. The severity and form of expression of each variable within the five levels on the phonology parameter can interact in complex ways with variables in any one or more of the three domains on the developmental correlates parameter during each period of phonological development at each age on the speech-sound normalization parameter.

Alternative Classification Approaches

Historical review of the many ways that developmental phonological disorders have been classified in the United States since the 1930s reveals a diversity of typological and nosological approaches (cf. Shriberg, 1982). With reference to the complexities created by the variables on the three axes in Figure 1, it is useful to contrast the etiologic classification system to be described with two other approaches to classification in communicative disorders.

Descriptive linguistic typologies. The most common approach to classification of developmental phonological disorders has been to focus only on the phonology parameter, ignoring causal and/or maintaining factors in developmental correlates and ignoring onset/outcome differences in speech-sound normalization. Particularly during the past two decades, typologies based solely on alternative phonological descriptions have been viewed as sufficient for research and clinical goals. It is important to note that in descriptive linguistic typologies it has been necessary to ignore the phonetic-level data of clinical distortion errors because these linguistic behaviors are not well-handled by abstract phonological units such as distinctive features or phonological processes. Therefore, despite claims made for the descriptive adequacy of linguistic typologies, including emerging approaches based on nonlinear phonological theories, typologies based only on phonological data lack real-world correspondence to the clinical presentation of children with developmental speech disorders. Particularly from the perspective of potential genetic antecedents, where gene regulation issues require a thorough profile of speech development as a biobehavioral trait, classifications based solely on descriptive linguistics may be inadequate for an eventual explanation of the onset and normalization of disorders.

Quantitative-multivariate typologies. *Quantitative-multivariate* classification—a method that does attempt to account for all clinical information—is a second classification approach in developmental phonological disorders (Wilson and Risucci, 1986). Quantitative-multivariate analysis yields variable clusters or factor structures, which in turn are used to suggest classification categories

for applied use. Of the few reported multivariate studies that have attempted to analyze associations among case history and speech factors, none have provided the foundation for a classification system (cf. Shriberg, 1982).

Clinical-inferential typologies. The third classification method, termed *clinical-inferential* classification by Wilson and Risucci, is viewed as the most appropriate approach for etiological classification of developmental phonological disorders. Clinical-based classification systems are those in which categories are derived directly from the experience of service delivery providers. In phonological disorders, examples arising from clinical-inferential experience include such classificatory categories as children with "single-sound errors," "multiple articulation errors," "a persistent de-rhoticized /r/," and so forth. These might not have the formal elegance of categories proposed in descriptive linguistic typologies, or those generated from multivariate analyses, but they do reflect information on all three of the parameters in Figure 1. That is, clinical descriptions such as "a 9-year-old child with a lateral lisp" or "a 12-year-old child with persistent articulation errors associated with motor-speech deficits" convey the central phonological information about the disorder, the causal inference, normalization history, and current status. The following discussion posits five such clinical-inferential classifications and provides an overview of the kinds of evidence used to support each subgroup.

FIVE SUBTYPES OF DEVELOPMENTAL PHONOLOGICAL DISORDERS

Speech Delay (SD)

The fundamental form of a developmental phonological disorder is termed *speech delay* (Shriberg, 1980, 1993; cf. Shriberg, Kwiatkowski, and Gruber, in press, for discussion and empirical support for the concept of *delay*). Figure 2 is a representation of SD using the schema discussed in Figure 1. On the developmental correlates axis a "-" sign is entered under cognitive-linguistic involvement, with "*" signs indicating normal speech-hearing mechanism and psychosocial processes. As dis-

cussed below, this clinical-inferential description suggests that, for all children with SD, either transient or persistent involvement of cognitive-linguistic function is the central feature of the disorder. The degree of cognitive-linguistic deficits is, of course, not so severe as to warrant classificatory terms such as cognitive disability or profound linguistic impairment. As will be apparent in subsequent discussion, the characteristic that distinguishes children with SD from the other subgroups is that children with SD do not have clinical involvement of the speech-hearing mechanism or psychosocial processes.

In Figure 2, trends reflecting speech-sound normalization on the phonology axis are based on a metric of articulatory accuracy in conversational speech termed the Articulation Competence Index (ACI) (Shriberg, 1993). The ACI is a distortion-adjusted tally of the percentage of consonant sounds articulated correctly. Essentially, among speakers with similar percentages of consonants correct, those with proportionally fewer distortion errors and more deletion or substitution errors score lower on the ACI than those with proportionally more distortion errors. Note that the ACI metric is based on natural speech output, rather than on any of the other levels of phonological processing or forms of manifest speech represented on the phonology axis in Figure 1. The solid line segments of the ACI trends are based on data obtained in recent studies (Shriberg and Kwiatkowski, in press; Shriberg, Gruber, and Kwiatkowski, in press; Shriberg, Kwiatkowski, and Gruber, in press); the dashed lines are linear interpolations.

