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# THE EFFECTS OF DEVELOPMENTAL FACTORS ON IQ IN HEMIPLEGIC CHILDREN

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**Abstract**—The effects of early unilateral brain lesions on subsequent intellectual functioning were explored in hemiplegic children with congenital or acquired lesions. For congenital hemiplegics who sustained damage pre- or perinatally, lower intellectual functioning (IQ) was most highly associated with longer elapsed time since lesion. Moreover, including lesion size as an additional predictor of IQ did not account for significantly more variance than elapsed time since lesion alone. In contrast, for acquired hemiplegics who sustained damage after birth, lower intellectual functioning was highly asscociated with larger lesion size. In this group neither elapsed time since lesion nor age at testing accounted for significantly more IQ variance than lesion size alone. Possible effects of maturational factors and functional plasticity are considered in interpreting this pattern of results.

## INTRODUCTION

UNILATERAL cerebral lesions that occur perinatally or in early childhood are reported to have different effects on cognitive functioning than lesions acquired later in life. Certain functions, notably language comprehension and production abilities, are reported to be relatively spared by lesions early in life [1, 2, 11, 21, 28–30]. In contrast, other functions are found to be no more spared, or in some cases, even *more* impaired by early than later lesions (e.g. performance on hidden figures test) [5, 6, 14, 16, 20]. In an attempt to account for the variable effects of early vs later lesions, WOODS and TEUBER [16] have proposed that "the earlier the lesion, the greater the reorganization of neural mechanisms underlying behaviour". This reorganization is apparently beneficial to some behaviors but not to others.

ST JAMES-ROBERTS [17, 18] has proposed that factors other than greater reorganizational capacity may account for the less severe impairments observed in individuals who have undergone hemispherectomy early in life vs later in life. The greater time period for functional recovery (time between the onset of a lesion and the assessment of cognitive functions) typically available for patients with early lesions is a proposed alternative. It is difficult to determine the relative contributions of recovery time period and functional plasticity to differences in intellectual functioning between age groups because both factors predict that patients with early lesions should have higher intellectual functioning than those with later lesions.

In the present paper we attempt to disentangle this issue by examining the relative roles of

elapsed time since lesion, age at lesion, and age at testing on the intellectual functioning of hemiplegic children with congenital or early acquired lesions. One implication of ST JAMES-ROBERTS' [17] proposal might be that among a group of brain-damaged children who sustained lesions early in life, longer elapsed time since lesion should be associated with higher intellectual functioning. Although this prediction has not been investigated in brain-damaged children, it is not supported by findings in other primates. Indeed, when compared to age-matched controls, animals with early lesions show greater impairment, rather than greater recovery, with increasing time since lesion. In particular, GOLDMAN [4] reports that monkeys with early lesions of the dorsolateral frontal lobe (at 50 days of age) are more impaired on a delayed alternation task at 2 years of age than at 1 year of age, compared to age-matched controls. In contrast, monkeys with later lesions of the dorsolateral frontal lobe (at 18–24 months of age), are severely impaired on delayed alternation irrespective of the time of testing following lesion.

The findings [4] suggest that the impairments seen following dorsolateral frontal lobe lesions in monkeys are influenced by two types of developmental factors. First, there are effects of age at the time of the lesion, as is widely acknowledged. Second, there are effects of age at the time of the testing in early but not later lesioned monkeys. In particular, early-lesioned animals may appear to exhibit sparing of function because the cortical areas that sustained damage are functionally immature. Hence, they do not contribute to performance on delayed alternation tasks even in intact animals. Later during development when these cortical areas begin to contribute to task performance, deficits on delayed alternation emerge in monkeys who sustained brain damage earlier in life.

Whereas the effects of age at lesion have been widely investigated in humans [e.g. 1, 2, 5, 6, 11, 16, 20, 28–30], the effects of age at testing and elapsed time since lesion have received relatively little attention. In the present study we attempt to examine the roles of age at lesion, age at testing and elapsed time since lesion on subsequent cognitive functioning in two groups of hemiplegic children, one with congenital lesions and one with early acquired lesions. The lesions of children in the congenital group originated prenatally or during the early postnatal period ("perinatal" onset) though the exact timing of the lesion is not known for individual cases. Because the time between lesion onset and assessment of intellectual functions varies among the individual congenitally hemiplegic childen in our sample, we are able to investigate whether different periods of time since lesion onset are associated with different levels of cognitive functioning. However, in this group of patients, any relation found between level of intellectual functioning and time of testing may be attributable either to elapsed time since lesion (recovery time period) or to age at testing, since the two measures coincide. In contrast, for the acquired hemiplegics, age at lesion, age at testing, and elapsed time since lesion vary from individual to individual, thus providing the potential to disentangle the effects of these different factors.

