HANDEDNESS AS A FUNCTION OF NEUROLOGICAL INVOLVEMENT IN A CLINIC-REFERRED SAMPLE OF DISABLED LEARNERS

by

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ABSTRACT

The present study is concerned with the pattern of handedness among learning disabled children. Typically, clinic-referred learning disabled samples are more likely than those in general school samples to have a higher than average rate of non-dextrality. Reports also indicate that in populations of individuals with neuropathology, such as epileptics and retardates, the incidence of left-handedness is at least twice as high as that reported for the normal population. These excess, "pathological," left-handers are thought to be left-handed by virtue of neuropathology leading to a switch of manual preference, rather than by genetic determination. Higher rates of neuropathology in clinic samples could account for the failure to find differences in general school, but not in clinic, samples. It was thought that in a sample of clinic-referred learning disabled children, non-dextrality would increase as a function of degree of neuropathology.

Children between the ages of 7 and 13, referred to a neuropsychology clinic because of learning difficulties, were followed up an average of fifteen years later. On the basis

of a neurological examination administered at the time of referral, children were classified into three groups: those with definite neurological abnormalities, those with questionable neurological signs, and those with no neurological abnormalities. Subjects were administered a questionnaire concerning their manual preferences for each of seven tasks. They were also given a test of grip strength and asked to write their name with either hand, so that the relative performance advantage with either hand could be assessed. These tests were administered both at time of referral and at time of follow-up, fifteen years later. Due to factors such as inconsistencies in test administration and subject attrition, 106 subjects received all three tests at time of referral, 124 at the adult testing, and 83 received all three tests at both times. In addition, 52 matched controls, selected from local school district records, were tested at the follow-up assessment, receiving a neurological assessment at this time.

The main hypothesis was not confirmed. Non-dextrality did not increase as a function of neuropathology across the three classifications, either at time of referral or at young adulthood. It was thought that, although individuals generally become more dextral over time, groups with more neuropathology, and consequently more "pathological" left-handers, would be less likely to show the normal developmental shift. Change scores, analyzed to determine both magnitude and incidence of change towards dextrality, produced no significant effects. Since they did not show a greater dextral shift than the group with no signs, non-dextrality in subjects with soft signs cannot be considered as evidence of lagging maturation which eventually catches up.

The comparison of learning disabled subjects at follow-up with adult controls was meaningful, if not statistically significant. There were two to three times as many learning disabled subjects as controls who were identified as left-handed on a number of measures. Although the difference was not significant, the close parallel with studies of both clinic-referred learning disabled subjects, and of retardates and epileptics, suggests that clinic-referred learning disabled subjects are more sinistral than controls, regardless of degree of neuropathology. Clinic samples could be biased by a tendency of both physicians and teachers to view left-handeness as a "warning sign" of neuropathology leading to an over-representation of left-handers among referrals.

Alternatively, neuropathology, irrespective of degree, may contribute to sinistrality. Even learning disabled subjects with no neurological signs were more sinistral than controls, implying that they could have incurred some mild neurological damage. An analysis of the persistence of neurological signs (Hern,1983) indicated that by adulthood these subjects actually presented signs of neuropathology, suggesting that they, too, could be "pathologically" left-handed. Clinic samples of learning disabled subjects, then, may include individuals with varying degrees of neuropathology. These subjects are more likely to be left-handed than normal controls, irrespective of degree of neuropathology.

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To my parents, Joy and Lee, who have always been there, with love and support.

INTRODUCTION

Although the study of learning disabilities has only recently entered an era in which neuropsychology plays a prominent role (Hiscock and Kinsbourne, 1982), theories based on brain-behavior relationships are not new to the field. In the 1930's Samuel Orton presented a theory of cerebral lateralization in dyslexia. Although still unsupported, it set the stage for several decades of writers who considered lack of established cerebral dominance to be a significant factor in dyslexia.

Orton's theory is based on the notion that "engrams", or memory traces, of letters and words are laid down in both hemispheres, those in the right hemisphere being in mirror-image reverse order of those in the left. In most individuals the dominant left hemisphere suppresses the mirror image engrams of the right hemisphere, allowing reading and writing to occur normally. In some people dominance is incomplete, leading to incomplete suppression of the reversed engrams and confusions in reading and writing. Orton called this condition "strephosymbolia", meaning "twisted symbols" (Orton, 1937).

During the course of his work, Orton noticed that children with language disabilites displayed different patterns of lateral preferences from normal children, being more often mixed- or left-handed. Orton concluded that mixed-handedness was pathognomic of strephosymbolia; the dominant hemisphere is unable to completely supress manual preferences controlled by the nondominant hemisphere. As for left-handers, he writes:

> It is only those in whom the tendency towards some measure of left-sidedness is present, but not in sufficient strength to assure complete unilateral superiority of the right hemisphere of the brain, in whom trouble may ensue. (Orton, 1937, p.130)

According to this theory, left-handers are at risk for strephosymbolia as long as the right hemisphere is not strongly dominant. Most researchers consider left-handers at risk for defective lateralization because their lateral preferences are usually not as strongly established as those of right-handers (Vernon,1971).

There is no evidence to support the engram hypothesis, but many researchers have searched for a relationship between lateral preference and dyslexia, with inconsistent results. Early researchers often noted disproportionately large numbers of sinistrals in dyslexics. Investigators such as Dearborn (1931), Roudinesco, Trelat and Trelat (as cited in Critchley,1971), and Orton, Skydaard and Eutis, (as cited in Hecaen and de Ajuriaguerra, 1964), noted a high incidence of sinistrality, up to 75%, in their dyslexic patients.

Since these findings were based mostly on clinical impressions, their validity is suspect. Controlled studies are not always so conclusive. Harris (1957), gave tests of lateral dominance to groups of unselected school children and clinic-referred reading disabled children. He found more mixed-handedness among seven-year-old and more left-handedness among the nine-year-old dyslexics. Hecaen and de Ajuriaguerra (1964) found more mixed-handers among younger dyslexics (age seven to ten) than older dyslexics and normals. Naidoo (as cited in Vernon, 1971) found children with ambiguous hand preference more likely to have lower verbal IQ's and histories of slow speech development.

A number of other studies fail to show any relationship between lateral preference and learning disorders. Using Harris' Tests of Lateral Dominance, Coleman and Deutsch (1964) found that hand dominance did not differentiate between normals and dyslexics. Belmont and Birch (1965), also using Harris' Tests, found no differences between nine- and ten-year-old poor and good readers. Both of these studies used the same handedness measures as Harris (1957) but failed to replicate his findings.

Since these studies also looked at children older than in Harris' study, these authors suggested that early differences in laterality may reflect neural abberations affecting later reading ability. However, Barlow (1963), using the same measure with six-year-olds, also failed to find any relationship. Hence, the age of the subjects clearly is not the explanation for differences in findings. De Hirsch, Jansky and Langford (1966), in a study specifically designed to predict future reading disability based on kindergarden test performance, did not find that ill-defined lateralization was a predictor of later reading problems. Investigations by Sparrow and Satz (1970) and Lyle (1969) also failed to find support for the hypothesis.

The Measurement of Handedness

There seems to be only weak support, at best, for the notion of abberant lateral preference patterns among reading disabled children. While some current researchers are skeptical that a significant relationship exists (Hardyck and Petrinovich, 1977; Bourke, 1978), others feel the concept may still be viable (Kinsbourne and Hiscock, 1981). The question is difficult to resolve due to methodological inconsistencies and to the frequent lack of controls for relevant factors. One major problem in handedness research is the variety of ways both to measure and classify handedness. Many discrepancies in the literature, such as the number of left-handers in a population, may be artifacts of the particular categorization procedure used.

According to Annett (1970a), most earlier clinic studies of handedness used Rife's classification system. Individuals who performed any of a given set of unimanual activities with the left hand were called sinistrals. This definition results in an excessive number of left-handers, making it difficult to compare these studies with later ones.

Self-report, the most common measurement method, yields a dichotomous classification based on a single measure. Although straight-forward, this method is often based on the preferred writing hand, which is vulnerable to outside pressures from parents and teachers; it is probably not a very valid measure of true handedness (Annett, 1973).

Humphrey (1951) was the first researcher to use a questionnaire to assess handedness. Self-report data were not accurate, he found, since a number of self-proclaimed left-handers indicated they preferred the right hand for a majority of the activities listed.

A variety of questionnaires have since been developed to assess handedness (ie, Oldfield, 1971; Satz, Achenbach and Fennell, 1967; Raczkowski, Kalat and Nebes, 1974; Annett, 1970a). These questionnaires vary relatively little in the types of tasks surveyed. Some researchers have attempted to control for some of the biases inherent in randomly selecting manual behaviors by selecting only those items which discriminate between groups (Oldfield, 1971), or by weighting more highly those that discriminate best (Subirana, 1969). Questionnaires correlate highly with actual behavioral assessments of manual preference and are reliable, although left-handers are more inclined to be inconsistent (Raczkowski,et al, 1974).

