Otitis Media, Fluctuant Hearing Loss, and Speech-Language Outcomes: A Preliminary Structural Equation Model

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The goals of this study were to estimate the risk for lowered speech-language outcomes associated with early recurrent otitis media with effusion (OME) with and without hearing loss and to develop a preliminary descriptive-explanatory model for the findings. Three statistical approaches were used to assess associations among OME, hearing loss, and speech-language outcomes. Participants were a subsample of 70 children followed prospectively in the Dallas Cooperative Project on Early Hearing and Language Development (Friel-Patti & Finitzo, 1990). Findings indicated that hearing levels at 12–18 months were significantly associated with speech delay and low language outcomes at 3 years of age. The risk for subclinical or clinical speech delay at 3 years of age was 2% for children with less than 20 dB average hearing levels at 12–18 months and 33% for children with greater than 20 dB average hearing levels at 12–18 months. A structural equation model (Jöreskog & Sörbom, 1993) indicated that the significant and substantial effects of hearing levels at 12–18 months on speech status at 3 years were significantly mediated by language status at 3 years. Discussion includes implications of these findings for alternative speech perception models linking early OME and hearing loss to later speech-language disorder.

KEY WORDS: assessment, epidemiology, phonology, speech disorders

A recent volume by Roberts, Wallace, and Henderson (1997) provides detailed reviews of research in otitis media with effusion (OME) and communicative disorders, including international data on prevalence and epidemiology (Daly, 1997; Henderson & Roush, 1997; Maw & Counsell, 1997); auditory processing, speech-language, learning, and psychosocial variables (Gravel & Nozza, 1997; Hall, Grose, & Drake, 1997; Roberts & Wallace, 1997; Schwartz, Mody, & Petinou, 1997; Wallace & Hooper, 1997; Winston, Roberts, & Zeisel, 1997); and research needs (Paradise, 1997). In contrast to the mixed evidence of the previous two decades, findings emerging from several research groups using experimental and longitudinal designs with infants and toddlers (e.g., Abraham, Wallace, & Gravel, 1996; Nittrouer, 1996; Roberts, 1997; Rvachew, Slawinski, Williams, & Green, 1996a, 1996b, 1999) indicate that OME places a child at increased risk for a variety of developmental and educational sequelae, particularly as effect sizes of as little as one-third standard deviation are accepted as a legitimate clinical concern for long-term effects (e.g., Donahue, 1993; Feagans, Kipp, & Blood, 1994;

Friel-Patti, 1990; Gravel & Wallace, 1992, 1995; Paradise, 1997; Robb, Psak, & Pang-Ching, 1993; Shriberg et al., 1999). These trends (reviewed in Roberts et al., 1997) notwithstanding, a review of the more than two dozen OME studies since Holm and Kunze (1969) reporting clinical speech data indicates that there are no available studies that estimate the risk for speech disorder associated with early recurrent OME with and without hearing loss (cf. Shriberg et al., 2000).

Three constraints appear to explain the lack of risk data on OME, hearing loss, and speech disorder. First, the research designs used in the most widely cited OME-speech studies have typically included retrospective data on the frequency, type, and duration of episodes of otitis media, but not on hearing levels. Second, among the more recent prospective designs that do include hearing data, the classification of speech disorder has typically been based on limited speech measures that may be insensitive to subclinical levels of disorder, particularly with young children for whom more severe involvement may manifest later in development (cf. Shriberg et al., 1999). Third, the statistical analyses and models used to relate precursor to outcome variables in OME-speech studies have focused on association and group-comparison statistics (e.g., correlations, *t* tests) rather than biostatistical techniques to estimate per-child risk (cf. Sackett, Haynes, Guyatt, & Tugwell, 1991). Moreover, few have attempted to model direct, moderating, and mediating pathways (see Baron & Kenny, 1986; Brown, 1997) linking early OME with or without hearing loss to later speech-language disorder.

The primary goal of the present study was to estimate the risk for speech disorder at 3 years of age for children with early recurrent OME and hearing loss at 6–18 months. The secondary goal was to use a structural equation modeling technique to develop a preliminary explanatory model for the findings.

Sample

Dallas Cooperative Project on Early Hearing and Language Development. Participants were a subsample of 70 children from a total of 483 followed prospectively in the Dallas Cooperative Project on Early Hearing and Language Development (Friel-Patti & Finitzo, 1990). Children enrolled in the larger study were recruited from a private pediatric practice in Dallas, Texas. The socioeconomic status of all families was middle to high-middle on the Hollingshead scale (1975). Children enrolled in the Dallas project met the following criteria: (a) monolingual English homes, (b) full-term gestation (37 weeks or greater), (c) singleton birth, (d) birth weight greater than 2400 grams, (e) Apgar at least 7/8, (f) normal newborn examination, and (g) typically developing at time of enrollment (no noted neurological problems or major physical defects). Families with children meeting these criteria were approached in the pediatrician's office by a study representative at or before the infant's 3-month well-baby check; all participants were enrolled at or before the 6-month well-baby evaluation. A total of 256 boys (53%) and 227 girls (47%) were followed from 6 months to 3 years of age.

Subsample of the Dallas Data for the Present Study. For the goals of the present study, a subsample of 70 children from the Dallas project was eventually assembled using two inclusionary criteria. First, eligible participants had to have complete records available for all tests given in the Dallas study (i.e., participants had a good record of compliance), including the audiocassette tape of a spontaneous language sample obtained at 3 years of age. A total of 111 of the 483 children met this inclusionary criterion. Records for these children and the original tape recordings of the conversational language samples were forwarded to Madison for analysis. Second, eligible participants had to have speech samples suitable for the planned analyses. In Madison, two experienced research transcribers glossed the 111 language samples and made judgments of the suitability of the tapes for speech sampling based on the technical quality of the tapes (recorded in the mid-1980s) and on the linguistic content and length of the speech sample. Token frequency criteria developed in prior work in child phonology ensured that the speech samples would be appropriate for computer-based analyses of the speech samples (cf. Shriberg, 1986). These procedures yielded the 70 acceptable audiotaped conversational speech samples.

Otitis Media Assessment and Classification

OME was monitored in the Dallas project using both pneumatic otoscopy and tympanometry. Children were monitored in three settings: (a) home or day-care centers where tympanometry was performed, (b) well-child and sick-child visits to the pediatrician's office where both otoscopy and tympanometry were performed, and (c) twice yearly comprehensive assessments at the Callier Center for Communication Disorders where otoscopy, tympanometry, and age-appropriate hearing assessments were performed. For children 6–18 months of age, the minimum contact was an immittance check every 6 weeks, with 12 weeks as the maximum allowable time between visits.

Pneumatic Otoscopy. Otoscopy was performed by one of two otolaryngologists or a pediatrician, depending on the setting. Otoscopic findings from one otolaryngologist were validated to myringotomy with resultant sensitivity of 97% and specificity of 94%. These data were analyzed using Cohen's Kappa to correct for chance

agreement; the Kappa-corrected value was .91 (Cohen, 1960). Interexaminer reliability was performed yearly comparing the validated otoscopist to the other examiners. Agreement exceeded 85% at each check.

Tympanometry. Tympanometry screening was completed using an automated microprocessor-controlled, one-component admittance instrument (Maico 610) with a 226-Hz probe frequency. The tympanometric classification employed was similar to that described by Paradise, Smith, and Bluestone (1976). Identical variants could not be employed because of differences in the instruments used in the two studies (see Friel-Patti & Finitzo, 1990, for complete description). Tympanometry prior to myringotomy was performed on a sample of 150 ears to validate the procedures used to identify OME.

Algorithm for OME. Results of pneumatic otoscopy and tympanometry were combined into an algorithm originally described by Cantekin (1983) and updated by Brostoff and Cantekin (1988). The algorithm collapses multiple tympanometric and otoscopic variables into one variable. OME was absent when a Type A tympanogram was obtained and present when a Type B tympanogram was obtained. Otoscopic confirmation was not required. However, in keeping with tympanometric validation of myringotomy results, all other abnormal tympanograms required otoscopic confirmation before a diagnosis of OME was made. If otoscopy was not available (i.e., in the home or day-care center visits), the decision was based on immittance alone, although these data were analyzed separately. The otoscopic observation of liquid meniscus, even in the presence of a Type A tympanogram, always yielded a diagnosis of OME.

Otologic Classification. For the planned risk analyses in the present study, a procedure was needed to categorize the 70 children's otologic histories during each of three time periods: 6–12 months, 12–18 months, and 6–18 months. First, using the widely applied 29 days per episode criterion developed by the Boston study group (Teele, Klein, Rosner, & The Greater Boston Otitis Media Study Group, 1984), the number of effusion episodes was calculated for each child during each period. For the 6–12 month and 12–18 month periods, the total number of days with effusion during the period was divided by 29 to yield a three-category ordinal scale: 0.0–0.9 episodes, 1.0–1.9 episodes, and 2.0 or more episodes. A similar procedure was used for the 6–18 month period with two modifications. First, to accommodate the 100% increase in time frame, the criterion for each category was doubled (0.0–1.9 episodes, 2.0–3.9 episodes, and 4.0 or more episodes). Second, to ensure that effusion was chronic throughout the period, the middle category required at least 0.5 episode of effusion and the most involved category required at least 1.0 episode in each 6-month period.

The categorical criteria were then used to classify a child's OME history as affected (OME+) or nonaffected (OME–). Children classified as OME+ met the most involved criteria for days with effusion (i.e., 2 or more episodes during the 6-month period and 4 or more for the 12-month period). To address the likelihood of false negatives, analyses were completed using both the less and more stringent days with effusion criteria to classify a child as OME–.

The data on the left side of Table 1 summarize the number and gender of children in each developmental period whose history of otitis media with effusion met the less and more stringent criteria. Notice that, whereas the less stringent criteria includes fewer children meeting the criterion for OME– compared to the more stringent criteria, the number of children classified as OME+ is the same in both classifications. As shown in the bottom row of Table 1, two-tailed Fisher exact tests indicated that there were no statistically significant differences in the proportion of children of each gender meeting otitis media classification criteria for OME+ and OME– for the six comparisons.

