

PERSISTENCE AND CHANGES OF NEUROLOGICAL SIGNS  
IN LEARNING DISABLED SUBJECTS OVER A 15 YEAR PERIOD

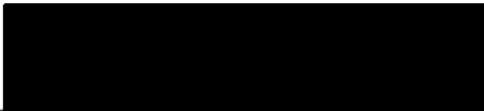
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### Abstract

Neurological data from the University of Victoria Longitudinal Study of Learning Disabled Children Growing Up (Hern, 1984; Spreen, 1987) was further analyzed to determine the nature of the changes which took place, and to determine if a pattern of change could be identified, based on the neurological systems potentially involved.

Subjects were 110 learning disabled persons, evaluated by neurological examination at Time 1 (mean age 9.6 years) and Time 2 (mean age 24.2 years). Each subject was evaluated on 25 neurological signs, rated by severity as hard or soft, and comparisons were made.

This thesis documents the extreme changeability in signs and subjects over time. Most signs found at Time 1 did not tend to persist, and many signs appeared at Time 2 in subjects who showed no evidence of them at Time 1. No subject who showed signs had the same profile of signs at both times, and 38% of the subjects with signs at Time 1 had a complete change of signs.

Of the subjects without neurological evidence of signs or damage at Time 1, 68% did show signs at Time 2. This suggests the possibility of subtle and perhaps undetectable (in childhood) damage in some learning disabled subjects.

Most researchers consider the presence of a single soft sign to be unimportant. This study found that while 14 of the 16 subjects with a single soft sign at Time 1 lost that sign, only 6 were sign-free at Time 2--the others gained different signs.

Signs which are considered to represent maturational lag were analyzed. While these signs often disappeared over time, other signs were usually found. There were no differences between older (age 10 and above) and younger (under 10) subjects in terms of Time 1 signs.


Signs were categorized in terms of possible neurological systems involved. It was predicted that signs gained over time would be likely to be gained within systems already implicated as damaged. This search for a predictive pattern of signs was not successful.

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## Introduction

### Defining and Classifying Hard and Soft Signs

Neurological signs are indicators of abnormalities in nervous system functions. They are commonly classified as "hard" or "soft". Hard neurological signs such as reflex abnormalities, ataxia, and paresis, when strongly and reliably elicited, are generally considered to be evidence of specific, localizable central nervous system damage or deficit (Tupper, 1987). Denkla (1978) refers to these as the "classic" neurological signs.

The etiology of hard signs is thought to be that of neurological insult resulting from such pre- or peri-natal events as anoxia, toxic or metabolic processes, structural damage, and direct traumatic injury such as that resulting from blows to the mother's abdomen or from obstetrical instruments. These signs may also result from events after birth of a traumatic, toxic/metabolic, or anoxic nature. Hard signs, therefore, are thought to be direct evidence of neurological insult.

Soft signs are often described as more subtle, more equivocal, and less stable than hard signs, and are usually considered not "localizable", ie. not signifying specific damage in a particular part of the CNS.

Many items have been classified as soft signs.

Peters, Romine, and Dyckman (1975) tested 80 "soft signs" in their neurological examination. These items ranged from choreiform movements and deep tendon reflexes, through to right-left orientation, winking, writing to dictation, and spelling. Such a broad definition of soft signs, in this and other studies, has caused much confusion, as it is difficult to discuss and evaluate soft signs if they cover the range from neurology to academic achievement and behavior.

Soft signs can be categorized, to reduce the level of confusion. The most basic level at which to study soft signs (beyond medical scans and EEG) is that of neurological signs. Other signs, involving components of reading and writing and of neuropsychological test batteries, require not only intact neurological systems, but also higher level functions. The use of such high-level signs can lead to the additional complication of dealing with several levels of potential interactions.

Restricting the definition of soft signs to only those with a proposed neurological basis, tested by a neurological examination, still allows room for several systems of categorization in the literature, with consequent disagreement. Rutter, Graham, and Yule (1970) in their Isle of Wight study provided the well-known grouping according to: 1) soft signs indicating

developmental delay, which disappear with age; 2) signs difficult to elicit and reproduce, therefore minor; and 3) signs which can also result from causes other than neurological damage (eg. strabismus, weak grip). Such a grouping tends to diminish the possible significance of soft signs, and does not encourage research. Spreen (1989) has responded to this critical grouping with counter-arguments based on his research with the University of Victoria Longitudinal Study of Learning Disabled Children Growing Up. His research supports the usefulness of using soft neurological signs in the evaluation of learning disabled subjects.

A more useful grouping for the purpose of exploring signs is proposed by several researchers. Denkla (1978) divides soft signs into two types: those considered to be classical neurological abnormalities but in a more subtle form, which implies some localization ("pastel classics"); and those which are age-referenced, or imply problems of developmental processes ("developmental signs"). She thinks of soft signs as "indicators of how the brain is organized in terms of space (pastel classics)...and time (developmental)" (p.236). Levine, Brooks, and Shonkoff (1980) make a similar division, referring to the two forms as equivocal hard signs and

developmental soft signs. Tupper (1987) calls them soft signs of abnormality and developmental soft signs.

While there appears to be agreement among many researchers on the type of division, not all of these researchers classify signs into the same categories. For example, Szatmari and Taylor (1984) consider the Babinski reflex to be developmental, since it is normal in infants; most others consider this to be a classic or minor hard sign. The classification of signs into these two categories will be explored later in this thesis.

#### Meaning and Importance of Soft Signs

The meaning and importance of soft signs has been debated for many years, and this search has been hampered by the problems of definition, as well as those of methodology; results to date are inconclusive (for a review see Hern, 1984; Levine et al., 1980; Spreen, 1987; Yule & Taylor, 1987). Some consider soft signs to be of no value (Schmitt, 1975). Others consider them to be important because of their prevalence in populations with disturbances of learning or behavior (Rie, 1987), but no one is sure of their specific effects. This search for the particular significance of soft signs has been the spur for much of the research. In fact, most studies of neurological signs specifically study soft signs, and exclude subjects with hard signs or definite evidence of

brain injury. The research has not yet been able to indicate causality in the connection between minor neurological deviation and soft signs, although this assumption is often made (H.G. Taylor, 1983; Tupper, 1987), and research in this area continues.

A difficulty with interpreting the relationship between soft signs and neurological damage is that soft signs are found in so-called "normal people" too, although not in the same quantity, nor in all signs. Hertzig, Bortner, and Birch (1969) found that one-third of the 15 control children in their study had soft signs, although none had more than one sign, and that was usually a slight abnormality of muscle tone. Hern (1984), in the Victoria Longitudinal Study, found that 58.7% of the adult controls showed soft signs, primarily asymmetry of skull or limb, abnormal tendon reflexes, synkinesia, and intention tremor. Gaddes (1980) suggests that the so-called "normal" learners who show soft signs may actually have some damage, but outside of the areas involved in academic activities. Touwen (1987) suggests that even though signs are also found in controls, we should consider soft signs meaningful unless it is discovered otherwise.

The finding of some signs in controls, the question of reliability (discussed later), and the lack of studies following signs through to maturity have caused many

neurologists and researchers to question the assumption that soft signs indicate damage. The evidence of a single sign is particularly suspect. Gardner and colleagues (1987) found in their study that a high proportion of both the borderline personality disorder group and the control group had one sign (76% vs 55%). Levine and colleagues (1980) suggested that a single sign may not be meaningful. Rutter et al. (1970) stated, based on the results of their study, that a mild sign of incoordination or weakness is more likely to be associated with normality than abnormality; however, a marked hard sign is probably indicative of neurological disorder. Touwen and Kalverboer (1973) suggested that with few exceptions no neurological sign has meaning when found in isolation.

An accumulation of signs may, however, be convincing evidence where one sign is not enough. Hertzog (1982) used as her definition of neurologically abnormal those who showed any localizing signs (hard signs), or two or more non-localizing (soft) signs. Gardner, Lucas, and Cowdry (1987) found two or more soft signs to be a useful cut-off, significantly differentiating borderline from control subjects (65% vs 32%). Gaddes (1980) considers subjects with at least three soft signs (from a list including nystagmus, tremor, athetoid or choreiform movements, asymmetry of skull or of sensation) to be

indicative of brain damage. Touwen and Kalverboer (1973) used a quantitative score, based on the assumption that the number of abnormal signs increases in proportion to the intensity and extent of cerebral dysfunction. All of the above studies used both pastel classic and developmental soft signs.

Rutter, Graham and Yule (1970) disagree with this summing, suggesting that it is the type and not the sum that matters.

As most of the research results to date have not found a correspondence between signs and specific damage, or signs and specific behavior, conclusions favour a general effect. Tupper (1987) states his expectation that soft signs will not serve as "specific neurological markers, but as nonspecific factors in behavioral or psychiatric disorders" (p. 12). Hertzog (1981) concludes from her work that soft signs reflect a primary disturbance in the organization of the CNS. Touwen (1987) considers signs to indicate a vulnerability of the nervous system, such that a child's ability to cope with the environment (in the broadest sense of the word) becomes endangered.

The neurological basis of soft signs is not known. Shaffer and colleagues (1983) suggest many origins, including perinatal trauma and genetic factors. Rutter

(1983) sees soft signs as a result of diffuse damage, of a genetic abnormality in biochemical metabolism, or possibly as a lesser variant of traumatic or infective brain damage. Szatmari and Taylor (1984) make a distinction between causes of classic and developmental soft signs, stating that developmental signs indicate delay, not necessarily structural damage. Speculation as to the etiology of soft signs, then, ranges from damage similar to that thought responsible for hard signs, through to developmental delay. This delay is considered by some to be caused by the damage outlined above, and by others to represent a natural variation in maturation rates.

#### Neurological Signs in Learning Disabled Populations

There is evidence of an increased incidence of neurological signs in various populations. This includes psychiatric populations such as schizophrenics (for a review see Heinrichs & Buchanan, 1988), borderline personality disorders (Gardner et al., 1987), behaviorally-defined groups such as attention deficit disorder/hyperactives, and the learning disabled (research on both latter groups is reviewed by Rie, 1987). Both hard and soft neurological signs are found often in learning disabled populations.

Brain dysfunction is accepted by many researchers to be a contributing factor in learning disabilities,

although not necessarily a direct cause (Deutsch & Schumer, 1970; Helper, 1980; Hertzog et al., 1969; Rie, 1987). The term Minimal Brain Damage or Dysfunction (MBD), has been used to describe this brain dysfunction, and the presence of soft signs has been used as one of the main bases on which to infer MBD (Tupper, 1987). However, the term and its usage are subjects of much controversy and confusion. The term MBD has included such behavioral expressions as hyperactivity, dyslexia, and attention deficit disorders, and these behaviors have been used to, in fact, diagnose MBD without associated neurological criteria. Denkla (1978) states that there is no such single entity as MBD, and no "MBD child", for the variance among those classified as MBD is greater than that among normal children, and "lumping together" a variety of syndromes and calling them one name is misleading. She argues not for abandoning the term, but for sharpening its definition to ensure that brain factors really are implicated, and calls it "MBD-learning disabled" (p.232).

The role of brain dysfunction/neurological signs in learning disabilities is not clear, and has turned out to be such a complex question that it may never be entirely answered. Most researchers recognize that learning disabled children are a diverse group, and that not all

learning disabilities can be attributed to neuropathological factors. Other potential causes involve issues of emotional stability, self-esteem, family situation, interpersonal difficulties, inherited ability, and combinations of the above.

For those learning disabled children who have neurological signs, there are several possible ways that the signs might affect the learning disability: 1) The signs may cause, or contribute directly to the disability. 2) Some underlying damage may cause the disability, and also cause the neurological signs. 3) The signs and the disability may be manifestations of two totally different processes or points of damage. 4) The sign may indirectly cause the disability, by its effect on the child's self-esteem, which then undermines his/her learning.

A group of learning disabled subjects presents the opportunity to study neurological signs occurring with a high enough frequency to allow some exploration and analysis. This is a population which has often been studied, and therefore comparisons with other studies are possible. This exploration may lead to hypotheses about the relationship between various neurological signs, which can then be applied to research involving not only learning disabled populations, but also other populations with evidence of neurological signs.

### Issues Associated with Neurological Signs

Five important issues related to the significance of, and the evaluation of neurological signs will be discussed. The first, directed more to the "pastel classic" neurological signs (minor or subtle manifestations of the classical signs), is that of the reliability of measurement. The second issue relates to the signs considered to be caused by maturational/developmental lag--do they go away? The third issue is that of the stability or persistence of signs over time; this can be discussed in terms of both hard and soft signs. The fourth issue is the complexity of assessing neurological change over time. The fifth issue relates to the question of whether there is a pattern of neurological signs that is predictive of change over time.

#### The reliability of neurological testing.

An important issue when analyzing the results of studies on neurological signs is that of reliability. Three questions of reliability are of particular concern when testing neurological signs. They are:

- 1) What is the level of inter-rater reliability? This involves neurologists' criteria--the threshold of what they will call abnormal--and methods of eliciting the sign.