As shown by the upper trend, labeled *normal acquisition*, children acquiring speech normally average approximately 75% articulate speech on the ACI by 3 years of age, moving upward at approximately 5% per year until reaching 100% at approximately 8½ years. In contrast, the bifurcated trend for children with SD indicates two normalization histories: *short-term speech-sound normalization*, which is the acquisition of developmentally appropriate speech by 6 years of age; and *long-term speech-sound normalization*, in which normalization occurs anytime after 6 years (for most SD children, by approximately 9 years). Finally, as

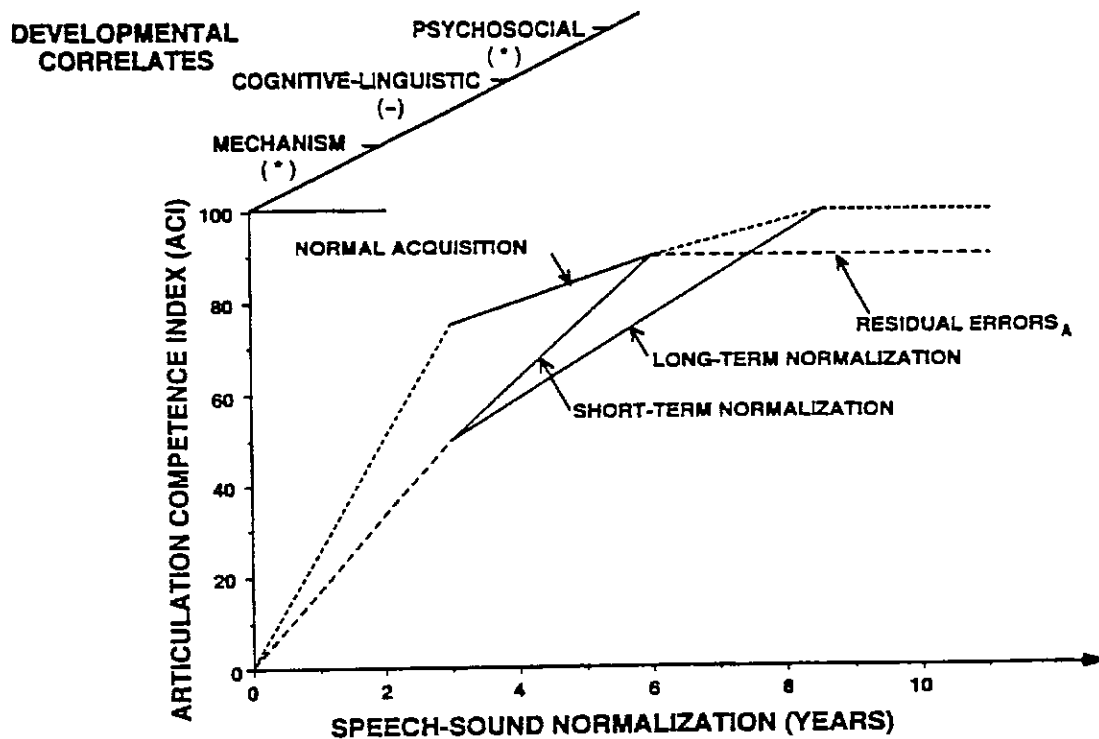


Figure 2. Three-parameter profile for speech delay (SD).

indicated by additional bifurcations in the two trends for children with SD, some children will retain clinically notable distortion errors, indicated in Figure 2 as *Residual Errors_A*. Later, such errors will be contrasted with those termed *Residual Errors_B*, with the latter designation applied to children with speech-sound distortion errors who do not have histories of SD.

Table 1 provides information on nine variables that contrast the five types of developmental phonological disorders to be discussed in sequence in this article. Some of the entries are based on records for approximately 350 children who received services in a university phonology clinic; additional information is taken from children recruited for a variety of studies (cf. Shriberg and Kwiatkowski, in press). Differences among the entries for each subgroup in Table 1 (no two columns reflect the same data for each row) support the hypothesis that the five subgroups have different etiologies.

Beginning with the first row, approximately 60% of the preschool children seen in our university phonology clinic meet the inclusionary and exclusionary criteria for SD. The primary inclusionary criterion for SD is an intelligibility problem that includes deletion and substitution errors that are not appropriate for a child's age (Shriberg, 1993). An additional criterion is that the phonological disorder not be associated with a significant clinical entity such as hearing loss, craniofacial dysmorphism or dysfunction, cognitive disability, or emotional disturbance. Based on a conservative estimate that the prevalence for all developmental phonological disorders in preschool children is 2.5% (Leske, 1981), the 60% sample estimate for SD suggests that the population prevalence for this subtype is 1.5%, or 1-2 children per hundred. Such epidemiologic information is central to behavioral genetics procedures in which estimates of a speaker's liability for a disorder are used to calculate the likelihood of familial aggregation.

Table 1. Descriptive data for five subtypes of developmental phonological disorders.