Table 1. Summary of cell sizes and gender characteristics of the 70 children classified by otitis media and hearing level histories from 6 to 18 months of age. See text for description of criteria used to classify children's otitis media histories as affected (OME+) or nonaffected (OME–) and their hearing histories as affected (HL+) or nonaffected (HL–).

	Otitis media history												Hearing level history					
	Less stringent classification criteria						More stringent classification criteria											
	6–12 mos		12–18 mos		6–18 mos		6–12 mos		12–18 mos		6–18 mos		6–12 mos		12–18 mos		6–18 mos	
	OME+	OME–	OME+	OME–	OME+	OME–	OME+	OME–	OME+	OME–	OME+	OME–	HL+	HL–	HL+	HL–	HL+	HL–
Boys	7	19	2	24	3	23	7	16	2	19	3	19	8	20	7	19	5	21
Girls	7	34	7	34	5	36	7	23	7	24	5	27	14	26	8	29	7	30
Total	14	53	9	58	8	59	14	39	9	43	8	46	22	46	15	48	12	51
p value[a]	.368 *ns*		.465 *ns*		1.000 *ns*		.754 *ns*		.283 *ns*		1.000 *ns*		.610 *ns*		.766 *ns*		1.000 *ns*	

[a]Fisher exact test.

Hearing Level Assessment and Classification

Hearing Levels. Age-appropriate measures of hearing in the Dallas project began at 6 months and were collected at 6-month intervals. Auditory brainstem responses were obtained at 6 months to rule out mild sensorineural hearing loss at enrollment. At 12 months of age, sound field visual reinforcement audiometry was performed using speech (Speech Awareness Threshold) and warble tone stimuli (cf. Friel-Patti & Finitzo, 1990) at 500 Hz and 2 kHz; at 18 months, sound field hearing levels were obtained for 500 Hz, 1 kHz, and 2 kHz. The ABR thresholds for each ear were averaged to obtain one value at 6 months and the sound field measure at each of the other ages reflected hearing in the better of the two ears. Hearing level measures and SATs were averaged across the frequencies obtained for each period, and the beginning and ending average values for each developmental period were averaged. These procedures yielded one value representing children's average hearing levels for each developmental period (6–12 months, 12–18 months, and 6–18 months).

Hearing Level Classification. Hearing level averages were used for categorical classification of children's hearing during each of the three developmental periods, using the cutoff point defined in Friel-Patti and Finitzo (1990). Average hearing levels greater than 20 dB during each period were classified as affected (HL+), and average hearing levels of 20 dB or less during each period were classified as nonaffected (HL–). The data on the right side of Table 1 indicate the number of boys and girls who were classified as HL+ and HL– during each period. Totals do not always sum to 70 as there were missing hearing data for some subgroups. As indicated in the bottom row of Table 1, Fisher exact tests of the proportions of children by gender in each classification were nonsignificant.

Language Assessment and Classification

The Sequenced Inventory of Communication Development (SICD) (Hedrick, Prather, & Tobin, 1975) was used to assess receptive and expressive language skills in the children beginning at or near the first birthday and at 6-month intervals thereafter. Samples of spontaneous language were also collected every 6 months from the time the children reached 24 months of age. These conversational samples were audiorecorded by speech-language pathologists following the guidelines described by Miller (1981). The children were all evaluated using the Peabody Picture Vocabulary Test–Revised (PPVT-R; Dunn & Dunn, 1981) at the 36-month evaluation.

For the present study, scores from the language measures obtained at the 36-month testing were used because that is the first age at which the conversational language samples could be processed for speech analysis. PPVT-R raw scores were converted to standard scores (M = 100, SD = 15), and raw scores on the receptive (SICD-R) and expressive (SICD-E) composites of the SICD were converted to age-equivalent scores. Examination of group-average scores on the language instruments in both the original Dallas project and the subsample of 70 children for the present purposes indicated significantly above-average performance. For the 70-children subsample, the obtained group mean on the PPVT-R was 111.9, with a standard deviation of 11.0, with only 1 child (1.4%) scoring below 85 (i.e., more than one standard deviation below the normative sample mean). On the SICD-R, the group mean was 41.9 months, with a standard deviation of 4.5 months; only 4 of the 70 children scored below 36 months (the chronological age of all the children). On the SICD-E, the group mean was 42.8 months with a standard deviation of 4.3 months; none of the 70 children had age-equivalent scores below 36 months.

Classification of Language Disorder. To address the above findings, and to maximize sensitivity to potential differences associated with OME and hearing level histories, the distributions of language scores for the 70 children in the present study were used to set cutoff levels for *low language*. Thus, as in the Dallas project, low language is defined as performance at the low end of the obtained range on the language measures (cf. Friel-Patti & Finitzo, 1990). For each measure—the PPVT-R, SICD-R, and SICD-E—children with scores more than one standard deviation below the mean performance of the subsample of 70 children were classified as low language.

Speech Assessment and Classification

Transcription, Computer Formatting, and Reliability. The 70 conversational samples selected for the present study were transcribed by a research assistant with 8 years of experience transcribing speech samples of children with speech-language disorders. Transcription was accomplished using a Dictaphone Model 2600 playback device and a system of narrow-phonetic transcription conventions developed for research in child phonology (Shriberg & Kent, 1995; Shriberg, Kwiatkowski, & Hoffmann, 1984). The transcriber was provided only the age and gender of the speaker on each audiocassette tape. Another research assistant error checked the transcripts and formatted them for computer analysis using recent enhancements to the PEPPER system (Shriberg, 1986, 1993). Descriptive analyses with PEPPER programs indicated that the

distributional characteristics of the conversational samples (e.g., word forms, phonemes) were similar to distributions in samples obtained in prior research and hence appropriate for the planned speech analyses.

Interjudge and Intrajudge Transcription Agreement. A total of six conversational samples, three each from children classified as HL+ and HL– during the 6–18 month period, were randomly selected to assess transcription reliability. Approximately 1 month after completing transcription of the original tapes the transcriber retranscribed the six tapes, yielding intrajudge information on a total of 2,443 consonants and 1,651 vowels/diphthongs. Another experienced research transcriber also transcribed the six tapes. The PEPAGREE program (Shriberg & Olson, 1988) was used to calculate broad and narrow transcription agreement for intrajudge and interjudge comparisons for each sound class. Mean intrajudge transcription agreement for consonants was 91.6% for broad transcription and 87.6% for narrow transcription; agreement for vowels/diphthongs was 93.5% for broad transcription and 90.0% for narrow transcription. Mean interjudge agreement for consonants was 83.6% for broad transcription and 75.5% for narrow transcription; agreement for vowels/diphthongs was 86.8% for broad transcription and 76.5% for narrow transcription. These estimates of transcription reliability are consistent with those reported in other studies in child phonology (cf. McSweeny & Shriberg, 1995; Shriberg & Lof, 1991) and are adequate for the questions addressed in this report.

Description of Speech Measures. The PEPPER program was used to compute scores on a series of measures for each of the 70 conversational samples, including the 10 speech metrics of articulatory competence described in Shriberg, Austin, Lewis, McSweeny, and Wilson (1997a, 1997b). These speech measures are considered to be more well developed than the language measures for the goals of the present study, reflecting better sensitivity and specificity to clinical and subclinical levels of disorder (cf. Shriberg, 1993). Nine of the 10 speech measures treat articulatory competence as a criterion-referenced continuous trait, with 100% on a metric reflecting maximum articulatory competence. The 10th measure provides a categorical classification of a child's speech status using a 20-category, hierarchical typology. Descriptions of the following 10 speech measures are provided in Shriberg et al. (1997a, 1997b) and in the previous paper in this series (Shriberg et al., 1999); for space considerations, definitions are not repeated in the present paper: (1) Percentage of Consonants Correct (PCC), (2) Percentage of Consonants Correct–Adjusted (PCC-A), (3) Percentage of Consonants Correct–Revised (PCC-R), (4) Percentage of Consonants in Inventory (PCI), (5) Percentage of Vowels/Diphthongs Correct (PVC), (6) Percentage of Vowels/Diphthongs Correct–Revised (PVC-R), (7) Percentage of Phonemes Correct (PPC), (8) Percentage of Phonemes Correct–Revised (PPC-R), (9) Intelligibility Index, and (10) Speech Disorders Classification System (SDCS).

Classification of Speech Disorder. In prior work in otitis media and speech with young children, it has been useful to set criteria for subclinical as well as clinical levels of speech disorder following two rationales. First, because measures used in OME research may lack sensitivity to small but reliable effects, it is useful to be sensitive to all levels of potential disorder. Second, a major theme in recent otitis media research is that what is manifested as a subclinical disorder at the earliest observed ages may develop into a clear clinical disorder later in growth and development (cf. Roberts et al., 1997).

Cutoff criteria for subclinical and clinical disorder similar to those used in Shriberg et al. (1999) were used to classify children's status on each of the nine continuous speech metrics. However, because these children's Intelligibility Index and other speech scores were lower than scores for children of comparable ages and gender in the reference database used in Shriberg et al. (1999)—likely due to the reduced quality of the audiocassette recordings—*z* scores for all measures were based on the distributions of scores of the 70 children. Examination of skew and kurtosis values for each measure indicated that transformations of the percentage data were not needed. *Subclinical* disorder was defined as scores from 1.3 to 1.9 standard deviation units from the mean of the 70-children subsample. *Clinical* disorder was defined as scores 2.0 or more standard deviation units from the mean of the subsample.

Statistical Approach

Prior studies using the 10 speech metrics have described distributional considerations leading to the use of both parametric and nonparametric statistics for inferential statistical testing as well as rationale for setting alpha levels (cf. Shriberg, 1993). Preliminary analyses of the current data indicated the need for similar approaches. Because of constraints in the psychometric characteristics of the speech and language scores (e.g., skew and kurtosis, standard deviation ratios, correlations between means and standard deviations, percentage of 100% scores, small and/or unbalanced cell sizes), nonparametric inferential statistics (Wilcoxon-Mann-Whitney rank order statistics [Siegel & Castellan, 1988]) were selected for analyses when measures were treated as continuous variables; parametric statistics (means, standard deviations) were used for descriptive purposes. Correlational analyses were completed with both parametric and nonparametric statistics, reporting only the parametric findings when the nonparametric coefficients were essentially similar.