## METHOD

*Subjects*

Psychological testing, neurological examinations, EEG readings and CT scans were obtained on 41 children seen in the pediatric neurology clinic at Wyler Children's Hospital of the University of Chicago between 1976 and 1984. Every hemiplegic child referred to the clinic was considered a potential subject pending confirmation by a CT scan that the lesion was unilateral. Because these children typically are scheduled for pediatric neurology appointments at least once a year, regardless of the degree of their impairment, over the 8-year period of our study it is unlikely that our sample was biased toward selecting cases with more severe problems. Tables 1 and 2 summarize the laterality of lesion, sex and age at time of psychological testing for children with congenital and acquired hemiplegia,

Table 1. Characteristics of cases with congenital lesions

| Case | Sex | Age at testing (months)* | Aetiology |
|---|---|---|---|
| Right-hemisphere lesion | | | |
| R1 | M | 71 | Mostly uncertain |
| R2 | M | 57 | |
| R3 | M | 216 | |
| R4 | M | 63 | |
| R5 | F | 71 | |
| R6 | F | 75 | |
| Left-hemisphere lesion | | | |
| L1 | M | 77 | Mostly uncertain |
| L2 | M | 73 | |
| L3 | M | 78 | |
| L4 | M | 59 | |
| L5 | M | 40 | |
| L6 | M | 75 | |
| L7 | M | 81 | |
| L8 | M | 67 | |
| L9 | M | 139 | |
| L10 | F | 177 | |
| L11 | F | 63 | |
| L12 | F | 91 | |
| L13 | F | 51 | |
| L14 | F | 159 | |
| L15 | F | 120 | |
| L16 | F | 180 | |
| L17 | F | 196 | |
| L18 | F | 106 | |
| L19 | F | 146 | |

*Equivalent to elapsed time since lesion for the congenital group since lesion onset is considered to be birth.

respectively. In addition, for the children with acquired lesions, the age at which the lesion was acquired and the time between onset of lesion and testing (elapsed time since lesion) are reported.

About half of the hemiplegic children had lesions confined to cortical areas, mainly to the frontal, parietal, and/or temporal lobes, with most cases having some frontal lobe involvement and only a few having occipital lobe involvement. The majority of the remaining patients had both cortical and subcortical lesions, with cortical involvement in frontal, parietal and/or temporal areas, and subcortical involvement of the basal ganglia and/or internal capsule. Only two patients had lesions confined to subcortical areas. No effects of lesion location on level or pattern of intellectual functioning were found in a previous examination of this population (see [12] for further information on these patients).

*Procedure*

Psychological testing was obtained on all patients in the study. This testing included administration of a standardized intelligence test. Depending on the age of the subject at the time of testing, either the Wechsler Preschool and Primary Scale of Intelligence (WPPSI) [24], the Wechsler Intelligence Test for Children, Revised (WISC-R) [23], the Wechsler Adult Intelligence Scale (WAIS) [22] or the Stanford-Binet, Form L-M [19] was administered.

For each subject, computerized axial tomography of the brain (CT scan) was used to identify the locus of the lesion and also to estimate its size. Subjects for whom the CT scan revealed a bilateral lesion were not included in the study. Size of lesion was estimated by selecting the slice on which the lesion was largest, and calculating the ratio of its maximal anterior–posterior extent to the anterior–posterior diameter of the slice (maximal diameter ratio). Second, the vertical extent of the lesion was estimated from the number of sections, in standard CT planes of 1 cm thickness, on which the lesion was evident. Based on these two estimates of lesion size, lesions were assigned to one of four categories as follows: 0—no detectable lesion; 1—lesions visible in only 1 or 2 sections and maximal diameter ratio below 0.2; 2—lesions visible in 3 or 4 sections, and maximal diameter ratio between 0.2 and 0.4; 3—lesions visible in 5 or more sections and maximal diameter ratio above 0.4. Two cases that did not fit within these limits were