2) What is the level of intra-rater reliability? How consistent is a neurologist or other rater over time?

3) What factors affect the subject and his/her performance? Rutter, Graham, and Yule (1970) conducted reliability tests within their comprehensive Isle of Wight study. They found most hard signs to be very reliable, and the soft signs, particularly those which are classic, or minor versions of hard signs, not to be very reliable. They noted that the testing of reflexes, for example, can be affected by the state of relaxation of the subject, by the room temperature, and the method of elicitation. Their finding regarding the question of inter-rater reliability was that with adequate standardization and training, a satisfactory level of reliability could be reached on most items (but not all). They cautioned against putting too much emphasis on slight isolated abnormalities, and also pointed out the danger of comparing studies because of the differing thresholds.

Stokman and colleagues (1986) performed a set of reliability studies on data from the National Collaborative Perinatal Project (Shafer et al., 1986; Shaffer et al., 1985). Consistency of sign (test-retest reliability) was found to be marginal to good. Based on their other results, they attributed this to within-subject factors rather than to problems with the raters.

Inter-rater reliability was tested both in the field and on videotape, with reliability judged to be acceptable overall. Intra-rater reliability was good. Kuzma and colleagues (1969) conducted extensive research on the reliability of standard neurological examinations, finding that most items were reliably measured, in terms of inter- and intra-rater reliability.

Peters and colleagues (1975), in their discussion of subject factors affecting performance, cite developmental factors, passing phenomena, and behavioral epiphenomena such as anxiety. Other possible factors are alertness (Stokman et al., 1986), motivation, and compliance (Marcus, Hans, Lewow, Wilkinson & Burack, 1985).

Shapiro, Burkes, Petti, and Ranz (1978) indicate that most soft signs can be reliably identified and quantified.

The conclusion of the researchers cited above is that most signs can be rated quite reliably, given some planning and training of evaluators.

There is a difference between what can be done, and what is done. For example, McMahon and Greenberg (1977) found that signs varied remarkably from one exam to another with hyperactive subjects. Many studies do not include reliability tests. However, for all studies which carefully define their criteria and train their raters, it

appears that an acceptable level of reliability can be reached.

Finally, there are random errors of measurement which occur when testing signs. Shafer and colleagues (1983) suggest that such errors would lower consistency effects, rather than spuriously raising them. They suggest that the bigger problem is in missing something that is there.

#### Maturation lag.

The question of maturational or developmental lag occurs with those signs which are appropriate and normal at an earlier age, but usually disappear with maturation. These are signs such as dysdiadochokinesia and synkinesia. When the signs are seen in an older child, they are considered abnormal, and it is often presumed that maturation has been delayed. To some researchers (eg. Rutter et al., 1970), this means that the individual will eventually mature and that the sign or signs will go away. However, to others (Denkla, 1978; Deuel & Robinson, 1987) delay and lag do not necessarily imply a catching-up, but only identify signs which would go away in the course of normal development.

This area has been studied from both a cross-sectional and a longitudinal perspective. Research results are mixed.

The results of cross-sectional studies are as follows:

1) Peters et al. (1975) found a significant and negative correlation between age and number of signs in learning disabled subjects aged 8-11.

2) Hertzig (1982), studied 156 children in special education placement, divided into four age groups, ranging from mean age 9 years 5 months in the youngest group to 15 years 6 months in the oldest. She found that there was a significant decrease in the incidence of incoordination and graphesthesia with age.

3) Shaywitz, Shaywitz, McGraw, and Groll (1984) studied three groups of students: normal (n=32), gifted (n=37), and learning disabled (n=35), ages 9.5 to 14.1 years. They found that the number of neuromaturational signs decreased with age for the normal and gifted subjects, but stayed relatively the same for the learning disabled subjects. Age groups used were <11, 11-12, and 12+.

4) Erickson (1977) in her study of the relationship between reading disability and MBD tested all 155 second grade students in one public school (age range 82-105 months, mean age 7.5 years). Eleven neurological test items were administered. None of the neurological variables was significantly correlated with chronological age. She speculated that the restricted age-range

contributed to the generally very low correlations. Comparisons made by dividing the subjects into reading disabled and control also failed to reveal significant differences.

The findings of the longitudinal studies are as follows:

1) Shaffer and colleagues (1983) found that more than half of their subjects with dysdiadochokinesis and synkinesis at age seven still showed these signs 10 years later.

2) In the longitudinal portion of Hertzig's (1982) study, 53 of the children were seen at mean ages 10.5 and 14.8. She found that they showed a mean of 4.47 signs at Time 1, and 2.70 signs at Time 2. However, most of those with 2 or more soft signs at Time 1, still had 2 or more at Time 2.

Both the cross-sectional and the longitudinal studies were split evenly between those supporting at least some signs which could be said to improve with age (and presumably maturation), and those finding no support. The studies used different cut-off points for their age groups, and this makes comparison between studies difficult. Some compared the mean number of signs in these age groups, and others ran correlations between age and signs.

Different signs have different ages at which normal maturation is said usually to be complete. Shapiro and colleagues (1978) suggest that signs follow maturational curves which reach the adult level in normal children by the age of approximately 8 years. Wolff, Gunnoe, and Cohen (1985) suggest that age 10 (at the latest) is the age of maturational completion for motor signs. They also found a greater relative frequency of signs in younger than older children (Wolff et al., 1983, 1985), as did Connolly & Stratton (1968).

Persistence of signs, and neurological outcome.

A distinction should be made between the terms persistence and stability. Persistence refers to the tendency of a sign which exists at one point in time (sometimes called a positive sign), to continue to exist over a certain period of time. Stability refers to the tendency of the sign to remain positive or negative (ie. either present or absent) over two or more testings. This distinction is important because the terms are related but not synonymous. If the time period between testings is so short that neither neurological improvement nor deterioration is likely to have occurred, then the reliability of measurement is considered to be the issue, rather than persistence or stability. The time between measurements in persistence or stability studies varies

greatly. Hertzog (1982) used a four-year time-frame, Shaffer and colleagues (1983) re-tested after 10 years, and Hern (1984) re-tested after an average of 15 years.

Persistence can be examined from the perspective of the signs themselves (eg. Does ataxia tend to persist over time?), or from the point of view of the persons (eg. Does this person or group of persons persist in having neurological signs over time?).

From the perspective of the signs, the expectation of many neurologists and researchers is that hard signs persist, and that soft signs do not. Of the soft signs, the minor or classic soft signs are criticized as unreliable (as discussed earlier), and the maturational signs are often expected to be outgrown. Behind these expectations is the assumption that if signs do not persist, then they are not so serious, and it is assumed that the child or adult has improved. Research on the signs has been discussed earlier; research from the perspective of the subjects--the neurological outcome of learning disabled subjects--follows.

Gillberg (1985) reported on her three-year follow-up of children diagnosed at age seven. There were 6 with motor/perception disorder (MPD group), and 38 with MPD and Attention Deficit Disorder. Many no longer showed abnormal neurological signs at age 10 (4/6 of the MPD

group, and 17/38 of the combined group). However, most of these students, as well as those who still had abnormal neurological signs, continued to show behavioral and/or school achievement problems at age 10. This suggests that the signs were not necessarily specifically responsible for the learning and behavior problems.

Menkes, Rowe and Menkes (1967) in their 25 year follow-up of 11 hyperkinetic subjects with MBD, found that all 6 who had evidence of definite neurological abnormalities at Time 1 also had them at Time 2. Of the other 5 subjects who had "probable" brain dysfunction at Time 1: 3 were abnormal at Time 2, and 2 were psychotic. Overall, Menkes, Rowe, and Menkes concluded that there was a trend to crystallization or worsening of neurological signs over time.

The finding of psychoticism as a possible outcome of some subjects diagnosed with MBD and neurological signs, brings up the whole question of emotional outcome. A review by Shaffer (1978), suggests that children with neurological dysfunction--showing a number of soft signs and emotional disturbance--may develop specific psychiatric syndromes in later life. However, he cautions about attributing cause or partial cause to neurological soft signs until more research is done.

Hern (1984) found that many neurological signs were unstable, and that hard signs were no more likely to persist than soft signs. Learning disabled subjects did not, for the most part, "grow out of" these signs; in fact, the tendency was for more signs to appear in early adulthood (age 25). Many subjects who were classified as learning disabled with no neurological hard or soft signs at the time of the first evaluation (mean age 10), developed signs over the course of approximately 15 years.

The prognosis for learning disabled students is generally much poorer than for normal students (Spren, 1987). The role of neurological signs in these outcomes is difficult to interpret, although the overall impression is that of greater difficulties with greater neurological impairment.

Neurological outcome measures: How to evaluate signs.

What is a "good neurological outcome" from a research point of view? Fewer signs is perhaps a rough indicator, especially if no new signs are gained. A common method of analysis is to sum signs and compare subjects to their own scores at two points in time, or to compare the mean scores of groups. However, simple summing can conceal more than it reveals (Yule & Taylor, 1987). For example, is the person who has dysarthria and abnormal tendon

reflexes at Time 1 and ataxia at Time 2 actually better, because of fewer signs?

For group comparisons, the discovery of the higher frequency of signs in behavioral, psychiatric, and learning-disabled groups compared to controls is useful. However, even though a group has a higher mean number of signs than controls, there is often overlap. While summing can usually differentiate groups of children with problems from control groups, Rutter et al. (1970) concluded that the total composite number was not useful, as it did not (in their study) differentiate children with different types of problems (eg. mental retardation from epilepsy or reading retardation). For this reason Yule and Taylor (1987) suggested using signs as screening devices only. If signs are to be more informative than this, another way of evaluating them, rather than simple summing, has to be found.

From the neurological perspective, one can look at the type of sign. Sensory signs are considered more "soft" than motor signs (Denkla, 1978), and therefore are often considered less serious. Some signs, such as an abnormal Babinski reflex, are considered hallmarks of damage by some neurologists (Van Allen, 1969). However, other signs are hard to rank because of differences of degree, and the necessity of evaluating the signs in the

context of history, of presenting problems, and of other accompanying signs and test results.

One can also look at the degree of abnormality. If a sign becomes less severe, then there is improvement in the sign (although not necessarily improvement in the subject's overall condition). However, with a change of signs between times, the picture becomes more complex.

Therefore, looking at the signs separately, especially the signs considered to be "soft", offers a limited picture. It is also necessary to look at the interrelationships between signs and to look at the different possible configurations (Reitan, 1974). Also, dealing with hard and the soft signs as if they were totally separate entities, and eliminating subjects with hard signs from many studies may be obscuring important connections.

#### The search for patterns of signs.

The next logical step is to group the signs. This is the search for a pattern that makes sense, and which can be backed up by research.

Much subgrouping of a functional nature has been done. Yule and Taylor (1987) provide a comprehensive overview of different attempts at classification. Some of the attempts at classification are based on patterns of behavior, some on functional learning problems, and others

on physiological measures or on coordination. They also include their own unsuccessful attempt, using a factor analysis of the data from two of Taylor's studies (1985, cited in Yule & Taylor). Their conclusion is that no empirical attempts at classifying soft signs has worked to date.

### Purpose of this Study

#### Background

Hern (1984) examined the neurological data from the Victoria Longitudinal Study of Learning Disabled Children, to answer several questions relating to neurological signs, including determining the persistence of neurological signs in learning disabled subjects.

Hern found many changes in neurological function over the time of the two evaluations (mean ages 10 and 25). Changes occurred in overall neurological status, in the number of signs per subject, and in the signs themselves. Only a few subjects improved, and overall the trend was toward an increase in neurological signs with age.

Hern concluded that there is little evidence from her results to support the widely held belief that learning disabled children with soft signs will "catch up" in neurological maturity or CNS integrity.

### The Need and Opportunity for Further Study

Currently, those who regard soft signs as useful consider them to be overall indicators of neurological dysfunction (for screening), or they use sums of scores as the basis for comparison between groups. Both the number of signs and the particular types of signs need to be taken into consideration to obtain a useful perspective. However, many subjects have more than one sign to evaluate, and the various combinations of possible signs is very complex. So far no useful pattern of signs has been determined.

The search for a connection between brain damage and behavior has led to more questions, and to controversy. It is a complex area, and the results of many studies are contradictory. An important step in the process of discovering the role of signs in brain damage, and their relation to behavior is to determine the course of those signs over time. This is not to say that signs which do not persist over time are unimportant. Rather, it explores the connection between the damage and the signs, before that of the signs and behavior.

The data collected for the Victoria Longitudinal Study offer an opportunity to investigate changes in neurological signs over time, on an individual level. Hern conducted several important analyses related to the

frequencies of signs in the groups studied. However, the number of subjects and signs involved, and the time span of approximately 15 years between examinations, encourages more questions. The data allow some interesting research which could not be done, or has not been done, in other studies.

What is the course of these signs? Can the signs which persist be differentiated from those which do not? Is there a pattern of signs which can tell us something about neurological outcome? What happens to the subjects over time?

