

Consonant Accuracy After Severe Pediatric Traumatic Brain Injury: A Prospective Cohort Study

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Purpose: The authors sought to describe longitudinal changes in Percentage of Consonants Correct—Revised (PCC–R) after severe pediatric traumatic brain injury (TBI), to compare the odds of normal-range PCC–R in children injured at older and younger ages, and to correlate predictor variables and PCC–R outcomes.

Method: In 56 children injured between age 1 month and 11 years, PCC–R was calculated over 12 monthly sessions beginning when the child produced ≥ 10 words. At each session, the authors compared odds of normal-range PCC–R in children injured at younger (≤ 60 months) and older (> 60 months) ages. Correlations were calculated between final PCC–R and age at injury, injury mechanism, gender, maternal education, residence, treatment, Glasgow Coma Score, and intact brain volume.

Results: PCC–Rs varied within and between children. Odds of normal-range PCC–R were significantly higher for the older than for the younger group at all sessions but the first; odds of normal-range PCC–R were 9 to 33 times higher in the older group in sessions 3 to 12. Age at injury was significantly correlated with final PCC–R.

Conclusion: Over a 12-month period, severe TBI had more adverse effects for children whose ages placed them in the most intensive phase of PCC–R development than for children injured later.

Key Words: neurologic disorders, pediatric traumatic brain injury, consonant production, speech sound disorders, speech recovery, neuroimaging, phonology

Traumatic brain injury (TBI) affects approximately 1.4 million U.S. children per year, and questions about a child's prospects for intelligible speech after TBI are among those most frequently voiced by family members (Campbell, Dollaghan, & Janosky, 2010). Some evidence exists concerning the impact of TBI on speech production in school-age children and adolescents. In line with findings after TBI in adulthood (Kuruville, Murdoch, & Goozè, 2007; 2008), persistent dysarthria is the most commonly reported speech deficit after TBI sustained during the school age and adolescent years. For example, in a

sample of 24 participants injured between ages 5 and 16 years whose speech was assessed using perceptual and instrumental measures from 6 to 101 months later, Cahill, Murdoch, and Theodoros (2005) reported substantial articulatory dysfunction with clinically significant consonant imprecision in 37% of cases and significantly poorer overall intelligibility in the children with TBI than in uninjured controls. In a longitudinal study of nine children injured between ages 5 and 16 years, Campbell and Dollaghan (1990) reported significant individual variability on a measure of speech intelligibility, and in their subsequent analyses, Campbell and Dollaghan (1994) showed clinically significant voice and/or prosody deficits in all participants 12 or more months postinjury.

Much less is known, however, about the impact of TBI on children injured during the preschool period, the time in which children are in the process of acquiring an inventory of consonant phonemes. Injuries are common in this age range; according to Langlois, Rutland-Brown, and Thomas (2006), the rate of emergency room visits after TBI is highest in children from birth to 4 years of age. In the few studies reporting on speech after TBI during the preschool years, the measures employed have typically conflated speech production with other aspects of communication (e.g., Anderson, Catroppa, Morse, Haritou, & Rosenfeld, 2005; Barlow, Thomson, Johnson, & Minns, 2005; Ewing-Cobbs, Prasad, & Hasan, 2008; Hanten et al., 2009). No studies have tracked consonant production longitudinally in a manner that would

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enable comparisons between the developmental trajectories of injured and uninjured children or of children injured at different points in development.

Understanding the impact of TBI on young children's speech development is of particular interest in light of recent evidence from other developmental domains suggesting that "younger is not always better" with respect to recovery from TBI (e.g., Giza & Prins, 2006). Investigators have reported that mortality rates (Luerssen, Klauber, & Marshall, 1988; Tokutomi et al., 2008), cognitive outcomes (Anderson et al., 2005; Ewing-Cobbs, Barnes, & Fletcher, 2003; Lah, Epps, Levick, & Parry, 2011), social outcomes (Sonnenberg, Dupuis, & Rumney, 2010), language outcomes (Hanten et al., 2009), behavioral outcomes (Wetherington, Hooper, Keenan, Nocera, & Runyan, 2010), and long-term functional outcomes (Bagnato & Feldman, 1989; Koskiniemi, Kyykkä, Nybo, & Jarho, 1995) are poorer in younger children after TBI than in older children, particularly when the injury is severe.

Some recent behavioral evidence suggests that developmental skills undergoing rapid change at the time of injury are more susceptible to serious disruption than skills that are more fully developed (Anderson et al., 2005; Ewing-Cobbs, Prasad, & Hasan, 2008). Ewing-Cobbs et al. (2006) reported that IQ recovery curves during the first year after moderate-severe TBI indicated less recovery in infants and preschoolers than in older children. Catroppa et al. (2009) found significantly worse reading outcomes—up to 7 years after the injury—for children injured from age 3 to 7 years than for those injured later. Some investigators have reported relatively poorer outcomes on measures of later-developing discourse and pragmatic language skills in cross-sectional samples of children injured at younger ages (≤ 4 years) than in those injured at older ages (Chapman, Levin, Wanek, Weyrauch, & Kufera, 1998; Ewing-Cobbs, Miner, Fletcher, & Levin, 1989). However, Ewing-Cobbs and colleagues (2003) noted that strong inferences about the extent to which outcomes vary in children injured at different points in the acquisition of a skill require a well-specified model of its developmental trajectory, ideally based on a single measure appropriate throughout the developmental curve.

