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Associative learning in children with perinatal brain injury

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Abstract

Associate learning for visual nonverbal and auditory verbal items was examined in 21 children with spastic diplegic cerebral palsy (SDCP) and 28 healthy children using four paired associate tasks. SDCP children showed poorer performance than the comparison group for learning pairs that required visual nonverbal responses, regardless of the stimulus modality. Within the SDCP group, lesion severity was assessed in 17 of the children. Lesion severity was related to the level of performance on paired associate tasks requiring visual nonverbal responses; lesion severity did not reach statistical significance for tasks requiring auditory verbal responses. The study suggests: (1) periventricular white matter regions are important for the development of basic learning processes, such as associative learning, and (2) learning of visual nonverbal material is disproportionately affected following white matter injury early in life. (*JINS*, 1997, *3*, 521–527.)

Keywords: Cerebral palsy, Cognitive development, Periventricular leukomalacia, Magnetic resonance

INTRODUCTION

Associative learning is a fundamental aspect of memory and learning that underlies many aspects of higher level cognitive activity. Differences in basic associative memory capacities can account for differential performance on a wide range of higher cognitive functions, including conceptual and problem solving skills (Siegler, 1991). For example, Siegler (1988) has demonstrated the importance of a child's associative learning ability for their choice of strategies and performance when solving mathematical problems. Studies of adult humans and animals have identified several anatomical regions of particular importance for associative learning processes. These areas include medial temporal regions and the posterior portion of dorsolateral prefrontal cortex (Petrides, 1982, 1985, 1990).

Associative learning in children with brain dysfunction has primarily been studied in patients with focal seizure disorders. Jambaque et al. (1993) have reported performance for children with epilepsy on a range of memory tasks that included verbal and visual paired associate learning. These authors included subgroups of children with either idiopathic generalized, partial extratemporal, left temporal, right temporal, or bilateral temporal epilepsy. The partial extratemporal group was primarily composed of children with seizure foci in the frontal lobes. Verbal paired associative learning scores were lowered compared to the control group for the partial nontemporal, left temporal, and bilateral temporal subgroups. Visual paired associate learning scores were lowered in all five of the subgroups. Right temporal lobe epilepsy groups were similar for verbal and visual paired associate learning. This study indicated that deficits in associate learning are sometimes present in children with extratemporal lobe epilepsy. Jambaque and colleagues attributed these deficits to frontal lobe contributions to memory functions, which may impact some aspects of planning and organizing information during encoding. In addition, the authors noted their paired associate tasks were similar to conditional associative learning tasks, which have been shown to be disrupted by frontal lobe lesions in adults and nonhuman primates (Petrides, 1982, 1985, 1990).

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Table 1. Comparisons of means and standard deviations (age-based standardized score (S.S.), $M = 100$, $SD = 15$) on measures of vocabulary and spatial analysis

	Neurologically normal		SDCP group		
Test	*M*	*SD*	*M*	*SD*	*t*(43)
PPVT–R	93.2 (S.S. = 94)	19.2	82.0 (S.S. = 82)	39.1	1.32
Spatial Relations	18.4 (S.S. = 109)	3.7	10.5 (S.S. = 71)	7.4	4.89*

*$p < .01$.

The role of extratemporal injury in the development of associative learning processes, though mentioned in the study by Jambaque, has not been well characterized in children with nonepileptic brain insults. Disruption of associative learning processes early in life may have a particularly negative impact on cognitive development, as it may interfere with the development of higher level cognitive skills. The present study examined associate learning in a group of children with perinatal brain injury resulting in spastic diplegic cerebral palsy (SDCP).

SDCP is a nonprogressive developmental disorder typically resulting from premature birth and factors associated with perinatal asphyxia such as intrauterine complications (Volpe, 1976). The most common neuropathology associated with SDCP is periventricular leukomalacia, which tends to occur bilaterally and in a generally symmetrical pattern (Park et al., 1989; Shouman-Claeys et al., 1989; Yokochi et al., 1991; Truwit et al., 1992). Additional anatomical changes can include ventricular enlargement, thinning of the corpus collosum, and cortical scalloping, whereas damage to subcortical gray matter is unusual (Park et al., 1989; Shouman-Claeys et al., 1989; Yokochi et al., 1991; Truwit et al., 1992). The SDCP population may be useful for understanding the effects of extratemporal brain injury on associative learning, because of the relative sparing of medial temporal structures. In addition, the early onset of injury in SDCP makes it a good population for understanding possible developmental differences between early *versus* late disruption of associative learning processes.