The large number of statistical tests within and across questions warranted a rationale for setting the alpha levels required for statistical significance. Post hoc power estimates were not completed because of the varying cell sizes between and within the numerous analyses. Given the theoretical and applied goals, it was considered equally important to avoid both Type I and Type II errors. Rather than using arbitrary family-wise criteria to set significance levels, the decision was to acknowledge all obtained *p* values at the .05 level or less as statistically significant. In a study with similar measurement constraints and descriptive-explanatory goals, Lahey and Edwards (1995) provide a well-reasoned rationale for using an even more liberal alpha level (.10) to minimize the possibility of a Type II error (cf. Shriberg et al., 1999).

Results

Preliminary Analyses

Two preliminary analyses series were completed to inform the primary analyses that follow. The first series inspected associations between and among the precursor variables, and the second assessed the strength of association between and among the outcome variables.

Precursor Variables: Days With Effusion and Hearing Levels

Continuous Data. The first series of preliminary analyses assessed the strength of association between and among days with effusion and hearing loss during the two 6-month periods from 6–18 months of age and the total 12-month period. Days with effusion and hearing loss were treated as continuous variables. As shown by the vertical arrows in Figure 1, Pearson correlational data indicated virtually no linear association between number of days with effusion (DWE) and hearing levels (HL) at 6–12 months (r = .12), 12–18 months (r = .05), or for the 12-month period from 6–18 months (r = .10). As also indicated in Figure 1, the association between days with effusion across the two developmental periods, 6–12 months and 12–18 months, is only low positive (r = .31), accounting for less than 10% of common variance. Cross-lagged coefficients between days with effusion and hearing loss across the two periods (r = .19, r = .11) are also only low positive. The association between hearing levels at each age period is moderately positive (r = .57), accounting for approximately 33% of common variance.

Categorical Data. Figure 2 provides the per-child concordance analyses for the same variables assessed in Figure 1. The percentages for each arrow indicate the percentage of those children clinically positive for the variable at the origin of the arrow (OME+, HL+) who are also clinically positive for that variable at the head of the arrow. Two summary observations are based on the patterns of percentages in Figure 2.

First, as indicated by the percentages for the four horizontal arrows in Figure 2, the stability of involvement in each domain across the two developmental periods ranged from 14% to 67%. As with the continuous data in Figure 1, concordance in involvement across the two developmental periods was lower for OME than for

Figure 1. Associations between and among number of days with effusion (DWE) and hearing levels (HL) at 6–12 months, 12–18 months, and 6–18 months.

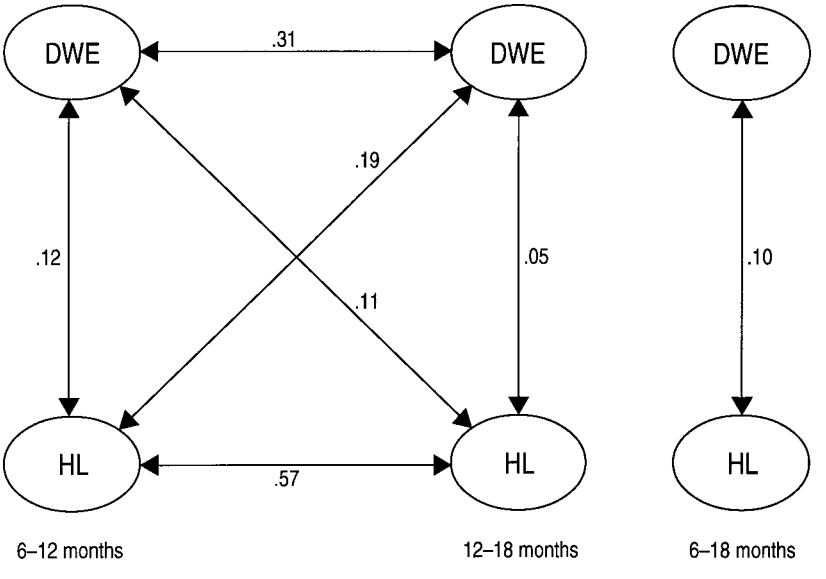
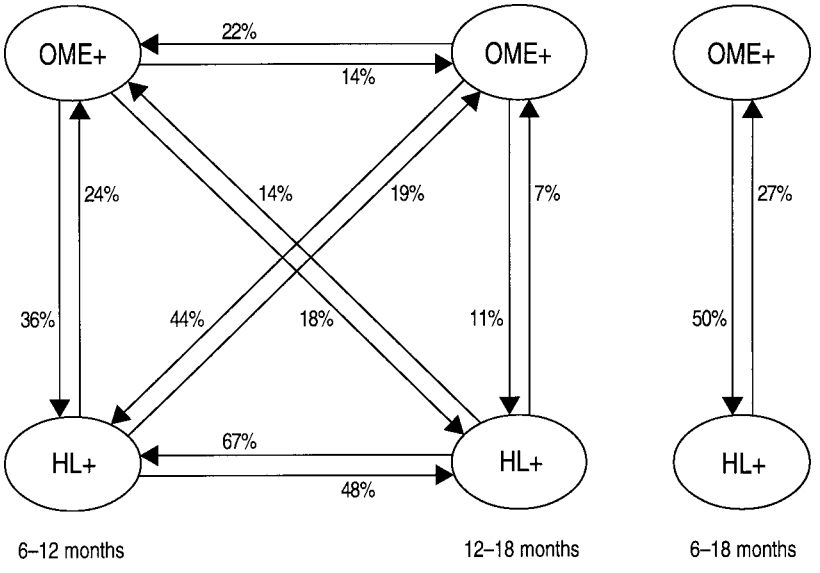


Figure 2. Percentage of children who are clinically positive for both otitis media with effusion (OME+) and hearing loss (HL+). See text for definitions of involvement for each variable.



HL. Fewer than one fourth (14%, 22%) of the children meeting the criterion for OME+ in one period also met the criterion for OME+ in the other period. Approximately one half (48%) to two thirds (67%) of the children who met the criterion for clinically significant hearing loss (HL+) at one developmental period also met the criterion for HL+ in the other developmental period. These data indicate the need to address otitis media and hearing loss individually in each of the two 6-month developmental periods from 6 to 18 months because many children evidently met the criterion for involvement in only one of the two periods.

Second, as calculated using the categorical procedures for OME+ and HL+, the per-child concordances between otitis media and hearing loss domains are relatively low. As shown by the percentages associated with the six vertical arrows in Figure 2, concordance in both domains during the same 6-month period ranged from 7% (percentage of HL+ children from 12 to 18 months who were also OME+) to 50% (percentage of OME+ children from 6 to 18 months who were also HL+). Thus, whether assessed as continuous (Figure 1) or categorical (Figure 2) variables, otitis media with effusion is not strongly associated with hearing loss. Clearly, not all children with middle ear disease would be expected to have associated hearing loss. However, it is expected that nearly all children with documented conductive hearing loss will have had earlier or concurrent middle ear disease.

These data underscore the limitations in the sensitivity of the measures used in this study and others to identify and classify episodes of otitis media with effusion in young children. The disparity between documented hearing loss and documented days with effusion indicates that a significant number of episodes of middle ear disease go undetected (cf. Mehta, 1990; Paparella, Shea, Meyerhoff, & Goycoolea, 1980; Roland et al., 1989). In the present context, these data suggest that in comparison with the days with effusion data, information on a child's hearing loss will be both conceptually more valid and empirically more reliable as a risk factor for later speech-language delay.

Outcome Variables: Language and Speech Competence

The second series of preliminary analyses inspected associations between and among the language and speech measures to determine the degree of concordance among outcome variables. Table 2 includes Pearson correlation coefficients expressing the linear associations between and among measures in each linguistic domain.

Language Measures. As shown in the first two rows of Table 2, PPVT-R scores have low positive association with the receptive (r = .38) and expressive (r = .36) scales of the SICD measures, which have a moderately positive (r = .60) correlation with each other. As none of the three correlations accounts for more than 50% of the variance in the other, and because both comprehension and expression domains are of individual interest, these data supported a decision to include findings from all three measures of language in the primary analyses to follow.

Speech Measures. The pattern of intercorrelations among the nine speech measures in Table 2 reflects the rationale for their use in descriptive-explanatory studies

Table 2. Intercorrelation matrix for the three language measures and nine speech measures at 3 years of age.

	Language measures			Speech measures							
	Receptive		Expressive								
	PPVT-R	SICD-R	SICD-E	PCC	PCC-A	PCC-R	PCI	PVC	PVC-R	PPC	PPC-R
SICD-R	.38										
SICD-E	.36	.60									
PCC	.18	.31	.48								
PCC-A	.24	.33	.48	.93							
PCC-R	.24	.33	.47	.93	.99						
PCI	.18	.10	.19	.53	.54	.51					
PVC	.03	.22	.25	.56	.59	.58	.34				
PVC-R	.07	.24	.24	.59	.62	.61	.31	.94			
PPC	.17	.32	.47	.98	.93	.92	.52	.69	.71		
PPC-R	.22	.34	.46	.92	.98	.99	.51	.69	.73	.94	
II	.18	.23	.49	.48	.52	.51	.18	.17	.14	.45	.47

Note. PPVT-R = Peabody Picture Vocabulary Test–Revised; SICD-R = Sequenced Inventory of Communicative Development–Receptive; SICD-E = Sequenced Inventory of Communicative Development–Expressive; PCC = Percentage of Consonants Correct; PCC-A = Percentage of Consonants Correct–Adjusted; PCC-R = Percentage of Consonants Correct–Revised; PCI = Percentage of Consonants in the Inventory; PVC = Percentage of Vowels Correct; PVC-R = Percentage of Vowels Correct–Revised; PPC = Percentage of Phonemes Correct; PPC-R = Percentage of Phonemes Correct–Revised; II = Intelligibility Index.

of child speech disorders (Shriberg et al., 1997a). As expected, there was considerable statistical overlap (.90s) among the three consonants measures (PCC, PCC-A, PCC-R) and between the two vowels/diphthongs measures (PVC, PVC-R). The consonants measures were only moderately correlated (.50s–.60s) with the vowels/diphthongs measures. Considering the conceptual and applied importance of information on consonant versus vowel/diphthong indices, and also important differences between indices reflecting only deletion-substitution errors (PCC-R, PVC-R, PPC-R) versus information from indices that treat common distortions (PCC-A) or all distortions (PCC, PVC, PPC) as errors, the primary analyses will need to report findings from each of these seven measures. The PCI and Intelligibility Index were weakly to moderately (.14–.54) associated with the seven other measures, supporting their inclusion also in the primary analyses. For the structural equation modeling to follow, the data in Table 2 provide strong support for treating Speech as a latent variable, with no one of the nine speech measures wholly capturing the concept of speech competence.