Questionnaires, sampling from a variety of tasks, demonstrate that handedness is best represented as a continuum from strong right- to strong left-handed preferences. The actual distribution is J-shaped, with the majority of individuals using the right hand for all or almost all activities, very few claiming equal preference, slightly more moderate left-handers, and even more who are strongly left-handed (Annett, 1972).

Although handedness is a continuum, it is often divided into discrete categories. There are a number of approaches that can be taken. Some dichotomize data into "predominantly left" or "predominantly right" (Raczkowski et al, 1974, Oldfield, 1971). Others trichotomize handedness, into right, mixed, and left, although the divisions are very arbitrary. Some choose arbitrary cut-off points based on the distribution of the data (Satz et al, 1967) or call all individuals ambilateral unless they are completely consistent for every item (Annett, 1970a). Handedness can also be divided into four, five or even six different categories, based on arbitrary cut-off points (Harris, 1957, Teng, Lee, Yang and

Cheng, 1979) or by subdividing right- and left-handers into "pure" and "mixed" forms (Annett, 1970b).

Annett (1970a) notes that much of the ambiguity in the studies relating handedness to learning disabilities could be due to researchers treating a continuous distribution as if it were discrete. Reports of the incidence of left-handedness in the past have ranged from one to thirty percent depending upon how left-handedness has been described (Hardyck and Petrinovich, 1977). As we have seen, a lot of the variability is due to inconsistent peformance of left-handers which makes it difficult to know where to draw the line. For researchers, the choice of criteria should be based on the theoretical and empirical questions which guide their investigations. It should be emphasized that differences between studies could be based more on methodological than actual differences.

Handedness may not only be described by questionnaires. Hildreth (as cited by Palmer, 1964), was one of the first to stress the need for an approach to handedness which would include other manifestations of motor activity. Many researchers have since begun using both performance and preference measures. Hardyck and Petrinovich (1977) noted that when performance measures are included, estimates of left-handedness become more stable, at nine to ten percent rather than the one to thirty percent figures noted earlier.

Empirical investigations suggest that performance measures pick up variability among left-handers that is not evident with other assessment methods. Benton, Meyers and Polder (1962) and Satz et al (1967) found that left-handers were more variable than right-handers in the extent of dominant-hand superiority on a variety of performance measures. Both sets of investigators discovered that slightly less than fifty percent of all self-proclaimed or questionnaire-classified left-handers had a right-hand superiority on performance measures.

It has been suggested that strength, skill and preference may be orthogonal dimensions (Porac and Coren 1981). A few of the tasks underlying these dimensions bear discussion.

Grip strength has long been used as a measure of dominance, but even Orton (1937) has noted that there seems to be no relationship between grip strength and manual preference. More recently, Porac and Coren (1981) found that, summing across several studies, the Dynamometer has only a 59% agreement with preference classification, slightly better than chance.

Different tests of dexterity, a component of manual skill, have been used with variable success. Some researchers (ie, Zurif and Carson, 1970, Benton et al, 1962), have looked at scissor use, which is affected more by environmental than genetic capacity. Small parts manipulation, another popular measure, (ie, Shankweiler and Studdert-Kennedy, 1975, Satz et al, 1967) shows a 74% correspondence with preference measures. Rate of finger tapping, another dexterity measure, also is not highly correlated with lateral preferece (Porac and Coren, 1981).

Manual performance is not a unitary concept, and it is not surprising that different performance measures are neither highly correlated with preference measures, nor very highly intercorrelated. Porac and Coren (1981) found the mean percent agreement between eight performance measures to be only 59%. Part of the discrepancy may be caused by very low test-retest reliability (Shankweiler and Studdert-Kennedy, 1975). Additionally, factor analytic studies suggest that handedness is multifactorial (Fleischman, 1972, Barnsley and Rabinovich, 1970), but it is not clear which factors are relevant to differences in cerebral organization (Kinsbourne and Hiscock, 1981). When considering at least ten independent dimensions of unimanual performance, it is difficult to know which ones to sample (Hicks and Kinsbourne, 1978).

Unlike manual preference, manual performance has a complex, multidimensional structure. The two assessment procedures do not produce a high degree of overlap; rather, manual performance seems to provide additional information about inconsistent left-handers. Selection of performance measures can be based on theoretically-distinct aspects of handedness, such as preference, speed, dexterity and pointing (Miller, 1982). Alternatively, manual performance can be considered to be the result of a mixture of both practice effects and genetic capacity. Since highly practiced skills, such as handwriting speed, may yield the most reliable differences between hands, some investigators suggest that they be used as measures of manual laterality (ie Shankweiler and Studdert-Kennedy, 1975, Provins and Cunliffe, 1972). Kinsbourne and Hicks (1981) disagree with this recommendation, noting that while highly practiced skills may yield the strongest and most reliable differences between hands, the magnitude and reliability probably reflect differential practice, and that even novel tasks may involve some transfer from more common activities. On the other hand, Annett (1970b) argues that if the differences between the hands in speed are a _product_, rather than a _cause_, of differential practice, changes in relative speed would occur as the individual matures and gains more experience. She found, however, that asymmetries of both preference and speed are equivalent at early childhood and early adolescence.

While a considerable amount of research has gone into developing valid and reliable questionnaires to assess lateral preferences, the dimensions of performance are much more elusive. The choice of performance measures to sample as an adjunct to a questionnaire is sometimes as much a matter of individual predilection as anything else. It is probably correct to assume that on most manual tasks right-handers will be more skillful with the right-hand, whereas handedness will be variable in left-handers, both in terms of magnitude and direction of manual advantage (Hicks and Kinsbourne, 1978). While many studies of laterality in learning disabled children rely primarily on questionnaire data, some utilize performance measures as well (ie, Annett, 1974, Satz and Sparrow, 1970). Since performance measures may give a different breakdown among left-handers than questionnaires alone, differences in methods of assessment may account for some of the variability among studies.

Unselected Versus Clinic Samples

A number of studies have been done on unselected samples of school children; almost all fail to find any differences in laterality between good and poor readers. Epidemiological studies by Clark (1970) in Scotland, Rutter, Graham and Yule (1970) on the Isle of Wight, Belmont and Birch (1965) in Scotland, and Malmquist (1958) in Sweden, all failed to find any relationship between aberrant lateral preferences and learning disability. A more recent study by Richardson and Firlej (1979) of public school children, none of whom had been referred for remedial teaching, also failed to find any relationship between reading or spelling performance and any of several laterality measures.

With clinical cases of dyslexia, left- or mixed-handedness seem to occur more frequently. Harris' study, cited earlier, compared clinic-referred and unselected children, and found differences between groups in children under ten. Perlo and Rak (1971), in a study of adult dyslexics referred to a language disorder clinic, found that one-quarter were left-handed. Although there was no control group, sinistrality in the general population is usually placed at eight to ten percent (Vernon, 1971). Wold (1968) found more left-handers among a clinic than a general school population. Bender (1968) and Zangwill (1960) report mixed- or left-handedness as a characteristic feature of dyslexia in their clinical experience.

The discrepancy between clinical and general samples suggests one possible explanation for inconsistent findings. According to Clark (1970), clinical populations contain more left-handers because of the tendency to consider left-handers with reading problems as a special category. These children are referred to physicians or other specialists because teachers see nondextrality as pathognomic of neurological disorder (Accardo, 1980).

In the past ten years physicians have been de-emphasizing the importance of laterality as a pathological sign. Nelson, Welker and Hobbs (1982) found no differences between right- and left-handed children on measures of motor proficiency and dexterity. They concluded that, while findings of left-handedness or ambidexterity should not be dismissed as of no consequence, they should not be considered abnormal neurological signs without additional evidence. Touwen (1972) has also asserted that no causal relationship between nondextrality and neurological disorders can be inferred without further evidence. Voeller (1981), however, notes that if a child manifests unequivocal hand preference before age one, it is likely that unilateral cerebral injury has been sustained.

While fifteen years ago members of the medical community suggested that nondextrality may be a correlate of neurological injury (ie, Benson and Geschwind, 1968, Paine and Oppe, 1966), current attitudes are more conservative. Whether or not nondextrality is truly an indicator of neurological damage, if viewed as such by either the medical or educational communities, nondextral dyslexics may be more likely to be referred to clinic settings.

A bias towards nondextrals in clinic referrals may not be the only explanation. It is possible that the effect of lateral preference is strong enough to appear in selected samples with high base rates, but is much weaker in general populations. Annett (1970b) looked at both a general school population and a subgroup of this sample selected for low scores on a verbal intelligence test. Although no relationship between laterality and verbal intelligence was noted for the general population, mixed-handers were more numerous in the low verbal intelligence subgroup. In a subsequent study, Annett (1974) found no lateral preference effect in a general school population, but found more left-handers in a subgroup of children with reading quotients significantly lower than their verbal intelligence scores.

Annett suggests that her underachieving subgroups are likely to be part of the population generally referred to clinics. However, since they were not part of clinic samples, they are not susceptible to the criticism of having been selected on the basis of nondextrality. Naidoo (1972) found that boys in a general school sample who were behind in reading by at least two years were less likely to be dextrals than those behind by only one year. Again, cases of more severe school failure are more likely to be referred to a clinic than the less severely disabled.