METHOD

Research Participants

The 21 children (11 female, 10 male) with perinatal injury resulting in spastic diplegic cerebral palsy who participated in the study were recruited through the Department of Neurosurgery at St. Louis Children's Hospital. All cerebral palsy participants but 1 were premature, and the neurological insult presumed to have produced their condition was believed to have occurred during the perinatal period. Eleven SDCP participants experienced additional neurological difficulties during the first 8 weeks after birth. Three experienced hydrocephalus that was shunted, 6 had neonatal seizures, and 7 had an intraventricular hemorrhage.[1] No child had a history of additional brain insult after 8 weeks of age, though 1 child had a single generalized seizure at 5 years of age. For the purpose of the present study, all participants had an estimated IQ above 75, normal or corrected normal visual acuity, and normal hearing based on clinical records.

Children with SDCP were compared to 28 neurologically normal children (14 female, 14 male) who were recruited from elementary schools in the St. Louis community. The comparison group was selected to match the historical age and sex distribution of children seen in the clinic from which the children with spastic diplegia were recruited. Developmental histories were not available for the comparison children, though parents were asked not to have their child participate if the child had a neurological condition. To assess more general cognitive ability, all children received the Peabody Picture Vocabulary Test– Revised (PPVT–R; Dunn & Dunn, 1984), a measure of receptive vocabulary, and the Spatial Relations test of the Woodcock-Johnson Psycho-Educational Battery– Revised (WJ–R; Woodcock & Johnson, 1990), a measure of fluid reasoning and spatial analysis. The mean age of the SDCP group was 8.6 years ($SD = 3.0$, range = 5.6–16.0) and the mean age of the neurologically normal group was 8.5 years ($SD = 3.1$, range = 5.5–16.0). *T* tests revealed no difference between groups in age [$t(47) = 0.38$, $p > .05$]. Performance on the PPVT–R and WJ–R Spatial Relations Tests are shown in Table 1.

Paired Associate Learning Tasks

Four paired associate (PA) tasks were designed with stimulus–response pairs for each of the four possible combinations of auditory verbal and visual nonverbal items (i.e., verbal stimulus–verbal response, verbal stimulus–nonverbal response, nonverbal stimulus–verbal response, and nonver-

[1] For children with a history of hydrocephalus or an intraventricular hemorrhage, additional brain injury may have occurred in the postnatal or neonatal period rather than the perinatal period. These conditions may also cause injury to periventricular white matter, including cystic formations and white matter loss that is similar to that seen in SDCP without these complications (Hochwald, 1985; Hale et al., 1992). Thus, all children sustained injury prior to 2 months of age, though the precise period at which the white matter injuries were sustained may vary between children. The white matter injury also may have occurred in a subset of the children, due to a variety of mechanisms, such as initial perinatal ischemia and later pressure effects.

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bal stimulus–nonverbal response). Each paired associate list contained six stimulus–response pairs. Verbal items were 24 nouns with an expected age of acquisition of less than 3 years of age (Carroll & White, 1973; Gilhooly & Logie, 1980). Nonverbal items consisted of 24 geometrical forms that could not be readily associated with a verbal label similar to those in Kimura's Recurring Figures Test (Kimura, 1963). Verbal and nonverbal items were each divided into four groups of six items. Stimulus–response pairings were determined by free random assignment without replacement, pairing each stimulus item with a response item. The ordering of pairs for presentation on testing trials, and the choice of foils used for the testing trials, were also determined by free random assignment without replacement.

Administration of the four paired associate learning tasks was conducted in a randomized order across participants to control for order of administration of tasks. Each child was provided instructions similar to the Wechsler Memory Scale–Revised paired associates before being administered each task. All verbal items were presented orally, and all nonverbal items were presented on an 22 × 28 cm inch card. For each paired associate list, three learning trials were given in which the to-be-learned pairs were presented. A testing trial was given after each of the three learning trials, for a total of 18 possible correct responses per list (six pairs multiplied by three trials).

For the learning trials, children were presented with the pairs at an approximate rate of 1 s per word and approximately 2 s per design. The presentation rates were determined from pilot work with non-brain-injured children, in order to equate performance on verbal and nonverbal items as closely as possible. For the testing trials, the child was given the stimulus items one at a time, with three foils for each stimulus item, in a multiple choice, recognition format. Feedback indicating the correct response was given after the child chose a foil.