Table 2. Characteristics of cases with acquired lesions

| Case | Sex | Age at testing (months) | Age at lesion (months) | Elapsed time since lesion (months) | Aetiology |
|---|---|---|---|---|---|
| Right-hemisphere lesion | | | | | |
| R7 | M | 102 | 36 | 66 | Head trauma |
| R8 | M | 114 | 114 | 0 | St. Louis encephalitis with stroke |
| R9 | M | 32 | 19 | 13 | Vasculitis |
| R10 | M | 127 | 72 | 55 | Sickle cell disease, stroke |
| R11 | M | 63 | 60 | 3 | Moya Moya disease, stroke |
| R12 | F | 90 | 60 | 30 | Sickle cell disease, stroke |
| R13 | F | 74 | 7 | 67 | Stroke with febrile illness |
| R14 | M | 124 | 8 | 116 | Head trauma |
| Left-hemisphere lesion | | | | | |
| L20 | M | 108 | 11 | 97 | Astrocytoma |
| L21 | M | 57 | 9 | 48 | Head trauma |
| L22 | M | 145 | 1.5 | 143.5 | Meningitis |
| L23 | M | 99 | 7 | 92 | Embolic stroke during cardiac catheterization |
| L24 | M | 62 | 30 | 32 | Stroke |
| L25 | M | 123 | 108 | 15 | Head trauma |
| L26 | F | 216 | 72 | 144 | Chronic focal encephalitis |
| L27 | F | 78 | 11 | 67 | Sickle cell disease, stroke |

classified as follows: a case in which 4 sections were involved but with a maximal diameter ratio of only 0.14 was classified as category 2, and a case with 5 abnormal sections and maximal diameter ratio of 0.32 was classified as category 3. Cases with ventricular enlargement as the only finding were classified according to the number of sections that showed the enlarged portion of the ventricle (see [12] for further information on the measurement of lesion size).

## RESULTS

### *Congenital hemiplegics*

The correlations of Verbal IQ (VIQ), Performance IQ (PIQ) and Full Scale IQ (FIQ) with age at testing and lesion size were calculated for the congenital hemiplegics ($N=25$). The correlation of age at testing (which is equivalent to elapsed time since lesion for this group) with VIQ, PIQ, and FIQ were all highly significant ($P<0.01$), but in a negative direction (see Table 3). Thus, the longer the elapsed time since the lesion, the lower the level of intellectual functioning as measured by both the Verbal and Performance IQ scales. There was also a significant negative correlation of lesion size with VIQ ($P<0.10$), but not with PIQ or FIQ (see Table 3).

A step-wise multiple regression with length of recovery period and lesion size entered as potential predictors of FIQ was performed to determine which variables contributed significantly to the prediction of IQ (Multiple $R^2=0.56$). Results showed that length of recovery period was the best predictor of IQ ($F=21.27$, d.f. $=1, 21$, $P<0.01$). Moreover, the amount of IQ variance accounted for was not significantly increased by lesion size. In fact, even with lesion size partialled out, the correlation of FIQ and length of recovery period was large and negative ($r=-0.61$, d.f. $=24$, $P<0.01$). Thus, for the congenital group, elapsed time since lesion which is equivalent to age at testing, is a significant predictor of FIQ whereas lesion size is not (see Figs 1a and b).

Inspection of Fig. 1 suggested that the negative correlation of elapsed time since lesion (age at testing) and FIQ is mediated mainly by older subjects, in particular, those who were older

Table 3. Congenital cases: correlations of VIQ, PIQ, and FIQ with elapsed time since lesion and lesion size

| | Elapsed time since lesion (=Age at testing) | Lesion size |
|---|---|---|
| VIQ | −0.782<br>d.f.=22*<br>$P<0.01$† | −0.417<br>d.f.=22<br>$P<0.10$ |
| PIQ | −0.610<br>d.f.=22<br>$P<0.01$ | −0.212<br>d.f.=22<br>n.s. |
| FIQ | −0.648<br>d.f.=23<br>$P<0.01$ | −0.283<br>d.f.=23<br>n.s. |

*d.f. is one less for VIQ and PIQ than FIQ as one subject was administered the Stanford-Binet because of age, and VIQ and PIQ are not provided by this test.
†$P$ values are two-tailed.

than 6–8 years of age at the time of testing. Regardless of the exact point between 6 and 8 years of age at which one divides subjects into older and younger groups, the correlation of elapsed time since lesion is much larger for the older (e.g. *older than 6 years*: $r=-0.803$, d.f.=13, $P<0.001$; *older than 8 years*: $r=-0.513$, d.f.=7, $P=0.15$) than the younger group (*less than 6 years*: $r=-0.240$, d.f.=8, n.s.; *less than 8 years*: $r=0.217$, d.f.=14, n.s.).