#### Purpose and Details of this Study

This project, then, is a further analysis of the data first evaluated by Hern. This study proposes to look at signs from several angles, in an attempt to answer a few of the many questions. First, an analysis of the individual signs will be conducted; second, an analysis of the subjects and their signs will be done. While it is recognized that there are limitations to the one-dimensional approach of this part of the study (ie. each sign is followed separately, even though subjects have different combinations of signs), an attempt will be made to look at the data from several angles, and to provide a different perspective from those of previous studies.

Third, an analysis of the findings related to maturational lag will be performed.

Then the major premise of the study will be explored. This involves the search for a different type of pattern--one that does not appear to have been previously explored. Hern did not detect any obvious patterns of signs during her analysis; however a detailed examination, following signs from Time 1 to Time 2 on an individual basis, was not within the scope of her dissertation. This search involves an analysis of neurological signs in terms of the involvement of specific neurological systems. That is, signs, both hard and soft, will be considered indicators of damage within specific neurological systems (eg. corticospinal, cerebellar, etc.).

The hypothesis is as follows: Damage to a particular system may result in the display of certain signs at one point in time. However, at another point in time, because of neurological development, changed demands on the system, or deterioration, different but related signs might appear. This might account for the "disappearance" of signs in studies which attribute this disappearance to maturation or improvement, even though other problems remain. The damage may still be there, but expressed differently. This also could be an explanation for the appearance of signs which were not present earlier.

The neurological perspective of this study is that hard and soft signs are indicators of damage in the CNS, and are on a continuum (Spreeen, 1987). The signs have been graded by severity. Hard signs are considered to be severe, and soft signs are considered to be less severe, or moderate, or sometimes bilateral instead of unilateral evidence of the same signs, as determined by the project neurologist. Some analyses will take these gradations into account, while others will use only the dichotomy of presence/absence. In comparisons of these data to other studies, adjustments will be made and noted (eg. synkinesia is considered by most other studies to be a soft sign, no matter what the severity, so a comparison would be made using both levels of severity from this study).

The following guiding questions are posed.

1. Signs: What happens to the individual signs between Time 1 and Time 2? Which signs are most likely to appear at Time 2? Which signs are most likely to disappear?

Based on the literature, signs thought to represent maturational or developmental lag would be most likely to disappear. Signs considered to be those of maturational lag, and comparable to signs to be tested in this study, are: graphesthesia and incoordination (Hertzog, 1982); dysdiadochokinesia and synkinesia (Levine et al., 1980;

Rie, Rie, Stewart & Rettemnier, 1978); dysdiadochokinesia, synkinesia, mild ataxia (lower body incoordination), mild incoordination (upper body), dyspraxia of tongue, and graphesthesia (Peters et al., 1975); dysarthria and simultagnosia (Rutter et al., 1970); dysdiadochokinesia, synkinesia, graphesthesia, choreiform movements (Shafer et al., 1983); dysdiadochokinesis, synkinesis, and choreiform movements (Shaywitz et al., 1984).

2. Subjects: What happens to the subjects between Time 1 and Time 2 in terms of number of signs observed, and the number of signs which are retained vs the number lost or gained?

3. The results of several studies indicate that signs considered to be developmental are found more frequently in younger children than in older children. These results are used to support the concept of maturational lag. Other studies have found no difference. There is some evidence to suggest that age 10 is the normal upper limit of neurological maturation for most signs. Age 10 will be used to separate the subjects into two groups to answer the following questions. Is there an age difference affecting the number of signs found? Which signs are most frequently found?

4. Is there a pattern of signs occurring, which could account for the instability and conflicting results found in most studies, and which would provide some predictive guidelines?

Working from the theory outlined above--assigning signs to the neurological system or systems most likely implicated, the expectation is as follows: for subjects with signs in one or more neurological systems at Time 1, the signs gained at Time 2 will most often be signs of the same system or systems.

#### Method

The base data for the current study comes from the University of Victoria Longitudinal Study of Learning Disabled Children Growing Up (Spren, 1987).

#### Subjects

The learning disabled subjects had originally been referred to the University of Victoria Neuropsychology Lab and/or the Special Counsellor's Office of the Nanaimo School District #68, between 1966 and 1972. They were referred because of learning difficulties, and not by a strict definition of learning disability. They were selected retrospectively for the longitudinal study on the basis of their age at the time of their original referral, the absence of primary sensory loss and primary emotional

disturbance, and an IQ score of at least 70 on the verbal or the performance subscale of the WISC. This original referral to the Neuropsychological Laboratory took place when the subjects were in the age range 7.5 to 12.5 years, but the neurological examination, performed by a qualified neurologist, usually took place prior to the referral, and in fact many of the referrals were from the neurologist. The mean age of subjects at the time of the neurological evaluation was 9.6 years, with a range from 6.7 to 13.1 years.

Subjects in the laboratory were administered a battery of neuropsychological tests, a neurological examination by a qualified neurologist if this had not already been done, and the WISC. The period of original assessment is referred to as Time 1, for the purposes of this thesis.

Subjects were called back 15 years later (on average) to participate in a second phase (here referred to as Time 2) of the Longitudinal Study of Learning Disabilities. (While many subjects also participated in Phase I, 7 years after the original referral, no neurological testing was done at this time and therefore this phase was not of interest in the present study.) During Time 2, these learning disabled subjects were administered another battery of tests, including the WAIS-R, the MMPI, and a

20-minute neurological examination by a qualified neurologist. They also participated in a structured interview exploring their educational, medical, social, relationship, occupational, and spare time activity history and current status. The mean age of subjects was 24.2 years, with a range from 17.9 to 29.2 years.

Subjects were selected from the data base for the current study if they had participated in both Time 1 and Time 2, with full neurological data available for both times. Their interviews were screened for the occurrence of any intervening disease or accidents which could have resulted in brain damage and thus affected the results.

Of the subjects who took part at Time 1, 4 had inadequate Time 1 neurological information, 35 did not return for Time 2, 11 of those who did return did not participate in the neurological evaluation at Time 2, 5 had injuries or diseases between testings affecting the neurological evaluation at Time 2, and 10 had one or more head injuries between testings which could have affected Time 2 testing (unconsciousness for a period of several hours, or coma, or concussion with effects lasting a week or more). Those subjects meeting the criteria to be included consisted of 110 learning disabled subjects: 27 females (24.5%) and 83 males (75.5%).

Subjects were also classified on the basis of the neurologist's report at Time 1 into three groups: Group 1) those with definite evidence of significant brain damage or dysfunction; Group 2) those with questionable or minimal brain dysfunction; and Group 3) those with no evidence of brain dysfunction. Not all subjects with the diagnosis of brain damage or minimal brain damage showed the specific signs tested in this study: diagnoses were made by the neurologist based on examination, history, and EEG reports.

For the purposes of this study, and for comparability with other studies, it was decided to further subdivide the groups into the following:

- 1A) diagnosed brain damage with hard signs (and often soft signs), abbreviated as: BD-hard signs, n=28;
- 1B) diagnosed brain damage with soft signs only: BD-soft signs, n=8;
- 1C) diagnosed brain damage with no signs: BD-no signs, n=2;
- 2A) minimal damage with soft signs: MBD-soft signs, n=35;
- 2B) minimal damage with no signs: MBD-no signs n=15; and
- 3) no neurological evidence of damage and no signs: LD, n=22.

These groupings refer to status at Time 1.

There is controversy about labelling children as brain-damaged or MBD. However, the abbreviations used in this study represent the diagnosis by the neurologist, or in the case of MBD-soft signs, the presence of signs with or without other minimal factors. It is also recognized that the brain damage, particularly MBD, may in some cases represent brain dysfunction, rather than necessarily structural damage.

#### Procedure

Twenty-six neurological functions which are usually tested during a neurological examination were placed on a checklist prepared by Hern (1984) in consultation with the project neurologist. This checklist was used for the Time 2 evaluations, which were performed by the project neurologist. The examinations were performed in the office of the project neurologist, and lasted approximately 20 minutes. The examination was done "blind" as much as possible. That is, the neurologist was given no history of the subjects, and the subjects were instructed beforehand not to reveal any history, and not to indicate whether they were members of the learning disabled group or the control group (control subjects were tested at this time, but since they had not been tested at Time 1, they were not included in this study). The project neurologist was one of the two neurologists who

had performed almost all of the evaluations at Time 1; however, the other neurologist performed the majority of those examinations. The neurologists' written reports and records from Time 1 were coded retrospectively, with the help of the two neurologists, using the checklist criteria.

The examination items, and brief definitions are listed below. Information on how the signs were tested, and the criteria for determining whether they were hard or soft signs, as used in the checklist, are detailed in Appendix A.

Definitions of signs.

Anaesthesia (or Anesthesia): "absence of normal sensation" (Mosby, 1986, p. 60).

Ankle clonus: "Repetitive extention-flexion movement of muscles of ankle, associated with increased muscle tonus...Reflex elicited by quick, vigorous dorsiflexion of the foot while the knee is held in a flexed position, resulting in repeated clonic movement of the foot". (Taber, 1981, p. 88).

Anosmia: "loss or impairment of the sense of smell" (Mosby, 1986, p. 66).

Asymmetry of skull or limb: an abnormal variation in size compared to normal, or (for limbs) compared to the opposite side.

Ataxia: "impaired ability to coordinate movement", seen as a "staggering gait and postural imbalance" (Mosby, 1986, p. 100).

Babinski reflex: "dorsiflexion of the big toe with extention and fanning of the other toes elicited by firmly stroking the lateral aspect of the foot. It is normal in newborn infants and abnormal in children and adults." (Mosby, 1986, p. 112).

Choreiform/athetoid movements: Choreiform movements are irregular, nonrhythmic, rapid movements of short duration which may appear to be coordinated, but are involuntary; athetoid movements are slow, writhing, involuntary movements (Dorland, 1985; Van Allen, 1969).

Diminished or hyperactive tendon reflexes: (are referred to in this thesis as "tendon reflexes" or "abnormal tendon reflexes"). This sign is a "deep reflex obtained by sharply tapping skin over tendon of a muscle" (Taber, 1981, p. 1434).

Diplopia: "double vision" (Mosby, 1986, p. 353).

Dysarthria: "difficult, poorly articulated speech" (Mosby, 1986, p. 371).

Dysdiadochokinesia: "difficulty in executing of rapid alternating movements" (Nichols, 1987, p. 181).

Dyspraxia of tongue movement: Dyspraxia is defined as "partial loss of ability to perform coordinated acts" (Dorland, 1985). In this study dyspraxia of the tongue was tested by having the subject move his/her tongue rapidly from side to side (Hern, 1984).

Graphaesthesia: This sign is determined by the inability to identify numbers traced on the palm of the hand (Hern, 1984).

Heel-knee sign: This sign is revealed by the inability to smoothly stroke the leg from knee to ankle with the opposite foot (Hern, 1984).

Incoordination: "Inability to produce harmonious, rhythmic, muscular action, but not due to weakness." (Taber, 1981, p. 715). It is defined for the purposes of this study as upper body incoordination, and is evaluated with the finger-nose and finger-toe tests (see Appendix A). Lower-body incoordination in this study is defined as ataxia.

Intention tremor: "Tremor when voluntary motion is attempted." (Taber, 1981, p. 1482).

Nystagmus: "involuntary, rhythmic movements of the eyes" (Mosby, 1986, p. 790).

Paresis: "partial paralysis" (Mosby, 1986, p. 842).

Position sense: the ability to perceive the position of body parts with the eyes closed (ie. kinesthetically), (Dorland, 1985).

Resting muscle tone: Muscle tone (tonus) is "the normal state of balanced tension" in the muscles (Mosby, 1986, p. 1132 ). Resting muscle tone is evaluated while the limbs are at rest (limp) to see if there is an increased or decreased amount of resistance to the passive rotation and movement of major joints (Hern, 1984).

Resting tremor: "Tremor present when the involved part is at rest but absent or diminished when active movements are attempted." (Taber, 1981, p. 1482).

Saccadic eye movements: "rapid, intermittent movements...of the eye" (Taber, 1981, p. 1265).

Simultagnosia: This is the loss of the ability to identify stimulation presented simultaneously at two locations (in this case, to opposite sides of the body). A positive sign is indicated when the stimulation to one side is ignored.

Strabismus: "crossed eyes" (Mosby, 1986, p. 1074).

Synkinesia: An involuntary movement which occurs while a voluntary movement is being carried out; for example, when the subject is asked to tap a finger of the right hand, its counterpart on the the left taps too (Deuel & Robinson, 1987).

Visual field defect: Cortical blindness in one-half, or a circumscribed portion, of the visual field.

For the current study, the neurological reports and all relevant data in the subjects' files and in the data base were reviewed and cross-checked. This was done to obtain a better understanding of the data, to clarify interpretations, and to ensure accurate and uniform coding. The ages at Time 1 were adjusted to reflect the date of the neurological examination, rather than the age at the time of the neuropsychological assessment. No cases of anosmia were noted at Time 1, and this sign was found in only one subject at Time 2; therefore it was dropped from further analysis. The 25 signs provide a reasonably full profile, including the major features of a neurological exam.