Magnetic Resonance Imaging Analysis

MR studies were available for 17 of the 21 children with SDCP. MR exams were obtained for clinical purposes on either 1.0 or 1.5 Tesla MR imaging units. The imaging parameters were T1 weighted images, TR 48 to 600 ms, TE 16 to 26 ms; proton density weighted images TR 2000 to 3000 ms, TE 20 to 40 ms, T2 weighted images TR 2000 to 3000 ms, TE 80 ms; slice thickness 5.0 mm with 1 to 2.5 mm interslice interval. Most sequences were performed with two acquisitions. Lesion site and severity were rated by one of the investigators who is a neuroradiologist (M.K.), and who was blind to clinical history and cognitive test results. The lesion patterns for the group were typical of SDCP, with the primary finding of periventricular leukomalacia (PVL). The PVL evident on the MR exams was bilateral and generally symmetric as is typical for SDCP (see Figure 1). In cases where the PVL included parenchymal loss, ventricular enlargement was present. In 2 cases with hydrocephalus, ventricular enlargement was present, and was attributed to the hydrocephalus. No abnormalities of the medial temporal region were noted on the exams. One child had apparent changes in deep white matter regions, which represented either an abnormality or signal artifact. Gray matter in the exams was free from focal injury, though in several cases more subtle signs of cortical scalloping were present.

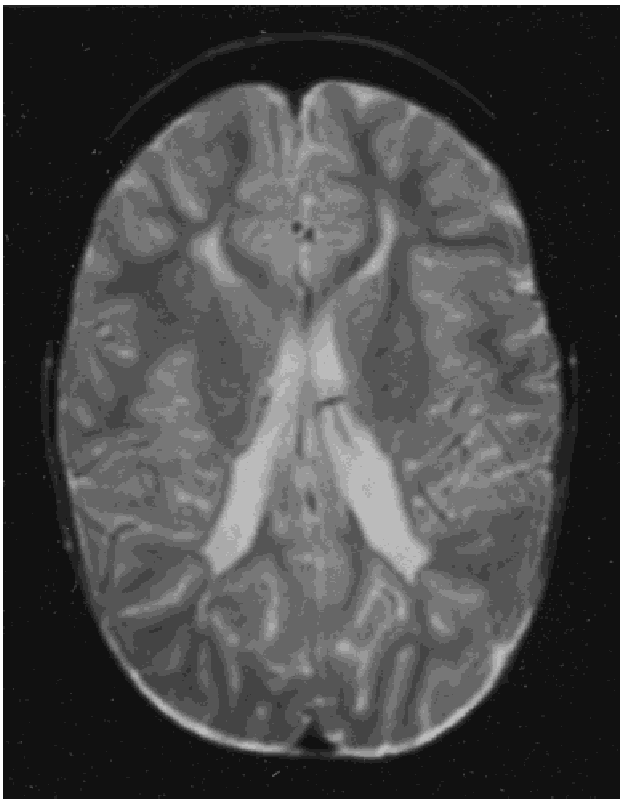

Fig. 1. MR scan showing periventricular leukomalacia, including moderate white matter loss in the trigone and body region and mild to moderate white matter signal abnormality in the trigone, body, and frontal periventricular regions.

The severity of periventricular abnormalities was rated on a 4-point scale (*no apparent*, *mild*, *moderate*, or *severe abnormality*) separately for the regions surrounding the trigone, body, and frontal regions of the lateral ventricles. These regions were chosen because they are physiologically discrete portions of the periventricular region, readily identifiable on MR exams, and frequently involved in SDCP. The 4-point scale was applied to periventricular white matter changes and periventricular parenchymal loss. This 4-point scale has previously shown acceptable interrater reliability and correlation with the extent of physical disability in a series of over 200 MR studies of children (Koby et al., 1993, 1996). A rating of zero represented normal appearance. A rating of 1 represented minimal abnormality with involvement of less than 10% of the white matter substance. A rating of 2 represented moderate abnormality with involvement of more than 10% of the white matter substance but less than 70%, without involvement of the intergyral U fibers. A

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rating of 3 represented a severe abnormality, with involvement of 70 percent or more of the white matter substance or involvement of the intergyral U fibers. The percent of white matter substance involved was determined by measuring the size of the normal and abnormal regions with tracings and calipers. The parenchymal loss was evaluated on the T1 weighted images and white matter signal abnormalities were measured primarily from the proton density weighted images, as they offer the best contrast between cerebrospinal fluid and normal and abnormal white matter.