A bias in referrals, then, may not be the only explanation; there may be a real difference between clinic and non-clinic samples which results in less dextrals in clinic groups. Generally, clinic samples are composed of a more severely disabled subgroup of the general population, and are therefore more likely to present signs of neurological damage. If nonright-handedness does signal some minor neurological dysfunction, then the unreliability of a trend towards increasing nonright-handedness among learning disabled populations may in fact reflect the neurological integrity of the experimental sample, rather than being a correlate of learning disabilities per se.

Pathological Left-Handedness

It has long been noted that many populations of brain damaged individuals contain an excessive number of left-handers (ie,Silva and Satz, 1979, Subirana,1969). Obviously, early paresis of the preferred limb will, in most cases, lead to a shift of manual dominance (Benson and Geschwind, 1968). More importantly, early damage affecting the motor cortex of the dominant hemisphere may lead to a shift in manual dominance. The nonpreferred hand, during the course of motor development, may therefore become preferred, because of its comparatively greater ability. Since the majority of the population is right-handed, left-sided lesions are likely to produce a shift to right hemisphere dominance for handedness and consequently a "pathological", that is, not genetically determined, left handedness. Conversely, right-sided lesions, in most individuals, would not affect handedness. Populations of brain damaged individuals, then, are likely to contain an excessive number of left-handers (Benson and Geschwind, 1968).

Pasamanick and Knobloch (1966) speculate that since environmental pressures towards right-handedness are great, some brain damaged individuals might resort instead to a clumsy type of ambidexterity. Typically researchers have discussed the increased incidence of left-handedness, rather than ambidexterity, among brain damaged populations, but, as noted before, the definition and measurement of left-handedness varies from researcher to researcher. Since left-handedness is so variable, according to the measurement used, it is quite possible that some researchers are including mixed-handers among their left-handers. In fact some researchers (ie, Satz, 1979) explicitly combine the two groups for analyses. Perhaps then, "pathological left-handedness" is really "pathological nonright-handedness".

Brain Damaged Populations

A higher incidence of left-handedness among brain damaged individuals was noted as far back as 1890 by Sachs and Peterson (as cited by Porac and Coren, 1981) who reported that 48% of their 156 hemiplegic patients were left-handed. In an investigation of left-handedness among retarded school children, Gordon (1920) noted that not only was the percentage of left-handers higher than in normal children, but, in the case of left- and right-handed twins, the left-handed twin is more likely to be retarded. He assumed that the same lesion not only affected the proper functioning of the dominant hand, but interferred with higher intellectual centers as well.

More recent studies have shown an increased incidence of left-handedness in a variety of neurologically impaired populations. Lonton (1976), found increased incidences of left- and mixed-handedness among children with myelomeningocele and hydrocephalus. Rutter, Graham and Yule (1970), in an epidemiological study of children on the Isle of Wight, found that left-handedness and left-footedness are significantly more common in children with a variety of neurological disorders, including cerebral palsy and brain stem lesions. Interestingly, they did not find an increase in left- and mixed-handedness among children with reading problems but without neurological disorders, supporting the view that neurological dysfunction, not reading problems per se, is the crucial variable.

Epileptics, too, seem to have a raised incidence of manifest left-handedness. Roberts (1955) found that 17% of the cases of traumatic epilepsy and encephalopathy operated on at the Montreal Neurological Institute were left-handers. When the lesions were present before the age of two, the percentage of left-handedness went up to 41%, which clearly emphasizes the fact that a pathological shift in handeness is the result of an early lesion. Satz, Baymur and Van der Vlugt (1979) found a raised incidence of left-handers among epileptics from four culturally distinct populations. Redlich (as cited in Hecaen and Ajuriaguerra, 1964) found an incidence of 17.5 percent in a group of epileptics as contrasted with eight percent in a group of psychiatric patients.

As an exception, Mc Manus (1980), reviewing data from a large prospective study of 12,000 British children, saw no relationship between left-handedness and epilepsy. In light of all the evidence supporting the relationship, this finding is quite surprising. Mc Manus, however, admits that there were very few severe epileptics among his population and that in such a group an association may exist.

Studies of retardates, in whom neurological damage is not necessarily limited to one or the other hemisphere, also find increased incidences of left-handedness. As previously cited, Gordon (1920) studied almost 8,000 normal and retarded children and found incidence rates of 7.3% and 18.2% respectively. Porac, Coren and Duncan (1980a) concluded that retardates showed significantly more left-sidedness than either their chronological peers or their mental age peers (preschool children). Silva and Satz (1979) studied 1409 retardates and found the frequency of left-handedness to be 17.8%. They found that individuals with abnormal EEGs had a higher incidence of left-handedness than those with normal EEGs, suggesting that the probability of an individual being right-handed decreases with poorer levels of cortical functioning. Hicks and Barton (1975) reported that right-hand preference is less common among the severely and profoundly retarded than among the mildly and moderately retarded, indicating that there may be a positive relationship between degree of retardation (and concommitant brain damage) and incidence of left-handedness.

Satz's Model

Although the concept of pathological left-handedness has been in the literature for over eighty years, it has recently been focused on by researchers who seek to explain the phenomenon more fully. Satz and collaborators (1972a,1972b,1979,1979) have put forth an arithmetic model to account for this observation. Satz noted that most explanations of pathological left-handedness focus on the fact that early left hemisphere damage can cause a mild dysfunction of the dominant hand in right-handers, causing the individual to switch to the left hand for manual activities. This explains the occurence of pathological left handedness, but doesn't take into account pathological <u>right</u> handedness, which would be caused by a rightsided lesion in a natural left hander. Satz noted that most studies of epileptic and retardate populations report the incidence of left-handedness as about 17%. He then developed a model which is based on assumptions of the equal probability of a lesion affecting either side (sometimes producing pathological right-handers), and the probability that such random lesions would lead to an overall 17% incidence rate of left-handedness.

Satz assumed that not all brain lesions contralateral to the genetically preferred hand would result in manual transfer. Using 8 and 17 percent as the empirically-based incidence rates of left-handedness in normal and brain damaged populations, respectively, Satz (1972a) determined that the probability of switching handedness following a given lesion contralateral to the dominant hand is about .21. Given equal probabilities of side of lesion, if 21% of the natural left-handers become pathological right-handers and 21% of the natural right-handers become pathological left-handers, then knowing the manifest handedness of an individual with a unilateral lesion, the probability of laterality of that lesion can be determined. When the appropriate calculations are made, the probability of a manifestly left-handed retardate or epileptic having a left-sided lesion would be .81 and the probability of a manifest right-hander having a left-sided lesion would be .44. Satz (1972a) confirmed his hypothesis by reviewing Penfield and Roberts' handedness data for a large group of epileptic patients undergoing surgery. He found that 75% of the left-handers had left hemisphere lesions, while only 41% of the right-handers had left hemisphere lesions. Thus the expected values of .81 and .44 were approximated; the data supported his contention that unilateral lesions can lead to a pathological switching of handedness which can be described by exact probabilities.

Satz went on to validate his theory (Satz, Baymur and Van der Vlugt, 1979), exploring the relationship between unilateral and bilateral lesions and handedness in four different clinical populations of epileptics and retardates. For each individual both handedness and side of lesion (i.e., nature of EEG abnormality--unilateral or bilateral) was known. He found that the incidence of manifest left-handedness among cases of bilateral EEG abnormality was substantially lower than among those with unilateral abnormality. He felt that in the case of bilateral damage, bimanual dysfunction would not lead to an advantage, and subsequent shift to, the non-dominant hand. However, in a separate study of retardates (Silva and Satz,1979), he found that the incidence of sinistrality in individuals with EEG dysfunction was abnormally high regardless of whether EEG involvement was asymmetric or bilaterally symmetric, but was higher than in those with normal EEGs. He concluded that two separate factors influence handedness in retarded populations: the presence of unilateral lesions which lead to an early switching of preference, and the presence of a general decrease in cortical functioning, as indicated by bilateral EEG abnormality.

Although Satz's model assumes that the probability of injury to the two hemispheres is equal, neurophysiological evidence suggests that the left hemisphere is especially vulnerable. Left occipito-anterior presentation of the fetal head during birth, the most common fetal orientation, leaves the left hemisphere more vulnerable to the pathological effects of increased extra-cranial pressure. In addition, the left hemisphere is apparently more vulnerable to vascular insufficiency because the left carotid artery supplies the left hemisphere relatively indirectly (Kinsbourne and Hiscock, 1981). Also, even with an equal likelihood of damage to both hemispheres, since the functions of the right hemisphere are more diffusely represented than those of the left, functioning may be more likely to switch to the relatively less differentiated right hemisphere (Porac and Coren, 1981).