Variable	Speech Delay + (SD +)				
	Speech Delay (SD)	Speech Delay + Otitis Media with Effusion (SD + OME)	Speech Delay + Developmental Apraxia of Speech (SD + DAS)	Speech Delay + Developmental Psychosocial Involvement (SD + DPI)	Residual Errors (RE)
Prevalance					
Clinical sample ^a	60%	30%	3-5%	5-7%	—
Population estimate ^b	1-2/100	7-8/1000	1-2/1000	1-2/1000	5/100
Gender	75% boys	?	80% boys	75% boys	equal?
Language					
Comprehension delay	10-40%	?	yes?	?	0%
Production delay	50-75%	?	yes?	?	0%
Speech-sound Normalization					
Short-term	75%	?	0%?	?	0%
Long-term	25%	?	100%?	?	100%
Retain RE _A ^c	?	?	yes?	?	—
Nuclear Family Aggregation	24-46%	?	yes?	?	?

^aBased on approximately 350 primarily preschool children referred for intelligibility problems of unknown origin. Older children with RE were seen only in special circumstances.

^bBased on an estimated prevalence of 2.5% for SD+ and 5% for RE (see text).

^cSee text for explanation of this term.

A second statistic central to classification issues is the gender ratio observed in children with SD. As shown in Table 1, ratios calculated from several studies range from 2:1 to 3:1, averaging 2.75 boys with SD for every 1 girl (Shriberg and Kwiatkowski, in press). As discussed below, this consistent finding contrasts SD and the other proposed subgroups of developmental phonological disorders.

A third set of descriptive findings indicated in Table 1 concerns the language status of children with SD when they are first evaluated in a clinical setting. Approximately 10-40% have language comprehension problems and approximately 50-75% have language production problems (Shriberg and Kwiatkowski, in press). These findings provide the primary support for the hypothesis of at least transient cognitive-linguistic involvement in children with SD, as indicated on the developmental correlates axis in Figure 2. The apparent

absence of language involvements in 25-50% of children with SD could reflect several methodological factors, including the sensitivity of measures to subtle language involvements and age of assessment. Specifically, based on findings reported by several research groups studying "late talkers," approximately 50% of 3- to 5-year-old children may have had earlier, undocumented language deficits that normalized by the time they were referred for speech evaluation (cf. Paul, 1991).

A fourth set of statistics show that approximately 75% of preschool children seen for intervention achieve short-term speech-sound normalization (Shriberg and Kwiatkowski, in process). Thus, by 6 years of age the majority of children who receive diverse forms of speech intervention achieve a level of speech-sound accuracy that no longer qualifies them as having SD. By definition, however, most still have speech-sound errors considered to be within the normal range for their age

(as described in the next section). Note that this figure for normalization is generally consistent with the information in Table 1 on the percentage of speech-delayed children with language comprehension involvements. Specifically, all follow-up studies have reported that children with language comprehension involvement normalize later than children without comprehension involvement. Thus, the remaining children who have long-term speech-sound normalization (i.e., typically by 9 years), constitute 25% of the sample, a figure that falls exactly in the middle of the 10–40% estimated prevalence of speech-delayed children who also have delays in language comprehension.

Finally, because we lack appropriate longitudinal data, it is not possible to estimate the percentages of children with either short-term or long-term speech-sound normalization who retain residual articulation errors (Residual Errors_A). The computer-based analysis that makes the classification is based on a narrowly transcribed sample of conversational speech (cf. *Speech Disorders Classification System* in Shriberg, 1993) and has not been used with a sufficient number of children older than 6 years. A study in progress will provide such information based on continuous speech samples from 3- to 60-year-old speakers in 60 families (Shriberg and Lewis, in process).

Causal origins. Recent findings from several research groups suggest that the traditional concept of developmental phonological disorders as functional disorders of unknown origin may be incorrect. Several independent studies support familial aggregation, which means that the origins could be found in a common genotype or common environment (cf. Lewis, Cox, and Byard, 1993), most likely the former, considering the weight of the evidence for genetic factors in a variety of other childhood areas (e.g., reading disability, psychological problems). Descriptive data such as those in Table 1 will play a central role in an eventual understanding of the mode of genetic transmission of speech-language delay (Plomin, 1990; Smith, 1992).

As shown in the fifth variable in Table 1, estimates of the aggregation of speech disorders in families are in the range of 24–46% (Lewis, Cox, and Byard, 1993). Most studies rely solely on pa-

rental reports of speech disorders that could be associated with a bias toward over-reporting speech delay. Nevertheless, these prevalence rates in siblings and parents of affected children are significantly higher than the 2.5% prevalence data for preschool children discussed above. Our parental report data suggest that as many as 60% of children with SD have one or more nuclear family members who have or have had a speech disorder (Shriberg and Kwiatkowski, in press).

Alternative phenotypes for SD. The hypothesis of genetic transmission of developmental phonological disorders makes three alternative claims about the form of the phenotype. In curricula and professional affairs within the discipline of communicative disorders, a developmental phonological delay is treated as an autonomous clinical disorder. In research and clinical contexts, however, phonology is modeled within language production processes (e.g., Bock, 1991). Therefore, in phenotype characterization, relationships among speech, language, and cognitive processes must be addressed specifically. Is the appropriate phenotype for the SD subtype of a developmental phonological disorder best modeled as a *general developmental delay*, a *specific speech-language delay*, or a *specific speech delay*?