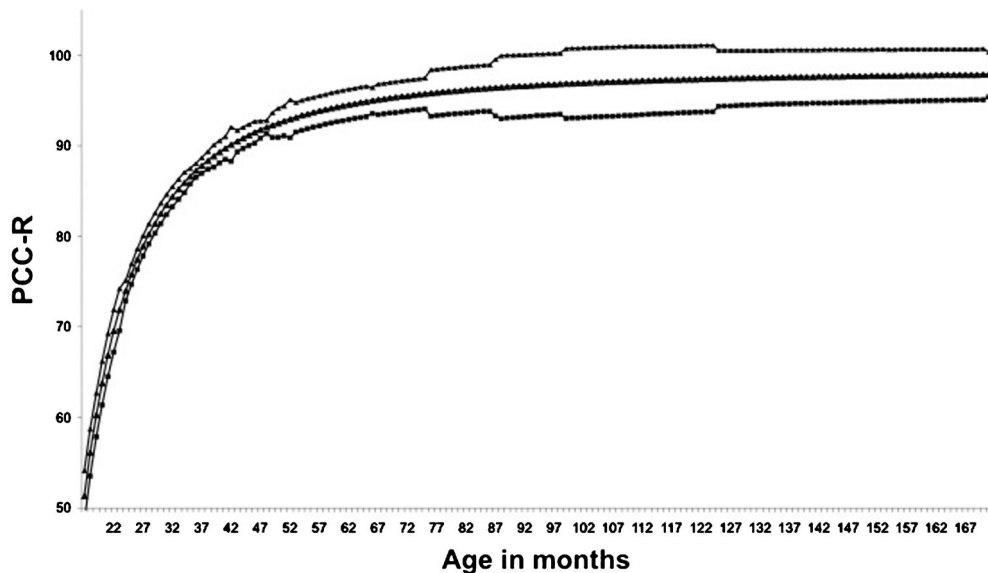
Such a developmental model and metric have become available for one important speech skill in English-speaking children: the ability to produce recognizable consonants. The metric, the Percentage of Consonants Correct—Revised (PCC-R; Shriberg, Austin, Lewis, McSweeney, & Wilson, 1997a), is well suited for testing the hypothesis that skills undergoing intensive development at the time of a childhood TBI will be more seriously affected than well-practiced skills. PCC-R is one of a suite of articulation measures that, having been derived from samples of connected conversational speech, are appropriate for children ranging in age from 18 months through adolescence; these measures are not vulnerable to the practice effects that can invalidate more structured tests on repeated administrations (Campbell, Dollaghan, Janosky, & Adelson, 2007). PCC-R reflects the number of consonants produced correctly, defined as not being omitted or substituted, relative to the total number of consonants in the words spoken by the child in a sample of conversational

speech. In contrast with metrics such as the Percentage of Consonants Correct (PCC) and the Percentage of Consonants Correct—Adjusted (PCC-A; Shriberg et al., 1997a), in calculating the PCC-R, researchers do not treat consonant distortions as errors. Accordingly, PCC-R reflects the basic parameter of consonant accuracy, independent of abnormalities that occur during production of a consonant but do not alter its phoneme category. Such distortion errors have been observed after TBI in school-age children (Campbell & Dollaghan, 1994; 1995), but because these errors are relatively common during the early phases of speech development in uninjured children, it is difficult to gauge their importance in young children after TBI. Shriberg et al. (1997a) recommended PCC-R for comparisons involving speakers of diverse ages and diverse speech status (p. 720); in addition, Shriberg, Austin, Lewis, McSweeney, and Wilson (1997b) reported that PCC-R is "the one best measure of articulation competence" (p. 731) for distinguishing between normal and abnormal speech development in children ages 3–8 years.

In addition to its appropriateness for children across a wide age span, PCC-R has a well-specified developmental model. As a prerequisite to the present study, Campbell et al. (2007) constructed a developmental function for expected growth in PCC-R at a fine-grained monthly level by applying curve-fitting procedures to PCC-R data obtained from several investigations (Paradise et al., 2001; Shriberg et al., 1997a; Stoel-Gammon, Kelly, Tinsley, & Kellogg, 1987) of typically developing children ($N = 1,858$) who were examined between the ages of 18 and 172 months. As detailed in Campbell et al., from among several thousand models generated from these compiled empirical data, a developmentally plausible and parsimonious model with $R^2 > .98$ ($p < .0005$) was selected as the basis for generating the expected PCC-R, *SD*, *SE*, and 99% confidence interval (CI) at each month of age in this age range. Figure 1, adapted from Campbell et al., shows the normal performance curve for PCC-R with the upper and lower bounds of its 99% CI for each monthly age. As illustrated in this figure, PCC-R does not increase at a constant linear rate. Its trajectory is relatively steep, initially, but its rate of increase slows substantially by around 60 months, the approximate age at which English-speaking children are expected to have acquired a nearly complete inventory of consonant phonemes (Grunwell, 1987; James, van Doorn, & McLeod, 2002; Porter & Hodson, 2001; Smit, Hand, Freilinger, Bernthal, & Bird, 1990). The normal performance curve thus provides an opportunity to evaluate PCC-R in children injured at different points in its developmental trajectory as well as an objective definition of the threshold for normal-range performance in the form of the lower bound of the 99% CI for PCC-R at each monthly age. Campbell et al. (2010) provided evidence that this definition distinguishes between preschool children diagnosed with normal speech acquisition or with speech disorder, supporting its use to determine whether a child's PCC-R falls above the threshold for the normal range at each monthly age.

