Excessive Postrotary Nystagmus Duration in Learning-Disabled Children

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The Southern California Sensory Integration Test results of 109 learning-disabled children were reviewed and analyzed to investigate the hypothesis that learning-disabled children with excessive postrotary nystagmus durations display greater neuropsychological impairment than learning-disabled children with normal or depressed postrotary nystagmus. The findings supported the hypothesis that learning-disabled children judged to have relatively greater neuropsychological involvement exhibited significantly longer postrotary nystagmus durations. Data analysis revealed that six Southern California Sensory Integration Tests assumed to measure cortical level functions shared significant variance with excessive Southern California Postrotary Nystagmus Test (SCPNT) scores. A similar relationship was not found for normal or depressed SCPNT scores. A connection between cortical level versus brainstem level central nervous system dysfunction, nystagmus, and learning disorder is discussed.

Recent neuropsychological research revealed dysfunction of the vestibular system in significant numbers of children identified as learning disabled (1-8). The vestibular system, together with other proprioceptors and the cerebellum, serves to regulate posture, muscle tone, body equilibrium, spatial orientation, and eye-head coordination (4). Parents, educators, and therapists have noted for years that some children identified as learning disabled exhibit varying degrees of impairment in areas related to vestibular functions, including deficiencies in postural reactions, coordination and body awareness. Ayres recognized (in the 1960s) that some learning-disabled children evidenced vestibular and proprioceptive disorders including a syndrome referred to as disorders in "postural and bilateral integration" (5, 6). This syndrome, more recently termed vestibular bilateral integration, is partially characterized by poor equilibrium reactions, poor oculomotor control (the presence of) remnants of primitive postural reflexes, hypotonic muscle tone, and abnormal responses to vestibular stimulation. All of these symptoms may be directly related to function or dysfunction of the vestibular and proprioceptive systems. de Quiros has also identified vestibular-related disorders in learning-disabled children (8, 7). One syndrome referred to as vestibular proprioceptive disintegration is characterized by muscle hypoto-
nia, poorly integrated postural mechanisms, delays in motor and language development, and deficient vestibulo-ocular reflex to neolauralbryinthine (caloric) testing. Quiros and Schrager (2) recently identified another related syndrome termed vestibular-ocular-motor split, which is characterized by impaired ocular fixation and scanning abilities, and poor eye-head coordination.

Both Ayres (1, 5, 8) and deQuiros (2, 3, 7) have repeatedly found significant numbers of children identified as learning disabled who display depressed or abnormal vestibular-ocular reflexes referred to as nystagmus, following rotatory or caloric vestibular stimulation. Independent researchers have conducted studies that tend to confirm the existence of vestibular dysfunction within the learning-disabled population. These investigations have shown that children with vestibular processing disorders display characteristic soft or nonfocal neurological signs (9, 10), a higher portion of certain language deficits (11), and lower scores on tests of visual-motor integration and reading achievement (12-14). Recently, it has been shown that learning-disabled children may exhibit differential changes in postrotary nystagmus duration as a function of sensory integrative therapy (15).

Introduction of the Southern California Postrotary Nystagmus Test (SCPNT) by Ayres has provided a quick, quantifiable measure of vestibular-ocular reflex following rotation in children 5-9 years of age (16). The vestibular-ocular reflex following rotation is commonly referred to as postrotary nystagmus (PRN). Ayres' most recent research revealed that 50 percent of the learning-disabled children under study has depressed PRN when tested with the SCPNT (1, 8). Thirty-seven percent had PRN within normal limits and 13 percent had PRN that was considered excessive. SCPNT scores of less than -1.0 SD or greater than +1.0 SD from the standardized mean are interpreted as abnormal, although probably measuring different aspects of neurovestibular function (16). In the research referred to above (Ayres), learning-disabled children with excessive SCPNT scores (> +1.0 SD) scored consistently lower on neuropsychological and achievement tests than learning-disabled children with normal or depressed PRN. Ayres stated that “Children with prolonged duration nystagmus, while fewer in number in this population, appear to have greater and more extensive neurological involvement as measured by this study” (1, p 25).

Occupational therapists treat children with sensory integrative disorders that may include dysfunction in the processing of vestibular information. The therapist who is aware of the nuances of vestibular dysfunction is in a better position to assist in differential diagnosis and treatment of children with learning disorders. The purpose of this study was to broaden the diagnostic and clinical significance of vestibular dysfunction in learning-disabled children by investigating the relationship of nystagmus duration to the degree of neuropsychological dysfunction in a sample of learning-disabled children.

Procedures
This was a causal comparative study in the sense that information concerning the variables under investigation was gathered ex post facto. Subjects consisted of 71 male and 88 female children ranging in age from 58 to 116 months. All subjects had medical and educational diagnoses of learning disability and were referred to a children’s rehabilitation center for diagnostic testing and treatment. Subjects’ IQs were within a normal range (80-120) as far as could be determined from psychological and school reports. The 109 children were administered a battery of informal and formal assessment procedures including the Southern California Sensory Integration Tests (SCSIT) and the SCPNT (16, 17). The tests were administered by two therapists experienced in pediatric assessment, one of whom was certified in the administration of the SCSIT. The SCPNT was administered in a well-lighted room in accordance with the standardized instructions. The conditions of administration were the same for all subjects. A SCPNT score of > +1.0 SD was considered indicative of excessive PRN.