Other researchers take issue with Satz over the need for gross signs of cortical damage in populations with increases in sinistrality. Bishop (1980) argues that mild unilaterally- or bilaterally-asymmetric abnormalities can be associated with increased sinistrality as well. He attempted to identify children with presumptive mild brain abnormalities by examining those who did particularly poorly on a square-tracing task with the nonpreferred hand. Children particularly clumsy with the nonpreferred hand were assumed to have some hemisphere abnormality contralateral to this hand which interferes with its functioning. With this measure, Bishop selected the worst 20%, out of 170 school children, predicting that this group would have a higher base rate of pathological sinistrality. Not only did the group show more sinistrality, but they also had a higher incidence of neurological disorders in childhood and showed significantly lower WISC-R and reading ability scores. Since Bishop feels that sinistrality in this group may be the expression of some mild brain abnormalities, rather than gross lesions, he calls it "Extended Pathological Left Handedness".

Not all researchers agree that cortical lesions are necessary for pathological left-handedness. Lonton (1976), in a study of myelomeningocele and handedness, found that asymmetrical damage in and around the upper part of the spinal cord can cause mixed-handedness. Liederman and Coryell (1982) studied the preferred direction of head turning in infants with and without a history of perinatal complications. They found that children with pregnancy complications were less likely to show a right-sided head turning preference than the control group. Liederman et al suggested that since differences are noted before the development of fine motor skill, injuries sustained during gestation and/or delivery may affect a laterally organized subcortical center, rather than just the motor cortex.

Bakan's Model

The studies previously discussed all concern the incidence of left-handedness among individuals with known pathology, assuming that the dysfunction causing the pathology is directly related to the dysfunction which caused the left-handedness. There is a substantial amount of empirical support for this hypothesis. Bakan (1977) however, took the concept of pathological sinistrality one step further and proposed that all nondextrality is pathological in origin. Bakan stated that pre- or perinatal events leading to hypoxia can adversely affect the pyramidal cells in the left motor cortex, which he felt are especially vulnerable to hypoxia (Bakan, 1977).

Bakan listed a number of correlates of left-handedness, including an increased frequency among males, who are more vulnerable to birth stress, and among twins, in which hypoxia-related birth complications are more frequent (Bakan, 1978). He suggested that deviations from right-handedness should be added to the "continuum of reproductive casualty", originally proposed by Pasamanick and Knobloch, (1966) as an explanatory model of the range of effects of pathology. Various neurologically-related disorders, ranging from learning disorders to still-births, are thought to result from a parallel continuum of pre- and perinatal trauma, varying in the extent of pathological outcome.

Bakan proposed that those infants who survive the effects of pregnancy and birth complications are more likely to be left-handed or ambilateral. This is why left-handedness is found in excess among those affected by other neuropathological conditions (Bakan, 1975).

Bakan went on to support his hypothesis by studying the relationship between birth order, and maternal age and sinistrality, citing evidence that the former two are correlated with birth and pregnancy complications (Bakan, Dibb and Reed, 1973). In samples of post-secondary students and university students he found significantly more sinistrals among high risk birth orders (first and fourth or later) than low risk birth orders (second and third) (Bakan, 1971, 1977). In another sample he found that self-reported birth stress and maternal age of thirty or over, were correlated with sinistrality (Bakan et al, 1973).

Bakan's speculations sparked a lively debate among researchers which still continues. Most accepted Satz's contention of pathologically-based sinistrality in individuals suffering from neuropathology, but few could accept the notion that all sinistrality is pathological. Schwartz (1977) noted that Bakan is not extending, but rather ignoring, Satz's model, which is based on the existence of both pathological and natural left-handers even in brain damaged populations.

Several studies question Bakan's findings of a relationship between sinistrality and birth order or maternal age. Hubbard's (1971) results directly contradict Bakan's, finding left-handedness to be greater in the low risk birth orders (second- and third-born). Schwartz (1977) found no relationship to birth order and proposed that differences in results are possibly due to classification methods, with questionnaires and self-report measures classifying left-handers differently.

Bakan (1977) argued that Hubbard's sample of private university students were likely to have received better prenatal care than Bakan's public university sample, leading to different base rates of pregnancy complications. Schwartz's sample, he contended, was from a Canadian provincial university with a higher mortality rate than the U.S., hence those individuals with pregnancy complications may be more severely handicapped and not present in a university sample.

Bakan's findings seem to have little generalizability to other populations. In a study of sinistrality among Taiwanese students, Teng, Lee, Yang and Chang (1976) found no effect of birth order or maternal age. Ashton's (1982) epidemiological study of 1818 Hawaiian families found no effect of maternal age or birth order on incidence of left-handedness. One would expect that an effect presumably based on pathology in every left-hander would be easily detectable in other populations.

Noting that Bakan (1971) demonstrated left-handedness to be more likely for males who were of "high risk" birth orders, Hicks, Elliot, Garbesi and Martin (1979) attempted to maximize the effects of risk factors by grouping individuals according to mother's age at birth, birth order, and sex into "high risk" and "low risk" groups, but were unable to find significant results. However Coren and Porac (1980), found that two of the birth risk factors, maternal age and birth order, interact; older mothers had fewer right-handed offspring who were second or third born (low risk position). The significance of this interaction, which is present in both sexes, is not clear, but since low-risk birth order is correlated with increased sinistrality, these results only partially confirm Bakan's findings.

Only Leviton and Kilty (1976) support Bakan's birth order hypothesis. They found a "dose-response" relationship between birth order and handedness in boys, with sinistrality increasing as a function of degree of perinatal risk. That is, the risk of left-handedness is relatively high in both first and fourth or higher birth order males and low in second and third birth orders, with the most left-handers found among the highest birth orders. This dose-response relationship indirectly supports the view that sinistrality belongs to the minor pathology end of the continuum of reproductive casualty mentioned earlier. However, Leviton and Kilty did not report their statistical analysis and state that one-sixth of their sample consisted of fifth grade children who had repeated a grade, suggesting they may be including learning disabled subjects, which would confound their results.

It is obvious that studying birth order and maternal age and handedness is one step removed from actually studying the relationship between birth stress and handedness. Several researchers have tried to study the incidence of birth complications more directly by asking individuals about the nature of their delivery. Bakan, Dibb and Reed (1973) asked college students about a variety of birth stress conditions and found that nonright-handed subjects reported birth stress about twice as often as right-handers. Schwartz (1977) asked college students more generally if their pregnancy was known to be normal, without mentioning specific types of complications, and found no relationship with sinistrality. Although the two measures of birth complications appear to be similar (college student self-report), perhaps

Schwartz's single, general question discouraged students from adequately considering the issue, increasing the chances of getting negative results.

Since self-report of birth complications is by its very nature based on second-hand information of questionable accuracy, Coren and Porac (1980) decided that a more valid method would be to contact mothers directly. Looking at four aspects of lateral preference (hand, foot, eye and ear preference), and birth stress, they found a relationship only between left hand preference and birth stress in males. Conversely, Ashton (1982), in a study of over 1800 Hawaiian families, found a significant effect of maternal birth stress in offspring hand preference for females only. Comparing his results with those of Coren and Porac, Ashton noted the sex difference, but suggested that if it is accepted that the chance of birth trauma is likely to be independent of the sex of the infant, then the sex difference may be considered fortuitous and the data should be pooled. When pooled, left-handed children are about 28% more likely, when birth is accompanied by stress. However it must be noted that there is a sex difference between the two studies which may or may not be fortuitous.

Relying on a mothers' report of birth complications is subject to inaccuracies as well. Not all mothers are aware of or remember all of the events surrounding each child's birth (Chamberlain and Johnstone, 1975). Smart (1980) examined hospital birth records and sent out questionnaires to the mothers of these children, who were by then six or seven, to ascertain the child's handedness. The only significant effect related to birth complications was that breech-delivered boys were more likely to become nonright-handed than Caesarian-delivered boys. This study provides minimal support at best for Bakan's hypothesis. Churchill, Igna and Senf (1962) found a weak, but significant relationship between atypical birth presentation and hand preference at age two, with left-handers more likely to be born with atypical presentations. Although this might suggest a relationship between perinatal events and subsequent handedness, children's hand preference at age two is not highly predictive of adult handedness (Gesell and Ames, 1947).

The data so far presented provide only weak support for a relationship between birth complications and handedness. However none of the studies are prospective in nature. Even the study by Smart relied on hospital records which were not compiled specifically for research purposes and thus lack standardization. Two longitudinal studies have been performed so far, one supporting, the other flatly refuting Bakan. Barnes (1975) provided some indirect support for Bakan. She found that the time a baby took to establish regular breathing was a good predictor of handedness at age three, with nonright-handers taking longer to establish breathing. Barnes did not interpret these results as supporting Bakan's anoxia theory, because she felt that all children established breathing before any damage could occur. Rather, she proposed that left-handed babies are more sensitive to the rigours of the birth process and therefore are more likely to have a stressful birth, rather than the stress causing left-handedness. However, whether or not these children suffered even mild anoxia is not known. Bakan at least, has cited Barnes as supporting his theory (Bakan, 1978) of hypoxia in sinistrals. Although her study can be commended for being prospective in nature, Barnes measured handedness when the children were only three years old. Her results would have more significance if she had waited a few years until handedness was fully established.