A researcher's position on the anticipated behavioral phenotype for a genetically transmitted disorder dictates the inclusionary and exclusionary criteria used to identify affected persons (the index case and affected relatives). In behavioral genetics parlance, a general developmental delay could be viewed as the "broad form" of SD; alternatively, the genotype could be envisaged as coding more narrowly for a specific speech-language delay; or the genotype could code only for a specific speech delay. If phonology is viewed as one parameter within a larger domain termed a *verbal trait*, then all persons with speech, language, spelling, and/or reading deficits can be classified as having alternative levels of involvement for this trait. In contrast, if phonology is more narrowly defined by its acoustic-articulatory requisites and products, then its genetic antecedents can be envisioned as significantly different from those for language.

Note also that, within the phonology parameter itself, several alternative views on the phenotype

are possible. The verbal trait perspective could classify persons with lowered performance on speech-sound awareness tasks as having a phonological deficit, even if there were no manifest speech errors. Moreover, a verbal trait perspective would classify persons with only single-sound distortion errors (e.g., a dental lisp) as evidencing the "narrow form" of the deficit.

The major problem with the general developmental delay phenotype is lack of evidence indicating that children with SD have general developmental delays. Case histories and performance profiles do indicate a variety of questionable developmental correlates, but most children with even significant speech involvement seem to be well within the normal range on all other developmental correlates (Shriberg and Kwiatkowski, in press).

There is also ample evidence against the specific speech delay hypothesis. Three findings are relevant: most significant, as indicated in Table 1, is the high percentage of children with speech delay who also have language involvements. A second finding inconsistent with the specific speech delay hypothesis is that speech profiles for speech-delayed children both with and without current language delays appear to be similar (Shriberg and Kwiatkowski, in press). Although it is possible that children with language production and particularly language comprehension involvement have more severe delays and therefore more speech-sound deletions, their overall error pattern is not qualitatively different from the error patterns of speech-delayed children without concurrent language involvement. A third finding provides counter-evidence for the specific speech delay phenotype: many studies report high comorbidity for fluency and voice disorders among preschool children identified as having a developmental phonological disorder (see reviews in Ruscello, St. Louis, and Mason, 1991; St. Louis, Hansen, Buch, and Oliver, 1992; Wolk, Conture, and Edwards, 1990).

These several considerations suggest that neither a general developmental delay nor a specific speech delay is the appropriate perspective for the phenotype associated with the subgroup of children termed speech delay. Rather, the hypothesis most

consistent with data on children who meet inclusionary and exclusionary criteria for this subtype is that the appropriate phenotype for speech delay is a specific speech-language delay. As discussed later, a nongenetic causal explanation posited for the development of RE_B also eliminates this subgroup from consideration as meeting phenotype criteria for specific speech delay.

Speech Delay + (SD +)

The class of developmental phonological disorders termed *speech delay +* (SD +) includes three subtypes that appear to have causal origins that differ from the presumed genetic origins for SD and that differ from one another. The decision to aggregate these subtypes under the cover term *speech delay +* denotes one or more "additional" factors observed in the speech-language profiles of certain children who otherwise qualify for SD. As shown in Figure 3, the deficit is indicated in the developmental correlates data, where, in addition to involvement in cognitive-linguistic processes, there is some specific historical or current information documenting involvement of the speech-hearing mechanism and/or psychosocial processes. As in cognitive-linguistic involvement, the "-" sign does not suggest that such differences are so significant as to merit a clinical diagnostic category such as dysarthria or autism. Rather, children with *speech delay +* (i.e., *speech delay +* "plus" a deficit, indicated by a "-" sign) do not fall within the normal age range on criterial measures in these domains. The following sections summarize emerging information on three subtypes of developmental phonological disorders subsumed by the classification SD +.

Speech Delay Associated with Otitis Media with Effusion (SD + OME)

In addition to meeting the inclusionary criteria for SD described above, children with *speech delay +* and otitis media with effusion (SD + OME) must have at least six episodes of recurrent otitis media with effusion in the first three years of life. Typically, in case history summaries, most of these children also have ample additional evidence indicating fluctuant hearing loss (cf. Shriberg and