*Acquired hemiplegics*

For the acquired hemiplegics, we examined the correlations of VIQ, PIQ, and FIQ with age at testing, age at lesion, elapsed time since lesion and lesion size (see Table 4). For these children, the correlations of lesion size with VIQ, PIQ, and FIQ were highly significant ($P<0.01$). The only other correlations approaching significance were those of length of recovery period with PIQ and length of recovery period with FIQ ($P<0.10$ and $P<0.15$ respectively). As was found for the congenital hemiplegics, these correlations were negative in direction.

A step-wise multiple regression was performed to determine which factors predicted a significant amount of the variance in the IQ of the acquired hemiplegics. The potential predictors were elapsed time since lesion, age at lesion, age at testing and lesion size (Multiple $R^2=0.51$). Results show that lesion size was the best predictor of IQ ($F=12.80$, d.f.=1,12, $P<0.01$) and the amount of IQ variance accounted for was not significantly increased by the other factors. Thus, in contrast to the congenital group, for the acquired group lesion size, but not elapsed time since lesion, was a significant predictor of IQ (see Figs 2a, b).

It should be noted that for the acquired hemiplegics, age at lesion and lesion size are significantly negatively correlated ($r=-0.67$, d.f.=14, $P<0.02$) such that children who sustained lesions at younger ages tended to have larger lesions. This finding may be attributable to more trans-synaptic degeneration when lesions are acquired at an early age, and damaged regions of the brain are not yet mature [3]. Alternatively, a lesion of a particular size that is acquired at a later age may have more devastating functional effects than the same size lesion acquired at an earlier age (Goldman-Rakic, personal communication), perhaps decreasing the probability that patients who sustain large lesions later in childhood would be seen in our clinic.

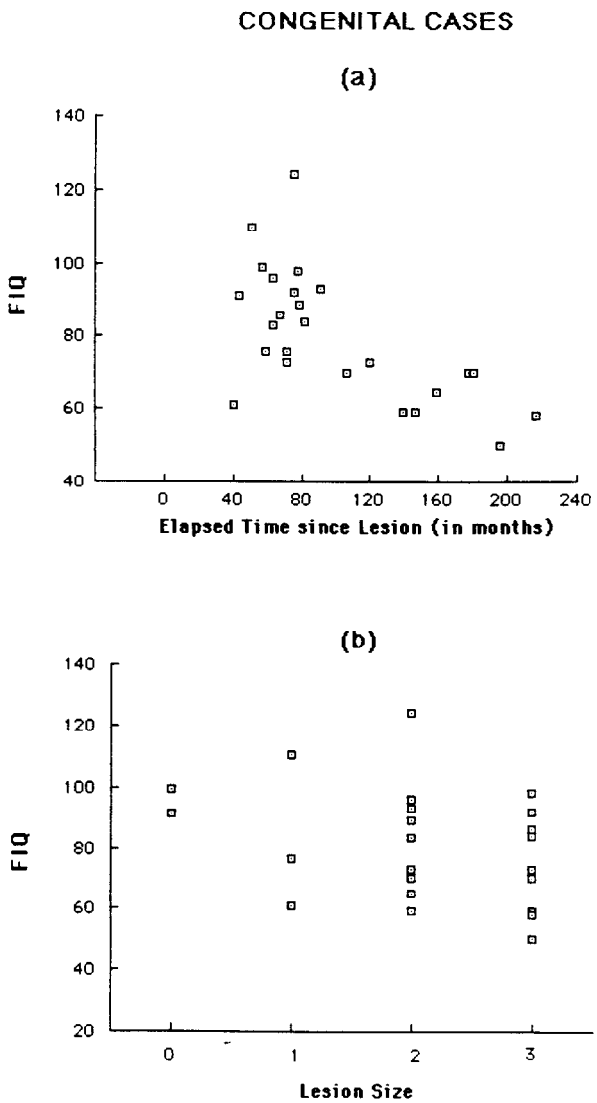


Fig. 1(a): FIQ vs elapsed time since lesion (age at testing) in months for congenital hemiplegics ($r = -0.648$). (b): FIQ vs lesion size for congenital hemiplegics ($r = -0.283$).

Our data suggest that age at testing (which is equivalent to time since onset of lesion for the congenital group) is a significant predictor of IQ among congenital hemiplegics older than 6 years of age. In order to investigate whether the falloff in IQ with elapsed time since lesion is best characterized as a loss of previously acquired knowledge, as an arrest in the acquisition of new knowledge, or as a decreased rate of acquisition of new knowledge, we examined subjects' WISC-R raw scores. Raw scores (rather than scaled scores) were used to address this question because they are not adjusted for age. It was necessary to confine ourselves to subjects who had all taken the same IQ test, so we considered only those children who had taken the WISC-R (27 of the 41 subjects in our samples).