Language and Speech Measures. The final data of interest in Table 2 are the intercorrelations between and among the three language measures and each of the nine speech measures, as shown in the first three columns, beginning in the third row. The 27 coefficients range from .03 to .49, indicating that speech and language acquisition, as measured in this study, are not closely associated. As expected, there is a trend for the expressive language measure to be more strongly associated with the speech measures than are either of the two receptive language measures. For the present purpose, these preliminary analyses of the strength of associations among the 12 measures—three alternative measures of language capability and nine alternative measures of speech capability—provide statistical support for a decision to include each measure as a potentially informative dependent variable in the univariate analyses to follow. Moreover, because none of the individual language measures shares more than 25% common variance with any of the individual speech measures, the constructs of language and speech are each candidates for latent variables in the planned structural equation modeling.

Primary Analyses

Because the preliminary analyses indicated only low positive association between early days with effusion and hearing loss, the first of two series of primary analyses assessed each variable as an independent precursor of later language and speech status. Analyses were performed using three approaches to measurement levels: (a) treating both precursor and outcome measures as continuous variables, (b) treating precursor measures as categorical variables and outcome measures as continuous variables, and (c) treating both precursor and outcome measures as categorical variables. A univariate approach was used for each of the three analyses to maintain maximum sensitivity to both the precursor and outcome variables. The group-means approaches (a and b) make full use of the parametric data for descriptive-explanatory questions, whereas the categorical approach

(c) provides the sensitivity to individual children needed for clinical-predictive issues.

Precursor and Outcome Measures Treated as Continuous Variables

Table 3 includes the Pearson correlation coefficients indicating the strength of association between the number of days with effusion and hearing levels from 6 to 18 months and the 12 continuous measures of language and speech at 3 years of age. The low magnitudes of these coefficients indicate little linear association between early otologic and hearing status and later language and speech performance. The highest coefficients were for the Intelligibility Index scores with days with effusion (coefficients of .10 to .34; maximum of less than 12% of common variance) and hearing levels (coefficients of –.22 to –.27; maximum of less than 8% of common variance). Thus, when assessed as continuous variables, days with effusion and average hearing levels have little-to-weak linear association with independent measures of later language and speech status.

Precursor Measures Dichotomized; Outcome Measures Treated as Continuous Variables

Tables 4 and 5 include findings for the second analytic approach in which the precursor variables are dichotomized using the procedures described previously and the outcome variables are continuous. As described, the statistical approach uses nonparametric Wilcoxon-Mann-Whitney rank-order tests, with parametric statistics (means, standard deviations) in Tables 4 and 5 providing a more useful description of between-group differences.

Otitis Media. Table 4 includes speech-language outcome findings for children whose days with effusion histories are dichotomized using the less stringent criterion (fewer than 2 episodes) and the more stringent criterion (fewer than 1 episode) for OME– within each 6-month period. Of the 36 between-group comparisons for each of the OME classification criteria in Table 4, only two comparisons were statistically significant at the .05 alpha level. Compared to children meeting the less stringent criterion for OME–, the 14 children who met OME+ criteria for the 6–12 month period had significantly higher Intelligibility Index scores (Less Stringent: p = .011; More Stringent: p = .007), averaging approximately 5–6% higher. Other than the possibility of occurring by chance, there is no explanation for these counterintuitive findings.

Hearing Loss. Table 5 includes speech-language outcomes for children whose hearing levels are dichotomized using the criterion for HL+ of greater than 20 dB HL during each of the developmental periods. A total of 4 of the 36 comparisons were statistically significant at or beyond the .05 level. Compared to children whose hearing levels did not meet the 20 dB average hearing loss criterion during the 12–18 month period (HL–), children who met criteria for HL+ had significantly lower scores on the SICD-E measure at 3 years of age (HL+: 40.4; HL–: 43.6; p = .017) as well as significantly lower scores on the Intelligibility Index (HL+: 81.5; HL–: 88.0; p = .004). For the total 6–18 month period, HL+ children scored significantly lower than HL– children on the SICD-E measure (HL+: 40.6; HL–: 43.4; p = .048). Trends for 26 of the 37 comparisons (70%) indicated lower average language and speech scores for HL+ children.

Precursor and Outcome Measures Treated as Categorical Variables

Findings for the third series of analyses are shown in Table 6 and Table 7. As described previously, the criterion for delay on the language measures is a score below one standard deviation unit from the mean of the 70 children (i.e., 15th percentile or below). For the nine

Table 3. Pearson correlation coeffecients for days with effusion and hearing levels from 6 to 18 months, and language and speech measures at 3 years of age.

	Language measures			Speech measures								
	PPVT-R	SICD-R	SICD-E	PCC	PCC-A	PCC-R	PCI	PVC	PVC-R	PPC	PPC-R	II
Days with Effusion:												
6–12 mos	.20	.17	.13	.08	.17	.17	–.11	–.10	–.12	.05	.13	.34
12–18 mos	.18	.20	.03	.20	.23	.23	.02	.05	.12	.18	.23	.10
6–18 mos	.23	.23	.11	.16	.24	.24	–.06	–.04	–.01	.14	.21	.29
Hearing Levels (dB):												
6–12 mos	.07	.14	–.05	–.11	–.12	–.14	–.03	.03	.02	–.08	–.11	–.23
12–18 mos	.03	.00	–.07	.04	.06	.04	.04	.12	.13	.06	.06	–.27
6–18 mos	.06	–.03	–.12	.04	.08	.07	.02	.08	.10	.05	.08	–.22

Note. See Table 2 for key to abbreviations.

Table 4. Language and speech outcomes at 3 years for children whose otitis media histories were dichotomized as OME+ and OME– (see text).

		Less stringent classification criteria					More stringent classification criteria				
		OME+		OME–			OME+		OME–		
Period	Measure	*M*	*SD*	*M*	*SD*	*p*[a]	*M*	*SD*	*M*	*SD*	*p*[a]
		2+ episodes		<2 episodes			2+ episodes		<1 episode		
6–12 mos		(*n* = 14)		(*n* = 53)			(*n* = 14)		(*n* = 39)		
	Language:										
	PPVT-R	116.5	8.7	111.1	11.1	.108	116.5	8.7	110.5	11.9	.102
	SICD-R	43.1	3.2	41.9	4.5	.230	43.1	3.2	41.6	4.6	.196
	SICD-E	43.4	3.1	42.8	4.6	.637	43.4	3.1	42.5	4.4	.442
	Speech:										
	PCC	82.8	3.9	82.8	5.9	.811	82.8	3.9	82.5	5.7	.808
	PCC-A	86.6	3.3	85.8	5.2	.739	86.6	3.3	85.3	5.2	.571
	PCC-R	87.1	3.1	86.3	5.0	.722	87.1	3.1	85.9	5.0	.530
	PCI	95.5	5.5	96.4	3.4	.850	95.5	5.5	96.3	3.7	.773
	PVC	97.3	1.8	97.8	1.7	.224	97.3	1.8	97.9	1.8	.211
	PVC-R	97.9	1.3	98.4	1.4	.140	97.9	1.3	98.4	1.4	.134
	PPC	88.6	2.2	88.9	4.0	.486	88.6	2.2	88.7	3.8	.440
	PPC-R	91.5	1.9	91.2	3.3	.932	91.5	1.9	91.0	3.3	.816
	II	90.3	5.3	84.9	7.8	.011*	90.3	5.3	84.1	8.3	.007**
12–18 mos		(*n* = 9)		(*n* = 58)			(*n* = 9)		(*n* = 43)		
	Language:										
	PPVT-R	116.0	9.2	111.6	11.0	.140	116.0	9.2	110.8	11.6	.103
	SICD-R	43.8	4.9	41.9	4.1	.301	43.8	4.9	41.5	4.2	.246
	SICD-E	43.3	4.2	42.9	4.3	.891	43.3	4.2	42.7	4.5	.815
	Speech:										
	PCC	83.6	5.5	82.6	5.5	.898	83.6	5.5	81.5	5.3	.610
	PCC-A	86.9	4.1	85.8	5.0	.586	86.9	4.1	84.7	5.0	.264
	PCC-R	87.4	4.0	86.3	4.8	.665	87.4	4.0	85.3	4.7	.264
	PCI	95.8	5.1	96.3	3.7	.881	95.8	5.1	96.1	4.0	.991
	PVC	98.2	1.1	97.6	1.8	.677	98.2	1.1	97.6	1.8	.517
	PVC-R	98.7	0.8	98.2	1.4	.287	98.7	0.8	98.1	1.5	.216
	PPC	89.5	3.5	88.7	3.7	.811	89.5	3.5	88.1	3.7	.559
	PPC-R	92.0	2.5	91.1	3.2	.370	92.0	2.5	90.5	3.2	.151
	II	87.1	6.9	85.8	7.8	.748	87.1	6.9	85.0	8.2	.611
		4+ episodes		<4 episodes			4+ episodes		<2 episodes		
6–18 mos		(*n* = 8)		(*n* = 59)			(*n* = 8)		(*n* = 46)		
	Language:										
	PPVT-R	113.0	10.9	112.1	10.8	.739	113.0	10.9	111.0	11.5	.535
	SICD-R	43.0	4.1	42.0	4.3	.534	43.0	4.1	41.5	4.4	.360
	SICD-E	42.0	3.0	43.1	4.4	.452	42.0	3.0	42.5	4.3	.752
	Speech:										
	PCC	82.6	4.3	82.8	5.7	.656	82.6	4.3	81.6	5.4	.913
	PCC-A	86.8	3.8	85.8	5.0	.684	86.8	3.8	84.8	4.9	.322
	PCC-R	87.4	3.6	86.3	4.8	.504	87.4	3.6	85.4	4.7	.222
	PCI	95.1	5.1	96.3	3.7	.424	95.1	5.1	96.0	3.9	.603
	PVC	97.6	2.1	97.7	1.7	.882	97.6	2.1	97.6	1.8	.754
	PVC-R	98.6	0.9	98.2	1.4	.575	98.6	0.9	98.2	1.5	.490
	PPC	88.7	2.3	88.8	3.8	.607	88.7	2.3	88.1	3.7	.952
	PPC-R	91.9	2.1	91.2	3.2	.478	91.9	2.1	90.6	3.2	.207
	II	87.8	6.6	85.8	7.8	.622	87.8	6.6	84.6	8.3	.361

Note. See Table 2 for key to abbreviations. [a]Wilcoxon-Mann-Whitney test.
*$p < .05$. **$p < .01$.