In order to be able to test the theory of neurological system involvement, the signs were classified according to the neurological systems for which they are considered indicators of damage. This was done with reference to textbooks on neurological examination procedures (DeMyer, 1974; Van Allen, 1969), and in consultation with Dr. Otfried Spreen, director of the University of Victoria Longitudinal Study and supervisor

of this thesis. Six systems were defined: Cerebellar (CB); Cortico-spinal (CS); Basal Ganglia (BG); Posterior Column (PC); Cranial nerves (CN); and Miscellaneous (MISC). Table 9, on page 56 lists the neurological signs and the systems to which they were assigned.

It is the premise of this thesis that hard and soft signs are neurological signs on a continuum. Therefore, unless otherwise specified, the data below refer to the combined data of hard and soft signs, thereby evaluating signs in terms of presence or absence.

## Results

Where significance levels have been calculated, an alpha probability level of  $p < .05$  has been considered statistically significant.

### Question 1

What happens to the individual signs between Time 1 and Time 2? Which signs are most likely to disappear? Which signs are most likely to appear at Time 2?

#### Overview.

Table 1 gives an overview of what has occurred over time. Examination of Columns 2 and 3 reveals that of the 25 signs examined, 16 increased in absolute frequency, 4 kept the same number, and 5 decreased. The amount of increase varied greatly. The 2 signs for which the overall frequency increased the most were Synkinesia, with a frequency of 1 at Time 1 and 40 at Time 2, and Intention Tremor, which increased from 2 to 36 occurrences. Resting Tremor, Position Sense, and Saccadic Eye Movement were not found at Time 1, and their Time 2 occurrences were infrequent. The other 11 signs which increased in frequency increased by as much as three times (eg. Asymmetry of Skull and/or Limb from 9 to 31 occurrences), or by as "little" as 1 occurrence (Diplopia). Of the 5 signs which decreased in frequency, only Choreiform-

Athetoid Movements decreased by more than 3 occurrences, from 23 to 13.

The most frequently occurring signs at Time 1, involving approximately one-quarter or more of the subjects, were: Incoordination, Ataxia, and Tendon Reflexes. The most frequently occurring signs at Time 2 were: Tendon Reflexes, Synkinesia, Intention Tremor, Asymmetry of Skull or Limb, Incoordination, Ataxia, and Resting Muscle Tone.

Table 1

Frequencies of Occurrence and Change Over Time (N=110)

Sign	Frequency		Lost	Retain	Gain
	Time 1	Time 2	Sign	Sign	Sign
Incoordination	31	28	17	14	14
Ataxia	29	28	12	17	11
Tendon reflexes	26	45	11	15	30
Choreiform-Athetoid	23	13	17	6	7
Dysarthria	18	22	4	14	8
Graphesthesia	17	22	14	3	19
Resting Muscle Tone	11	27	1	10	17
Paresis	10	13	3	7	6
Asymmetry (skull/limb)	9	31	2	7	24
Babinski reflex	9	17	4	5	12
Heel-knee	7	25	1	6	19
Dysdiadochokinesia	7	22	5	2	20
Ankle Clonus	7	15	7	0	15
Dyspraxia of Tongue	6	5	3	3	2
Strabismus	5	5	2	3	2
Anaesthesia	4	3	3	1	2
Simultagnosia	3	3	3	0	3
Intention Tremor	2	36	0	2	34
Nystagmus	2	2	1	1	1
Visual Field Defect	2	2	2	0	2
Synkinesia	1	40	1	0	40
Diplopia	1	2	1	0	2
Resting Tremor	0	4			4
Position Sense	0	3			3
Saccadic Eye Mvmt	0	1			1

Column 4 lists the number of subjects who had the sign at Time 1, but lost it. Column 5 lists the number

Column 4 lists the number of subjects who had the sign at Time 1, but lost it. Column 5 lists the number who had the sign at Time 1 and retained it. Column 6 lists the number of subjects gaining the sign at Time 2. An examination of these three columns reveals a great deal of change over time.

To confirm the amount of change over time, and to determine to what extent signs could be predictors of their own presence over time, correlations of signs with themselves at the 2 times were calculated (eg. the presence of Ataxia at Time 1 was correlated with its presence at Time 2). While many correlations were statistically significant, only 5 signs had correlations accounting for more than 25% of the variance from Time 1 to Time 2. These signs, followed by the percent of variance accounted for, were: Dysarthria, 41%, Strabismus, 34%, Paresis, 32%, Dyspraxia of Tongue, 27%, and Resting Muscle Tone, 26%.

#### Persistence of signs and the appearance of new signs.

A more fine-tuned perspective on the changes taking place can be seen by evaluating the signs in terms of those which tend to persist, and those which tend to appear for the first time at Time 2. Table 2 lists the signs in increasing order of persistence over time. The frequencies at Time 1 and Time 2 are repeated in this

table, in order to maintain perspective. Column 2 represents the percentage of occurrences which persisted over time, and was calculated as follows: number retaining the sign / frequency at Time 1, multiplied by 100. (Percentage figures are rounded to the nearest whole percent.)

Column 3 lists the percentage of subjects in which the sign was found for the first time at Time 2. It was calculated by comparing the number gaining it to the number who did not have it at Time 1: number gaining the sign at Time 2 / (110 - frequency at Time 1) x 100 (and rounded).

Table 2

Persistence of Signs and Signs Gained Over Time (N=110)

Sign	% Retain Sign (Persist.)	% Gain Sign	<u>Frequency</u>	
			Time 1	Time 2
Visual Field Defect	0%	2%	2	2
Diplopia	0%	2%	1	2
Ankle Clonus	0%	15%	7	15
Synkinesia	0%	37%	1	40
Simultagnosia	0%	3%	3	3
Graphesthesia	18%	22%	17	22
Anaesthesia	25%	2%	4	3
Choreiform-Athetoid	26%	8%	23	13
Dysdiadochokinesia	29%	19%	7	22
Incoordination	45%	18%	31	28
Nystagmus	50%	1%	2	2
Dyspraxia of Tongue	50%	2%	6	5
.....				
Babinski reflex	56%	12%	9	17
Tendon reflexes	58%	36%	26	45
Ataxia	59%	14%	29	28
Strabismus	60%	2%	5	5
Paresis	70%	6%	10	13
Asymmetry (skull/limb)	78%	24%	9	31
Dysarthria	78%	9%	18	22
Heel-knee	86%	18%	7	25
Resting Muscle Tone	91%	17%	11	27
Intention Tremor	100%	31%	2	36
Saccadic Eye Mvmt		1%	0	1
Resting Tremor		4%	0	4
Position Sense		3%	0	3

Note. All percentage figures are rounded.

Soft signs and maturational lag.

The soft signs representing maturational lag in the literature were selected to test the findings of some studies which indicated that these signs would disappear over time. The question was:

What happens to the soft signs of Time 1? Which "go hard" (ie. become more severe, to the extent that they meet the criteria for hard signs)? Which "go normal" (ie. improve to the extent that there is no evidence of abnormality)?

As almost all detailed research on soft signs is done with groups screened for definite neurological abnormality, the following data is presented for the 35 subjects in the MBD group who showed soft signs at Time 1. This group represents an approximate match to the comparison groups in other studies.

The top half of Table 3 lists the signs considered to be developmental soft signs, and the bottom half of the table lists the signs considered to be classic or minor soft signs. The signs at Time 2 are categorized under: the percentage of signs which stayed the same (soft), the percentage of those which deteriorated to become hard signs, and those which improved to become classified as normal. The last column shows the number of subjects in which the sign appears new in Time 2, to provide

perspective. Only 15 of the 25 signs are represented; the other 10 signs were not found in this group at Time 1.

Table 3

The Course of Soft Signs in the MBD Group With Signs

(n=35)

Sign	Time 1	Time 2			
	Frequency	Same	Hard	Normal	Appear
<u>Developmental signs:</u>					
Choreiform-Ath.	12	0%	0%	100%	0
Dysdiadochokinesis	3	0%	0%	100%	8
Synkinesia	1	0%	0%	100%	10
Dyspraxia of Tongue	1	0%	0%	100%	1
Graphaesthesia	9	22%	0%	78%	3
Mild Incoord.	9	33%	0%	67%	5
Mild Ataxia	8	63%	0%	38%	2
Dysarthria	7	43%	29%	29%	4
.....					
<u>Classic soft signs:</u>					
Ankle Clonus	4	0%	0%	100%	2
Paresis	1	0%	0%	100%	2
Anaesthesia	1	0%	0%	100%	1
Tendon Reflexes	8	50%	0%	50%	11
Asymmetry	3	67%	0%	33%	13
Strabismus	3	33%	33%	33%	0
Heel-Knee	1	100%	0%	0%	6

Note. Percentage figures are rounded.

Question 2

Subjects: What happens to the subjects between Time 1 and Time 2 in terms of number of signs observed, and the number of signs which are retained vs the number lost or gained?

Overview and results for subjects as grouped by neurological diagnosis.

The number of signs for each subject at Time 1 was correlated with the number of signs at Time 2, to determine to what extent this Time 1 frequency was predictive of the Time 2 frequency. The correlation was  $r=.74$ ,  $p < .001$ .

There was great variability between subjects in the numbers of signs at Time 1 and Time 2, and in the changes taking place between the two times, as measured by the numbers of signs lost, retained, and gained. Table 4 lists group statistics for these numbers, using separate sections for each category. In sections A) to D), frequency at Time 1, and the numbers of signs retained, lost, and gained are listed for the three groups who had signs at Time 1. The frequency at Time 2 (section E) is given for all six groups.

Table 4

Group Data on Subject Change Between Time 1 and Time 2A) Frequency Time 1

<u>Group</u>	<u>Mean</u>	<u>Std. Dev.</u>	<u>Range</u>	<u>n</u>
1A) BD-hard signs	5.2	3.3	1-13	28
1B) BD-soft signs	1.6	1.1	1-4	8
2A) MBD-soft signs	2.0	1.4	1-6	35

B) Signs Retained

<u>Group</u>	<u>Mean</u>	<u>Std. Dev.</u>	<u>Range</u>	<u>n</u>
1A) BD-hard signs	3.1	3.3	0-11	28
1B) BD-soft signs	0.5	0.8	0-2	8
2A) MBD-soft signs	0.7	1.1	0-5	35

C) Signs Lost

<u>Group</u>	<u>Mean</u>	<u>Std. Dev.</u>	<u>Range</u>	<u>n</u>
1A) BD-hard signs	2.1	1.8	0-8	28
1B) BD-soft signs	1.1	0.8	0-2	8
2A) MBD-soft signs	1.3	0.8	0-3	35

D) Signs Gained

<u>Group</u>	<u>Mean</u>	<u>Std. Dev.</u>	<u>Range</u>	<u>n</u>
1A) BD-hard signs	3.9	2.8	0-12	28
1B) BD-soft signs	3.1	2.9	0-9	8
2A) MBD-soft signs	2.7	2.6	0-9	35

.....  
E) Frequency Time 2

<u>Group</u>	<u>Mean</u>	<u>Std. Dev.</u>	<u>Range</u>	<u>n</u>
1A) BD-hard signs	7.1	5.3	0-17	28
1B) BD-soft signs	3.6	3.5	0-10	8
1C) BD-no signs	3.5	0.7	3-4	2
2A) MBD-soft signs	3.4	3.3	0-12	35
2B) MBD-no signs	1.5	1.2	0-4	15
3) LD-no signs	1.7	2.0	0-6	22

.....

The frequencies of signs at Time 1 for groups 1A and 2A were compared, and were significantly different (Mann-Whitney U Test,  $p < .001$ ). A comparison of the frequencies of groups 1B and 2A was not statistically significant (Mann-Whitney U Test). This indicates that those with only soft signs had similar frequencies at Time 1, whether or not definite brain damage was diagnosed.

Frequency at Time 1 can be broken down into components, as seen in the next two sections of the table: Signs Retained plus Signs Lost. The sections representing Signs Retained and Signs Gained, when combined, equal the frequency at Time 2 (there is rounding error).

At Time 2, Group 1A had significantly more signs than group 2A (Mann-Whitney U Test,  $p < .01$ ). Other comparisons (Mann-Whitney) revealed that there were again no

significant differences between the two soft signs groups--one diagnosed with definite brain damage (Group 1B), and the other as MBD (2A). The MBD-soft signs group and the MBD-no signs group appeared to be different, but the analysis was not significant ( $p < .07$ , Mann-Whitney U Test). The MBD-no signs group and the LD group were also not significantly different (Mann-Whitney U Test).

Grouping subjects based on changes over time.

Another way to look at what happens to the subjects is in terms of changes in signs over time. Seventy-one of the 110 subjects showed signs at Time 1, while 39 showed no signs. When re-examined at Time 2, of the 71 with signs, 27% showed a decrease in signs, 15% had the same number of signs, and 58% showed an increase. Some of these subjects lost all signs, some lost a few signs, others lost some and gained some, and yet others had a complete change of signs. Of the 39 subjects with no signs at Time 1, 74% showed signs at Time 2.