Table 4. Acquired cases: correlations of VIQ, PIQ and FIQ with elapsed time since lesion, lesion size, age at lesion and age at testing

| | Age at lesion | Age at testing | Elapsed time since lesion | Lesion size |
|---|---|---|---|---|
| VIQ | −0.128<br>d.f. = 13*<br>n.s. | −0.220<br>d.f. = 13<br>n.s. | −0.298<br>d.f. = 13<br>n.s. | −0.649<br>d.f. = 13<br>$P<0.012$† |
| PIQ | +0.332<br>d.f. = 13<br>n.s. | −0.267<br>d.f. = 13<br>n.s. | −0.507<br>d.f. = 13<br>$P<0.10$ | −0.747<br>d.f. = 13<br>$P<0.01$ |
| FIQ | +0.176<br>d.f. = 14<br>n.s. | −0.334<br>d.f. = 14<br>n.s. | −0.451<br>d.f. = 14<br>$P<0.15$ | −0.669<br>d.f. = 14<br>$P<0.01$ |

*d.f. is one less for VIQ and PIQ than FIQ as again one subject was administered the Stanford-Binet because of age, and VIQ and PIQ are not provided by this test.
†$P$ values are two-tailed.

Mean raw scores for each age group included in the normative sample were plotted for each subtest. These normative data are provided in the WISC-R manual. These scores were then compared to those for each child in the congenital group and to those for each child in the acquired group. The pattern of the plots across subtests were quite similar. In general, scores of the children with congenital hemiplegia remain quite close to the age-appropriate norms until about the age of 6. After this age, their raw scores continue to increase, but at a slower rate than for children in the normative sample. Thus, our data suggest that a longitudinal study would find a decreased rate of acquisition of new knowledge in congenital hemiplegics older than 6 years of age. In contrast, scores of the children with acquired hemiplegia appear to vary more randomly with age. This would be expected since lesion size rather than age at testing or time since onset of lesion best predicts IQ for children in this group. The plots for the WISC-R Vocabulary and Block Design subtests, the best measures of *g* for the Verbal and Performance subscales respectively, are presented in Fig. 3 (a–d) for both congenital and acquired hemiplegics.

*Socioeconomic variables*

Possible effects of socioeconomic status on our results were examined since this variable has been reported to affect functional recovery in brain damaged patients [13, 15, 27]. In particular, higher socioeconomic status has been reported to be associated with improved intellectual functioning over time in brain-damaged patients whereas lower socioeconomic status has been reported to be associated with decreased intellectual functioning over time. As a rough index of socioeconomic status, we used available information on whether the parent(s) and/or legal guardian(s) of the child was working or receiving public aid at the time of the child's psychological testing. We first compared the mean FIQ of children whose parents worked with those whose parents were receiving aid, partialling out the effect of lesion size. Lesion size was significantly larger in the "on-aid" group ($P<0.005$). This analysis revealed no significant differences in IQ between the two groups once the effect of lesion size was removed ($F<1$). (Mean FIQs adjusted for lesion size: on-aid: 75.5; working: 80.7.)

In order to determine whether the negative correlation between elapsed time since lesion and IQ in our sample was mediated by unfavorable socioeconomic conditions, the