Table 5. Language and speech outcomes at 3 years for children whose hearing loss histories at 6–18 months were dichotomized as HL+ and HL– (see text).

	6–12 months					12–18 months					6–18 months				
	HL+ (*n* = 22)		HL– (*n* = 46)			HL+ (*n* = 15)		HL– (*n* = 48)			HL+ (*n* = 12)		HL– (*n* = 51)		
Measures	***M***	***SD***	***M***	***SD***	***p*[a]**	***M***	***SD***	***M***	***SD***	***p*[a]**	***M***	***SD***	***M***	***SD***	***p*[a]**
Language:															
PPVT-R	112.7	8.7	111.3	12.1	.715	113.2	8.5	111.3	11.8	.719	114.7	9.0	111.1	11.5	.293
SICD-R	42.6	4.1	41.6	4.8	.651	41.3	4.0	42.1	4.9	.278	42.0	4.1	41.9	4.9	.769
SICD-E	42.3	4.9	43.1	3.9	.433	40.4	4.0	43.6	4.2	.017*	40.6	4.2	43.4	4.2	.048*
Speech:															
PCC	81.8	6.5	82.9	5.1	.503	80.1	6.8	83.5	4.8	.043*	80.1	7.4	83.1	5.1	.150
PCC-A	84.6	6.3	86.1	4.7	.477	83.6	6.8	86.4	4.3	.126	84.2	7.7	85.8	4.6	.793
PCC-R	85.1	6.2	86.6	4.4	.537	84.0	6.7	86.9	4.0	.123	84.7	7.6	86.4	4.4	.896
PCI	95.7	4.3	96.4	3.7	.477	96.3	3.6	96.4	3.8	.843	96.1	3.6	96.2	4.1	.707
PVC	97.8	1.7	97.5	2.1	.731	97.5	2.0	97.8	1.7	.842	97.2	2.1	97.7	1.9	.439
PVC-R	98.3	1.5	98.1	1.7	.503	98.1	1.7	98.2	1.6	.968	97.8	1.8	98.2	1.7	.659
PPC	88.3	4.3	88.8	3.5	.645	87.2	4.6	89.3	3.3	.078	87.1	5.1	89.0	3.5	.203
PPC-R	90.5	4.1	91.3	3.1	.616	89.8	4.6	91.5	2.8	.223	90.0	5.2	91.2	3.0	.972
II	84.1	8.7	87.1	6.8	.131	81.5	8.5	88.0	5.5	.004**	83.5	6.6	86.5	7.6	.092

Note. See Table 2 for key to abbreviations. [a]Wilcoxon–Mann–Whitney test.
*$p < .05$. **$p < .01$.

continuous speech measures, the criterion for a subclinical or clinical disorder was a score of 1.3 or more standard deviation units from the mean of the subgroup of 70 children (i.e., 10th percentile or below). For the SDCS measure, the criterion was Speech Delay (SD), as defined by the SDCS computer program (Shriberg et al., 1997b).

Otitis Media. As shown in Table 6, odds ratios were used when relative risk ratios could not be used because of zero frequencies in at least two cells (Kahn & Sempos, 1989). None of the 39 risk analyses using either the less or more stringent classifications of OME– yielded statistically significant findings, which would be indicated by a confidence interval that did not include 1.

Hearing Loss. Table 7 is a summary of findings testing the association between early hearing loss and later speech-language status. All comparisons for each of the developmental periods were obtained using relative risk ratios, as the cohort of 70 children was dichotomized into the two hearing history groups for each comparison. Of the 39 tests, 10 (25.6%) were statistically significant at or less than the .05 level, as indicated by Wilcoxon-Mann-Whitney test values. For each of the 10 statistically significant comparisons, more children classified as positive for hearing loss (HL+) at 12–18 months or the total 6–18 month period had subclinical or clinical speech disorder at 3 years of age compared to children classified as HL–. Relative risk ratios for the 10 statistically significant analyses ranged from 9.60:1 to 21.25:1, indicating that children classified as HL+ at 6–18 months of age were at approximately 10 to 21 times increased risk for subclinical or clinical speech disorder at 3 years of age. The lower bound confidence limits for these comparisons ranged from just over 1 to just under 3; upper bound confidence limits ranged from approximately 86 to 166. Thus, although the 10–21 times increased risk for speech disorder was significantly greater than chance in these comparisons, the relatively large confidence limits likely associated with small cell sizes prohibit reliable generalizations about the magnitude of these risk estimates.

The pattern of significant findings in Table 7 can be interpreted as follows. First, effects were clearly associated with consonant development (PCC, PCC-A, PCC-R) rather than vowel/diphthong development (PVC, PVC-R), reflected as well in the metrics that included articulation of both consonants and vowels/diphthongs (PPC, PPC-R). Second, effects were not only limited to distortions of consonants (PCC, PPC) or just the noncommon clinical distortions of consonants (PCC), but also reflected deletions and substitutions of consonants (PCC-R, PPC-R). Finally, the pattern of significant findings indicates that the 12–18 month period of development is most highly associated with later clinical or subclinical speech disorder. There were no statistically significant findings for the 6–12 months comparisons, and statistical findings for the total 6–18 month period mirrored those found for the 12–18 month period.

Table 6. Risk estimates (relative risk ratio and odds ratio analyses) for otitis media histories at 6–18 months and language-speech status at 3 years (see text for definition of affected for the language and speech measures).

		Less stringent classification criteria					More stringent classification criteria				
		% Affected			Confidence limits		% Affected			Confidence limits	
Period	Measure	OME+	OME–	Relative risk	Lower	Upper	OME+	OME–	Odds ratio	Lower	Upper
6–12 mos[a]											
	Language:										
	PPVT-R	0	19	0.00[d]	0.00	1.56	0	23	0.00	0.00	1.29
	SICD-R	7	12	0.62	0.08	4.73	7	13	0.52	0.01	5.42
	SICD-E	0	16	0.00[d]	0.00	2.08	0	15	0.00	0.00	2.32
	Speech:										
	PCC	0	11	0.00[d]	0.00	3.24	0	10	0.00	0.00	4.27
	PCC-A	0	8	0.00	0.00	5.89	0	10	0.00	0.00	4.27
	PCC-R	0	8	0.00[d]	0.00	5.89	0	10	0.00	0.00	4.27
	PCI	21	11	1.89	0.54	6.64	21	15	1.50	0.21	8.52
	PVC	14	8	1.89	0.39	9.30	14	8	2.00	0.15	19.46
	PVC-R	7	6	1.26	0.14	11.22	7	5	1.42	0.02	29.31
	PPC	0	11	0.00[d]	0.00	3.24	0	10	0.00	0.00	4.27
	PPC-R	0	8	0.00[d]	0.00	5.89	0	10	0.00	0.00	4.27
	II	7	8	0.95	0.11	7.81	7	10	0.67	0.01	7.74
	SDCS	14	11	1.51	0.33	7.00	14	13	1.13	0.10	8.12
12–18 mos[b]											
	Language:										
	PPVT-R	11	16	0.70	0.10	4.91	11	19	0.53	0.01	5.13
	SICD-R	11	11	1.06	0.14	7.78	11	12	0.93	0.02	10.15
	SICD-E	0	14	0.00[d]	0.00	3.80	0	17	0.00	0.00	3.22
	Speech:										
	PCC	0	10	0.00[d]	0.00	5.85	0	14	0.00	0.00	4.22
	PCC-A	0	7	0.00[d]	0.00	10.47	0	9	0.00	0.00	7.66
	PCC-R	0	7	0.00[d]	0.00	10.47	0	9	0.00	0.00	7.66
	PCI	11	14	0.81	0.11	5.70	11	14	0.77	0.02	7.96
	PVC	0	10	0.00[d]	0.00	5.85	0	9	0.00	0.00	7.66
	PVC-R	0	7	0.00[d]	0.00	10.47	0	9	0.00	0.00	7.66
	PPC	0	10	0.00[d]	0.00	5.85	0	14	0.00	0.00	4.22
	PPC-R	0	7	0.00[d]	0.00	10.47	0	9	0.00	0.00	7.66
	II	0	9	0.00[d]	0.00	7.55	0	9	0.00	0.00	7.66
	SDCS	11	10	1.07	0.15	7.91	11	9	1.22	0.02	14.68
6–18 mos[c]											
	Language:										
	PPVT-R	13	14	0.91	0.13	6.33	13	18	0.66	0.01	6.54
	SICD-R	13	10	1.21	0.17	8.78	13	13	0.93	0.02	9.82
	SICD-E	0	14	0.00[d]	0.00	4.45	0	16	0.00	0.00	4.00
	Speech:										
	PCC	0	10	0.00[d]	0.00	6.82	0	13	0.00	0.00	5.21
	PCC-A	0	7	0.00[d]	0.00	12.17	0	9	0.00	0.00	9.39
	PCC-R	0	7	0.00[d]	0.00	12.17	0	9	0.00	0.00	9.39
	PCI	13	14	0.92	0.13	6.44	13	15	0.80	0.02	8.09
	PVC	13	8	1.48	0.20	11.08	13	9	1.50	0.03	18.39
	PVC-R	0	7	0.00[d]	0.00	12.17	0	7	0.00	0.00	14.87
	PPC	0	10	0.00[d]	0.00	6.82	0	13	0.00	0.00	5.21
	PPC-R	0	7	0.00[d]	0.00	12.17	0	9	0.00	0.00	9.39
	II	13	7	1.84	0.23	14.51	13	9	1.50	0.03	18.39
	SDCS	25	8	2.95	0.68	12.76	25	11	2.73	0.21	21.80

Note. See Table 2 for key to abbreviations. SDCS = Speech Disorders Classification System. [a]Classification A, OME+ *n* = 14; Classification A, OME– *n*s: 51–53; Classification B, OME+ *n* = 14; Classification B, OME– *n* = 39. [b]Classification A, OME+ *n* = 9; Classification A, OME– *n*s: 56–58; Classification B, OME+ *n* = 9; Classification B, OME– *n*s: 41–43. [c]Classification A, OME+ *n* = 8; Classification A, OME– *n*s: 57–59; Classification B, OME+ *n* = 8; Classification B, OME– *n*s: 44–46. [d]risk estimated using odds ratio.