Overall severity of abnormality for each of the three regions was calculated to provide a more general measure of pathology. Because white matter loss is indicative of more severe pathology than signal abnormality (manifest as increased signal on proton density and T2 weighted images), white matter loss was given a weighting of 2 and white matter signal abnormality was given a weighting of 1 to compute overall severity. This resulted in scores for the degree of abnormality which varied from zero (*no abnormality*) to 9 (*severe white matter loss* and *signal abnormality*).

RESULTS

To examine differences between the SDCP and healthy comparison groups for the paired associate tasks, a mixed factor repeated measures analysis of variance (ANOVA) procedure was used with group (SDCP or nonneurologically involved) as a between-subjects factor, and modality of the stimulus (verbal or nonverbal) and response (verbal or nonverbal) as within-subjects factors. The ANOVA indicated a significant main effect for group [$F(1,47) = 9.65$, $p < .01$], stimulus type [$F(1,47) = 10.05$, $p < .01$], and response type [$F(1,47) = 6.08$, $p < .05$]. Two-way interactions were found between group and response type [$F(1,47) = 5.61$, $p < .05$], and stimulus type and response type [$F(1,47) = 59.91$, $p < .01$].

To examine the nature of these interactions, *post hoc* analyses were conducted. Pairwise *t* tests comparing each task within participants revealed cross modal paired associate tasks were easier than tasks with one modality of stimuli and responses [$t(48) = 4.15$–11.19, all ps $< .01$] and children scored higher on verbal–verbal pairs than on nonverbal–nonverbal pairs [$t(49) = 3.29$, $p < .01$]. Performance on verbal–nonverbal and nonverbal–verbal tasks were comparable [$t(49) = 0.39$, $p > .05$]. After collapsing across stimulus type, a one-way ANOVA revealed the Group × Response Type interaction was due to children with SDCP scoring lower than the comparison group when the responses were nonverbal items; pairwise comparisons within the SDCP group showed poorer performance when nonverbal responses were required compared to verbal responses (see Figure 2). An examination of Figure 2 shows that the comparison group had equivalent performance across the two response modalities. This equivalent level of performance across the two conditions suggests that, when the tasks are grouped by response modality, the overall level of difficulty of the two sets of tasks is similar.

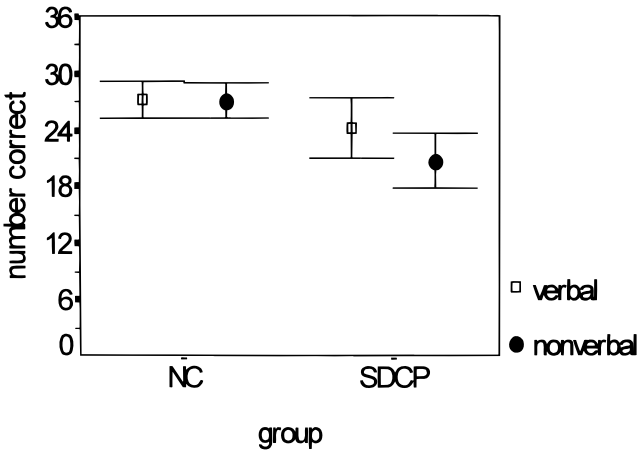

Fig. 2. Mean and 95% confidence interval around paired associate scores for normal comparison (NC) and spastic diplegic cerebral palsy (SDCP) children. For the SDCP group, performance on visual nonverbal response trials was lower than for auditory verbal response trials [$t(20) = 3.12$, $p < .01$], and lower than the NC group [$F(1,47) = 15.96$, $p < .01$]. Auditory verbal responses did not differ between groups [$F(1,47) = 2.83$, $p > .05$]. The comparison group showed equivalent performance across the two response modalities [$t(27) = 0.08$, $p > .05$].

Because of the presence of significantly lower Spatial Relations scores in the SDCP group, it was possible that the difficulties with learning the nonverbal responses was due to a more general deficit in visual spatial ability, rather than a specific difficulty with nonverbal learning. To address this possibility, the performance for verbal and nonverbal response conditions from the paired associate tasks were examined after statistically controlling for performance differences on the Spatial Relations test. Linear regression was used with Spatial Relations scores as the independent variable to create residual scores for the verbal and nonverbal response conditions of the paired associate tasks. These residual scores were used in a mixed-factor repeated measures ANOVA with group as the between-subjects factor (SDCP or nonneurologically involved) and modality of the response on the paired associate tasks (verbal or nonverbal) as the within-subjects factor. The ANOVA indicated no main effects for group [$F(1,47) = 0.00$, $p > .05$], or for response type [$F(1,47) = 0.08$, $p > .05$]. Similar to the analysis without controlling for Spatial Relations scores, a two-way interaction was present between group and response type [$F(1,47) = 4.08$, $p < .05$]. An examination of residual scores indicated the SDCP group's relative difficulty with nonverbal responses was present after statistically controlling for more general nonverbal ability.