Mc Manus (1981), acknowledging that the only way to resolve the question is with a large prospective study, analyzed data collected by the National Child Development Study in Great Britain. The study was set up to examine the effects of a wide range of obstetric factors on over 18,000 children. Handedness was assessed at ages seven and eleven. The relationship of handedness to twenty-eight different factors which might be associated with birth stress was evaluated. Although a few perinatal factors were significantly related to handedness, the vast majority were not. Mc

Manus concludes that the magnitude of the study precludes any further discussion of Bakan's theory.

In sum, it is clear that the issue of pathological left-handedness has been hotly debated. There is a fair body of literature which suggests that populations with neuropathology often have a higher proportion of nonright-handers. Often this is the result of unilateral lesions, but general decreases in cortical functioning or noncortical lesions may play a part. There is evidence that the degree of dysfunction and amount of left-handedness are on a "continuum of reproductive casualty" at least for populations with known pathology, such as retardates. That all left-handedness originates from birth trauma is highly doubtful.

The whole debate has great relevance for learning disabilities research. Major neurological trauma results in increased sinistrality, while with minor trauma this is less likely. Mentioned earlier was the observation that learning disabled children in clinic populations are more likely to be left-handed than those in more general populations. It is possible then, that those children in clinic populations with signs of neurological damage are more likely to be left-handed, and further, that the incidence of left-handedness increases as a function of the degree of damage.

Neurological Signs

According to Rutter (1977), dyslexia can be differentiated along several dimensions, including the presence or absence of brain damage. Although it is often difficult to differentiate along this dimension in the absence of overt neurological disease or disorder, the distinction is a meaningful one. Brain damage, whether of a post- or perinatal onset, may cause specific reading and spelling difficulties, in cases where there is no impairment of general intelligence.

Learning disabled children labeled "brain damaged" often do have evidence of central nervous system abnormality, although the range of signs is great, and ranges from obvious cases of CNS damage (as in cerebral palsy), to cases where the only neurological sign is incoordination. In one study of the meaningfulness of the label "brain damaged" in a school for neurologically-handicapped youngsters, Hertzig et al (1969) found that only one-third of the children demonstrated hard signs of neurological damage and 90% had soft signs, with one-quarter showing both hard and soft signs of neurological damage.

The distinction between "hard" and "soft" signs is both qualitative and quantitative. "Hard" signs are findings that, according to Hertzig et al (1969), "have been classically employed in neurologic diagnosis and include abnormalities in reflex, cranial nerve, and motor organization, lateralized dysfunctions and the presence of pathological reflexes (p.440)".

"Soft signs" are considered "soft" because their interpretation and meaning are somewhat uncertain. There are two major types of soft signs. The first are subtle manifestations of classical neurological abnormalities which, if stronger and more reliable, would suggest definite presence of brain damage. According to Levine et al. (1980), " these findings were felt to reflect evidence of a mild, but discrete brain deficit, which allowed the clinician to make a diagnosis of a localized lesion, albeit a 'minimal' one" (p.41) . The second type consists of signs which are considered normal within a certain age group, disappearing with time, but when they occur in older children, are indicative of a developmental delay (Levine et al, 1980). Accardo (1980) notes that one-third to one-half of all learning disabled children have soft signs, suggestive of either maturational delay or mild brain damage.

Although a soft sign is interpreted without any knowledge of causal events, neuropathological evidence suggests that minimal cerebral lesions do occur at birth and may be responsible for the syndrome of minimal brain damage. Towbin (1971) stated that mild hypoxia incurred during the fetal-neonatal period often results in focal or diffuse neuronal damage with consequent minimal neurologic symptoms that can be later expressed as learning, behavior, or motor disorders. The presence of soft neurological signs is usually taken to characterize minimal brain dysfunction, while hard signs indicate more gross brain damage (Nichols and Chen, 1981). Since some soft signs are mild forms of hard signs (ie, mild reflex abnormalities), minimal brain damage and brain damage may be part of a continuum of neurological dysfunction.

As mentioned before, the idea of a "continuum of reproductive casualty" was first explored by Pasamanick and Knobloch (1966) who studied the neurological development of premature and full-term infants and found among the preterm sample a wide range of severity of cerebral damage. They postulated that the effects of damage to the brain during the pre- and perinatal period could vary greatly, from clearcut neurological disorders resulting from severe damage, to a predispositon to minor behavioral abnormalities resulting from mild damage. Minimal brain injury, then, was qualitatively, but not quantitatively, similar to more severe brain injury (Pasamanick and Knobloch, 1966).

In a recent review of the continuum concept, Rutter (1982) concluded that while there is no firm evidence suporting it, the idea probably does have some validity. Although there is no doubt that subclinical brain damage can produce psychological sequelae, he feels that rather severe damage is needed to give rise to persistent behavioral and cognitive deficits. Rutter notes that studies of perinatal brain injury suggest that pregnancy and birth complications can lead to psychological sequelae even when there is no overt neurological disorder, but these results are often confounded with socioeconomic disadvantage. Rutter concludes that the idea of minimal brain dysfunction as belonging on a continuum of reproductive casualty does have validity, but remains unproven.

The concept provides a good framework from which to consider the question of increased nonright-handedness among learning disabled individuals. If minimal brain dysfunction is a lesser variant of gross brain damage, then individuals with soft neurological signs are likely to suffer from minor neurological damage, such as that discussed by Towbin (1971). Since, as was previously stated, the incidence of left-handedness increases as a function of the degree of neurological damage, it seems plausable that children with neurological soft signs would be more likely to be left-handed than children with normal neurological examinations, but less likely than those with hard signs of neurological damage.

Alternatively, minimal brain damage may not be truly minimal in nature. Benton (1973) discussed evidence that cerebral lesions in children must be quite extensive or have specific disorganizing properties to produce behavioral abnormalities. He felt that there is no evidence that the damage in "minimal brain damage" is actually less extensive that in lesions underlying cerebral palsy and mental retardation.

There is still debate over just what soft signs mean. According to Rutter et al (1970), although there is evidence that soft signs may be due to neurological damage, arising from, for example, pre- or perinatal complications, such signs appear quite often in normal children who experienced no such complications. If soft signs reflect such a heterogeneous state of affairs, then not every individual with soft signs is necessarily brain damaged. If soft signs indicate brain damage in some, but not all, individuals, then even if the brain damage is not "minimal", the predictions of handedness can still be made: children with hard signs would be most frequently left-handed, followed by those with soft signs, and finally, those with no neurological signs, who would have the lowest incidence of left-handedness. Left-handedness could increase as a function of either the magnitude or probability of neurological damage.

There is experimental evidence that subclinical brain damage can lead to increases in sinistrality. Dugdale and Jeffrey (1981) noted that there were more left-handers among children who had had meningitis during their first year than among sibling controls. Although the authors speculate that during early bouts of meningitis these children may have incurred left hemisphere damage, leading to a switching of manual dominance, none of these children showed any major neurological or psychological findings. These children had only their neurological history as indirect indications of possible brain damage. This is similar to the case with soft signs, which can be thought of as merely indicators of possible neurological dysfunction.

Children with minimal brain dysfunction (MBD), which is sometimes, but not always, based on "soft" neurological signs (Beaumont, 1976), often have a high incidence of non-right-handedness. According to the National Institute of Neurological Disease and Blindness task force (Clements, 1966), a high incidence of left and mixed laterality is one of the specific neurologic indicators of minimal brain dysfunction. Beaumont (1976), in a study of children diagnosed as minimally brain damaged, found that they were not only less right-sided than the control group, but on retesting they were less stable in their preferences, indicating mixed-handedness. These differences were found with measures of lateral preference. On measures of lateral performance (manual speed), MBD children did not show the significant difference between preferred and nonpreferred hands that characterized normals; performance with either hand was exceptionally poor. Beaumont interpreted this as support for the idea that in MBD children lateralization is less clearly established.

There is an alternative explanation, however, which fits the pathological left-handedness model. Since environmental pressures towards right-handedness are great, these children may be resorting to the "clumsy type of ambidexterity" suggested by Pasamanick and Knobloch (1966) as an alternative to adopting a clean left-hand preference. Lateral preference would then be more unreliable, and performance measures would fail to show a clear hand advantage.

The Collaborative Perinatal Project (Nichols & Chen, 1981) a followup of almost 30,000 subjects, is probably the most extensive prospective study of minimal brain dysfunction ever done. Based on a series of neurological, psychological and behavioral examinations, children were categorized according to the presence of neurological soft signs, learning disorders or hyperactivity. Those with gross signs of neurological damage, such as cerebral palsy, were not included in the study. Manual dominance was examined when the children were four and seven years of age. At both ages, children with left- or mixed-handedness had significantly more neurological signs than the rest of the cohort. By age seven, nearly 16% of those with neurological soft signs had left- or mixed-hand dominance as compared to 11% of the controls (Nichols & Chen, 1981).