The extent to which children's speech outcomes after severe TBI can be predicted is another question of considerable

Figure 1. Normal performance curve for PCC-R, with the upper and lower bounds of its 99% confidence interval (CI) for each monthly age. Adapted from Figure 1 in T. Campbell, C. Dollaghan, J. Janosky, & P. D. Adelson (2007), "A performance curve for assessing change in Percentage of Consonants Correct—Revised (PCC-R)," *Journal of Speech, Language, and Hearing Research*, Vol. 50, Issue 4, p. 1113.



interest. A number of variables have been linked, albeit inconsistently, to outcomes after TBI in other domains of development. Variables including gender, race/ethnicity, socioeconomic status, and indicators of family functioning have been reported to mediate behavioral and neuropsychological outcomes following severe TBI in some studies (e.g., Anderson, Morse, Catroppa, Haritou, & Rosenfeld, 2004; Haider et al., 2007; Keenan, Hooper, Wetherington, Nocera, & Runyan, 2007) but not in others (e.g., Anderson, Catroppa, Morse, Haritou, & Rosenfeld, 2009; Ewing-Cobbs et al., 2006; Yeates, Taylor, Walz, Stancin, & Wade, 2010). Studies of the association between injury severity and outcomes after pediatric TBI have also yielded inconsistent results, due at least in part to the difficulty of classifying the diffuse, extensive, and widely varying neurological sequelae of TBI (Kochanek, Bell, & Bayir, 2010; O'Connor, Smyth, & Gilchrist, 2011). Behavioral measures of severity such as Glasgow Coma Scale scores and duration of posttraumatic amnesia have been linked to outcomes in adults, but their value for children has been questioned (e.g., Forsyth & Waugh, 2010; Shore et al., 2007). A wide variety of potential severity indicators for children based on neuroimaging and neurophysiological protocols have been investigated in recent years, with varying results (e.g., Ashwal, Holshouser, & Tong, 2006; Beauchamp et al., 2011; Ewing-Cobbs, Prasad, Swank, et al., 2008; Power, Catroppa, Coleman, Ditchfield, & Anderson, 2007; Sigmund et al., 2007). No evidence exists on the relationship of any such factors to speech outcomes after severe pediatric TBI.

In the present study, we examined consonant production in a prospective cohort of 56 children who sustained a severe TBI between ages 0;1 (years;months) and 10;6. For

each child, PCC-R scores were calculated each month over a 12-month period, beginning when the child produced at least 10 recognizable words. The study had three specific aims. The first was to describe the longitudinal changes in PCC-R scores for individual participants with severe TBI and for the group as a whole. The second was to determine whether the odds of normal-range PCC-R scores differed significantly for children injured at or before 60 months of age and in children injured from 61 to 126 months of age. The third was to determine whether any of eight potential predictor variables were significantly associated with PCC-R values at the end of the 12-month sampling period.

Method

Participants

We recruited participants in two phases. In the first phase, we identified potentially eligible participants from among children admitted to a Level 1 Pediatric Trauma Center between 1999 and 2004. Shortly after admission, the first author met with the parent(s) or legal guardian(s) to describe the study and to obtain consent to screen the child for possible inclusion. Parents who consented were interviewed to obtain information on the child's date of birth, languages spoken in the home, level of education of the child's mother, and whether the child had been diagnosed previously with any neurodevelopmental, speech, or language deficits. In addition, a research nurse reviewed the medical chart of consented children and entered the following information into the study database: date and type of injury, lowest (worst) Glasgow Coma Scale score during the acute

period, and whether the child's clinical CT scan during this time was positive for neurological damage.

We included in the second phase of the recruitment process all participants who met the following criteria: (a) severe TBI, defined as a Glasgow Coma Scale score ≤ 8 and a positive CT scan; (b) age at injury < 11 years; (c) injury not known or suspected to have resulted from abuse; and (d) monolingual English home environment and no previously diagnosed neurodevelopmental, speech, or language deficits according to parent report. The verbal output of these participants was monitored to identify the point at which speech sampling could begin, defined as the production of at least 10 recognizable words. In the acute care setting, study personnel monitored participants' word production daily; upon discharge, participants' parent(s) or guardians(s) were asked to keep a daily list of words produced by the child and to contact study personnel as soon as the child began speaking. Study personnel contacted parents by telephone every 2 weeks thereafter; the first speech sampling session was scheduled as soon as possible after the family reported that the child had begun speaking.