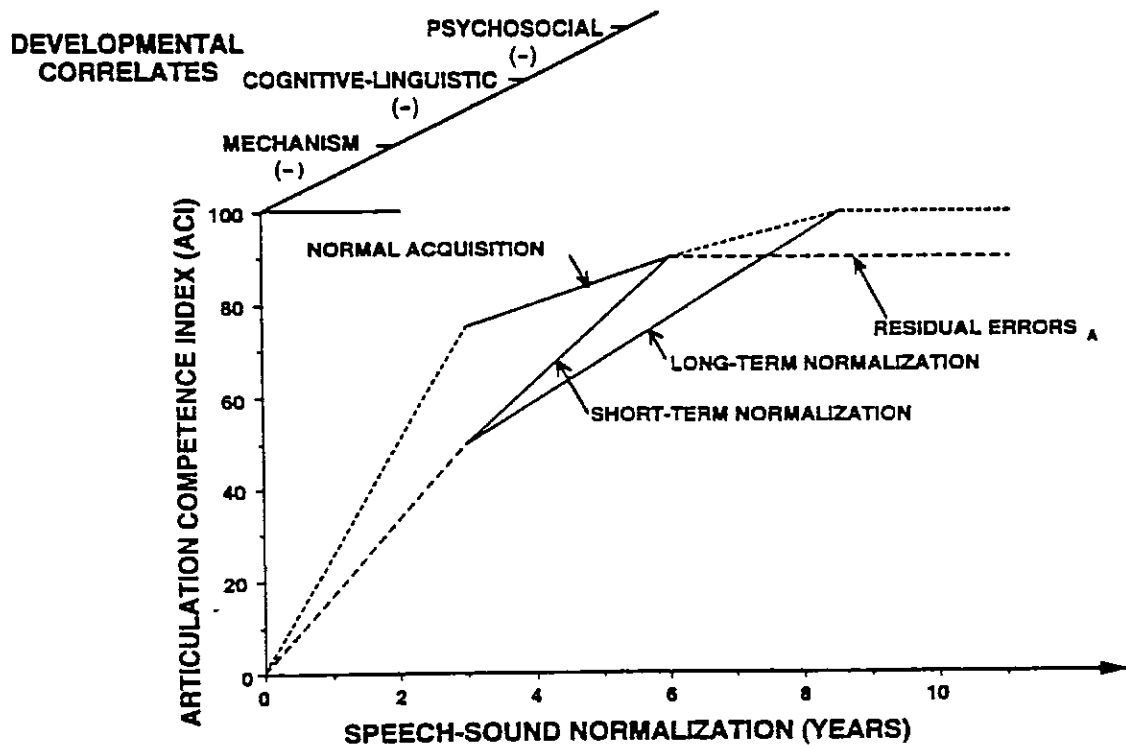


Figure 3. Three-parameter profile for SD+.

Kwiatkowski, in press). Exclusionary criteria for SD+OME are similar to that for SD; that is, there are no other primary involvements.

Two hypotheses about the effects of early recurrent otitis media with effusion on speech development have been pursued in research studies. The weak hypothesis asserts that the fluctuant hearing loss and all other factors associated with certain types of middle ear disease provide sufficient cause for delay in the onset and/or rate of subsequent speech development. The strong hypothesis asserts that not only does the fluctuant hearing loss and other factors cause developmental delay in rate and/or onset, but also these recurrent problems underlie a unique profile of phonological errors that is pathognomic (i.e., distinctly characteristic) of children with SD+OME. Although validation of either claim would be sufficient to establish otitis media as a causal subgroup, implications for assessment and intervention differ sharply depending upon which of the two hypotheses is correct.

Our own position on these two hypotheses is that children with speech disorders owing to early recurrent otitis media with effusion do have a specific speech-error profile reflecting the phonological consequences of the ages at which they experienced fluctuant hearing loss (Shriberg, 1987; Shriberg and Smith, 1983). As noted, however, it is beyond the scope of this article to describe the speech and prosody-voice profiles that discriminate SD+OME from SD profiles and profiles for each of the other two SD+ subtypes. A study in progress attempts to cross-validate prior findings and assess whether children with SD+OME have different short-term and long-term speech-sound normalization histories than do children with SD or other forms of SD+ (Shriberg and Friel-Patti, in process).

We estimate that preschool children with SD+OME constitute approximately 30% of referrals for intelligibility problems of unknown origin (see Table 1). Of all preschool children 2.5% have developmental phonological disorders; therefore,

30% indicates that 0.75, or 7-8 children per thousand, have speech sequelae associated with early recurrent otitis media with effusion. It is important to underscore the many demographic limitations and potential ascertainment biases affecting estimates based on clinical referral samples. Our prevalence estimate for SD + OME might be considered low relative to the number of children who experience recurrent middle ear problems in the first three years of life. We believe, however, given the many ways in which observant caregivers compensate for the communicative effects of fluctuant hearing loss and the efficacy of aggressive medical regimens, that this estimate from a community with access to excellent health care is not unduly conservative.

Causal origins. As indicated by the question marks for language status, speech-sound normalization, and familial aggregation in Table 1, the relevant descriptive and causal origin data for SD + OME are not yet available. Although the speech findings for children with histories of early recurrent otitis media with effusion are reviewed in several places (e.g., Friel-Patti, 1990; Hasenstab, 1989; Lonigan, Fischel, Whitehurst, Arnold, and Valdez-Menchaca, 1992; Roberts, Burchinal, Hoch, Footo, and Henderson, 1988), studies to date have not provided the type and specificity of information proposed as relevant in the present article. A large-scale study in progress should yield valid population statistics for the occurrence of speech-language effects as sequelae of early recurrent otitis media with effusion (Paradise et al., 1993).

One available statistic, that plays a large role in etiologic modeling, is gender. In comparison to the approximately 3:1 ratio of boys to girls for SD, gender ratios for children with SD + OME may not be nearly as high. Klein (1986) notes that in most epidemiologic studies the incidence of acute episodes of otitis media in boys is not significantly different from that in girls, although maleness was found to be a significant risk factor in the Greater Boston Otitis Media Study (Teele, Klein, Rosner, and the Greater Boston Otitis Media Study Group, 1989). The prospective study by Paradise and colleagues cited above should provide the phonological data needed to determine per-age gender ratios for SD + OME. Such data may turn out to be

crucial to the hypothesis of SD + OME as an etiologic subgroup of developmental phonological disorders.