Table 7. Risk estimates (relative risk ratio and odds ratio analyses) for hearing loss histories at 6–18 months and language-speech status at 3 years (see text for definition of affected for the language and speech measures)

Period	Measure	% Affected HL+	% Affected HL–	Risk estimate	Confidence limits Lower	Confidence limits Upper
6–12 mos[a]						
	Language:					
	PPVT-R	14	18	0.77	0.23	2.61
	SICD-R	9	16	0.58	0.13	2.58
	SICD-E	18	9	2.00	0.55	7.25
	Speech:					
	PCC	14	9	1.57	0.38	6.41
	PCC-A	14	4	3.14	0.56	17.44
	PCC-R	14	4	3.14	0.56	17.44
	PCI	14	13	1.02	0.28	3.71
	PVC	5	13	0.35	0.04	2.72
	PVC-R	5	9	0.52	0.06	4.41
	PPC	18	7	2.79	0.68	11.40
	PPC-R	14	4	3.14	0.56	17.44
	II	9	7	1.39	0.25	7.75
	SDCS	18	11	1.67	0.50	5.62
12–18 mos[b]						
	Language:					
	PPVT-R	14	17	0.84	0.20	3.51
	SICD-R	14	13	1.12	0.25	4.94
	SICD-E	29	9	3.36	0.96	11.73
	Speech:					
	PCC	33	2	16.00	2.02	126.46**
	PCC-A	20	2	9.60	1.08	85.58*
	PCC-R	20	2	9.60	1.08	85.58*
	PCI	13	13	1.07	0.24	4.74
	PVC	13	6	2.13	0.39	11.59
	PVC-R	7	6	1.07	0.12	9.51
	PPC	33	2	16.00	2.02	126.46**
	PPC-R	20	2	9.60	1.08	85.58*
	II	13	2	6.40	0.62	65.74
	SDCS	20	10	1.92	0.52	7.11
6–18 mos[c]						
	Language:					
	PPVT-R	9	18	0.51	0.07	3.59
	SICD-R	9	14	0.65	0.09	4.76
	SICD-E	27	10	2.73	0.76	9.75
	Speech:					
	PCC	42	2	21.25	2.73	165.55**
	PCC-A	25	2	12.75	1.45	112.13*
	PCC-R	25	2	12.75	1.45	112.13*
	PCI	8	14	0.61	0.08	4.48
	PVC	17	6	2.83	0.53	15.13
	PVC-R	8	6	1.42	0.16	12.46
	PPC	42	2	21.25	2.73	165.55**
	PPC-R	25	2	12.75	1.45	112.13*
	II	8	4	2.13	0.21	21.55
	SDCS	17	12	1.42	0.33	6.17

Note. See Table 2 for key to abbreviations. SDCS = Speech Disorders Classification System. [a]HL+ *n* = 22; HL– *n*s: 44–46. [b]HL+ *n*s: 14–15; HL– *n*s: 47–48. [c]HL+ *n*s: 11–12; H– *n*s: 50–51.
*$p < .05$. **$p < .01$. (Wilcoxon-Mann-Whitney test).

To summarize, the data in Table 7 provide strong evidence for the association of hearing status during 12–18 months and speech status at 3 years of age. Statistically significant differences were obtained on 5 of the 10 conceptually different speech measures, each indicating increased risk for subclinical or clinical speech disorder for children who met the criterion for hearing loss at 12–18 months. Based on the PCC-R and PPC-R data, the most conservative summary is that children with hearing loss as defined in this study had approximately 10 times the risk for speech disorder. As shown in Table 7, one fifth (20%) to one third (33.3%) of children with 20 dB or greater hearing losses at 12–18 months (HL+) had a subclinical or clinical speech disorder at 3 years of age (termed the positive predictive value), compared to a 2% affected rate for children who did not meet the 20 dB hearing loss criterion (HL–). The 2% rate in children classified as HL– is consistent with prior estimates of 2–3% prevalence rates of speech disorder in preschool children (Leske, 1981; Shriberg & Austin, 1998; Shriberg & Kwiatkowski, 1994; Winitz & Darley, 1980). However, it is lower than the recent estimate of approximately 14% in 3-year-old children based on extrapolation from data obtained on 6-year-old children (Shriberg, Tomblin, & McSweeny, 1999). Even if based on the more recent prevalence estimate, the present findings of speech delay at 3 years of age in 20% to 33% of children with HL+ histories would implicate HL+ as a significant risk factor for later speech delay.

It should be noted that the magnitude of a positive predictive value is greatly influenced by the prevalence rate in the sample. In the present sample, there were only 6 children of the 63 classified who were positive for speech disorder. Sensitivity (detection of true positives for later speech disorder) of HL+ at 12–18 months was 83.3%; of the 6 children positive for speech disorder at 3 years, 5 were detected as HL+ at 12–18 months. Specificity (rejection of true negatives for later speech disorder) for HL– at 12–18 months was 81.0%; of the 58 children negative for speech disorder at 3 years, 47 were classified as HL– at 12–18 months.

Structural Equation Modeling

Overview

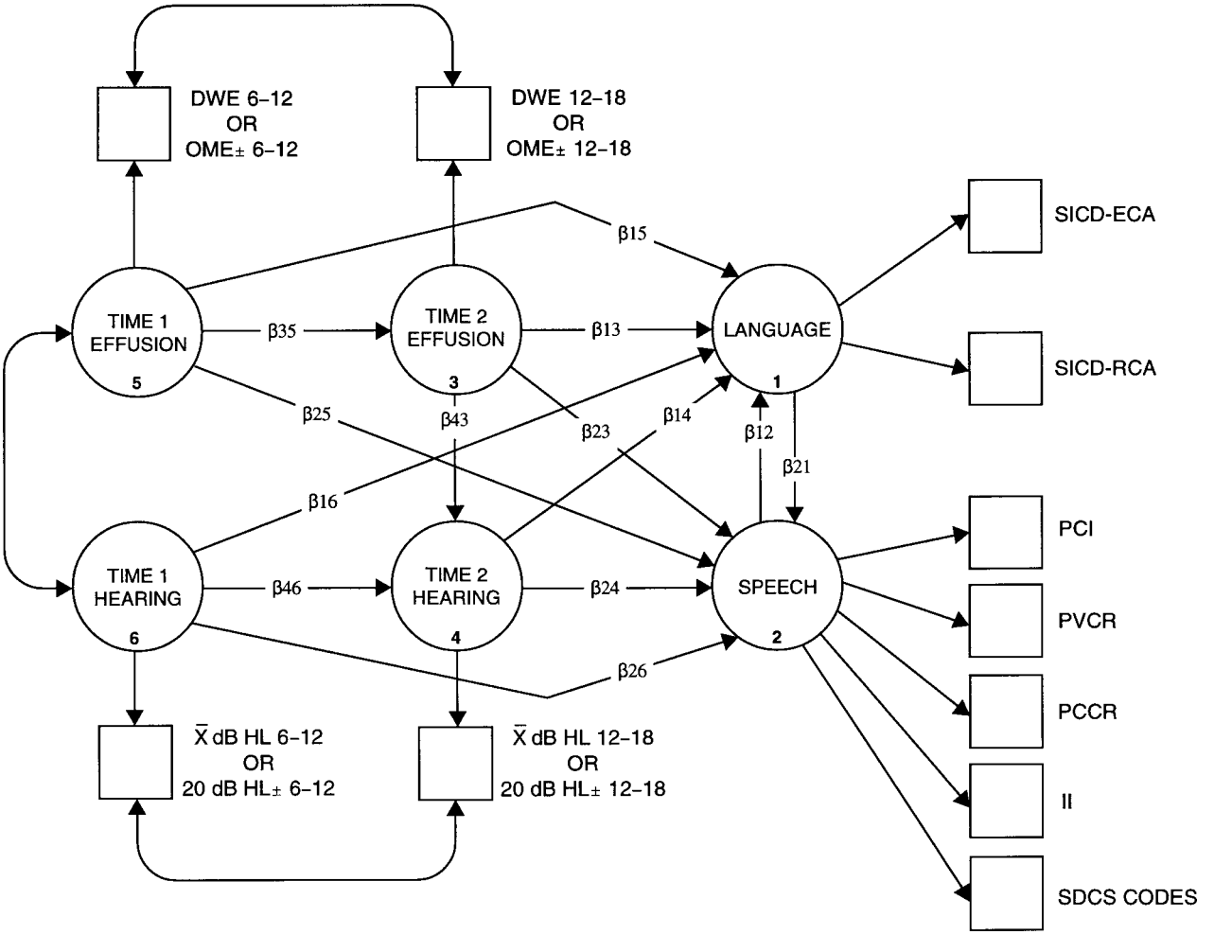
Background. Of several implementations of structural equation modeling (e.g., AMOS [Arbuckle, 1997]; EQS [Bentler, 1993]; LISCOMP [Muthén, 1988]), LISREL (Jöreskog & Sörbom, 1993) was used to develop a preliminary explanatory model linking precursor to outcome variables. The primary task in structural equation modeling (SEM) is to develop and solve two main sets of equations: measurement equations and structural equations. In SEM technique, theoretical constructs can be treated as latent variables, measurement

error variance can be separated from the measurement structure, and precursor or risk variables can be tested for direct or indirect (mediating) effects on outcome variables. The Appendix provides a technical description (see also Roberts et al., 1995).