Of 99 children who were screened for possible inclusion in the study, 60 (61%) met the initial enrollment criteria and agreed to participate. Four of these 60 children were excluded from the study: two who failed to produce 10 recognizable words within the study's duration and two others whose parents had not reported a history of speech or language deficits during the initial intake screening in the trauma center shortly after injury, but later recalled that their child had been treated for articulation deficits in school. Accordingly, the study sample included 56 participants.

Procedure

Speech sampling sessions. The 12 speech sampling sessions were scheduled to occur monthly, beginning at the point when the child was observed or reported to be speaking at least 10 intelligible words. As noted below, the interval between the child's injury and the time when speech sampling could begin varied considerably, but no child was younger than 20 months of age at the first speech sampling session. At each sampling session a 15-min spontaneous connected speech sample was audio-recorded as the child and a trained examiner played and/or conversed in the presence of a standardized set of toys (including kitchen utensils, food items, and miniature characters, vehicles, and furniture). In addition, at the first session the child's hearing was screened by earphones or in a sound field, as appropriate, to ensure thresholds ≤ 20 dB at 1, 2, and 4 kHz.

Recording and processing of speech samples. Speech samples were recorded using a portable audiocassette recorder (Marantz PMD 402 or 502) and associated microphone (Shure UHF wireless system or Radio Shack 33-3003). Research assistants, all of whom had completed graduate-level coursework and advanced training in phonetic transcription, identified and transcribed phonetically the first 100 word types (i.e., unique words) using the transcription consensus procedures described in Shriberg, Kwiatkowski, and Hoffmann (1984). Trained research assistants entered

phonetic transcriptions into computer files for analysis using Programs to Examine Phonetic and Phonological Evaluation Records software (Shriberg, Allen, McSweeney, & Wilson, 2000).

To calculate interobserver reliability for phonetic transcription, a second, blinded, research assistant randomly selected and independently transcribed 70 conversational speech samples (10% of all samples recorded). The percentage of phoneme-by-phoneme agreement was 93%, a value consistent with those reported in other studies of children's speech production (Campbell et al., 2003; Shriberg, Tomblin, & McSweeney, 1999).

Measures

Speech. The speech metric calculated at each sampling session was PCC-R (Shriberg et al., 1997a), a measure of segmental consonant accuracy. As described previously, PCC-R is calculated by dividing the total number of correct consonants produced, defined as the sum of consonants not omitted or substituted, by the total number of consonants in the words spoken by the child.

To identify sampling sessions at which an individual child's PCC-R fell above the threshold for normal-range performance, his or her PCC-R was compared to the lower bound of the 99% CI for PCC-R expected of children of that age in months as derived from the normal performance curve shown in Figure 1 (Campbell et al., 2007). PCC-R values exceeding this threshold were defined as being within the normal range at that monthly session.

Associated variables. Nonparametric (Spearman) rank-order correlation coefficients were calculated between eight independent variables and standardized PCC-R (z) scores at the 12th and final sampling session. PCC-R z scores were used for this analysis because raw PCC-R scores vary with age; standardized scores enabled associations to be examined regardless of children's ages at the final session.

Four variables were reported by parents: the child's age at the time of injury, the child's gender, the mother's educational level (less than high school graduate, high school graduate, some college, or college graduate), and place of residence (urban, suburban, or rural). Two other potential correlates were ascertained from the medical record: the mechanism of the child's injury (motor vehicle-related, fall, or other blunt trauma) as recorded in the study database by the research nurse and the lowest Glasgow Coma Scale score within the first 24 hours following injury in the pediatric trauma center, as recorded by a neurosurgical resident or attending physician. Note that the range of possible Glasgow Coma Scale scores was restricted because a score ≤ 8 was a condition of participation in the study. One potential correlate, the total number of hours of treatment the child received for communication deficits during the 12-month speech-sampling period, was determined according to written logs of the duration of all such treatment sessions maintained by the child's parent and/or the treating speech-language pathologist.

The final variable considered as a potential correlate of PCC-R z score at the final sampling session was intact brain

volume as calculated from MRI. Although we performed all image processing on clinical MRI scans rather than high-resolution volumes, we chose to perform coarse morphometric analysis as a way to quantify the volume of intact brain instead of trying to quantify the degree of lost brain (lesion volume). This approach avoided the pitfalls that commonly accompany quantification of injury in clinical images in which the methods are most accurate in estimating the volumes of focal lesions of significant size but lose accuracy in accounting for ventricular enlargement and brain atrophy, which commonly accompany TBI and contribute significantly to the overall outcome of such individuals (Bigler, 2007).