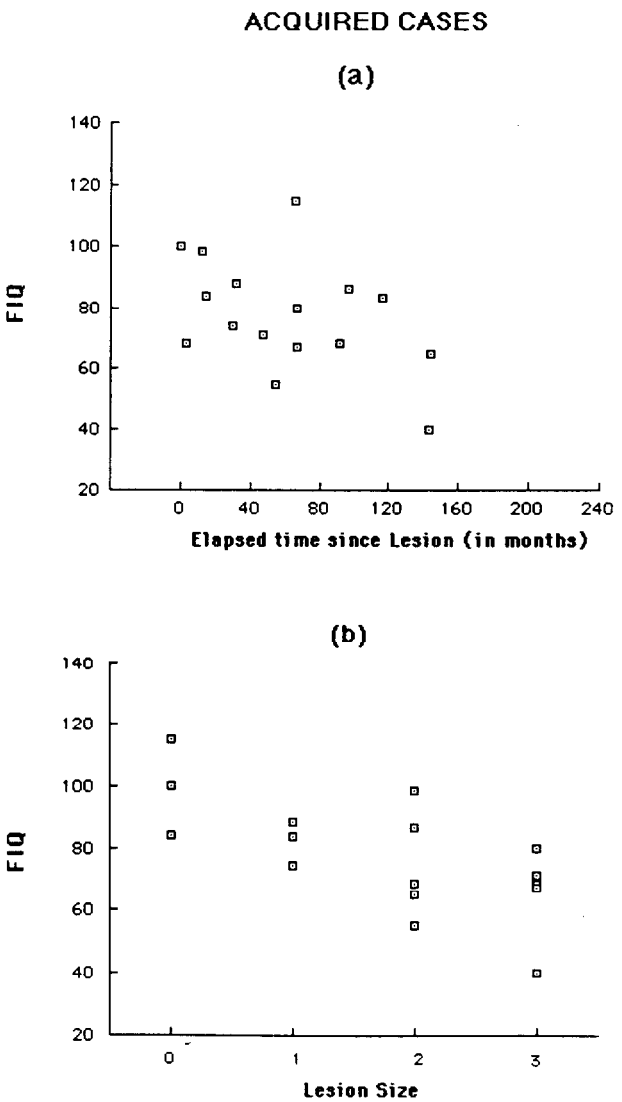


Fig. 2(a): FIQ vs elapsed time since lesions in months for acquired hemiplegic ($r = -0.451$). (b): FIQ vs lesion size for acquired hemiplegics ($r = -0.669$).

correlation of IQ and elapsed time since lesion was calculated separately for the on-aid and working groups. This correlation was highly significant for the group with working parents ($r = -0.67$, d.f. $= 27$, $P < 0.01$) but not for the group with parents receiving aid ($r = -0.31$, d.f. $= 10$, n.s.). Thus, it does not appear that the negative correlation between IQ and recovery period is primarily mediated by low socioeconomic status. It should be noted, however, that the lower correlation in the on-aid group may be attributable to a higher proportion of acquired cases in this group (8/12) than in the working parent group (8/29) ($X^2 = 5.46$, $P < 0.05$) (recall that elapsed time since lesion is a more significant predictor of IQ for children with congenital than acquired lesions).