Speech Delay Associated with Developmental Apraxia of Speech (SD + DAS)

A persisting clinical-inferential conclusion for clinicians who work with young children with SD is that a small number have some type of associated motor-speech problem. In addition to having developmental errors similar to those made by children with SD, these children seem to have problems selecting and sequencing speech sounds and different prosody patterns. Such error patterns resemble those of adults with acquired apraxia of speech. Recent literature reviews of this historically controversial category of speech disorders, including terminological issues associated with *verbal apraxia* versus *apraxia of speech*, are provided by Crary (1993); Hall, Jordan, and Robin (1993); Marquardt and Sussman (1991); Stackhouse (1992); and Velleman and Strand (in press).

Our inclusionary criteria for children with speech delay + developmental apraxia of speech (SD + DAS) are based on these children's deviant prosody-voice characteristics in conversational speech. As illustrated by data from a study of 14 such children compared to data for children with SD (cf. Shriberg, 1993, Figure 12), a primary difference in these children's communicative profile is posited to be in their difficulties with lexical and sentential stress.

Notwithstanding the significant divergence of opinion on the nosology for and validity of this clinical classification, several findings shown in Table 1 support its candidacy as a subtype. Our clinical research data suggest a 3-5% prevalence of SD + DAS among children referred for developmental phonological disorders. Using the 2.5% figure for developmental phonological disorders and the maximum 5% sample estimate, population prevalence is thus estimated to be 0.125%, or 1-2 preschool children per thousand.

Six of the seven remaining entries for SD + DAS in Table 1 are qualified by question marks. Although there are several dozen clinical reports in the literature, ascertainment (i.e., sampling) issues

and other methodological limitations warrant a conservative perspective on each variable.

Gender ratios for SD + DAS are similar to gender ratios for SD, averaging approximately 80% boys. Children with SD + DAS have been reported to have both language comprehension and language production delays, providing a primary impetus for the use of the alternative term *developmental verbal dyspraxia*.

The speech-sound normalization entries in Table 1 are viewed as important for the discussion of causal origins below. Compared to the estimated 75% of SD children who experience short-term speech-sound normalization, all children with SD + DAS are reported to have long-term speech-sound normalization (100%). Lack of progress in intervention is, of course, a prime inclusionary criterion for SD + DAS, although it has not generally been a sufficient criterion. Rather, it is the perceived difference in the quality of a child's speech that warrants clinical identification of SD + DAS, characterized by a profile of uncommon speech errors and prosody-voice differences that will be described in a forthcoming report.

A salient difference between SD + DAS and each of the other four subtypes described in Table 1 is in the temporal dimension, including speech-sound onset and/or normalization histories. Children with SD + DAS can retain into adulthood errors of speech precision that are more extensive than the residual errors of children with histories of speech delay (RE_A), such as later acquisition of adult-like voice onset times. Finally, as indicated in Table 1, findings from several studies suggest that SD + DAS aggregates in families.

Causal origins. The primary feature of both causal and clinical interest in SD + DAS is the negative history of short-term speech-sound normalization. In comparison with the estimated 75% rate of short-term normalization for children with SD, these normalization findings might be interpreted as suggesting significant biological origins different from those associated with the essentially temporary delay seen in SD. Although the family aggregation data strongly suggest genetic transmission, whether the involvement in SD + DAS is genetically transmitted or is associated with non-

genetically based deficits in the developmental neurobiology of speech-language remains a question for research. There is much work to do in first identifying the inclusionary criteria that are pathognomic for this classification before behavioral and molecular genetic studies can proceed. Like other disorders studied internationally, SD + DAS will require collaborative research efforts in order to assemble appropriately sized databases for programmatic study (e.g., see Epstein et al., 1991).

Speech Delay Associated with Developmental Psychosocial Involvement (SD + DPI)

The third subtype within the general class of developmental phonological disorders termed SD + includes children whose speech delays appear to be causally associated and maintained by developmental psychosocial involvements (SD + DPI). Among the five subtypes delineated here, SD + DPI is perhaps the most speculative as a distinct clinical entity. Psychosocial contexts influence normal speech-language development, and speech delays of all types have psychosocial consequences. For certain children, however, the clinical-inferential proposal denoted by the term SD + DPI is that developmental psychosocial issues are sufficient to cause speech delay. Similar to the other two groups of children with SD +, the strong claim is that these issues result in specific speech and prosody-voice errors that are distinct from those that make up the core speech delay. Inclusionary information to identify such children is taken from concurrent case history information and social work reports, as well as concurrent or follow-up information on the types of exceptional educational services that were provided in the schools. This classification is supported only by clinical experience; there are few research findings, and they are only suggestive (cf. Baker and Cantwell, 1982, 1987; Baker, Cantwell, and Matison, 1980; Beitchman, Hood, Rochon, Peterson, Mantini and Majumdar, 1989; Beitchman, Nair, Clegg, Ferguson and Patel, 1986).