Clinical MRI images, taken according to a standard protocol (axial T1, axial T2, axial T2*, coronal FLAIR, and axial proton density [PD]) within 2 months of the final speech sampling session, were available for those 48 participants who returned for follow up imaging. Because images were obtained for clinical decision making and not yet standardized, the acquisitions were not completely consistent across individuals; some were missing sequences and some had heterogeneity in planes, slice thickness, and field of view. As a result, the images could not be as straightforwardly combined (e.g., for multispectral analyses), as they are in studies with more controlled research acquisitions.

As a surrogate measure of premorbid brain volume, we manually measured the calvarial volume. Then, for each hemisphere, we measured the volumes of the fluid spaces of the brain, including the ventricles (and extensions due to the trauma), the sulci (assessing shrinkage), and regions of encephalomalacia (from traumatic lesions). By calculating the difference between calvarial volume and the sum of these nontissue volumes, we gained an informed estimate of posttraumatic brain tissue volume. We also counted the number of punctate lesions revealed in long TR sequences (as an approximate measure of axonal shear injury).

As an initial estimate of calvarial volume, we submitted the T2* axial volume as an input to the automask function in AFNI whole-brain mask (Cox, 1996), then refined the mask manually by using the aligned PD and T2 axial volumes as a reference underlay. Ventricular volume was calculated by first drawing a rough initial mask around the lateral and third ventricles on the T2 images. This initial mask was then refined by a three-step semi-automatic segmentation procedure: The first step in this procedure was to calculate image intensity histograms for white matter (WM) and cerebrospinal fluid (CSF) at six control points on the borders of ventricles (again using the T2 volume) to establish a cutoff value to discriminate between WM and CSF. In the second step, we used this cutoff threshold to create a binary signal-intensity-based mask. In the third (final) step, we multiplied the binary mask with the initial T2 mask to create the final image containing precise ventricular volumes. The same histogram-based process was used to create the sulcal mask.

Lesion volume was calculated by manually delimiting lesion borders using PD and T2 scans as guides, establishing

signal intensity borders using histograms over multiple slices in both scan types (where lesion tissue is hyperintense in T2 and either hyper- or hypointense in PD, relative to healthy white matter or grey matter), then refining the manually drawn lesion mask using these thresholds. Ventricles were also excluded from lesion masks; indeterminate cases were further defined by hand using the coronal FLAIR images as a guide. To identify and count punctate lesions in WM, small hypointensities were tagged in each slice using the T2* axial volumes. Finally, intact brain volume was calculated as the sum of ventricular, sulcal, and lesion volume subtracted from the calvarial volume.

Analyses

To describe longitudinal changes in PCC-R scores, we calculated group *M*s and ranges for PCC-R scores and PCC-R *z* scores at each of the 12 sampling sessions. Cuzick's nonparametric test for trend (two-sided) was employed to test for a significant linear trend in group *M* PCC-R *z* scores.

To determine whether the odds of normal-range PCC-R scores differed significantly for children injured at different points in the normal performance curve, we divided the sample into younger and older groups. Children in the younger group ($n = 23$) were injured during the relatively steeply rising portion of the normal performance curve, at or before 60 months of age. Children in the older group ($n = 33$) were injured after 60 months, a point after which PCC-R scores change minimally. The number of PCC-R scores falling above the threshold of the normal range was determined for each group at each session, and we calculated odds ratios and associated 95% CI for this binary outcome. An odds ratio of 1 would indicate that the odds of normal range PCC-R in the older and younger group did not differ. An odds ratio of 2 would indicate that the odds of normal-range PCC-R were twice as high in the older than in the younger group. Odds ratios of 2 or more have been defined as clinically significant by some investigators (e.g., Sackett, Haynes, Guyatt, & Tugwell, 1991), particularly if the lower bound of the 95% CI also exceeds this level.

To address the third objective of the study, we calculated univariate Spearman rank-order correlation coefficients between the eight independent variables and PCC-R *z* scores at the final sampling session standardized to the *M* and *SD* of the normal performance curve.

Results

Information on the 56 participants is summarized in the Appendix. Age of injury ranged from 1 to 126 months ($M = 71$, $SD = 38$), and age at the first speech sampling session ranged from 20 to 127 months ($M = 75$, $SD = 35$). Speech sampling sessions began within 3 months of injury for most of the participants (84%), but for 11% of participants, more than 13 months elapsed before the child produced the requisite 10 words such that speech sampling sessions could begin.

Descriptive analyses. Complete PCC-R data sets were available from all 56 participants at Sessions 1 to 8;

Table 1. Mean and range of PCC–R and PCC–R *z* scores by monthly sampling session.

Session ^a	PCC–R score		PCC–R <i>z</i> score	
	<i>M</i>	Range	<i>M</i>	Range
1	87	47 to 100	–0.68	–6.59 to 4.09
2	88	43 to 100	–0.53	–7.12 to 2.98
3	90	43 to 100	–0.32	–6.89 to 1.90
4	90	47 to 100	–0.33	–7.22 to 1.68
5	90	46 to 100	–0.41	–7.83 to 2.03
6	91	58 to 100	–0.37	–4.91 to 1.12
7	91	59 to 100	–0.37	–5.38 to 2.06
8	91	53 to 100	–0.51	–6.64 to 2.20
9	92	60 to 100	–0.44	–6.11 to 1.77
10	92	56 to 100	–0.38	–6.44 to 1.85
11	92	65 to 100	–0.50	–5.21 to 0.48
12	92	60 to 100	–0.51	–5.90 to 1.74

Note. Percentage of Consonants Correct—Revised (PCC–R) *z* scores were calculated for each participant using the *M* and *SD* for his or her age in months on the normal performance curve.