Description of the Model. Figure 3 is a nonrecursive structural equation model developed to test the direct and mediated effects of OME and hearing levels at 6–12 months and 12–18 months on speech and language at 3 years of age. Both the measurement model and the structural equation model were conducted using a two-stage least-squares (2SLS) estimation procedure from the SIMPLIS computer program (Jöreskog & Sörbom, 1986). The 2SLS procedure is a noniterative method that is robust to many violations and performs similarly to optimal iterative methods (Brown, 1990; Hägglund, 1982). Several 2SLS models were run using data from 59 of the 70 children with complete hearing loss and language data. To support findings based on the small sample size, SEM technique models were compared using both continuous and categorical classification of the precursor and outcome variables.

The labels in the top and bottom rows of Figure 3 indicate the continuous (DWE, HL) and categorical (OME±, HL±) SEM models. The labeled arrows indicate the direct effects of each effect path using customary LISREL notation. The variables in the six circles are labeled in pairs from right to left (i.e., Language = 1, Speech = 2; Time 2 Effusion = 3, Time 2 Hearing = 4; Time 1 Effusion = 5, and Time 1 Hearing = 6). Thus, for example, the equation representing the direct effect of Time 1 Effusion on Language is designated *β15* (i.e., the effect on endogenous variable *1* (Language) of exogenous variable *5 (*Time 1 Effusion). The abbreviations on the right side of Figure 3 are for the two language measures and five of the nine speech measures described previously. The following sections describe procedures and findings used to support the preliminary model in Figure 3.

Figure 3. A structural equation model for the precursor and outcome variables in the Dallas subsample.



Measurement Structure

Procedure. The first SEM analysis tested the measurement structures for language and speech shown in Figure 3. The question was whether language and speech should be treated as separate or combined domains and which measures should be used for each domain. The following six widely accepted fit indices were used to test how well the measurement structure met goodness of fit criteria: (a) likelihood ratio test, which represents a good fit by a nonsignificant chi-square value; (b) the ratio of the chi-square to the degrees of freedom in the model, with a ratio below 3:00 indicating a good fit; (c) the Goodness of Fit Index (GFI); (d) the Adjusted Goodness of Fit Index (AGFI); (e) the Normed Fit Index (NFI); and (f) the Non-Normed Fit Index (NNFI). Values greater than .90 are desirable for the last four indices; Mulaik et al. (1989) provide details on the NFI and NNFI, and Jöreskog and Sörbom (1993) provide details on GFI and AGFI.

Findings. Statistical assessment of the entire set of measures indicated that the two dimensional measurement model shown in Figure 3 was the best measurement structure, including combining the two SICD modalities—receptive and expressive language—as one latent trait. Assessment of the measurement model met all fit criteria as reflected in the following fit index findings: $\chi^2 = 13.57$, $df = 16$, $pr = .63$, $\chi^2/df = .848$, GFI = .99, AGFI = .97, NFI = .98, and NNFI = .99. The two language and five speech measures did not contribute equally to their latent structures based on comparisons of the contribution of each measure's standardized value (Jöreskog & Sörbom, 1993): $\chi^2 = 71.70$, $df = 21$, $pr = .00$, $\chi^2/df = 3.41$, GFI = .92, AGFI = .90, NFI = .90, and NNFI = .93. Loadings for the receptive and expressive language scales of the SICD were approximately similar, but loadings for the five speech measures did not meet equivalence criteria, with the PCI contributing most to Speech. The obtained Speech model is readily supported conceptually, with each of the five measures reflecting a unique aspect of speech competence.

Effects of Early OME and Hearing Loss on Speech-Language at 3 Years

Decomposition of variance in a nonrecursive SEM is not an additive approach, but rather depends on the complex interactions in the system under study. A series of two-wave nonrecursive models was constructed for the model shown in Figure 3. As shown, the direct effects on Speech and Language include the four paths from the precursor variables OME (Time 1 and Time 2) and Hearing (Time 1 and Time 2), plus a path from the other communicative domain (Speech or Language). The remaining links in Figure 3 contribute indirectly to the total variance in the system. A two-part analysis was completed to compute total and direct effects and to determine if any one or more variables was substantially mediating the direct effects of a variable.

Total and Direct Effects on Speech and Language at 3 Years. Table 8 is a summary of the variance decomposition for the five sources of influence linking OME and Hearing Loss to Language and Speech. As shown in the first row, the overall R^2s for language and speech from the continuous and categorical models ranged from .402 to .515. The values for each of the two Language models (continuous and categorical) were within approximately 3 points of one another, and those for Speech within approximately 9 points of one another, indicating fairly good agreement for the two SEM approaches. As the SEM model based on continuous data is presumably more sensitive to individual differences in precursor and outcome variables, the continuous values will be used for the remaining discussion.

Continuing discussion of the overall R^2 values, these data indicate that the structural model of OME and hearing loss at the two time periods shown in Figure 3 accounts for approximately 52% of the variance in Language scores and approximately 40% of the variance in Speech scores at 3 years of age. The magnitude of this total variance is considerably higher than the zero-order correlations for OME and hearing levels with each of the individual language and speech measures (see Table 3). However, the R^2 values of approximately 52% and 40% for Language and Speech, respectively, are consistent with two of the intercorrelations for the SICD-E with speech measures shown in Table 2. As indicated in the third column from the left in Table 2, the PCC-R and Intelligibility Index have Pearson correlations of .46 and .49, respectively, with the SICD-E. Thus, a question is whether language and speech status at 3 years of age is responsible for the major effect of the system of influences on the total R^2s in the first row of Table 8.

To address this question, the other entries in Table 8

Table 8. Variance decomposition for the overall effects on language and speech for continuous and categorical structural equation models.

	Language		Speech	
	Continuous	Categorical	Continuous	Categorical
Overall R^2	.515	.484	.402	.489
R^2 difference with variable link removed				
Language			.256	.269
Speech	.209	.273		
OME 6–12	.050	.000	.001	.001
OME 12–18	.070	.016	.014	.068
Hearing 6–12	.095	.076	.095	.102
Hearing 12–18	.259	.123	.098	.018

provide information on the contribution of each of the five sources to the total R^2 values for Language and Speech. These values are the differences in the R^2 values between the overall R^2 and the R^2 with the variable link removed from the system. The higher the value in each column, the greater the contribution of that variable to the total R^2 in the first row of each column. Beginning with Language, hearing levels at 12–18 months (25.9%) are associated with approximately half of the total R^2 value for the entire system (.259/.515 = 50.3%), Speech another approximately 41% (.209/.515), and the other three precursors each from 10% to 19% (.050–.095/.515). For Speech, Language contributes approximately 64% to the total R^2 (.256/.402), hearing levels at 6–12 months (.095/.402) and 12–18 months (.098/.402) approximately 24% each, and the OME variables less than 1% (OME 6–12: .001/.402) and approximately 4% (OME 12–18: .014/.402). Thus, the values in Table 8 indicate that it is not the case that all of the variance in Language and Speech at 3 years of age is due to the association between domains. Rather, hearing levels at 12–18 months have an almost 10% greater effect (50.3%) on Language at 3 years than does Speech at 3 years (40.6%). For Speech at 3 years, however, hearing levels at 12–18 months contribute only 24.4% to the total variance, with Language accounting for the substantial remainder (63.7%). As with the zero-order correlations in Table 2, these findings are not interpretable further because they could mask the effects of mediator variables somewhere in the system—the question addressed in the final analysis of the SEM data. What can be supported to this point is that language and speech competence at 3 years of age appeared to be (a) substantially influenced by each other, (b) differentially influenced by hearing levels at 12–18 months, and (c) little influenced by frequency of OME at either 6–12 months or 12–18 months.

Direct and Mediated Effects on Speech and Language. Table 9 is a summary of the effects on language and speech at 3 years decomposed into direct and mediated influences (Chen, 1983). It is useful to begin in the middle of the table where the total effects of hearing levels at 12–18 months on Language at 3 years is indicated as –.510. As effects are given in standardized values, this indicates that a negative change in Language of approximately one half of a standard deviation unit occurs for each one standard deviation elevation in hearing levels at 12–18 months. As hearing levels at 12–18 months are also mediated by speech, this total effect can be decomposed into two effects in the system. One is the direct effect of Hearing at 12–18 months on Language (–.820), which accounts for 72.6% of the total effect (see fourth column from left in Table 9). The second is the mediated effect of Speech at 3 years, which, at .309 standardized units, accounts for the remaining 27.4% of the effect. Thus, on an absolute basis, direct effects of elevated hearing levels accounted for nearly three fourths of the total effects on language scores at 3 years (72.6%), with mediation by speech accounting for the remaining approximately one fourth (27.4%).

Turning to the effects of hearing levels at 12–18 months on Speech at 3 years, examination of the decomposition of the total effects indicates a puzzling finding. Although the standardized total effect of hearing levels at 12–18 months on Speech at 3 years is only .118, the positive algebraic sign indicates a positive association between elevated hearing levels (i.e., hearing loss) and speech competence. Decomposition into direct and mediated effects indicates approximately equal contributions

Table 9. Direct and mediated influences of hearing levels at 6–12 months and 12–18 months on language and speech status at 3 years of age.

	Language			Speech		
	Path(s)	Standardized effect	Absolute % of effect	Path(s)	Standardized effect	Absolute % of effect
Hearing 6–12 months influence						
Total effect	β16 + β46β24β12 + β46β14 + β26β12	–.100	100%	β26 + β46β14β21 + β46β24 + β16β21	–.139	100%
Direct effects	β16	.273	31.8%	β26	–.280	33.8%
Mediated effects	β46β24β12	.105	12.2%	β46β14β21	–.170	20.5%
Mediated effects	β26β12	–.201	23.4%	β46β24	.210	25.4%
Mediated effects	β46β14	–.278	32.6%	β16β21	.166	20.3%
Hearing 12–18 months influence						
Total effect	β14 + β24β12	–.510	100%	β24 + β14β21	.118	100%
Direct effect	β14	–.820	72.6%	β24	.618	55.3%
Mediated effects	β24β12	.309	27.4%	β14β21	–.500	44.7%

Note. It must be remembered that all paths in this model are mediated to some degree by speech and language since this is a nonrecursive model structure with a reciprocal link between language and speech. See Figure 3 for key to paths.

from direct effects (.618 standard deviation units, 53.3% of the total effect) and indirect effects (–.500 standard deviation units, 44.7% of the total effect). Thus, the mediated effects are appropriately in the negative direction, but the positive direct effects suggest that elevated hearing levels are associated with better speech scores. Such trends could reflect measurement problems with speech or, if reliable, could warrant examination for understanding the potential protective factors associated with environmental response to elevated hearing levels.