The presence of neuropsychological dysfunction in a population of learning-disabled children is controversial and must be regarded as a relative question. The criterion employed to operationally define greater or more extensive neuropsychological involvement for purposes of this study was a combined SCSIT mean score < -1.0 SD. Any subject meeting this criterion on the SCSIT was placed in Group I, designated as having relatively greater or more extensive neuropsychological involvement. All other subjects were placed in Group II, designated as having relatively less neuropsychological involvement. All of the SCSIT subtests, except “Double Tactile Stimulation,” not routinely given to all subjects and therefore not included in the analysis, were used to determine the overall SCSIT mean score for each subject.
A multiple regression equation was generated to determine which of the neuropsychological variables as measured by the SCSIT differentiated those children with hyper-responsive PRN. Multiple regression analysis allows the researcher to predict the outcome of one dependent or criterion variable from several independent or predictor variables considered simultaneously. The SCSIT scores for the 15 children with hyper-responsive PRN served as the independent or predictor variables, and SCPNT scores > +1 SD served as the dependent or criterion variable. Variable coding was performed according to Cohen and Cohen (18), using a program of subsets developed by Schatzoff and others (19). The generation of a regression equation yields an \( R^2 \) value that equals the proportion of the variance of the dependent variable (excessive PRN) accounted for by the independent variables (SCSIT scores). The \( R^2 \) for the equation was .5912 and the standard error of estimate was .21. Thus, the regression model accounted for approximately 59 percent of the variance in the dependent variable (excessive PRN) in terms of the SCSIT scores. The computation of regression equations and accompanying \( R^2 \) values when the sample size is not at least three times larger than the number of independent variables \( k \) is regarded by some authorities as controversial (20) and may result in an inflated \( R^2 \) value. Cohen and Cohen (18) argue that increasing \( N \) does not necessarily resolve this difficulty when a large number of independent (predictor) variables \( (> 10) \) are present. The \( df \) will be larger with an increased \( N \), which, taken by itself, increases power. However, the type I error rate depends on the number of statistical hypotheses, and not on \( N \). Even the potentially higher power conferred by a large \( N \) may be dissipated by a large \( k \) and the inflated \( R^2 \) that large \( k \) may produce. The large number of independent variables in this study (16) was necessary to include the subtests of the SCSIT battery. The results of the chi-square and ANOVA indicate a significant effect between the two SCSIT response groups based on SCPNT scores. The regression analysis was employed with consideration of the large \( k \) as an adjunctive procedure to further substantiate the findings of the previous statistical analyses and to help clarify the relation of individual SCSIT scores to nystagmus-responding categories. The regression coefficients and not the \( R^2 \) value were of primary importance in such an analysis.

In the data analytical spirit (21) of this investigation, multiple regression analysis was felt to be the most sophisticated and sensitive method of providing relative indications regarding which SCSIT subtests were the best predictors of hyper-responsive PRN. In order to accomplish this, the individual regression coefficients for each of the SCSIT subtests were mathematically converted to beta weights. (Beta weights can be thought of as the regression coefficients that would have been obtained if the various regression coefficients were equal to each other in terms of means and standard deviations.) The independent or predictor variable with the largest absolute beta weight is the best predictor of the dependent, or criterion, variable. The calculated beta weights and their corresponding t-values of the independent variables for each of the SCSITs appear in Table 2.

Respective regression equations were also generated for the 45 sub-
Table 2
Beta Weights and t-Values for SCSIT Scores of Children with Excessive Prn