Analyses were also conducted relating paired associate task performance to MR ratings for the 17 SDCP children with MR exams. The examination of paired associate performance in relation to specific location of lesions for the SDCP group was complicated by multicollinearity between the lesion severity ratings: There was a significant positive

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relationship between lesion severity among the trigone, body, and frontal regions surrounding the lateral ventricles (trigone and body, $r = .75$, $p < .01$; trigone and frontal, $r = .58$, $p < .05$; and body and frontal, $r = .60$, $p < .05$). This relationship is consistent with previous reports of lesion patterns in SDCP, as there is a tendency for lesions to occur posteriorly and extend anteriorly with increasing severity (Shouman-Claeys et al., 1989; Yokochi et al., 1991; Truwit et al., 1992). To examine the relationship between response modality and MR findings, two hierarchical linear regression analyses (Model I error) were used (Cohen & Cohen, 1983). Because the SDCP group's deficit appeared to be related to response type, scores on the paired associate tasks were collapsed across stimulus type. For the two hierarchical models, performance on tasks with verbal and visual responses were used as the dependent variable. Age was entered in the first step. In the second step, lesion severity in the frontal, body, and trigone regions were entered as a set in order to examine the effects of lesion severity after controlling for age. In the third step, the Age × Lesion Severity interaction terms were entered in order to examine whether lesion severity was dependent on age (see Table 2).

Tasks with verbal responses and those with visual responses both showed a significant positive relationship with age for SDCP children. Overall lesion severity was negatively related to performance for tasks with visual responses. There was a negative relationship between performance with verbal responses and lesion severity, which did not reach statistical significance ($p < .20$). For nonverbal responses, none of the β weights for the three periventricular regions reached statistical significance, suggesting a generalized effect for lesion severity. Similar hierarchical regression models conducted separately for verbal–nonverbal pairs and for nonverbal–nonverbal pairs showed comparable results to the model with scores collapsed across stimulus type. These two tasks differed in their pattern of β weights for the three separate regions: Nonverbal–nonverbal pairs showed a significant relationship with trigone injury [$t(12) = -2.26$, $p < .05$], and verbal–nonverbal pairs showed a significant relationship with frontal injury [$t(12) = -2.96$, $p < .05$].

For comparative purposes, PPVT–R and WJ–R Spatial Relations raw scores were related to lesion severity using similar linear regression procedures. PPVT–R scores were related to age [$R^2 = .76$; $F(1,15) = 47.51$, $p < .01$], and overall lesion severity [$IR^2 = .13$; $F(3,12) = 4.53$, $p < .05$], but not Age × Lesion Severity [$IR^2 = .01$; $F = 0.32$, $p > .05$]. For the PPVT–R, the β weight for the frontal region reached statistical significance [$t(12) = -3.03$, $p < .05$]. WJ–R Spatial Relations scores were also related to age [$R^2 = .58$; $F = (1,15)$, $p < .01$], and overall lesion severity [$IR^2 = .26$; $F(3,12) = 6.40$, $p < .01$], but not the interaction terms [$IR^2 = .01$; $F(3,9) = 0.26$, $p > .05$]. For WJ–R Spatial Relations, none of the β weights for the three separate regions reached statistical significance.

DISCUSSION

The present study examined paired associate learning in children with perinatal injury resulting in SDCP. Children with SDCP showed impaired associative learning when learning visual nonverbal responses regardless of the stimulus type. Performance when learning verbal responses was relatively more intact in SDCP children. In addition, lesion severity related to SDCP children's ability to learn nonverbal responses, but did not reach statistical significance for verbal responses. These results have implications for the role of early brain injury in the development of associative learning and the relationship between brain injury and cognitive development in SDCP.

The disruption of periventricular white matter early in life appears to have an impact on associative learning ability. The capacity for associative learning in SDCP seems to be dependent upon the type of material the child is learning. Material specific deficits in associative learning have been explored in the context of left *versus* right temporal lobe epilepsy (e.g., Cohen, 1992; Jambaque et al., 1993). The present study suggests material specific deficits in associative learning can be found with injury early in life that is relatively symmetric, bilateral, and lies outside of temporal lobe regions.