^a*N* = 56 for Sessions 1–8; *N* = 55 for Sessions 9–12.

because one participant moved to a different city after Session 8, there were 55 participants at Sessions 9 to 12. Table 1 shows group *M*s and ranges for PCC–R scores and PCC–R *z* scores at the 12 sampling sessions.

Figure 2 plots individual participants' PCC–R *z* scores and group *M*s at each session. As shown in this figure, individual PCC–R *z* scores varied widely. Group *M* PCC–R *z* scores fell between 0.32 and 0.68 *SD*s below the mean

across the 12 sessions, and a test for linear trend was nonsignificant (two-sided *p* = .87, Cuzick's nonparametric test for trend).

Figure 3 is a matrix indicating the PCC–R values that fell above the threshold for normal-range performance for each participant at each sampling session. Columns correspond to the 12 monthly testing sessions; rows correspond to individual participants, ordered by age at injury. Age at the first session is also provided. White cells indicate sessions at which a participant's PCC–R score fell above the lower bound of the 99% CI for his or her monthly age; black cells indicate PCC–Rs below this threshold. The visual impression conveyed by this graph suggests that PCC–R values were below the normal range more often in the younger children (approximately the top half of the graph) than in the older children, although some older children had low PCC–R values at some or even all of the sampling sessions.

Figure 3 also shows considerable variability between and within children in PCC–R scores over time. The percentage of participants whose PCC–R scores fell above the threshold of the normal range varied from 57% to 73% across the 12 sessions. Twenty participants (36%) had normal-range PCC–R scores at all sessions; seven participants (13%) had PCC–R scores that never fell within the normal range. Another eight children (14%) had PCC–R scores that were below the lower bound of the normal range at the first two sessions but above it thereafter. In 19 participants (34%) PCC–R scores did not change in a consistent fashion, crossing the lower bound threshold more than once over the 12 sessions. Finally, one child's PCC–R score was above the

Figure 2. Individual participants' PCC–R *z* scores (denoted by diamonds) and group means (denoted by filled circles) at each monthly speech sampling session.

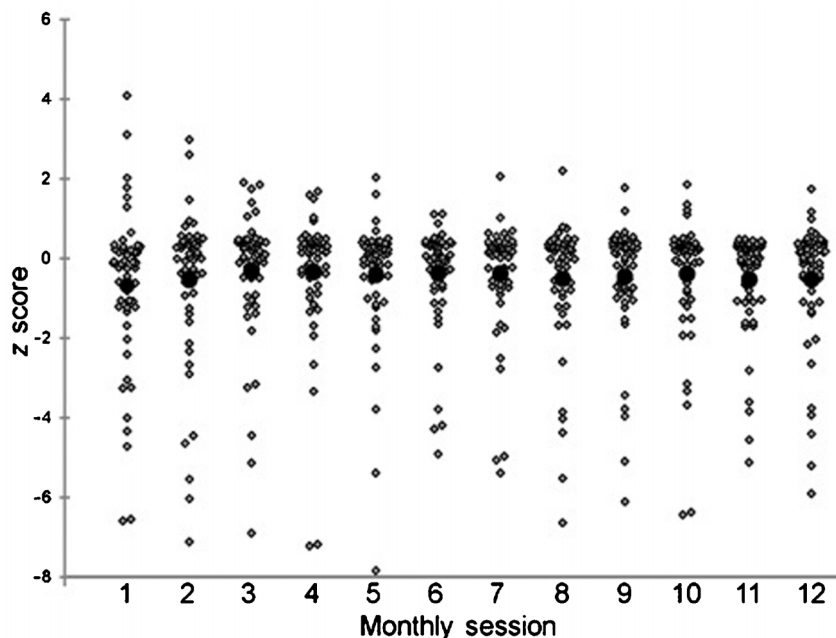


Figure 3. PCC-R values that fell above the threshold for normal-range performance for each participant at each sampling session. White cells indicate sessions at which PCC-R score fell above lower bound of 99% CI for his or her monthly age. Black cells indicate PCC-Rs below this threshold. ND = no data.

Age (mos) at injury	Age (mos) at Session1	Session number											
		1	2	3	4	5	6	7	8	9	10	11	12
1	21												
8	22									ND	ND	ND	ND
14	21												
19	20												
20	22												
21	43												
22	69												
22	22												
26	29												
27	56												
29	30												
30	43												
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lower bound at Session 1 but below this threshold at all remaining sessions.