Individuals with soft signs of neurological damage are also more left-handed. The 16% rate for children with soft signs can be compared to the 17% rate noted by Satz (1972a) for populations of brain-damaged individuals. Considering left-handedness as varying along a continuum of increasing degree or prevalence of neurological damage, one would expect that the two values would be further apart, reflecting the different degrees of neurological damage. However, since the incidence of left-handedness varies according to the assessment procedure used (Belmont and Birch, 1963), it is difficult to compare across studies. Additionally, handedness is not a stable entity; children become more right-sided as they mature (Porac, Coren and Duncan, 1980b). A 16% incidence rate in seven-year-olds may be somewhat smaller when they become adults. The question of whether or not left-handedness increases as a function of neurological damage can best be answered in a study designed specifically for that purpose.

Neuropathology and Nondextrality

Considering the contribution of neurological damage to nondextrality in dyslexia, there is some evidence that left- and mixed-handed dyslexics really do differ from their dextral counterparts. Based on extensive clinical experience, Zangwill (1960) wondered whether there might be two types of developmental dyslexics. Those who were left-handed or ambidextrous were frequently not only learning disabled, but had a higher incidence of retarded speech development, defects of spatial perception, motor clumsiness and other indicators of abnormal maturation. Right-handers, in comparison, showed a relatively pure dyslexia, without the concomitant symptomatology.

Based on these observations, Zangwill proposed three possible explanations. First, he thought that both poorly developed laterality and reading backwardness could be due to the effects of actual cerebral lesions. He notes in this regard that early damage to the left hemisphere can lead to either complete or partial shifts of hand preference (either left-handedness or ambidexterity). Focal neurological signs, found in some dyslexic children, support this contention.

A second explanation is that certain children with "ill-defined" laterality have an additional "constitutional weakness in maturation" or maturational lag. This is suggested by the presence of minimal signs of neurological dysfunction in these children. Zangwill included left-handers among those with "ill-defined" laterality because left-handers are generally less consistent in their patterns of lateral preference.

The third explanation is that ill-lateralized children are particularly vulnerable to the effects of stress, such as minimal brain injury at birth. Zangwill noted that a history of early brain disease or minor epilepsy is not uncommon among ill-lateralized dyslexics. These stressful events then, can ultimately disturb the proper development of academic and perceptual-motor skills.

In his first explanation, Zangwill explicitly accepts that overt neurological damage can lead to both learning problems and nondextrality. His third explanation, that vulnerability to stressful events is a correlate of both nondextrality and learning disabilities, is reminiscent of both Bakan and Pasamanick. Zangwill looks at causation differently, though; nondextrality predisposes the individual to be more affected by neurological insult, rather than being a result of that insult. Also, he never clearly states whether or not these stressful events lead to actual neurological damage. This may be because Zangwill interprets minor neurological signs as being evidence of abnormal maturation, as presented in his second explanation, rather than evidence of minor damage. As stated earlier, soft signs have been interpreted as representing either phenomenon.

Development of Handedness

Handedness is not immutable. Investigations of handedness in children have found distinct developmental trends; however there is no clearcut evidence of the age at which manual preference is established. Although most researchers agree on the instability of hand preference in a child's early years, they have not always agreed on the age by which unilateral preference is established. Reports range from age ten (Belmont and Birch,1963) to five (Sinclair, 1971) and even younger. Most research implies that handedness is established sometime in the early school years.

Some of the variability in these reports may be due to methods of assessment of handedness or sample variation. But, more importantly, even after handedness is established, individuals seem to become more right-handed with age. Cross-sectional studies, of groups ranging from preschoolers to senior citizens, consistently report a trend towards increasing right-sidedness over time.

In a survey of published studies, Porac and Coren (1981) compared incidence rates of lateral preference measures reported for various age groups to get a gross estimate of age-related shifts in handedness. They found that overall adult samples seem to be about 12% more right-handed than infant samples. Studying the question directly, Porac, Coren and Duncan (1980) measured lateral preference in 1964 subjects ranging from 8 to 100 years and found that individuals become more right-sided at the rate of .19% per year. They confirmed their results in another sample, compairing preschoolers to high school students, and found 12.4% more consistent dextrality among the older group. McGee and Cozad (1980) and Fleminger, Dalton and Standage (1977), also found increasing right-handedness with age.

The trend towards increasing dextrality is stable, but difficult to explain. Prior to 1930, attitudes towards handedness favored dextrality, and it is not surprising to find more right-handers among people raised during that era; but the phenomenon is not limited to individuals over fifty. Other explanations are either environmental or maturational. It is possible that covert pressures on a nondextral individual forced to cope with a dextral environment lead to a gradual shift towards dextrality. Alternatively, increasing right-handedness may be secondary to physiological changes, such as the progression of myelination (Porac and Coren, 1981).

Regardless of the underlying mechanism, the dextral shift can provide us with more information about pathological nondextrality. An individual forced to switch handedness because of some minor neurological insult will be much less than likely to switch back over time. Theoretically, environmental or maturational pressures towards dextrality would be insufficient to overcome an innate neurological bias against it. A long term study of changes in handedness should show that individuals with clear signs of neurological damage are less likely to become more dextral over time. Those with soft signs of neurological damage, if we view soft signs as indicators of either a lower probability or lesser degree of neurological damage, would show a slightly greater likelihood of becoming dextral. Keeping in line with previous research, individuals with no neurological signs will become comparatively more dextral over time.

Indirect evidence suggests that, at least for individuals with hard signs of neurological damage, these predictions are valid. In their investigation of several clinical populations Satz, Baymur and Van der Vlugt (1979) compared manifest left-handedness in two adult and one child sample of epileptics. Although different assessment methods were used (questionnaire versus dextrality measures), the percentage of left-handers remained at 17% in each study. It is not certain what criteria were used for differentiating left- from right-handers, but the consistency between studies is noteworthy.

Maturational Lag

Nondextrality, as characteristic of younger individuals, could, alternatively, be symptomatic of a general maturational lag, responsible for both the learning disorder and the handedness pattern. Over time, those maturationally delayed individuals could "catch up," becoming more dextral. Lauretta Bender was one of the first proponents of the theory that developmental dyslexia is due to a general maturational lag. According to Bender (1956), a maturational lag signifies a slowing in cortical differentiation (Bender, 1956). Maturational lag models range from postulating a delay in the differentiation of the two hemispheres from equipotentiality (Orton, 1937), to a delay in the maturation of the left hemisphere alone (Hiscock and Kinsbourne, 1981). In any case, children are thought to be "maturationally delayed" because the level of their abilities is that of a much younger child (Kinsbourne, 1973).

As noted earlier, many "soft" neurological signs are suggestive of maturational delay in as much as they occur regularly in normal young children, but should not be present in older children. The similarity between immature neurological responses and immature academic performance is so striking that, as Levine, Brooks and Shonkoff (1980) explain:

> Through guilt by association, many clinicians postulate that minimal deviation or immature responses on an extended neurological examination reflect minimal deviation or immature development of high-

> er cortical processes necessary for successful learning. (p. 41)

The concept makes heuristic sense, but whether or not developmental delay is distinct from the many other causes (i.e., traumatic, socioeconomic, genetic) of learning disorders, including neurological factors, is debatable (Pasamanick, 1973). Some researchers (Thompson, 1973; Critchly, 1970) consider maturational lags to be the primary cause of reading disability, attributable to genetic, rather than neurological factors. Conversely, Kinsbourne (1973) considers developmental lags and frank neurological deficit to be on a continuum of neuropathology, a noxious influence producing either one or the other, depending on its severity and distribution. Rutter et al (1970) on the other hand, find that delays in some developmental functions have weak, and others much stronger, association with neurological disorder. According to Rutter, because strength of neurological associations with each type of developmental delay has not been determined, it is not easy to separate maturational delays due to neurological disorder from those which are purely developmental. Whether or not maturational delays are separate from, identical to, or part of a continuum of neurological disorder, is clearly a matter for speculation. Nonetheless, each of these researchers considers developmental soft signs to be meaningful indicators of maturational lag.

If these children do suffer from a maturational delay, do they eventually catch up? Drawing from her clinical experience, Bender states that lags in development tend to correct themselves by maturation. Critchley (1970) comments that a maturational lag does not imply that potentialities are limited, and maturation may eventually speed up. Dykman and Ackerman (1976) are less convinced because in their longitudinal studies a "developmental lag" continued well into adolescence.

The issue is complex. Satz, Fletcher, Clark and Morris (1981) describe several different possible models of developmental patterns for learning disabled children which explain how time of onset, rate and end points of development can covary. They feel there is no simple lag or deficit construct; factors such as the age of the sample can determine whether a learning disabled performance is classified as a deficit or delay.