<table>
<thead>
<tr>
<th>SCSIT*</th>
<th>Absolute Beta Wt.</th>
<th>t-value</th>
<th>Alpha Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Motor Accuracy (R + L)</td>
<td>.5614</td>
<td>3.41</td>
<td>*p &lt; .01</td>
</tr>
<tr>
<td>Imitation of Posture</td>
<td>.5017</td>
<td>3.01</td>
<td>*p &lt; .01</td>
</tr>
<tr>
<td>Bilateral Motor Coordination</td>
<td>.4664</td>
<td>2.17</td>
<td>*p &lt; .05</td>
</tr>
<tr>
<td>Standing Balance Eyes Closed</td>
<td>.2311</td>
<td>1.67</td>
<td>NS</td>
</tr>
<tr>
<td>Standing Balance Eyes Open</td>
<td>.0679</td>
<td>.89</td>
<td>NS</td>
</tr>
<tr>
<td>Space Visualization</td>
<td>.3011</td>
<td>2.06</td>
<td>*p &lt; .05</td>
</tr>
<tr>
<td>Figure Ground</td>
<td>.2291</td>
<td>1.91</td>
<td>NS</td>
</tr>
<tr>
<td>Position in Space</td>
<td>.3044</td>
<td>2.04</td>
<td>*p &lt; .05</td>
</tr>
<tr>
<td>Design Copy</td>
<td>.4129</td>
<td>2.81</td>
<td>*p &lt; .01</td>
</tr>
<tr>
<td>Kinesthesia</td>
<td>.0978</td>
<td>.92</td>
<td>NS</td>
</tr>
<tr>
<td>Manual Form Perception</td>
<td>.1871</td>
<td>1.37</td>
<td>NS</td>
</tr>
<tr>
<td>Finger Identification</td>
<td>.0672</td>
<td>.87</td>
<td>NS</td>
</tr>
<tr>
<td>Crossing the Midline</td>
<td>.1071</td>
<td>1.03</td>
<td>NS</td>
</tr>
<tr>
<td>Graphesthesiia</td>
<td>.0410</td>
<td>.47</td>
<td>NS</td>
</tr>
<tr>
<td>Localization of Tactile Discrimination</td>
<td>.0601</td>
<td>.81</td>
<td>NS</td>
</tr>
<tr>
<td>Right/Left Discrimination</td>
<td>2513</td>
<td>2.00</td>
<td>NS</td>
</tr>
</tbody>
</table>

Double tactile stimulation was not routinely given to all subjects and was not included in the analysis.

Subjects with normal SCPNT scores (-1.0 to +1.0 SD) and the 49 subjects with depressed SCPNT scores (< -1.0 SD), using normal and depressed SCSIT scores as dependent variables and SCSIT scores as independent variables. The $R^2$ for the normal SCPNT group was .0916, indicating that SCSIT scores accounted for less than 10 percent of the shared variance with normal SCPNT scores. The $R^2$ for the depressed SCPNT group was .2348, indicating that SCSIT scores predicted 23 percent of the shared variance with depressed SCPNT scores. Computation and comparison of beta weights revealed that almost the entire amount of variance for this equation was dependent on the "Standing Balance Eyes Closed and Open" tests of the SCSIT. This finding supports an earlier study in which the standing balance tests along with selected clinical observations were found to be related to depressed SCPNT scores (10).

The difference in mean ages between the two groups, although not statistically significant ($t = 1.55, p > .10, df/107$), indicates that Group I children tended to be younger than those children in Group II. Children with relatively greater neuropsychological involvement are more likely to be identified as having learning difficulties at an earlier age than those children with little or minimal neuropsychological dysfunction. This fact was believed to have contributed to the age difference between the two groups. A Pearson product moment correlation was computed between PRN duration and age. No relationship was found ($r = .16, p > .01$).

Discussion and Conclusion
Measurement of PRN has been one of the primary techniques used to differentiate children with vestibular processing dysfunction. Ayres has hypothesized that differential neurophysiological mechanisms may be operating as causative factors in depressed and excessive PRN (1, 16). Depressed PRN may result from overinhibition of the vestibular nuclei—possibly through connections with the cerebellum. Excessive PRN has been hypothesized to result from an insufficient amount of inhibition from higher level cortical centers acting on the vestibular nuclei and pontine reticular formation (16). Markham (22) has reported experiments on animals and humans, which indicate that an ablation or lesion of certain cortical structures significantly enhances or prolongs vestibular nystagmus. He concluded his review by stating,
“Most of these studies indicate that cortical lesions actually enhance or prolong vestibular nystagmus. This may suggest that the vestibulo-ocular arc is released from higher centers and supports the general concept of the cerebral cortex exerting an inhibitory type of modulation on lower centers.” (22, p 592).

Recently Takemori, Ono, and Maeda have demonstrated that the parietal lobe is of particular importance in the cortical control of vestibular nystagmus (23). Nystagmographic studies of children with overt cerebral dysfunction including developmental delay and cerebral palsy have revealed significantly longer PRN durations for these children than for normal controls (24-27), suggesting reduced cortical regulatory control over vestibular nystagmus.

The information reported in Table 2 indicates that those learning-disabled children exhibiting excessive PRN durations display lower scores on those Southern California Sensory Integration Tests that require higher level integrated cortical functioning: Motor Accuracy, Imitation of Posture, Design Copy, Bilateral Motor Coordination, Space Visualization, and Position in Space. In other words, the above tests of the SCSIT shared the most variance with, or were the best predictors of, excessive PRN.

The findings of this study provide tentative support for the Ayres assertion that learning-disabled children with excessive PRN display “greater or more extensive neurological involvement” than learning-disabled children with normal or depressed PRN (1, p 25). Inferences from this investigation and related literature indicate that learning-disabled children with excessive PRN (14 percent of this sample) may suffer from subtle Central Nervous System dysfunction of cortical origin. However, as noted previously, the question of neuropsychological involvement in a learning-disabled population is a relative one. The relationship between excessive PRN and degree of neuropsychological dysfunction found in this study must be regarded as tentative. The results of this investigation provide indications for further research.

Acknowledgments

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