Odds ratios for normal-range PCC-R scores in the younger and older groups. Twenty-three children were injured at or before 60 months of age; 33 children were injured at 61 months or later. Because one child in the younger group was not available for sessions 9 through 12, at these four sessions the younger group included 22 rather than 23 participants. Table 2 shows for each session the number and percentage of children in each group whose PCC-R scores fell above the threshold for the normal range and the corresponding odds ratio, CI, and *p* value. At every session after the first, the odds of normal-range PCC-R scores were significantly higher in children injured after 60 months than in children injured at earlier ages. From sessions 3 through 12, the odds of normal-range PCC-Rs were 9 to 33 times higher in the older than in the younger group, and lower bounds of all CIs exceeded 2.

Univariate correlations. Table 3 shows univariate nonparametric correlations between the eight independent variables and PCC-R *z* scores at the final sampling session. Only one statistically significant association, between age at injury and final PCC-R *z* score, was found ($\rho = .40$; $p = .001$); all other correlations had *p* values $> .05$.

Discussion

We studied PCC-R, a measure of accurate consonant production, in a prospective longitudinal cohort of 56 children who sustained a severe TBI between the ages of 1 month and 10 years. PCC-Rs varied considerably within and between children over 12 monthly speech-sampling sessions, with no significant linear trend over time. The odds of normal-range PCC-R scores were significantly higher in children injured after 60 months of age than in children

Table 2. The number and percentage of participants in each group whose PCC-R scores fell above the threshold for the normal range, as well as associated odds ratios (ORs) and 95% confidence intervals (CIs).

Session	Younger group ^a		Older group ^b		OR	95% CI
	<i>n</i>	%	<i>n</i>	%		
1	11	48	21	64	1.90	[0.65, 5.63]
2	9	39	25	76	4.86*	[1.53, 15.44]
3	10	43	29	88	9.43*	[2.49, 35.68]
4	9	39	29	88	11.28*	[2.95, 43.05]
5	11	48	30	91	10.91*	[2.58, 46.11]
6	7	30	30	91	22.86*	[5.19, 100.65]
7	10	43	29	88	13.00*	[3.06, 55.15]
8	8	35	28	85	10.50*	[2.92, 37.81]
9	6	27	29	88	19.33*	[4.75, 78.77]
10	7	32	31	94	33.21*	[6.58, 179.65]
11	7	32	29	88	15.54*	[3.92, 61.60]
12	7	32	30	91	21.43*	[4.84, 94.87]

* $p < .01$.

^a $N = 23$ for Sessions 1–8; $N = 22$ for Sessions 9–12. ^b $N = 33$ for all sessions.

Table 3. Univariate nonparametric correlations between the eight independent variables and PCC-R *z* scores at the final sampling session.

Independent variable	ρ	95% CI	<i>p</i>
Age at injury	.40	[.151, .598]	.001
Intact brain volume	.18	[-.112, .439]	.226
Glasgow Coma Score	-.17	[-.411, .100]	.218
Place of residence	-.16	[-.406, .106]	.234
Gender	-.12	[-.373, .145]	.367
Maternal education	-.09	[-.343, .178]	.513
Hours of treatment	-.04	[-.299, .225]	.772
Mechanism of injury	-.01	[-.276, .249]	.916

injured at younger ages at every session but the first; from the third to the 12th month of sampling, the odds of normal-range PCC-R were 9 to 33 times higher in the older than in the younger group. Of eight variables potentially associated with outcomes after TBI in childhood, only age at injury was significantly correlated, at a moderate level, with PCC-R at the final sampling session. These findings are consistent with other recent evidence contradicting the traditional expectation that children injured at younger ages will have better outcomes than children injured when older (Ewing-Cobbs, Prasad, Swank, et al., 2008); they also are consistent with the hypothesis that the functional consequences of severe pediatric TBI will differ for skills being developed at the time of injury and skills already established (Anderson et al., 2009).

The present study had a number of strengths, including its prospective longitudinal design, its sample size, and the inclusionary and exclusionary criteria that were used in an effort to reduce noninjury-related sources of heterogeneity among participants. The fact that speech was sampled longitudinally at consistent monthly intervals for a full year provided a more reliable perspective on intra-individual variability after TBI than would have resulted from less frequent sampling. The PCC-R measure also had several important advantages. First, by contrast with more contrived measures such as picture naming, PCC-R is derived from conversation, a context likely to be familiar to children of all ages. Second, because PCC-R is appropriate for children over the full age range we studied, inferences could be drawn from a single, consistent metric, avoiding the potential problems associated with comparing different measures at different ages. Third, the availability of a normative developmental trajectory for PCC-R enabled an objective means of determining, for each child at each monthly age, whether his or her score fell above the lower limit of the 99% CI for children of that age—a critically important advantage given that it is impossible to control the age at which a child is injured or the amount of time that elapses before he or she is able to speak after the injury.

The present investigation also had several limitations, some of which are inherent to the heterogeneous nature of the pediatric TBI population. First, although we attempted to exclude children who had developmental deficits prior to their injuries, we necessarily relied on parents as the source of

such information. Parent report is a well-accepted method for identifying young children at risk for developmental deficits at a particular point in time, but the validity of parents' retrospective judgments about developmental status is unknown. There appears to be no alternative to using retrospective parental reports to estimate preinjury status for the overwhelming majority of young children who have not undergone formal developmental assessments at the time when their injuries occur. However, evidence corroborating parental reports of normal development prior to a TBI could strengthen the inference that the relatively poorer PCC-R outcomes in the younger group can be attributed to age or developmental stage rather than to undetected developmental deficits that were present prior to injury.