Table 2. Results of hierarchical multiple regression procedures relating paired associate performance to lesion severity

Dependent variable	Independent variable(s)	R^2	IR^2	*df*	*F* value
Auditory verbal responses	Age	.57	.57	(1,15)	19.73**
	Lesion severity (frontal, body, & trigone)	.71	.14	(3,12)	2.05
	Age × Frontal, Age × Body, Age × Trigone	.73	.02	(3,9)	0.86
Visual nonverbal responses	Age	.20	.20	(1,15)	3.73
	Lesion severity (frontal, body, & trigone)	.61	.41	(3,12)	4.24*
	Age × Frontal, Age × Body, Age × Trigone	.66	.05	(3,9)	0.42

Note. IR^2 values represent the incremental increase in R^2 with the addition of independent variable(s) in the hierarchical regression model.
*$p < .05$.
**$p < .01$.

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The SDCP group's performance was notable for greater deficits when learning visual nonverbal responses was required. Difficulty with this type of encoding could be related to at least one of two aspects of brain injury in SDCP: First, it may be that early brain injury leads to difficulty with nonverbal encoding in a general, nonspecific manner; and second, it may be that the particular locus of injury in SDCP leads to deficits in visual nonverbal encoding. With regard to the first possibility, previous studies of brain injury in children have indicated the pervasiveness of nonverbal deficits following many forms of diffuse childhood brain injury, including premature birth, head injury, meningitis, seizure disorder, and cranial irradiation (Woods & Teuber, 1973; Ditka et al., 1975; Taylor et al., 1984; Fletcher & Copeland, 1988; Fletcher et al., 1990; Dorman & Katzir, 1994). This type of relationship could account for Jambaque et al.'s (1993) finding of deficits in visual paired associate learning regardless of the type or focus of seizure activity, while performance on verbal paired associate tasks varied across groups.

Lesion severity in particular locations in SDCP could also be important for the disruption of visual nonverbal learning. The extent of injury in the trigone region, which contains both the optic radiations and occipital–parietal white matter tracts, has previously been shown to relate to visual–perceptual skills in children with SDCP, and could relate to other nonverbal abilities (Koeda & Takeshita, 1992; Goto et al., 1994). Frontal lesions that affect conditional associative learning have been shown, with selective lesions, to result in modality-specific difficulties with learning, such as specific deficits in conditional learning for visual material (Petrides, 1987). The strong interrelationship between lesion severity in each of the regions examined in this study is not uncommon in SDCP, but limits the conclusions that can be reached about the importance of lesion location for the present set of findings. Beta weights from the regression models used in this study suggested lesion severity in specific regions may relate to particular aspects of the associative learning tasks, but the limited sample size and relative homogeneity of the distribution of lesions limits the interpretation of these results. Studies with larger samples of children with SDCP, or samples with greater variability in lesion distributions, could better address the impact of lesion size *versus* lesion location in this population.

The findings in the present study provide information about the relationship between lesion patterns and cognitive abilities in SDCP: As overall lesion severity increased, children showed poorer ability to learn visual nonverbal responses and performed more poorly on measures of general verbal and nonverbal ability. This is contrary to an earlier study that reported that ventricular dilation and white matter reduction on MR exams of children with cerebral palsy were related to the severity of motor disability, but not cognitive ability (Yokochi et al., 1991). An absence of a relationship between periventricular injury and cognitive ability has also been reported by Feldman et al. (1990). In both of these previous studies, however, the authors used limited categorizations of cognitive ability (e.g., abnormal *vs*. normal intelligence level), which likely accounts for their null findings. The fact that lesion severity in the present study related to both learning ability for nonverbal responses and to more general verbal and nonverbal ability highlights the multiple areas of deficit one can expect in cases of more extensive perinatal injury. Additional studies of children with SDCP with larger samples may better address the degree of sparing for auditory verbal learning with more extensive injury. The data from the present small sample suggests verbal learning ability is relatively less affected than nonverbal learning ability.

In conclusion, associative learning is an important basic ability required for normal cognitive development. This ability appears to be disrupted by perinatal brain injury in a modality-specific manner. The impact of this pattern of deficient associative learning may be important for understanding difficulties in higher level cognitive abilities in SDCP and other groups with early brain injury.

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