Can subjects be grouped based on such changes?

Inspection of the data revealed that the subjects fell quite naturally into six Result Groups. The first two groups consisted of those who showed no signs at Time 1, and the last four groups all had signs at Time 1. The Result Groups (RGs) are as follows:

RG1: No signs either time (-,-); n=10.

RG2: No signs at Time 1, but signs at Time 2, (-,+); n=29.

RG3: Signs at Time 1, but no signs at Time 2, (+,-); n=9.

RG4: Signs at Time 1; retained all signs at Time 2, and gained some, (+,+); n=10.

RG5: Signs at Time 1; retained some signs, lost some, gained some (1 exception did not gain), (+,+); n=25.

RG6: Signs at Time 1; lost all signs and had an entirely new set of signs at Time 2, (+,-\*); n=27.

Table 5 shows what happened to the original groups over time.

Table 5

Crosstabulation of Original Groups by Result Groups

Groups	RG1	RG2	RG3	RG4	RG5	RG6	N
	(-,-)	(-,+)	(+,-)	(+,+)	(+,+-)	(+,-*)	
1A) BD-hard signs			1	4	15	8	/28
1B) BD-soft signs			1	2	1	4	/ 8
1C) BD-no signs		2					/ 2
2A) MBD-signs			7	4	9	15	/35
2B) MBD-no signs	3	12					/15
3) LD-no signs	7	15					/22
<b>Total</b>	<b>10</b>	<b>29</b>	<b>9</b>	<b>10</b>	<b>25</b>	<b>27</b>	<b>/110</b>

The importance of a single sign.

A final frequency question relates to the importance of a single sign. The data in this study provided an opportunity to trace separately those who had only one sign at Time 1. The group diagnosed as MBD provided enough subjects (n=16) on which to meaningfully report this as an additional analysis. Table 6 shows the results for these subjects.

Table 6

Results for MBD Subjects With One Soft Sign at Time 1

(n=16)

Sign	<u>Time 1</u> Frequency	<u>Time 2</u>		
		No Sign	Kept Sign, Gained 1	Lost Sign, Gained More
Graphesthesia	7	4	1	2
Choreiform-Ath.	5	1		4
Tendon Reflex	2	1		1
Anaesthesia	1			1
Dysarthria	1		1	

Question 3

The results of several studies indicate that signs considered to be developmental are found more frequently

in younger children than in older children. These results are used to support the concept of maturational lag. Other studies have found no difference. There is some evidence to suggest that age 10 is the normal upper limit of neurological maturation for most signs. Age 10 will be used to separate the subjects into two groups to answer the following question: Is there an age difference affecting the number of signs found, or which signs are most frequently found?

The total population was divided into two groups, those below age 10, which included 64 subjects, and those age 10 and above, which included 46 subjects.

A comparison was made of the number of signs found in the two age groups. The results are in Table 7 below:

Table 7

Frequency of Signs at Time 1 by Age Group

Number of Signs	Age < 10	Age > or=10
	% (n=64)	% (n=46)
0	36%	35%
1	16%	28%
2	19%	17%
3-6	17%	15%
7-13	13%	4%

Note. Percentage figures are rounded.

A Chi-square comparison between groups was not significant.

To investigate which signs are more frequently found in each age group, Table 8 lists the signs, with the percentage of the group showing the sign.

Table 8

Signs Most Frequent at Time 1 by Age Group

<u>Sign</u>	<u>Age &lt; 10</u>	<u>Age &gt; or = 10</u>
Incoordination	33%	22%
Ataxia	30%	22%
Tendon Reflexes	27%	20%
Choreiform-Athetoid	20%	22%
Dysarthria	17%	15%
Graphesthesia	13% *	20%

(\* tied with Resting Muscle Tone)

Chi-square comparisons between groups for each of the signs showed no significant differences.

Question 4

All signs have been classified into one or more of six systems groups: Cerebellar (CB); Cortico-spinal (CS); Basal Ganglia (BG); Posterior Column (PC); Cranial nerves (CN); and Miscellaneous (MISC). These are outlined in Table 9.

Table 9

Neurological Signs Assigned to Neurological Systems

Sign	Neurological System					
	CB	CS	BG	PC	CN	MISC
Babinski reflex		CS				
Tendon reflexes		CS				
Ankle clonus		CS				
Resting muscle tone	CB	CS				
Ataxia	CB	CS		PC		
Incoordination	CB			PC		
Heel-Knee	CB					
Dysdiadochokinesis	CB					
Intention Tremor	CB					
Position Sense	CB			PC		
Graphesthesia				PC	CN	
Simultagnosia				PC	CN	
Anaesthesia				PC		
Dysarthria	CB		BG			
Nystagmus	CB				CN	
Diplopia		CS			CN	
Strabismus					CN	
Saccadic Eye Mvmt					CN	
Dyspraxia of Tongue					CN	
Choreiform-Athetoid			BG			
Resting Tremor			BG			
Visual Field Defect					CN	MISC
Paresis		CS				
Asymm. (Skull/Limb)						MISC
Synkinesia		CS				

Subjects were assigned a score on each of the systems, based on the signs present in that system at Time 1. Signs gained at Time 2 were compared with the Time 1 score. The prediction was that the majority of signs gained would be gained within existing systems. A complicating factor was that some signs are possible indicators of damage in more than one system (eg. Resting Muscle Tone may indicate cerebellar or cortico-spinal damage). Because of these overlaps, more systems might be implicated than were actually involved. Asymmetry of skull or limb was left out of the comparison, and Visual Field Defect was coded only as a cranial nerve problem, since the Miscellaneous category does not represent a specific neurological system. (It is recognized that a visual field defect may result from damage at the cortical level, but this classification system does not code cortical damage, other than the cortico-spinal system.)

From the total of 110 subjects, 57 met the criteria of having had signs at Time 1, and having gained new signs (excluding asymmetry) between Time 1 and Time 2.

For each subject, the number of signs gained in previously affected systems was compared to those gained in systems not previously implicated. Results are listed in Table 10.

Table 10

Pattern Search in Neurological Systems

<u>Result</u>	<u>No. of Subjects</u>
Greater number to new systems	14
Greater number to previously affected systems	10
Equal number to both	6
Unable to determine	27
All 5 systems implicated Time 1	6
4 systems implicated Time 1	19
Unclear	2
<hr/> Total	<hr/> 57

The theory of neurological signs, as assigned to systems, was not supported. In order to test the validity of the grouping by neurological systems in an empirical situation, separate factor analyses were conducted (post hoc) for the Time 1 and Time 2 signs. This was done to see if the signs would group as predicted. The Time 1 factors did not resemble the neurological groupings. The Time 2 factor analysis, however, showed several factors which grouped signs approximately as predicted. The Time 2 factor loadings are listed below in Table 11.

Signs which grouped together under Factor 1 appeared to be mostly those representing cerebellar signs. The group of signs which clustered under Factor 2 appeared to be mostly cortico-spinal signs. The highest loadings on Factor 3 were cranial nerve signs, but not all cranial nerves grouped here. The signs grouping on Factor 4 were overlapping signs, having the posterior column system in common. Factors 5 and 6 were a mix of signs, while Factors 7 and 8 appeared to relate particularly to one

sign each. The loadings which are underlined represent those which would be predicted, given the way the majority of signs grouped. For example, since so many of the cerebellar signs load highest on Factor 1, the loadings of all signs designated cerebellar (see Table 9) are underlined in that column, for ease of comparison. Loadings at or above .30 are reported (accounting for 9% or more of the variance). However, an exception is made for those which had been predicted, but did not reach .30; these are listed in parentheses. Loadings for cranial nerves and basal ganglia signs did not group as expected, so are not underlined. Similarly, other loadings under factors which were not predicted are not underlined. Loadings are rounded to the second decimal place. The loading squared represents the variance accounted for by that factor.

Table 11

Time 2 Factor Analysis: Factor Loadings (rounded)

Sign	Factors							
	1	2	3	4	5	6	7	8
	CB	CS		PC				
Babinski		<u>.66</u>						
Tend Reflex		<u>.64</u>						
Ankle Clon		<u>.73</u>						
R Msc Tone	<u>.34</u>	<u>.73</u>						
Ataxia	<u>.54</u>	<u>.47</u>		(.25)				
Incoord	<u>.81</u>			(.08)				
Heel-Knee	<u>.78</u>	.35						
Dysdiado	<u>.61</u>	.37			.30			
Inten Trem	<u>.63</u>							
Posn Sense	(.22)			<u>.67</u>	.34			
Graphaesth				<u>.63</u>				
Simult				<u>.73</u>				
Anaesthes				<u>.47</u>		.66		
Dysarth	<u>.50</u>			.38				
Nystag	(.05)		.88					
Diplopia		(.03)					.81	
Strabis			.78				.43	
Sacc Eye Mvm								.81
Dyspr Tongue			.32		.39	-.58		
Chor-Ath	.64							
Rest Trem					.54			
Vis Fd Def	.31		.56			.61		
Paresis	.44	<u>.57</u>			.30			
Asymm		.40			.38			.33
Synkinesia		(.12)			.78			

## Discussion

### Representativeness of the Sample

Spren (1987) reported on a sample taken from the Victoria School Board Learning Assistance Center to test the representativeness of subjects in the Victoria Longitudinal Study. The findings indicated that the subjects sampled in the University of Victoria Study were reasonably representative of the learning disabled children of the Greater Victoria area. Attrition did result in some loss of representativeness, however. An IQ comparison of the originally referred participants and non-participants revealed that the IQ of the participants was significantly lower than that of the non-participants.

The 110 subjects who are a part of the current study (Group A) were compared to the 35 subjects who participated in Time 1 and the adolescent phase, but who did not return for Time 2 (Group B). The comparison is outlined in Table 12.

Table 12

Comparison of Group in Current Study (A) With  
Non-Returning Group (B)

	Group A (n=110)	Group B (n=35)
% Males	75.5	71
Mean Age	9.6	9.9
Mean Full Scale IQ	93.5	104

It appears that the subjects who did not participate at Time 2 were similar in age and sex ratio to those who did participate. The non-participant group did have a higher mean IQ score, although the difference (10.5 points) is less than one standard deviation (15 points). A possible reason for the lower IQ of participants was discussed by Spreen (1987), and is applicable also to this study. He suggested that those who are able to overcome some of the handicaps of their learning disabilities, perhaps because of their higher IQs, are not eager to think of themselves as belonging to this group. This attrition does reduce the generalizability of the results, but cannot be avoided.

Reliability of measurement

Reliability is dealt with in the body of the discussion, at points where questions of reliability affecting the results arise.

### Question 1

The purpose of Question 1 was to examine what happened over time from the perspective of the individual signs, and to see which signs were more likely to appear or disappear. A sub-question deals with the issue of maturational lag.

#### Overall changes in frequencies.

As Table 1 on page 42 made clear, the majority of signs increased in frequency between Time 1 and Time 2, and some of these changes were substantial. The first question that is raised in such situations is that of the reliability of the measurement--could the signs have been missed at Time 1? As discussed in the Methods section, the Time 1 neurological examination was conducted as a regular examination, and the results were coded from the neurological report with input from both the project neurologist and the neurologist who had performed the majority of the examinations. The Time 2 neurological examination was conducted according to the checklist devised by Hern and the project neurologist. Hern suggests the possibility that not every item was checked routinely for every patient, although this is not likely to account for the magnitude of differences found, since these items are part of a normal neurological examination.

One sign, Synkinesia, was considered by the neurologist to be possible to overlook during a routine examination, so that may have accounted for its low frequency at Time 1. At Time 2 it was on the checklist, so it was specifically tested and observed. Hern also points out that there may have been a tendency for the neurologist using the checklist at Time 2 to find certain signs more frequently because of the prompting provided by the checklist. She also mentions the possibility that the neurologist may have used a higher baseline for these signs in children, considering a slightly inadequate performance to be within the normal range for a child, but considering it to be abnormal when found in an adult.

Another factor which might have led to the discrepancy between the number of signs at the two times is that neurologists are not eager to consider a sign abnormal unless the evidence is very strong (Goldstein & Tupper, 1987). This is a harder distinction to make with children, especially for the signs considered to be developmental. The examination at Time 2 was done for research purposes, with adults, and with a checklist so it was not only easier to determine abnormality, there may have been less reluctance to label a sign abnormal. The use of a checklist, giving what Goldstein and Tupper (1987) refer to as "anchor points", likely increased the

reliability at Time 2, as did having the ratings done "blind" as much as possible.

Finally, there could be inter-rater differences, since the project neurologist did not perform the majority of the examinations at Time 1.

Given the above-mentioned factors, it would seem that the greatest likelihood of error would be in missing signs at Time 1, rather than recording signs that were not there, or making the errors at Time 2. Even so, there were many signs found at Time 1. Also, many more signs were found at Time 2, whether they actually appeared over time, or were undetected, or undetectable, at Time 1.