As indicated in Table 1, we estimate that 5-7% of the children seen at our clinic meet case history

criteria for SD+DPI, yielding a population prevalence of 1-2 children per thousand. Gender ratios based on just our clinical sample are estimated at approximately 3:1, or 75% boys. The question marks in the remaining rows in Table 1 indicate that, like SD+OME, the data on language, speech-sound, and family aggregation are insufficient to warrant estimates for these variables. SD+DPI is proposed as a transient disorder reflecting a response to environmental stress; a forthcoming report will provide information on speech and prosody-voice characteristics.

Residual Articulation Errors (RE)

Figure 4 is the three-parameter description of the fifth subtype of developmental phonological disorders, termed residual errors (RE). As described, subscript qualifiers are used to indicate which persons with RE met criteria for SD or SD+ at

some earlier point in their lives (RE_A) and which had only the developmental form portrayed in Figure 4 (RE_B). The inclusionary criterion for RE_B is the retention of at least one articulation distortion error that is questionable at 6 years of age and persists past 9 years of age. In addition to the exclusionary speech criteria for RE_B (i.e., no history of SD or SD+), these speakers have normal histories for variables in each of the three developmental correlates domains, as indicated by the "*" symbols in Figure 4.

In the present pluralist culture, exactly which types of speech-sound distortions constitute a clinically relevant speech "error" is a matter of social consensus. There is consensus within our discipline that sibilant fricative (e.g., dental and lateral /s/) and liquid distortions (e.g., de-rhoticized /r/ and rhotic vowels) are speech errors; such behaviors are sometimes depicted in presumably humorous characterizations in the media (e.g., in cartoon

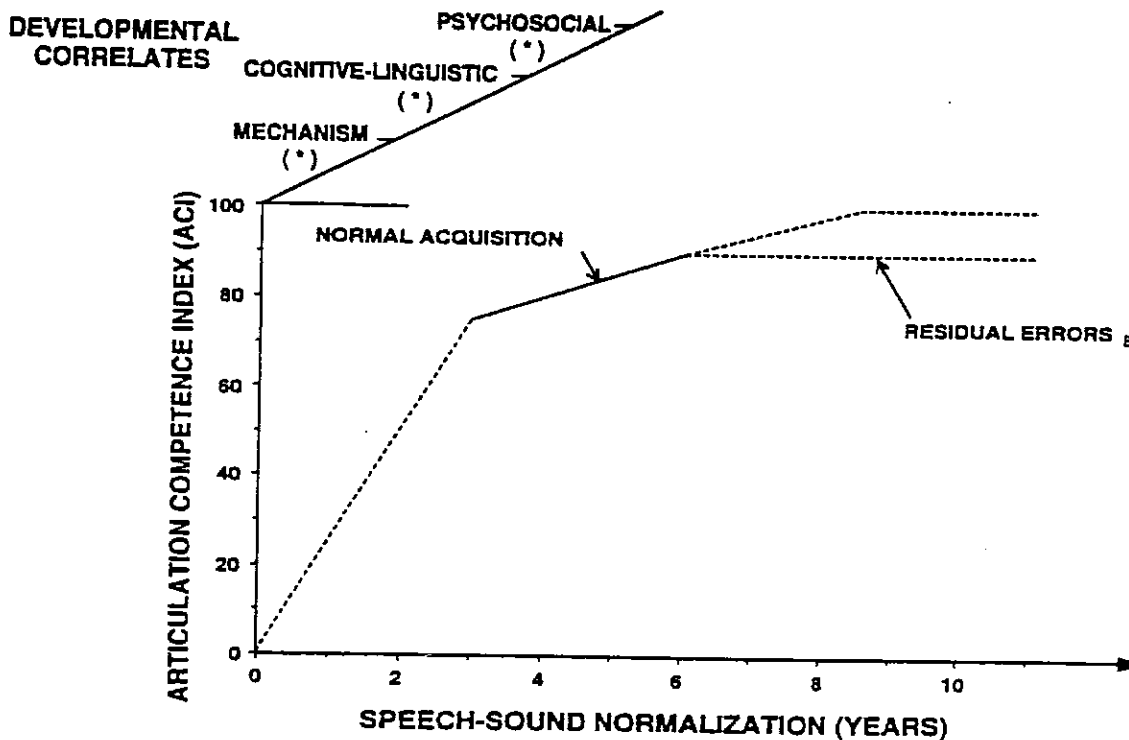


Figure 4. Three-parameter profile for residual errors (RE).

characters and celebrity imitations). It is the potential informativeness of distortions as biobehavioral events, rather than their social consequences, that makes them of central contemporary interest in speech-genetics and other subtyping research. Subtle-to-pronounced distortions of fricatives and liquids are frequent in English as well as in other languages (e.g., Fang and Ping-an, 1992; Qvarnström, Laine, and Jaroma, 1991).

Recall that RE_B are circumscribed speech errors that persist past the developmental period for speech (nominally 9 years). As described, the most frequent speech-sound distortions observed in native adult speakers of American English are either distortions of one or more of the sibilant fricatives or distortions of one of the two liquid consonants and the rhotic vowels.