A second limitation concerns the lack of information about the participants' outcomes after the 12th month of the sampling period. Although we followed each child for a full year, the longer-term impact of TBI could not be addressed within the resource constraints of this study. Accordingly, it is unknown whether the 18 children (33%) who did not have normal-range PCC-Rs at the final sampling session achieved this level at a later date.

Although the sample size of the present study was relatively large given the number of data points obtained longitudinally from each participant, it was insufficient to allow analyses of the extent to which PCC-R outcomes might vary systematically within narrower age ranges than the younger and older groups that were studied here. The binary cut that we imposed between 60 and 61 months is consistent with observations that the consonant inventory of English is essentially complete at age 5, and it also corresponds reasonably well with the point at which the PCC-R curve reaches asymptote. However, it could also be informative to examine narrower age ranges within the lower portion of the curve. For example, data at the final session were available for four of the five children in the present study who were injured before 21 months of age; 3 of these had normal range PCC-R scores at the final session. By contrast, of eight participants who were injured between 21 and 36 months—an interval during which the normal PCC-R curve increases sharply—none had a normal range PCC-R score at the final sampling session. It also would be of interest to compare the impact of TBI on specific consonants not yet acquired and those already mastered by an individual child, although this would require more detailed information on the child's preinjury speech production skills than is typically available. In any case, testing hypotheses about differential outcomes for children injured in narrower intervals, whether defined by age or by developmental stage for specific speech sounds, will require a larger number of participants than were available in the present sample.

As noted above, limited and inconsistent evidence has suggested that some of the eight variables we considered as potential predictors of PCC-R scores at the final session might contribute incrementally to cognitive and nonspeech linguistic outcomes (Anderson et al., 2009; Ewing-Cobbs, Prasad, & Hasan, 2008; Keenan et al., 2007; Yeates et al.,

2010). However, we found only one variable, age at injury, to be significantly associated with final PCC-R. The failure to find other significant associations could have resulted from a variety of factors. Efforts to identify measures of injury severity that are predictive of outcomes after severe TBI are ongoing (Oni et al., 2010; Shin et al., 2012; Wilde et al., 2010), and the lack of a significant correlation between intact brain volume and PCC-R outcome in the present study could reflect in part the limited capacity of clinical imaging to detect diffuse axonal damage after TBI (Ashwal et al., 2006). Alternatively, injury severity might better predict speech deficits at the motor-articulatory level, such as nasalization of nonnasal consonants or lateralization of sibilants (Cahill et al., 2005; Campbell & Dollaghan, 1994), than the basic ability to produce recognizable consonants that is reflected in PCC-R. Understanding the impact of the diffuse brain damage that accompanies severe TBI on the multiple levels of the phonological and speech production system as well as the relationship between disruptions of oral-motor control and injury sequelae in other motoric systems are questions for future research.

The present study represents a starting point for future longitudinal explorations of speech production after severe TBI in childhood. However, because we were able to study recovery longitudinally and prospectively in a relatively large sample of children, using a measure of consonant production that is derived from a naturalistic conversational context, appropriate across a broad age range, and for which a normal developmental function has been specified, the present study appears to provide the strongest evidence to date that severe pediatric TBI has more serious effects on skills that are not yet fully consolidated at the time of injury than on skills that are better established. Additional investigations employing longitudinal research designs, well-specified developmental trajectories for specific skills, and aggregated risk models are needed to improve our understanding of the long-term consequences of pediatric TBI on children's development in speech and other domains.

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Appendix

Descriptive Information on Participants ($N = 56$)

Gender

Male: 33
Female: 23

Race/Ethnicity

Caucasian: 47
African American: 5
Multiracial: 4

Maternal Education Level

< High school graduate: 5
High school graduate: 22
Some college: 24
College graduate: 5

Place of Residence

Urban: 25
Suburban: 15
Rural: 16

Mechanism of Injury

Motor vehicle-related: 40
Fall: 13
Other blunt trauma: 3

Glasgow Coma Score

Level 3–4: 10
Level 5–6: 18
Level 7–8: 28

Age (months) at Injury

M (SD): 71.14 (37.46)
Range: 1–126
 ≤ 60 months: 23
 ≥ 61 months: 33

Age (months) at First Sampling Session

M (SD): 74.84 (34.72)
Range: 20 – 127

Interval (months) Between Injury and First Sampling Session

M (SD): 3.70 (8.19)
Range: 0–47
Interval
0–3 months: 47
4–7 months: 3
 ≥ 13 months: 6

Number of Hours of Communication Treatment During the 12-Month Sampling Period

M (SD): 52.96 (51.05)
Range: 0–168

Intact Brain Volume ($N = 48$)

M (SD): 0.916 (0.035)
Range: 0.817–0.979