A maturational lag model can be used to explain inadequate performance in many different areas of cortical functioning, such as motor development, attention and cognition (Kinsbourne, 1973). The focus of the present discussion is manual laterality. The unresolved question here is whether reading disability can be caused by a maturational lag, which in turn is reflected by a slowdown in lateralization of manual functions. In their study of handedness in retardates mentioned earlier, Porac, Coren and Duncan (1980) found that retardates were more like preschoolers than their chronological age-mates. Porac et al point out that this could be either a maturational lag or maturational arrest, retardates either progressing at a slower rate, or prematurely ceasing development. Whether there is an ultimate "catch up" phase in a retarded population is debatable, but theoretically possible for those with learning disabilities.

Sparrow and Satz (1970) investigated laterality in nine- to twelve-year-old dyslexic children and found that while early-developing manifestations of laterality, such as hand preference, did not differentiate dyslexics from normals, later developing measures, such as right-left orientation, did. Since these children were older, Satz and Sparrow (1970) put forth a theoretical formulation which predicted that a lag in the maturation of the left hemisphere will differentially delay those skills which are in primary ascendency at a given age. Perceptual-motor skills then, will be delayed in younger children, while language and higher cognitive processes will be delayed in older children, who will have since caught up in perceptual-motor skills. In a test of his hypothesis, Satz found that a lag during kindergarten on certain perceptual-motor tests did discriminate children with learning problems, and consequently disappeared by grade two (Satz, Friel and Rudegeair, 1974). Hand preference however, did not discriminate between the groups at kindergarten age. Other researchers have looked at ambilaterality in young children (Kershner, 1978, DeHirsch, Jansky and Langford, 1966) as a predictor of later learning problems, speculating that if both are signs of a maturational lag, they should be correlated. However neither researcher found confirmation of this hypothesis.

Commenting on DeHirsch et al's negative findings, Satz and Sparrow (1970) suggested that mixed laterality may be greater in groups who are younger, or in populations with more severe maturational delay or neurological damage. Here the distinction between ambilaterality as a sign of neurological damage and as a sign of maturational delay becomes blurred. As was mentioned earlier, a major problem with the maturational delay hypothesis is that some researchers see it linked to, others totally separate from, neuropathology. Clinic populations of dyslexics, with more neurological aberrations, have more non-dextrals, but it is not totally clear whether nondextrality reflects a deficit or maturational delay. As previously noted, it all depends on what age group is sampled. A longitudinal study could shed some light on the matter. Given that the normal course of development is towards increasing dextrality (Porac and Coren, 1981), if nondextrality is sometimes due to patholcgy, then by adulthood these pathological nondextrals should still be nondextrals. However, if nondextrality is a sign of maturational delay, then by adulthood these individuals would have had sufficient time to catch up and would show an even greater rate of change than those developing normally.

This hypothesis rests on the assumption that a maturational lag implies a catch-up phase. As we have seen, there are numerous hypothetical maturational models. One way to differentiate between the lag and deficit models is to say that a deficit model precludes the possibility of a catch-up phase, whereas with a lag model a catch-up is possible. Silver and Hagin (1966) followed up on eighteen of their dyslexic clients, now in early adulthood, and found that ambidexterity persists into adulthood. Although their results indicate that ambidexterity may not mature to definite laterality, their sample was subdivided into "organic" and "developmental" groups of eight and ten subjects respectively, which is probably of insufficient size to detect any other than major changes in handedness. Silver and Hagin's subdivisions were of subjects with classical hard neurological signs and those with no gross clinical findings. It is interesting that they found more ambilaterals among the second group, the "developmental" reading group. Silver and Hagin did not look at developmental soft signs, those normal in young children but pathological in older children, but it is quite possible that their group with no gross findings did have these developmental signs. A longitudinal study with sufficient subjects could possibly find evidence of a maturational "catch-up" in subjects with these developmental signs.

Because of numerous weaknesses, this hypothesis can be only considered exploratory. First of all, there is no evidence that a maturational lag implies a "catch-up" phase. Even if learning disabled individuals remained nondextral into adulthood, it could not be concluded that they are not "maturationally delayed." Secondly, the failure of previous researchers to find any association between handedness and later learning problems could be because these studies were not done with clinical populations, where the severity of disability is likely to be greater, or, alternatively, because any maturational delay reflected in hand preference could have occured when the children were younger but is not present in older children.

Additional problems lie in the rationale for using handedness as a measure of neurodevelopmental immaturity. Many researchers (i.e., Kershner, 1978, Silver and Hagin, 1966) see mixed-handedness, rather than nondextrality, as a sign of immature development of lateralization, for two main reasons. First, following directly from the "developmental soft sign" reasoning, mixed preference patterns are thought to be more characteristic of a younger child. Secondly, less established manual preference is considered evidence of weaker cortical lateralization.

Neither line of reasoning is without its flaws. There is evidence that young ambilaterals most likely change towards dextrality over time (Steffen, 1975). Porac, Coren and Duncan (1980a) however, use both mixed- and left-handedness as signs of possible cortical immaturity because they found that young children are overall less dextral. As was stated before, it is difficult to separate sinistrals from ambilaterals because sinistrals are so inconsistent. It seems most parsimonious to use a very strict definition of sinistrality, considering as left-handed only those with consistent left-hand preferences, so more variable sinistrals will not be considered among the mixed-handers.

As for less-established manual preference patterns being related to weaker cerebral specialization, there is no evidence that ambidexterity per se is related to less complete lateralization (Kinsbourne and Hiscock, 1981). In addition, recent investigators suggest that cerebral specialization may not develop extensively after birth. Cerebral asymmetries of posture and perception are present even in neonates. If the two hemispheres do not progress from a diffuse to highly specialized state, then persisting ambilaterality cannot reflect maturational delay.

Despite many necessary qualifications, the maturational lag hypothesis is worth exploring as an alternative explanation for developmental trends that could present themselves in learning disabled individuals with varying degrees of neurological involvement.

Hypotheses

This investigation will consider the following hypotheses:

1. Nondextrality is increased in learning disabled samples as a function of neuropathological involvement. Children with hard signs of neurological damage have the highest incidence of nondextrality, followed in turn by children with soft signs, and no neurological signs.
2. Nondextrality persists into adulthood. The linear trend of increasing sinistrality with increasing neurological involvement will be present in the same sample of learning disabled children fifteen years later. Those with no neurological signs will be as dextral as normal learners.
3. Because pathological nondextrality is invariant, as they become adults, individuals with no neurological involvement will show a greater tendency to become dextral than those with neurological signs. Indivi-

duals with hard neurological signs will be less likely to become dextrals than those with soft signs.

4. As an exploratory hypothesis, individuals with neurological involvement of the "developmental soft sign" type are likely to show a _greater_ change towards dextrality as they become adults than learning disabled individuals with no neurological signs, because they have "caught up" maturationally.

METHODS

Subjects

The current analysis is part of a larger follow-up study of 203 children who were referred for psychological assessment to the University of Victoria Neuropsychology Clinic because of learning problems. To be included in the follow-up study, a child was required to have had a complete neurological and neuropsychological assessment, a verbal or performance IQ of at least 70, no brain damage acquired after the perinatal period, and no significant sensory impairments or primary emotional disorders. In addition, children included in the study must have been between eight and twelve years of age at time of referral for the first phase of the study, with a minimum of four years having elapsed before follow-up. The age criterion was relaxed to include subjects between seven and thirteen at time of referral, for the second phase of the study.

Subjects were assigned to one of three diagnostic categories, based on the neurological report received at the time of the assessment. Children were classified according to the presence of hard or soft neurological signs. A hard sign was defined as a sign which was considered to be definite evidence of brain damage in seven- to thirteen-year-old children. Soft signs are often considered to be equivocal indicators of brain damage and include both developmental soft signs, such as clumsiness; and minor forms of classical hard signs, such as slight reflex asymmetries or minor choreiform movements of fingers. Appendix 1 gives a list of the hard and soft neurological signs by which subjects were classified.

Based on the neurological examination, children were assigned to one of three categories.

1. Brain dysfunction (group 1): Learning disability in conjunction with one or more hard signs of neurological damage.
2. Questionable brain dysfunction (group 2): Learning disability in conjunction with one or more soft neurological signs.
3. No brain dysfunction (group 3): Learning disability with no hard or soft neurological signs.

A subject with one or more hard signs would be placed in group 1, even though he may have a number of soft signs as well.

A control group of 52 average learners (group 4) was selected from the records of two senior and one junior secondary school in the Greater Victoria School District. Students were included if they had neither experienced learning problems, nor had made exceptionally good progress in elementary school. Subjects were matched with the learning disabled sample for age and sex. The three schools were chosen to represent a mixture of socioeconomic backgrounds to best match the learning disabled groups.

The follow-up study was completed in two phases. Phase one was conducted when the learning disabled group was between 12 and 24 years (mean age 19). At this time, both students and parents were interviewed separately on a number of issues, including school experiences, health problems, and family relationships. In phase one, 48 of the original 203 learning disabled subjects were excluded because they failed to meet the criteria, or were either untraceable, unable or unwilling to participate, leaving 155 subjects at phase one, plus 52 controls.