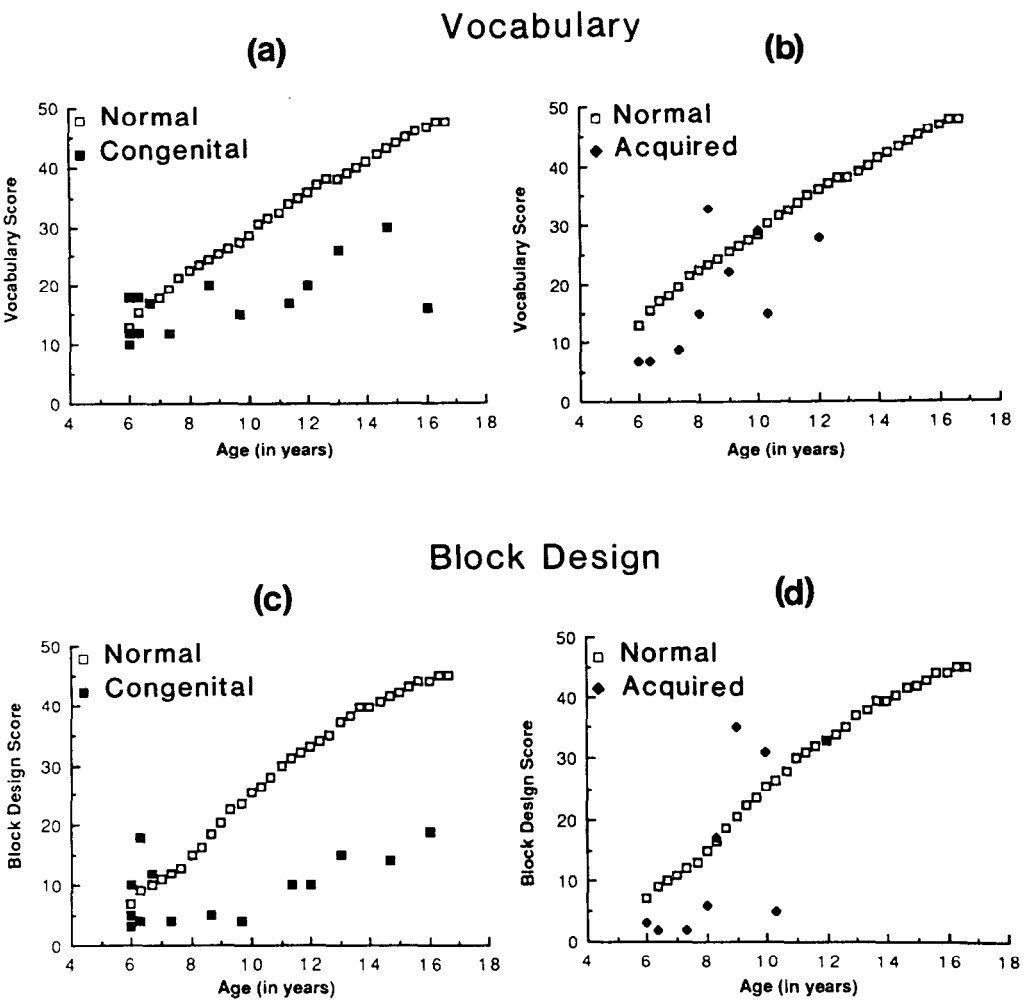


Fig. 3(a): Scaled WISC-R Vocabulary raw subtest scores vs age at testing for the congenital hemiplegics and normative sample. (b): Scaled WISC-R Vocabulary raw subtest scores vs age at testing for the acquired hemiplegics and normative sample. (c): Scaled WISC-R Block Design raw subtest scores vs age at testing for the congenital hemiplegics and normative sample. (d): Scaled WISC-R Block Design raw subtest scores vs age at testing for the acquired hemiplegics and normative sample.

## DISCUSSION

The results of our study suggest that length of recovery time period (equivalent to age at testing in this group) has a differentially greater effect on the intellectual functioning of children with congenital hemiplegia, and lesion size has a differentially greater effect on the intellectual functioning of children with acquired hemiplegia.

For congenital hemiplegic children, a falloff in intellectual functioning, relative to an age-matched normative sample, appears to begin when the child is about 6 to 8 years of age. This finding does not lend support to St James-Roberts' hypothesis that higher intellectual functioning of patients with early rather than later hemispherectomy is accounted for by a

longer time period between onset of lesion and testing [18]. At least among children with congenital hemiplegia, our results suggest the opposite; that is, measured IQ is higher when testing is done at shorter rather than longer time intervals following the lesion.

The present findings raise the question of why, particularly in the congenital group, overall IQ decreases with time since lesion onset. Our data suggest that after 6–8 years of age, children with early brain lesions are not able to gain cognitive skills at the same rate as children with intact brains. One possible explanation for the present results is related to GOLDMAN's [4] findings of significant effects of maturational status on deficits following prefrontal brain damage in early lesioned monkeys. Goldman's studies show that relative to age-matched controls, monkeys with early dorsolateral frontal lobe lesions show deficits on delayed alternation when tested at 2 but not 1 year of age. Similarly, IQ deficits in our congenital hemiplegics are larger when testing is done after rather than before age 6–8 years. Similar to Goldman's report that delayed alternation performance of monkeys with later lesions is not affected by age at testing, IQ deficits of our acquired hemiplegics are not related to age at testing (equivalent to time since onset of lesion). It should be noted, however, that the lack of a significant negative correlation between recovery time period and IQ in the acquired group may result because they have a shorter mean recovery period (mean = 61.8 months) than the congenital group (mean = 101.2 months) ($t = 2.46$, d.f. = 34, $P < 0.02$, two-tailed). Thus, if we re-tested the acquired group at longer time intervals from the onset of lesion, it is possible that we would find a significant negative correlation between their IQ scores and elapsed time since lesion.

Goldman has suggested that early lesioned monkeys may exhibit sparing of function for a time during development because of the functional immaturity of the brain in intact animals [4]. Thus, when lesions are acquired early in life, areas of the brain corresponding to the damaged areas of lesioned monkeys may not be contributing to task performance in normal monkeys. As the relevant cortical areas mature and begin to contribute to task performance in normal monkeys, deficits may emerge in the lesioned monkeys. In contrast, in later lesioned monkeys deficits may be present from the time of lesion onward because of the relative maturity of the brain in intact animals. Thus, when lesions are acquired later in life, areas of the brain corresponding to the damaged areas of lesioned monkeys may already be contributing to task performance in normal monkeys. A similar explanation may account for the falloff in IQ over time in the congenital, but not acquired hemiplegics.