The only sign which decreased appreciably was Choreiform-Athetoid Movements. This sign may have been an example of the developmental sign which is "outgrown", and will be discussed later in this regard; however, it should be noted that for 1 sign out of 25 to decrease to this degree is not out of the realm of chance occurrence.

Why did the majority of signs increase in overall frequency? The answer is not straightforward, and there are factors involving individual signs operating as well. For example, the project neurologist did note from his experience that the frequency of occurrence of Intention Tremor appears to increase with age. Unfortunately,

little is known about the course of signs over many years, so unknown factors may be at work here.

Comparing the frequency of occurrence of signs at Time 1 with their occurrence at Time 2 may be useful for looking at group results, but can be easily misinterpreted as soon as one tries to apply it to subjects. This will be clearly shown in the next section. However, in considering just this overall picture, a simple count, 230 total occurrences at Time 1, and 414 at Time 2, emphasises Hern's point in her dissertation (1984): overall, signs do not appear to decrease over time.

The most frequently occurring signs at Time 1, Incoordination, Ataxia, and abnormal Tendon Reflexes, are also among the most common signs at Time 2. These signs represent motor (as opposed to sensory) functions, and the first two signs may be indicators of damage in several areas. As such, these signs are more likely to appear with any damage.

Synkinesia and Intention Tremor, also found frequently at Time 2, were found by Hern (1984) to be common in adult controls, as were abnormal Tendon Reflexes. This does not mean that such signs are of no value when found in the learning disabled, nor that they indicate a normal neurological situation. However, it

does confound the question of the meaning of the presence of such signs.

Changes within signs over time.

The next step is to look more closely at the changes between times. The first thing that becomes apparent is the amount of change within signs. Many of the subjects who showed certain signs at Time 1 did not show those signs at Time 2; others appeared to develop the signs over time. For example, in Table 1, Incoordination appears in 31 subjects (28% of the total) at Time 1, and in 28 subjects (25%) at Time 2. Without a more detailed look, it would be easy to make the assumption that Incoordination is basically stable over time. However, what actually occurred, as seen in Columns 4-6, is that 17 subjects "lost" the sign, 14 retained it, and 14 different subjects gained it. This is not an uncommon example, as can be seen throughout Table 1.

This changeability is the reason why discussing overall gains can be deceptive. A group composed of subjects with a particular sign at Time 1 may have few members in common with the group having the sign at Time 2.

Stability and persistence.

This extreme changeability raises the question of the stability of signs over time. This can be approached from

more than one angle, depending upon the perspective from which the question is being asked. A method used by Hern (1984) and Hertzog (1982) accounts for both the presence and absence of signs, which means that the stability equation includes signs which are present at both times, as well as absent at both times. As an example, Graphesthesia is present in 17 subjects at Time 1, and 22 subjects at Time 2, with only 3 subjects retaining the sign; N=110. The Stability Rating would be  $3/110$  (2.7%) for those positive at both times, and  $74/110$  for those negative at both times (67.3%), for a stability total of 70%. The advantage of this method is that it gives a perspective for the whole population under study.

A different approach, used in this study, removes the absence-absence figures, and calculates stability based on the presence of the sign at one or both times. Using the same example as above, those with Graphesthesia at Time 1 who retained the sign, were  $3/17$ , or 17.6%. This is referred to here as persistence of the sign. Those who were not found to have Graphesthesia at Time 1, but who showed it at Time 2 were  $19/93$ , or 21.5%. The advantage of this method is that it specifically addresses the questions:

- 1) Of those subjects with the sign at Time 1, what percentage still had the sign at Time 2 (18%, rounded, for

Graphesthesia). Column 2 of Table 2 (page 45) answers this question for each sign.

2) Of those who did not have the sign at Time 1, what percentage had it at Time 2 (22%, rounded, for Graphesthesia). Column 3 of Table 2 gives these figures for each sign.

If one looks down Column 2 of Table 2, it can be seen that those signs in the top section of the table have a persistence in terms of presence-presence, of 50% or less. In other words, a subject in this study who had the sign at Time 1 showed a 50-50 chance or less of carrying it into adulthood. The bottom half of the chart shows those signs which were more stable over time. A subject with a sign in this section showed a better-than-even retention of the sign. It can be readily seen that few signs were persistent enough to encourage the idea of making predictions based on childhood signs.

Column 3 of Table 2 shows that those signs found most frequently for the first time in adulthood are a mix of signs considered classic neurological signs (hard and soft), and those considered to be developmental soft signs, both severe and relatively minor. This indicates that so-called "developmental" signs are not exclusively developmental--they may appear later on.

The question of which signs are most likely to appear or disappear is more complicated than it appears at first. By comparing Columns 2 and 3, it can be seen that the two aspects, persistence and gain, appear to operate independently. Signs which have a high rate of persistence, such as Asymmetry of skull or limb, may also have a high rate of being gained; or like Dysarthria, they may have a relatively low rate of gain. Similarly, low persistence does not predict a higher rate of gain.

Overall, from Table 2, it appears that signs such as Paresis, Asymmetry of Skull or Limb, Dysarthria, Heel-knee, Resting Muscle Tone, and Intention Tremor, when found in childhood, tended to persist. Paresis and Dysarthria did not tend to appear unexpectedly at Time 2, while the others had a relatively high rate of gain. The other signs were much less persistent over time, and signs such as Synkinesia, Graphesthesia, and abnormal Tendon Reflexes often appeared at Time 2.

Hertzig (1982) found a great variability in the stability of the non-focal (soft) signs over time. Coordination and graphesthesia were particularly unstable over time, as they were age-related in her study.

This current study, however, found a serious lack of persistence in 12 of the 22 signs which were found at Time 1, and a fairly high rate of gain (10% or more of the

subjects developing the sign) in 12 of the 25 signs found at Time 2.

Why is there so much change? This question will be addressed in the discussion of Question 2, as it relates particularly to subject factors and not just to signs alone.

Soft signs and maturational lag.

The signs which are thought to represent maturational or developmental lag are of particular interest, because they confirm or challenge the common expectation that such signs will often go away when the child matures--he or she is just thought to be slowly maturing. Also, the disappearance of a sign, while it does not necessarily mean that there was no underlying damage, or that the damage has been repaired/compensated for, tends to make the sign seem less serious. On the other hand, signs which persist, and the development of such signs between childhood and adolescence, tend to give the sign a more serious cast.

By comparing the top and bottom halves of Table 3 on page 47, it can be seen that both groups of signs have some which tend to disappear (ie. are rated as normal at Time 2), and others which do not. Generally, it appears from Column 2 that the developmental signs occur with a

higher frequency than the non-developmental (classic) soft signs.

Five signs had only one occurrence, so little can be said about them. Comments on the findings of the four signs with 3 or 4 occurrences are also questionable. The sign which does appear to give support to the theory of developmental lag signs which go away, is Choreiform-Athetoid Movements: all 12 cases became normal at Time 2, and no new cases appeared at Time 2. However, as will be seen later, these data must be tempered with the knowledge that the disappearance of one particular sign does not necessarily mean that the subject is now neurologically normal.

Dysdiadochokinesis and Graphesthesia are also often mentioned as developmental signs. The frequency of Dysdiadochokinesis in this group is very low, so any comments are tentative. However, for both of these signs, there appears to be some support for the theory that they might be developmental signs, but not exclusively so, as indicated by the rate of new appearances.

Mild Ataxia (comparable to incoordination of the lower limbs in other studies), and Dysarthria did not tend to disappear. Mild Incoordination (incoordination of the upper limbs) was slightly better in this regard. This does not mean that these three signs are not developmental

signs, but it gives support to those who suggest that "developmental" and "outgrow" are not necessarily synonymous. Also, Ataxia, Incoordination, and Dysarthria can be indicators of damage in several areas, and not just delay.

A problem in the evaluation of signs is that some of those which might be developmental signs can also be abnormal signs at an early age, as in the three signs discussed above. As Touwen (1987) says, it is hard to distinguish a developmental finger-nose test (Incoordination) or age-inadequate Dysdiadochokinesia from deviant performance on these signs. Therefore, one cannot tell if the presence of a sign is a manifestation of a delay, or is an abnormal sign. Some studies try to make this distinction by comparing the frequencies of occurrence in younger compared to older children, presuming that more signs in a younger group indicates developmental signs. This theory will be tested in Question 3.

When the non-developmental (classic) soft signs are considered, they do not appear to be substantially different from the developmental signs. However, the low occurrence rates make replication essential. Rutter and colleagues (1970) mentioned tendon reflexes (of which ankle clonus is one form) as affected by such things as

the state of relaxation of the subject and by room temperature. Such factors may have affected the disappearance and appearance of these signs, although there is no way to determine this.

This part of the study, then, provides some support for a theory of maturational lag in relation to Choreiform-Athetoid movements. Support for Dysdiadochokinesis, mild Incoordination, and Graphesthesia is weak. There is no support for mild Ataxia and Dysarthria as maturational signs which might be expected to go away. Maturational lag will be explored further, from the perspective of the subjects, in Questions 2 and 3.

### Question 2

This questions looks at changes from the perspective of the subjects--focussing on the number of signs observed, and the number of signs retained, lost and gained.

#### Overview and results for subjects as grouped by neurological diagnosis.

The .74 correlation of the frequencies at the two times indicates that a higher frequency of signs at Time 1 is associated with a higher frequency at Time 2. This correlation accounts for 55% of the variance. While such

a statistic is useful overall, it does not indicate the extent of the changes which have taken place.

It is difficult to convey the amount of change seen in the individual subjects in a concise manner, and still more difficult to make it comprehensible. Unless they are carefully and thoroughly outlined, group totals and means may inadvertently conceal more information than they reveal. In Table 4 on pages 49 and 50, it can be readily seen that the standard deviations sometimes exceed the means. It is clear from these data and the ranges that there is much overlap between groups, but also that on average, the group diagnosed as brain-damaged with at least one hard sign, has more signs than those groups with soft signs. This is not unexpected, and was outlined by Hern (1984).

The comparisons of group data in Table 4 indicate that the presence or absence of these signs and the severity of signs (hard or soft) are better indicators of the relative frequency of signs at Time 2 than are the diagnoses of brain damage or MBD without accompanying neurological signs. The absence of signs at Time 1 only indicated fewer signs at Time 2, it did not predict an absence of the signs. The similarity of results for the LD group compared to the MBD-no signs group (ie. both tended to gain signs) gives some support for the concept

of a neurological substrate for at least a substantial proportion of the LD subjects in this study.

Grouping subjects based on changes over time.

The outcome (Result) groups provide a different perspective on the changes. No subject had the same combination of signs at both times, and 27 of the 71 (38%) with signs at Time 1 had a completely different set of signs at Time 2. This is a level of change which to my knowledge has not been reported before in the literature. This analysis does not take into account changes in severity, which would have added yet another dimension of change.

From Table 5 on page 52, one might assume that subjects in Result Group 2 (those without signs at Time 1 who gained signs at Time 2) probably gained one sign, or two at most. One might also assume that those who lost all signs (Result Group 3), and those who had a complete change of signs (Result Group 6), probably had few signs to begin with. It is useful then, to look at these groups more closely.

Result Group 2 consisted primarily of 80% of the MBD group and 68% of the LD group who had no signs at Time 1, but showed them at Time 2. (Two of the BD group who had no signs at Time 1 gained signs at Time 2, but there are too few subjects to make comparisons.) An interesting

comparison can be made between the MBD and LD groups. The number of subjects gaining signs, compared to those not gaining, showed no significant difference between the two groups (Chi-square analysis). A comparison of the number of signs gained, as seen in Table 13 below, indicates that the results were slightly more severe for the LD group, although the numbers are small for a statistical comparison.

Table 13

Signs Gained in Two Groups Without Previous Signs

Signs Gained at Time 2	MBD Group	LD Group
1	6	7
2	2	2
3-4	4	3
5-6	0	3
	12	15

Approximately one-half of each group gained more than one sign at Time 2, and many of them gained 3-6 signs. This may represent a decline in CNS integrity in both of these groups who were previously without signs. It is possible that these subjects had some damage at Time 1, but for the LD it was not serious enough to show up on a neurological examination, and for the MBD-no signs group,

it was not manifested in the neurological signs at that time.

In Result Group 6 (those who had a complete change of signs) the individuals varied greatly. Their outcomes are summarized in Table 14 below. Results for the BD-hard signs and BD-soft signs groups are combined under the BD category.

Table 14

Comparison of the Number of Neurological Signs Lost and Gained in Two Groups

Signs Lost or Gained	BD (n=12)	MBD (n=15)
Lost 1-2, gained 1-2*	7	6
Lost 1-2, gained 3-4	3	6
Lost 1-2, gained 5-7	1	3
Lost 7, gained 3	1	

\* The mildest change, those who lost 1 sign and gained 1 different one, accounts for only 3 of the BD, and 1 of the MBD group.

It appears that the changes of signs are fairly extreme here. This lends weight to the concept of diffuse and perhaps mild damage, and also damage that does not usually improve.