Although RE_B prevalence is difficult to calculate because surveys often include persons who have residual errors associated with speech delay (i.e., RE_A), clearly it is much higher than that of either SD or any of the three subtypes of SD+. As indicated in Table 1, a conservative estimate of the population prevalence after the developmental period is 5%, although such often-cited figures have never been well documented (cf. Bernthal and Bankson, 1993). Gender ratios in RE are also quite different from those in SD and the three subtypes of SD+, suggesting a more equal distribution of women and men who have residual errors. Although not documented in the archival literature, clinical experience indicates that persons with lisps and de-rhoticized /r/s do not have histories of language involvement. By definition, they have long-term speech-sound normalization; some retaining distortions for life. Finally, there is no information on familial aggregation; a forthcoming study will provide prevalence data on RE_A and RE_B in adolescents and adults (Shriberg and Lewis, in process).

Causal origins. Each of the entries for RE in Table 1 contrast with those for SD, suggesting that causal origins for RE are different from the genetic origins posited for SD or the specific hearing, motor-speech, and psychosocial origins posited for SD + OME, SD + DAS, and SD + DPI. Because residual distortion errors occur so fre-

quently in persons who have no other developmental involvements, an alternative view is that they reflect inappropriate learning. Following is a summary of one possible environmental explanation that is being pursued in associated research.

In a series of studies of residual errors in school children, we found some interactions between speech-sound error type, birth order, and family size (Shriberg, 1975). Within samples of early elementary school children who had residual speech errors, children with /s/ errors were significantly more likely to be first-born, and children with /r/ errors were significantly more likely to be later-born and from larger families.

A social learning theory view (cf. Grusec, 1992) offers a possible explanation for these observations. Briefly, the proposed explanation is based on linguistic dispositions of first-borns versus later-borns in relation to the ontogenetic sequence of speech-sound acquisition, especially the crucial role of /s/ and /z/ sounds, which occur in over 70% of the 14 grammatical morphemes. In a variety of developmental studies, first-borns have been found to be eager for, and perhaps pressed for, language learning. Because the sibilant fricatives /s/ and /z/ are so important for syntactic development, eager young language learners tune in to the function of these sounds early—but they lack the motor development to say them correctly. The articulatory phonetics of sibilant fricatives result in a more anterior tongue placement, or dentalized productions. Thus, such distortions, which perforce are made early in life, are habituated, perhaps for life. Later-born children, lacking a particular press to tune in to language, have the most difficulty with what is arguably the most difficult class of English sounds: the /r/ consonant and rhoticized vowels. Children who delay tuning in and/or tuning up until the developmental window for speech acquisition begins to close can habituate a distortion error.

The point in reviewing the above speculations in this context is only to suggest at least one alternative explanation for the many differences between SD and RE in Table 1. Birth order is only a marker for responses to the ambient language that

children must acquire. A two-factor theory of phonological development is described elsewhere in which the constructs of capability and focus are posited to capture underlying processes. Briefly, capability reflects the cognitive-biological readiness for learning, and focus reflects the psychological disposition to attend to the stimuli needed to acquire or normalize speech (Kwiatkowski and Shriberg, 1993). Thus, the origin of RE is posited to be a mistiming in tuning in and tuning up, relative to the developmental timetable underlying normal auditory-articulatory learning.

SUMMARY

The primary goal of this article is to introduce a perspective on the possibility of etiologic subgroups among developmental phonological disorders. Four claims have been made.

First, emerging evidence supports the plausibility of five subtypes of developmental phonological disorders. The implication is that an etiologic perspective is more appropriate than the descriptive-linguistic perspective that has been the predominant paradigm in developmental phonological disorders for the past three decades. Compared to descriptive-linguistic typologies, an etiologically based classification system offers distinct advantages for assessment, intervention, and, ultimately, primary, secondary, and tertiary forms of prevention (American Speech-Language-Hearing Association, 1991).

A second claim addresses conceptual and methodological needs in speech-genetics research. The proposal is that a fruitful search for the appropriate phenotype for the most common form(s) of developmental phonological disorders would require explication among alternative levels of phonological processing, particularly the interfaces among speech, language, and other academic areas as levels of a more general verbal trait. Among alternative perspectives on the appropriate phenotype for SD as defined in the present typology, a specific speech-language delay is the best fit, based on associations and dissociations in the data to date.

A third claim is that there are specific etiologic factors that can result in three subtypes of speech delay, referred to as SD+OME, SD+DAS, and SD+DPI. Evidence for each subtype contrasted with one another and with SD includes differences in developmental correlates, prevalence, gender, speech-prosody profiles, and normalization histories. To date, there is only modest evidence to support each of the classification categories; future research should allow for direct statistical analysis of differences among subtypes.

The fourth claim is that the most common form of speech disorder, residual articulation distortions, arises solely as a response to the developmental context in which precise articulation is an acquired skill. The division between SD+ and RE is readily appreciated in clinical contexts but presents a conceptual problem for emerging genetics studies that must deliberate the appropriate inclusionary and exclusionary criteria for affected status (cf. Shriberg, 1993). Thus, whereas for many years single-sound errors have been of little clinical and research interest, their conceptual status as the most common form of a developmental phonological disorder requires new theoretical attention.

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