Finally, the data in Table 9 indicate that total effects of hearing levels at 6–12 months on Language (–.100 total standardized effect) and Speech (–.139 total standardized effect) were relatively small. Decomposition of the percentages indicates that only approximately one third of the effects of hearing levels at 6–12 months on later Language and Speech were direct, with approximately two thirds mediated by the three pathways shown in Figure 3.

Discussion

Findings from a prior and the present study indicate that OME and associated elevated hearing levels increase the risk for lowered speech-language outcomes at 3 years of age (Shriberg et al., 2000). Some measurement and design constraints in the present study include limitations in (a) the number of hearing assessments used to estimate hearing levels during the two developmental periods; (b) the language and speech measures, particularly the language measures, relative to other comprehension and production domains of contemporary interest; (c) the higher language status of the children in this sample relative to national norms; (d) the cell sizes, with power constraints affecting both the risk analyses and the structural equation modeling; and (e) the sociodemographic composition of the sample. Cross validation of the risk and SEM findings using more well-developed measures and larger and more sociodemographically diverse cohorts will be needed to support generalizations from the following observations.

Multifactorial Risk Models

A substantive concept emphasized in a previous report and suggested again by the present findings is the need for well-developed multifactorial models of risk precursors associated with early OME and later speech-language delay (Shriberg et al., 2000). As concluded by all researchers and reviewers of this literature, the speech-language sequelae of OME appear to depend on complex associations among child and environment variables. In the Shriberg et al. (2000) study, days with effusion was associated with significantly increased risk for subclinical or clinical speech disorder in only one of two samples. Early recurrent OME was associated with approximately 4.6 increased risk for later speech disorder in a sample of 50 children of Native American background enrolled in a Head Start program on a reservation but was not associated with significantly increased risk for speech disorder in a sample of 35 children followed since birth in a university pediatrics clinic. In the present study, using similar speech assessment methods, days with effusion was also not significantly associated with clinical or subclinical speech disorder, but hearing loss at 12–18 months was associated with approximately 10–21 times increased risk for later subclinical or clinical speech disorder. The sociodemographic composition of the 35 children in the pediatrics sample in the prior study was similar to the sociodemographic profile of the 70 children in the present sample followed from 2 months of age in a private pediatric practice. Moreover, the structural equation modeling indicated that although hearing levels predicted a sizeable proportion of the variance in language and speech at 3 years (as much as 52%), a substantial proportion of variance was unaccounted for by OME and hearing data at 6–18 months. Thus, a multifactorial model, allowing for potential protective, mitigating, and exacerbating variables moderating or mediating the effects of significant hearing loss on speech-language, appears to be the most appropriate current perspective on the risk findings.

Structural Equation Modeling

Findings from the second goal of this study suggest that the treatment of language and speech as independent latent traits is statistically well motivated and holds promise for conceptual and applied research needs. The most intriguing finding of the preliminary SEM analyses is that the significant and substantial effects of hearing levels at 12–18 months on speech at 3 years were significantly mediated by language at 3 years. One conceptual perspective is that the statistical data might reflect alternative views of how auditory deficits affect speech perception, which, in turn, affects the acquisition of phonological forms of the ambient language. The preliminary data from the SEM findings might be interpreted in relation to two alternative accounts.

Direct Effects, Acoustic-Phonetic Account. Speech acquisition models that emphasize the centrality of the acoustic-phonetic features of speech would predict that early recurrent fluctuant hearing loss would have specific consequences for the perception, discrimination, and representation of phonemes and their allophonic variations. Speech sounds with the lowest phonetic salience would be the most difficult to acquire correctly—specifically, those whose intensity-frequency characteristics are most consistent with the hearing loss configurations associated with OME (Dobie & Berlin,

1979; Groenen, Crul, & Maassen, 1992; Petinou & Schwartz, 1999). Incorrect or underspecified representations of these speech sounds would, in turn, manifest as specific speech production errors (cf. Shriberg & Smith, 1983). Crucially, such deficits could occur as relatively autonomous speech production phenomena directly related to the signal properties of the auditory input. Relative to the SEM model in Figure 3, the pathway from hearing loss at 12–18 months to speech status at 3 years ($\beta 24$) would appear to assess the strength of this association, wherein an early acoustic-phonetic processing constraint during this developmental period is viewed as the *direct* source of the later speech disorder.

Indirect (Mediated) Effects, Cognitive-Linguistic Account. Speech acquisition models that emphasize active discovery of linguistic forms, termed constructivist-type models (e.g., Aslin & Pisoni, 1980; Donahue, 1993; Goldinger, Pisoni, & Luce, 1996; Jusczyk, 1996; see also Ertmer & Newby, 1993) would predict less direct, substantially mediated effects of early fluctuant hearing loss on the acquisition of ambient linguistic forms. Rather than autonomous deficits based on acoustic-phonetic constraints, constructivist-type accounts predicate cognitive-linguistic sequelae of fluctuant hearing loss as the sources of concurrent and later deficits in speech-language and other learning domains. All indirect pathways in Figure 3, in which the effects of early hearing levels on later speech are mediated by language at 3 years, would appear to be consistent with this alternative to a direct effects, acoustic-phonetic model.

Support for the Mediated Account. Several findings in the present study and associated research support a mediated, rather than direct acoustic-phonetic, account of the effects of hearing on speech. First are the clear findings in the SEM data indicating that the apparently direct effects of hearing levels on these children's speech at 3 years were substantially mediated by their language status at 3 years. Second, as described previously, the obtained speech effects were not limited to only acoustically less salient sounds but rather were spread across all consonant features and included diverse error types at both phonemic (deletions, substitutions) and allophonic (distortions) levels. A third consideration is a finding from associated research indicating that, among 85 children referred for speech disorder of unknown origin, those with substantial histories of OME had significantly lower Intelligibility Index scores (Shriberg & Friel-Patti, 1997). In the present data, speech measures contributed less than 27% to the variance in Intelligibility Index scores (cf. Table 2), with language, prosody, and other sociolinguistic and paralinguistic variables likely additional sources of variance (cf. Jerger, Jerger, Alford, & Abrams, 1983; Kent, 1992; Shriberg & Kwiatkowski, 1982).

A fourth consideration supporting the mediated-effects model is the data referenced previously suggesting that multifactorial models are needed to account for the long-term effects of OME on linguistic, academic, and social skills. Roberts et al. (1995) report that measures of home environment substantially mediated the direct effects of hearing loss on several measures of language and general development. The present SEM model was designed to explore fewer variables than studied in Roberts et al. (1995), focusing on issues associated with speech outcomes. Following the findings of these investigators, it is quite possible that home environment variables mediate the direct and indirect hearing level findings in the present data. What can be suggested from the data on variables at hand is that these findings fail to support a direct-effects, acoustic-phonetic account for the speech sequelae of early recurrent fluctuant hearing loss. Rather, they are consistent with a mediated effects account in which speech sequelae arise from correlates of impairments in cognitive-linguistic processes.

Conclusion

The central clinical conclusion from the present study is that for infants with greater than 20 dB average hearing loss from 12 to 18 months of age, the prevalence of subclinical or clinical speech disorder on the Percentage of Phonemes Correct (PPC) metric at 3 years was 33%. In comparison, for infants with averaged hearing levels equal to or less than 20 dB HL from 12 to 18 months, the prevalence of subclinical or clinical speech disorder on the PPC at 3 years of age was 2%. For the Percentage of Phonemes Correct–Revised (PPC-R), which is a more conservative metric that scores only deletions and substitutions of consonants and vowels/diphthongs as errors, the comparable prevalence of speech disorder at 3 years for children classified as HL+ and HL– at 12–18 months, respectively, was 20% versus 2%. Preliminary attempts to model the significant explanatory pathways suggest that the increased risk for speech errors is substantially mediated by children's language acquisition, which, in turn, supports multifactorial pathway models linking early recurrent OME and hearing loss to diverse child and environment factors.

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Appendix A. Structural Equation Modeling.

Structural equation modeling includes two main sets of equations: measurement equations and structural equations. The measurement equations describe relationships among the measured variables and the theoretical constructs presumed to underlie them. The set of measurement equations allows the researcher to assess the accuracy of the proposed measurements. The structural equations express the hypothesized relationships among the theoretical constructs in the system under study. The structural model may be defined as the following latent dependent and independent variable linear model:

$$\underset{\sim}{\eta} = \underset{\sim}{B}\underset{\sim}{\eta} + \underset{\sim}{\Gamma}\underset{\sim}{\xi} + \underset{\sim}{\zeta} \quad (1)$$

where $\underset{\sim}{\eta}' = (\eta_1, \eta_2, \ldots, \eta_m)$ and $\underset{\sim}{\xi}' = (\xi_1, \xi_2, \ldots, \xi_n)$ are the latent dependent and independent variables, and $\underset{\sim}{B}(m \times m)$ and $\underset{\sim}{\Gamma}(m \times n)$ are coefficient matrices, with $\underset{\sim}{\zeta}' = (\zeta_1, \zeta_2, \ldots, \zeta_m)$ a vector of residuals. Equation (1) represents the structural equations. The vectors of η and ξ are not directly observed, but are reflective of observed vectors $\underset{\sim}{y}' = (y_1, y_2, \ldots, y_p)$ and $\underset{\sim}{x}' = (x_1, x_2, \ldots, x_q)$, such that:

$$\underset{\sim}{y} = \underset{\sim}{\Lambda}_y\underset{\sim}{\eta} + \underset{\sim}{\varepsilon}, \quad (2)$$

and

$$\underset{\sim}{x} = \underset{\sim}{\Lambda}_x\underset{\sim}{\xi} + \underset{\sim}{\delta}, \quad (3)$$

where $\underset{\sim}{\Lambda}_y$ and $\underset{\sim}{\Lambda}_x$ are measurement coefficient matrices, and $\underset{\sim}{\varepsilon}$ and $\underset{\sim}{\delta}$ are errors in measurement. These represent the measurement equations. For more details regarding structural equation modeling, the reader is referred to Bollen (1989).