### Importance of a single sign

How important is a single sign? Most of the subjects in Result Group 3 (with signs at Time 1, but none at Time 2) had just one sign at Time 1. In this case, the expectation is confirmed that those who "recover" at least in terms of signs, are usually not as seriously affected originally. However, this raises the question about the reverse situation. If those who recover tend to have only one sign, does that mean that those with only one sign tend to recover? The question of the importance of a single sign in subjects in this study is illustrated in Table 6 on page 53.

Overall, of the 16 subjects with one soft sign, 6 became "normal", 2 kept the sign and added another, and 8 lost the sign but gained 1-7 different signs, including hard signs.

If one were only looking to see if the sign had disappeared, it did, in all but two cases. However, 8 of those 16 had different, and often more serious signs at Time 2. All of the signs in Table 6 are considered to be developmental signs, except for Tendon Reflexes. Choreiform-Athetoid movements (primarily choreiform movements in this study), were seen in Question 1 to disappear over time. However, here it can be seen that of the five subjects with this sign as their only sign, only

one became sign-free at Time 2. Of the seven subjects with Graphesthesia as their only sign, four became sign-free, while the others gained different signs at Time 2. This indicates that signs of developmental delay, or maturational lag, are not benign, but may indicate a compromised nervous system. Because the signs go away does not mean there is no damage; it may only indicate that the damage does not show itself consistently in the same way over time.

While it is true that controls may have soft signs, and that one soft sign does not necessarily persist, it is also true in this study that one sign did not usually predict a positive neurological outcome.

It is possible that one sign in a control person has a different significance than one sign in a learning disabled person. The disability may act as another factor predicting a less-than-ideal outcome. Wolff and Hurwitz (1973) suggested that there may be some functional significance in a single sign (eg. choreiform movement) if it is correlated with behavioral disturbance. (They refer to Prechtl's syndrome which involves choreiform movements and three behavioral indices: impulsivity, emotional lability, and lack of persistence.) Therefore, as Wolff and Hurwitz see significance in a single sign combined with a behavioral disturbance, so too, one sign combined

with a learning disability may be a stronger indicator of damage than either alone.

Overall, then, even though many signs go away, as seen in Question 1 and in the literature, other signs often appear. Helper (1980) in his review of studies, also found that while some learning disabled subjects may "grow out of" symptoms or deficits, others seem to "grow into" some deficits.

Teuber and Rudel (1962) suggest that the changeability of the system is dependent on what behaviors or signs are being tested. They found evidence that some behaviors are impaired by brain injury at any age, other behaviors show the effects of early damage but the behavioral manifestation disappears with development, and still other behaviors show effects of earlier damage but only after a delay (ie. with maturation).

One can speculate as to what factors might account for losses of signs, such as spontaneous recovery, or compensation by one part of the brain taking over a function for a damaged area. The gaining of signs might be due to the measuring instrument, the persons measuring, or it might be a result of the effects of years of demand on an already compromised system. However, whatever the speculations are, the results of this study show that the hallmark of signs over time appears to be change. Many

signs are readily gained and lost, and lost signs do not appear to indicate that the subject has become "normal". In most cases it indicates only that he/she has picked up a new sign or set of signs--and often has gained more signs than were lost.

### Question 3

This question looks at maturational lag from the perspective of the age of the subjects, based on the concept that younger children would be more likely to show signs identified as "developmental" than older children. One-half of the studies reviewed found evidence of an age-difference in signs found, while the other half found no evidence to support this idea.

A comparison of the number of signs at Time 1 by age group (below age 10 vs age 10 and above) showed that the groups were not significantly different (Chi-square analysis).

It could be argued, however, that since the diagnosed brain damaged subjects are included with those with only mild signs or suspected MBD, the results might be confounded by this combination of subjects. Accordingly, another comparison was done, using only the MBD-soft signs group, on the frequencies of developmental signs (from Table 2, page 45) by age group. Only those signs with

more than one occurrence were included. Results are shown in Table 15 below.

Table 15

Frequency of Soft Signs at Time 1 in the MBD Group, by Age

Sign	Age < 10 (n=16)	Age >or=10 (n=19)
Choreiform-Athetoid	4	8
Dysdiadochokinesis	3	0
Graphesthesia	4	5
Mild Incoordination	5	4
Mild Ataxia	4	4
Dysarthria	4	3

It is clear from the table above that only the first two signs show what may be differences between the groups. This apparent difference between groups on the Choreiform-Athetoid sign was not significant (Chi-square analysis). The direction of the difference argues against this sign as developmental. Dysdiadochokinesis appeared only in the younger group; however, the low number of occurrences makes support for it as a developmental sign rather weak. Statistically, the difference between groups on this sign was not significant (Fisher's exact test,  $p = .086$ ).

Helper (1980) in his examination of 33 follow-up studies of children with MBD, found that older learning

disabled subjects showed a different constellation of deficits than younger ones. In this study, however, a comparison of signs in each age group revealed that the groups had remarkably similar rankings of sign frequencies. While the younger group did appear to show a higher proportion of the signs (Table 8, page 55), the differences were not significant (Chi-square).

Overall, there was no significant difference between signs found in younger compared to older subjects, either in the entire group, or in the MBD-soft signs subgroup.

#### Consolidation of the Findings Related to maturational Lag

At this point the findings from Questions 1, 2, and 3 can be combined, and some conclusions drawn. When the signs were analyzed separately, it appeared that there was good support for Choreiform-Athetoid Movement as a developmental sign, with weak support for Dysdiadochokinesis, Graphesthesia, and Mild Incoordination. There was no evidence to support Mild Ataxia (lower body incoordination) or Dysarthria as developmental signs.

However, when the signs were viewed in context (ie. subjects with single signs were followed over time), it was discovered that while subjects with the Choreiform-Athetoid sign often lost that sign, in almost all cases

they gained other signs. In subjects with Graphesthesia, four out of seven became sign-free at Time 2, but the other three showed signs.

A comparison by age group, to see whether the so-called maturational or developmental signs appeared more frequently in younger subjects (those under age 10), found no significant differences between the two age groups.

The concept of signs representing maturational lag which are seen as benign, and eventually are expected to be outgrown, is not supported in this study. The signs identified as maturational may have disappeared over time; however, the subject most often gained other signs. If there is such a thing as a lag, it is not benign, and the lag may itself be caused by damage. This corresponds to the position of Denkla (1978).

Two considerations as to the validity of the concept of maturational lag are: first, to prove that a lag takes place neurologically, and second, that this lag is responsible for the soft signs in question. Some EEG studies are making progress in measuring maturation in different brain functions (Chiarenza, Papakostopoulos, Giordana, & Guareschi-Cazzullo, 1983; Corning, Steffy, Anderson, & Bowers, 1986), but the research results are not yet at the stage of clinical application. The connection to soft signs therefore also awaits. A third

consideration for the learning disabled is to prove that the lag/soft signs affect learning, and this has not been done.

Contrary to how it is often presented, the question of damage or delay is not necessarily an either-or proposition. Dalby (1979), in his review of issues facing researchers studying brain processes and dyslexia, likens it to the nature-nurture controversy, where it eventually was shown that both play important parts. So too, damage and delay may be shown to interact in many persons.

#### Question 4

The search for a pattern of change in neurological systems was not successful. It was also more complicated than anticipated. The double- and triple-assignment of signs to systems was necessary in order to take into account possible relationships, but unfortunately these were the signs very frequently found in subjects, and found in numerous combinations. This posed problems at both ends of the analysis. For example, a subject who had Incoordination at Time 1 was considered to have two systems affected--cerebellar and posterior column systems. If he then had Graphesthesia at Time 2, the Graphesthesia was assigned to the cerebellar system (a change within the same system) and the cranial nerve system (an addition to

a new system). Such situations were common, and in fact since many subjects had several signs at each time, it often became even more complicated. The analysis therefore was done on a case-by-case basis, taking these factors into account.

As is evident in Table 10 (page 58), the greater number of signs gained went to new systems, rather than systems previously affected. There were 27 cases in which it was not possible to determine into which category the new signs should fall. When 4 or 5 (out of 5) systems are already affected, then the odds are "loaded". The evidence which implicated several systems in some of the subjects was based only on the fact that one of the signs overlapped several systems (ie. Ataxia alone implicated three systems). Therefore a selective approach, based on the weight of evidence was tried. In these cases, a system was not considered implicated if only one sign was attributed to it, and that sign was one which overlapped systems. To be considered, a system had to have one non-overlapping sign included, or two to three overlapping signs. For the 6 cases in which 5 systems had been implicated, these new criteria did not reduce the implicated systems by more than one system, which still put a heavy "load" in favour of these previously affected systems. For the 19 cases in which 4 systems had at first

been implicated, 9 cases were still obviously loaded in favour of previously affected systems, and 2 were undeterminable. Of the 8 others, 7 could be said to support the theory of signs added to previously implicated systems, while 1 favoured new systems. However, even adding these 8 subjects to the results would still not adequately support the theory, as the totals would be 17 in favour of the theory, and 15 against it.

One last approach was tried. If there is any validity or usefulness for the theory of signs being more often added to existing systems, it should be able to prove itself at the other end of the scale, that is, where 1 or 2 systems only are implicated, and the weight of chance is against it. Nine subjects had only one neurological system implicated, and of these, 1 favoured previously implicated systems, 6 favoured new systems, and the other 2 were equal. Another 9 subjects had two systems implicated, and of these, 2 favoured the existing, and 7 favoured the new. Clearly this theory was not supported, and a prediction based on the possible neurological systems involved at Time 1 does not work.

Even though no pattern of change was found, this does not necessarily negate the grouping of signs by neurological systems. While the factor analysis performed at Time 1 indicated that the signs showed little relation

to the predicted groupings, at Time 2 there was some support for those groupings. At Time 2, the signs appeared to cluster approximately along the lines of the cerebellar, corticospinal, and the posterior column systems as predicted (as seen in Table 11, page 60). Also, the distribution of factor loadings for Resting Muscle Tone and Ataxia gave support to the overlapping categories for these signs. The basal ganglia signs did not group separately. Dysarthria (an overlapping sign) appeared with the cerebellar signs, which would indicate that its appearance in this study was related more to cerebellar than to basal ganglia signs. (Basal ganglia damage appears to be less common in this study.) However, the appearance of Chorieform-Athetoid Movements in this group is not understood. The cranial nerves appeared to separate into distinctive factors, loading on Factors 3, 7, and 8, indicating their separability in terms of damage. Though not predicted, it does make sense in terms of different positions of the nerves along the brainstem. Nystagmus and Strabismus had high loadings on Factor 3; they can both be caused by damage to cranial nerves 3, 4, and 6, and so their co-grouping is explainable.

Factors 5 and 6 are not clear factors. Synkinesia loads highly (.78) on Factor 5, and this is also the highest loading for Resting Tremor (.54). This may be

related to their higher occurrence in the group of normal controls studied by Hern (1984), but this is not certain and is difficult to interpret. Factor 6 is also a mix of signs and is difficult to interpret.

Overall, though, the high and expected loadings on what appear to be cerebellar, cortico-spinal, and posterior column signs, as well as the separation of cranial nerve signs, provide support for the grouping of signs by neurological systems.

It is possible that that early evidence of damage, as seen through neurological signs, is expressed in non-specific ways, but that as the system matures, the signs "settle" into more specific patterns. This can only be answered by further study.

Often underlying this search for subtypes or patterns of dysfunction is the premise that the damage or the dysfunction is relatively circumscribed and discreet. Evidence from the National Collaborative Perinatal Project indicates that there are multiple etiologies for neurological signs (Nichols, 1987). Much damage is hard to classify, and studies of pre- and peri-natal damage indicate that while some damage is focal, much is multi-focal or diffuse (Towbin, 1987). Therefore, it appears that in many cases, particularly those involving soft signs, localizing damage in childhood, for the purpose of

predicting neurological outcome may not be possible. What happens to this damage over the course of time remains to be discovered.

Hertzog and colleagues (1969) point out the neurological heterogeneity in their study, and marked variability in findings. This heterogeneity, they suggest, may be due to the different locus and extent of damage, the time of life when it is sustained, and opportunities for development with which this damaged area is associated.

No pattern of change could be ascertained from this study. Perhaps with the development of more sophisticated imaging techniques, it may eventually be possible to work back from identified areas of focal or diffuse lesions to neurological signs, particularly soft signs. In this way the relationship between damage and the signs can be determined, and this relationship can be monitored over time. However, this is not possible at present.

Another interpretation of the failure to find a pattern predicting change over time is that the signs may not represent damage at all, but may be normal variations in performance. However, the firm association between signs, particularly the presence of several signs, in groups with neuropsychological, academic, and emotional problems, makes the assumption of some damage or

dysfunction feasible. Perhaps the damage affects areas responsible for neurotransmitters; this would tend to show more diffuse effects. There is also the possibility that rather than structural damage, there may be a genetically determined biochemical abnormality.