An alternative, but not mutually exclusive explanation is that the reorganizational capacity of the young lesioned brain is adequate to support early developing functions, but not more complex functions that typically develop later, as the normal brain matures. Early damage may attenuate the ability of the brain to undergo normal developmental changes, thus limiting the computational and/or storage capacities necessary to master the skills normally acquired during development. Assuming that the cognitive skills acquired at one point during development are necessary to maintain a normal rate of subsequent intellectual growth (scaffolding model), the disparity in intellectual functioning between children with early brain damage and normal, age-matched controls would be expected to increase with age.

Consistent with our finding of a significant relation between elapsed time since lesion and intellectual functioning in congenital hemiplegics, KOLB *et al.* [8, 10] report that relative to age-matched controls, rats who sustained lesions at a young age show a gradual decrease in brain weight that continues into adulthood. It is possible that such a process is related to the decrease in IQ over time in the congenital hemiplegics. In fact, the older children in our

sample tended to have larger lesions, which might be indicative of continued loss of neuronal mass. It is possible that the obtained relationship between age and lesion size might arise because hemiplegic children with larger lesions are more likely to continue to come to the pediatric neurology clinic throughout their development than those with smaller lesions. This does not seem very likely in our sample, as even the hemiplegic children with small lesions typically are scheduled for a yearly pediatric neurology appointment.

Whereas elapsed time since lesion has a greater impact on the intellectual functioning of congenital than acquired hemiplegics, the reverse is true of lesion size. This result is consistent with KORNHUBER *et al.*'s [7] report of a large negative correlation ($r = -0.73$) between lesion size and IQ in children who sustained brain damage after the age of 5. Relative immaturity of the brain at the time of lesion in congenital hemiplegics may allow for greater reorganization of functions than in acquired hemiplegics. As a result, the extent of damage may play a greater role in the subsequent intellectual functioning of acquired than congenital hemiplegics. In acquired hemiplegics, because many areas of the brain are already committed to particular functions at the time of lesion, it may be relatively difficult to compensate for large lesions with remaining intact areas. In congenital hemiplegics, even when lesions are large, uncommitted intact areas may be able to reorganize to subserve the functions that would have been carried out by the lesioned areas. Assuming there is a limit to the brain's reorganizational capacity, however, lesion size might become a significant predictor of IQ in congenital hemiplegics later during development.

In fact, certain aspects of our data tentatively suggest that this may be the case. Among the older half of congenital hemiplegic children (older than age $6\frac{1}{2}$ at the time of testing, $N = 13$) those with relatively large lesions (lesion size category $= 3$, $N = 5$) had a non-significantly lower mean IQ than those with relatively small lesions (lesion size category $= 2$, $N = 7$) (mean IQ: 64.2 vs 74.1). In contrast, among congenital hemiplegics who were younger at the time of testing (less than $6\frac{1}{2}$ years, $N = 12$, those with relatively large lesions (lesion size category 2 or 3, $N = 7$) had a non-significantly higher mean IQ (mean IQ $= 93.14$) than those with relatively small lesions (lesion size category 0 or 1, $N = 6$) (mean IQ: 93.1 vs 85.5).

In summary, analogous to Goldman's findings with monkeys [4], our findings with hemiplegic children indicate that the intellectual functioning of children with early brain lesions is related to age at the time of testing, as well as age at the time of the lesion [12]. Age at the time of testing was found to be an especially powerful predictor of subsequent intellectual functioning in hemiplegic children with congenital lesions. In particular, our findings suggest that the intellectual development of congenital hemiplegics remains close to the normal curve until 6–8 years of age, at which time their rate of development appears to slow down in comparison to normals. We attribute this pattern of development both to limits on the reorganizational capacity of the early lesioned brain and to the maturation of the nervous system in normals.

Of course, our findings await confirmation by a longitudinal study. Currently, we are obtaining long-term follow-up information on the intellectual functioning of the hemiplegic children in this study and thus far, the data are consistent with the cross-sectional findings. In this longitudinal study, we are investigating whether there are particular markers that predict the severity of the falloff in IQ. In fact, our preliminary longitudinal data suggest that the falloff is more severe in those children with more severe neurological problems, as indexed by larger lesion size, EEG abnormalities, and degree of hemiparesis. For those children who are identified as being at risk for a significant falloff in IQ at 6–8 years of age, it is important to address the question of whether intensive early intervention could diminish

the degree of falloff. In this regard, it is interesting to note that exposing rats with early brain lesions to an enriched environment has been reported to decrease behavioral dysfunction and to diminish decreases in cortical thickness [9, 25, 26].

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