The question of reliability of the findings cannot be dismissed. No reliability tests were conducted. It is possible that the reliability of the signs themselves, subject factors, inter-rater differences, and intra-rater factors could all have contributed. This is of more concern at Time 1 than at Time 2, since Time 2 was more structured and had one neurologist perform almost all of the examinations. Problems with reliability would explain the failure to find a pattern of change, and the failure to find a grouping at Time 1 according to neurological systems. However, given the qualifications and experience of the neurologists, and the vast amount of change over time, it seems more likely that the "signal" of great change comes through the "noise" of possible unreliability very strongly.

The most likely explanation for the findings is that the damage is diffuse or multi-focal, and often subtle. The damage is there, and does not seem to go away, but its effects show up in different tests at different times, depending upon the threshold of detection of the various

signs at the time they are tested. Whether this changeability continues into late adulthood and old age is not yet known. There is some evidence from this study that more signs appear with age, but the questions of reliability discussed above cloud this issue. Further study may indicate deterioration in the system over time, but this also remains to be studied.

Spren (1989) suggests that with these neurological signs perhaps we are seeing only the minor manifestations of more serious damage (ectopias, aplasias, etc.) which extends to higher cortical areas. The damage is revealed at the subcortical and cortico-spinal level in neurological signs, and at the higher cortical level (association and cognitive areas) in learning disabilities.

### Conclusions

1. The findings of Hern (1984) were confirmed in this smaller and more restricted group. The overall frequency of signs in this learning disabled group did not decrease over time. In fact, the frequency appeared to increase substantially.
2. A prominent characteristic of the course of signs over time was their extreme changeability. Twelve of the 22 signs occurring in Time 1 had a persistence ratio of 50-50

or less, and only 5 signs persisted in more than 75% of the cases over time.

3. While many signs did "go away", other signs often appeared. When the subjects were grouped in terms of changes over time, it was found that, with the exception of 10 subjects who had no signs at either time, no subject had the same profile of signs at both times. More specifically, 27/71 (38%) of the subjects who had signs at Time 1, had a complete change of signs at Time 2. Most of these subjects had changes involving several signs, rather than just one.

4. Of the 39 subjects with no signs at Time 1, 29 (74%) showed signs at Time 2. All but 2 of these were in the MBD and LD groups. Many of the MBD and LD subjects gained several signs, providing some support for the concept of neurological damage in these learning disabled subjects, even for those who in childhood showed no signs upon neurological examination.

5. The diagnosis of definite or minimal brain damage appeared to have less impact on the results than the type of signs found (hard, soft, or no signs). The BD-soft signs group was not significantly different than the MBD-soft signs group, in terms of the numbers of signs at Time 1 and Time 2. The MBD-no signs group was not

significantly different than the LD group, in the number of signs gained over time.

6. It is said in the literature that a single soft sign is not important. The results of this study show that while those who "lose" all signs over time usually had only one sign in the first place, the reverse is not necessarily true--a single sign did not usually precede a sign-free result. Of 16 subjects with only one sign at Time 1, 14 lost the sign. However, only 6 showed no signs at Time 2. Eight subjects lost their original sign, but gained 1-7 signs, including hard signs. Therefore, a cursory look at single signs might at first appear to indicate a favourable result; a closer look reveals that most subjects did not end up sign-free--they just changed signs.

7. Maturation lag: Signs which in the literature are purported to represent maturational lag were analyzed, using a group of MBD subjects. While some of these signs did disappear over time, other signs, often several signs, usually appeared. These signs also were among the signs gained at Time 2, so they are not exclusively developmental. If their presence at Time 1 did indicate delay, then it was not a benign delay, but one that appears to be related to underlying damage.

8. A pattern of signs, based on neurological system involvement, which might predict neurological outcome, was tested. It was not possible to predict outcome using this method. However, there was evidence of a grouping of signs according to neurological system involvement, at Time 2 only.

#### Further research

The testing of the predictive usefulness of a pattern of signs, based on neurological system involvement, was not successful. The most frequent signs at both times were those such as ataxia and incoordination which may be indicative of damage in several different systems. However, the factor analysis of the signs at Time 2 showed a grouping of signs comparable to the proposed neurological systems, while that at Time 1 did not. This may indicate that damage in childhood is expressed in non-specific ways, but that in adulthood, signs "settle" into a pattern. One way to test this is by a follow-up study of these subjects, to see if the signs and patterns hold over time. Valuable information which might answer several of the questions raised in this thesis could be obtained from a Time 3 study. Such a follow-up might also answer the controversial and important question of whether the subjects are mostly changing over time, or whether

they might be deteriorating, in a neurological sense. They could be compared in adulthood to the normal learners used as a control group at Time 2 by Hern.

Many studies in the literature follow learning disabled subjects, and other subjects with signs, only into adolescence. While it is difficult to find subjects once they leave the school system, it would be worth carrying some of these other studies into adulthood, to learn more about how neurological systems with some indications of damage change over time.

The finding that one sign alone may be an indicator of damage requires replication. This requires not only looking at the sign to see if it disappears (which it often did in this study), but testing other signs in the subject to see if he/she develops other signs over time.

There is a need for control subjects to be measured from childhood through to adulthood. We do not know much about what happens to a basically normal neurological system over time. There is also a great need for norms, at different age levels, for these signs.

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## Appendix A

The Testing of Neurological Signs, and Criteriafor Ranking

(from Hern, 1984)

Anaesthesia was tested by touching the cheeks, the backs of the hands, and the shins, while the subject had his/her eyes closed. The subject was asked if he/she felt a difference in the touch. A hard sign was diagnosed if the anaesthesia was very marked; moderate or slight anaesthesia was considered a soft sign.

Ankle clonus is a form of hyperactive reflex, but is separately noted for children. The neurologist was looking for a sustained (markedly increased reflex arc) reaction to diagnose as a hard sign, or unsustained for a soft sign.

Ataxia was tested by: 1) observing the subject's normal walk; 2) the heel-toe walk--having the subject place the heel of one foot against the toe of the other foot for five or more consecutive steps; and 3) hopping on each foot for five consecutive hops. A hard sign was diagnosed if the subject could not hop or showed a definitely asymmetrical hop, if he/she was grossly unsteady, or could not do the heel-toe walk. A soft sign was indicated by mild to medium asymmetry while hopping, by a deviation of the straight line hopping, or by a

slight wobbling (unsteadiness) or slowness on the heel-toe walk.

Asymmetry of skull or limbs was determined by "eyeballing" for obvious asymmetry; measurements were taken if there appeared to be a deviation from normal.

The Babinski reflex was tested by scratching the sole of the foot and the upper and outer surface of the foot separately. A positive sign is seen in the raising of the big toe and the fanning of the other digits. A soft sign was listed if there was a very mild or equivocal response.

Choreiform and athetoid movements: the subject was observed during normal interactions. They were formally tested by having the subject close his/her eyes and extend the hands in front of the body for 30 seconds. Athetoid, pseudo-athetoid, choreiform movements, and distonia were noted. Hard signs were diagnosed by marked movements, soft signs by slight movements.

Diplopia was tested by having the subject follow the examiner's finger through the full range (circle) of eye movements, including convergence. The subject was asked if he/she ever saw double.

Dysarthria: Attention was given to the subject's normal speech. A hard sign was indicated if the subject had an obvious speech difficulty (not including stuttering). The subject was asked to repeat the

following phrases after the examiner: Trudeau's constitution; static electricity; eleven benevolent elephants.

Dysdiadochokinesis was tested by having the subject alternate supination and pronation of each hand separately. This sign was rated hard if the subject was unable to do this, and soft if the action was disjointed and dysrhythmic.

Dyspraxia of tongue movement was tested by having subjects move their tongues from side to side rapidly. If there were other signs of spasticity and spastic speech, difficulty in tongue movement was not considered dyspraxic.

Graphesthesia was tested by tracing numbers (6, 9, 3, and 8) on each palm of the subject's hands while his eyes were closed. If the subject made errors, the number was retraced. The subject had to make a mistake on a number at least twice for this to be considered abnormal.

Heel/knee testing was done by stroking the leg from knee to ankle on each leg with the opposite foot. The neurologist was looking for asymmetry and incoordination. It was diagnosed as a hard sign if it was marked, grade 2, uni- or bilateral, and as a soft sign if it was slight, grade 1.

Incoordination was tested by the finger-nose test with the eyes open and closed, and by the toe-finger test with the eyes open and closed. This was done at least twice for each hand, and was rated as marked (hard sign) or slight (soft sign).

Intention tremor was determined by observation--marked for a hard sign, or slight for a soft sign. The neurologist was careful to distinguish between essential familial tremor which is fairly smooth throughout the range of movement, and true intention tremor which becomes more marked as the hand nears the target.

In testing for nystagmus, if there appeared to be a congenital (familial) nystagmus this was considered normal. A hard sign was indicated if the eyeball continued to quiver after the examiner's finger stopped moving when located out 30 degrees to either side, or up or down. As well, a hard sign was diagnosed if there was definite, sustained, and/or unilateral evidence. A soft sign was indicated if the quivering stopped within a couple of seconds after the examiner's finger stopped, and if the movement was bilateral.

Paresis was determined by testing the degree of resistance and any asymmetry by having the subject spread and hold rigid the fingers of each hand separately. The neurologist attempted to bend the fingers forward and push

the fingers together. The shoulder abductors, thigh flexors, and foot dorsiflexors were tested similarly. A hard sign was diagnosed for asymmetrical paresis, while a soft sign was diagnosed if there was a slight bilateral weakness.

Position sense was tested with the subject's eyes closed. The neurologist moved the subject's little finger and later the big toe up and down, and asked the subject the direction of the movement. It was rated as marked for a hard sign, and slight or mild for a soft sign.

Resting muscle tone was tested by rotating and moving the subject's elbows, wrists, hips, knees, and ankles while the limbs were limp. The neurologist looked for a normal, increased or decreased amount of resistance in these joints, and rated any spasticity or hypotonus as a hard sign if it was marked, or a soft sign for slight abnormalities.

Resting tremor was rated as marked or slight, for hard or soft signs, and was determined by observing the hands and head while in the resting position.

Saccadic eye movements were looked for during the testing for diplopia. A soft sign was indicated if the eyeball was jerky in the pursuit movement.

Simultagnosia was tested by simultaneously touching two points on the subject's hands, cheeks and shins while

his eyes were closed. Did he feel both or was one side ignored?

Strabismus was tested by observing the eyes at rest and in the test for diplopia.

Synkinesia was tested by having the subject move the fingers of one hand rapidly, while the neurologist looked for any movements of the other hand. It was also tested by having the subject tap one foot rapidly, while the neurologist observed the other foot.

Tendon reflexes, diminished or hyperactive: the reflexes of the knees, ankle, biceps and wrist (supinator) were tested. Finger flexion reflex (Hoffman's sign) was tested as well. A hard sign was indicated if the reflexes were rated as grade 2 unilateral, or grade 3 uni- or bilateral. A soft sign was indicated by a grading of 1 uni- or bilateral, or grade 2 bilateral.

Visual field defect was tested by simultaneous bilateral confrontation with fingers.

## Appendix B

Raw Data

## Data List:

The data for each subject is contained on two lines.

Line 1: BDGROUP 1, SEX 3, AGE1 6-8, AGE2 10-12,  
NX101 TO NX126 16-66.

Line 2: NX201 TO NX226 7-57.

## Variable and Value Labels:

BDGROUP: Grouping by diagnosis of brain damage;  
1=Brain damage, 2=MBD, 3=LD.

SEX: 0=Female, 1=Male.

AGE1: Age at Time 1, in months.

AGE2: Age at Time 2, in months.

NX101 TO NX126: Neurological exam items at Time 1;  
1=Hard Sign; 2=Soft Sign; 3=Normal.

NX201 TO NX226: Neurological exam items at Time 2;  
1=Hard Sign; 2=Soft Sign; 3=Normal.

NX101, NX201: Ataxia;

NX102, NX202: Asymmetry of skull or limb;

NX103, NX203: Anosmia; NX104, NX204: Visual field defect;

NX105, NX205: Diplopia; NX106, NX206: Strabismus;

NX107, NX207: Saccadic eye movement;

NX108, NX208: Nystagmus; NX109, NX209: Dysarthria;

NX110, NX210: Dyspraxia of tongue;

NX111, NX211: Choreiform-athetoid movements;

NX112, NX212: Resting tremor;  
NX113, NX213: Resting muscle tone; NX114, NX214: Paresis;  
NX115, NX215: Diminished or hyperactive tendon reflexes;  
NX116, NX216: Ankle clonus; NX117, NX217: Babinski reflex;  
NX118, NX218: Synkinesia; NX119, NX219: Incoordination;  
NX120, NX220: Heel-knee test;  
NX121, NX221: Intention tremor;  
NX122, NX222: Dysdiadochokinesia;  
NX123, NX223: Anaesthesia; NX124, NX224: Simultagnosia;  
NX125, NX225: Position sense; NX126, NX226: Graphesthesia.



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Title of Thesis

Persistence and Changes of Neurological Signs in Learning Disabled Subjects Over A 15 Year Period

Author



Brenda M. Lowick

June 26, 1989



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