The second phase was more extensive, including a second interview, a neurological examination, a neuropsychological test battery and the MMPI. By this time the subjects were between 18 and 29 (mean age 24). Of the 155 learning disabled subjects participating in phase 1, 119 participated in phase 2. The others either refused, were deceased, were untraceable, or lived too far away. Twenty-two additional subjects who met the criteria, relaxed to include subjects age seven to thirteen at referral, but who had not participated in phase 1 were added to this group, making a total of 141 learning disabled participants in phase 2. Of the original control subjects, 46 participated, the others either refused, were untraceable, or lived too far away. Five additional control subjects meeting the criteria were selected as replacements.

Procedures

Self-report measures of handedness were obtained from the first interview for each subject. During the phase 1 interview, subjects were asked if they were right- or left-handed and if they have always been so. Seven different responses were possible, depending on direction and degree of preference.[1]

The neuropsychological tests administered in phase 2 assessed a variety of cognitive abilities, including memory, school achievement, general intelligence and motor skills. Many of the tests were chosen because identical or similar versions had been administered in the initial childhood assessment and could provide direct comparison data. Lateral dominance was one of the functions measured at both times. Subjects were given the Lateral Dominance Examination, a revised version of the Harris Tests of Lateral Dominance (Har-

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[1] Strongly right, predominantly right, ambilateral, predominantly left, strongly left, left changed from right, and right changed from left.

ris,1958). The Lateral Dominance Examination is a fourteen item questionnaire, addressing eye, foot and hand preferences. Only the seven questions covering manual preferences were used for analysis. Subjects were asked to show the examiner how s/he would perform various manual tasks, such as cutting with a knife, or using an eraser (see Appendix 2). Each response was scored, giving ten points for a right-hand, five points for a both or either-hand and zero points for a left-hand preference. Total scores range from zero to seventy, with zero referring to an extreme left-hander and seventy an extreme right-hander.

Measures of manual performance, administered with the Lateral Dominance Examination, include a test of grip strength and a test of fine motor speed. To measure grip strength, the subjects were requested to squeeze a Smedley dynamometer as tightly as possible, registering the force exerted in kilograms. After a practice trial, two test trials were given for either hand, subject's score being the average of the two trials. To assess fine motor speed, subjects were asked to write their name with either hand, beginning with the preferred hand. Times for each hand were recorded separately.

For each subject a difference score was calculated, subtracting left hand from right hand performance and dividing by the sum: (r-l)/(r+l). Right minus left hand performance should give an indication of the strength of advantage of the dominant hand. Dividing by the sum reduces the chance that scores will be distorted because of overall intersubject differences in strength or name length.

Not all subjects in the second phase of the study received all three of the tests. Five were unable to write with the nondominant hand. Unavailability or failure of the dynamometer accounted for missing data for thirteen of the subjects, and two were not given all three tests, although no explanation was provided. A total of 171 of the 191 subjects in phase two received all three tests.

As noted earlier, a condition for being in the study, was that each learning disabled subject had received a neuropsychological assessment at the time of initial referral. The nature of the assessment varied according to the needs of the individual and not every child received the same tests. Although many subjects received the Lateral Dominance Examination, only 106 received all measures (e.g., writing time, grip strength, questionnaire). Considering each measure alone, 125 received the questionnaire, 111 the writing time measure, and 136 received the grip strength test. Table 1 shows the number of subjects in each group with all three tests at either the time of referral or at the adult assessment. Also included are the number of subjects with all three tests at <u>both</u> assessments.

TABLE 1

Subject Distribution for Each Assessment

Subjects With All Three Tests At Time Of Referral

Group	1	2	3
N	41	42	23

Total N=106

Subjects With All Three Tests For Adult Assessment

Group	1	2	3	4
N	46	54	24	47

Total N=171

Subjects With All Three Tests For Both Assessments

Group	1	2	3
N	33	34	16

Total N=83

Grip strength, a measure of gross motor strength, is often given in cases of suspected motor weakness, apart from its use as a handedness performance measure. It is not surprising then, that many subjects were administered this test only. The questionnaire and writing time measures are not often administered separately. Fourteen subjects received the lateral dominance questionnaire but not the writing time measure. Of these fourteen, two were hemiparetic, two were spastic hemiparetics and unable to write, three were too uncoordinated to write with the nondominant hand, three did not write their full name on one of the two trials, and for three subjects no explanation was made for missing data. A total of 106 subjects received all three tests.

RESULTS

Hypothesis One

Hypothesis one predicted that nondextrality would increase as a function of probable neuropathology in learning disabled children. Children with hard signs of brain damage (group 1) would be the least dextral, followed in turn by those with questionable brain damage (group 2) and those with no brain damage (group 3). As the following results show, the hypothesis was not supported.

Since handedness may change over time, it would be preferable to divide each category into two or three age groups, analyzing each separately. However, in this sample such a procedure would seriously reduce the number subjects in each cell. Preliminary analyses were performed to determine if it were feasible to collapse across age groups. Univariate analyses of variance were performed for each category with two levels of age (under ten years and ten or older). Only one comparison approached significance. For the brain dysfunction group (group 1), differences on the writing time measure were significant at $p<.065$ ($F=3.58$, $df=1/39$). This value is above the critical value of .05. Therefore, all groups were collapsed across age (see table 2).

TABLE 2

Analysis of Variance for Each Measure by Each Neurological Group

Comparing Older (10-0 to 13-11) with Younger (7-0 to 9-11) Subjects

Test	Group	F Ratio	df	F Prob.
Questionnaire	One	2.82	1/43	.100
	Two	.44	1/48	.508
	Three	.92	1/23	.346
Dynamometer	One	1.17	1/49	.283
	Two	.66	1/55	.420
	Three	.98	1/27	.331
Writing Time	One	3.58	1/40	.065
	Two	.84	1/40	.363
	Three	.54	1/22	.471

A multivariate analysis of variance, using Wilks Lambda, demonstrated no significant effect of category with the three test variables (F=.848, df=6/156, p<.534) at time of referral. The multivariate analysis was followed, post hoc, by individual chi square tests for each variable. Although there were no distributional differences, the possibility remained that classifying performances as right, left or ambilateral may produce frequency differences. For the lateral dominance questionnaire, a stringent classification criterion was employed. Subjects with scores of 70 (all right responses) were considered right-handers, those with scores of 0 (all left reponses) were identified as left-handers, and those with scores of 5 to 65 were considered as ambilaterals. This strict criterion required uniform responses for a classification of nonambidexterity and has been used previously by many researchers (ie, Annett, 1970b, Porac and Coren, 1981). Chi square analysis of subjects with data for all three measures (n=106) did not yield significant group differences (chi square=.195, df=4, p<.995).

For the performance measures, classification of right-, left- and mixed-hand performances was more arbitrary. Unlike the preference scores, which have a J-shaped distribution (Annett, 1970), difference scores of performance measures generally approximate the normal distribution, with the mean shifted towards the direction of a right-hand advantage.

Even those individuals with generally left-handed preferences often show a slight right-hand advantage for performance measures (Annett, 1970). As a criterion for ambilaterality, the mean and standard deviation of the control group at time two was used, and ambilaterality was defined as plus or minus one standard deviation from the mean, right- and left-handedness being all values above and below this range, respectively. Since the control group samples from the general population, they provide a good reference point from which to compare frequencies among the three learning disabled groups. Using those subjects with scores for all three tests (n=106), the chi square for writing time was not significant (chi square=6.40, df=4, p<.171). The criterion for ambilaterality was -.19 through -.65, right-handedness was set at -.65 through -1.0 and left-handedness at -.19 through +1.0. With the writing time measure a smaller right than left hand score indicates a right hand advantage, the formula (r-l)/(r+l) producing right-hand advantage values in the extreme negative range and left-hand advantage values in the positive range.

The same procedure was followed for the dynamometer, producing a marginally significant chi square (chi square=8.98, df=4, p<.061). Based on the mean and standard deviation of the control group, the criterion for ambilaterality was -.047 through .13, right-handedness was .13 through +1.0 and left-handedness -1.0 through -.047. Unlike the writing time measure, a smaller right than left hand score indicated a left hand advantage, so the signs are reversed. Examination of means (table 3) suggests that group 2 is less right-handed and more ambilateral than either group 1 or 3.

A second chi square was performed using an absolute criterion; a difference score greater than zero indicating a right hand advantage, a score below zero indicating a left hand advantage, and zero being ambilateral. This chi square was not significant for either the writing time (chi square=5.58, df=4, p<.23) or the dynamometer (chi square=2.3, df=4, p<.67) measures. Although for the dynamometer the first chi square was marginally significant, many of those who were ambilateral in the first analysis were considered right-handers in the second analysis, producing no group differences (see table 4).

TABLE 3

Chi Square for Dynamometer at Time of Referral

Criterion for Ambilaterality is Plus or Minus One Standard Deviation from the Mean of the Control Group

	Right	Ambilateral	Left
Group One	22.0% n=9	56.1% n=23	22.0% n=9
Group Two	2.4% n=1	81.0% n=34	16.7% n=7
Group Three	17.4% n=4	69.9% n=16	13.0% n=3

Total N=106

Raw Chi Square=8.98 df=4 p<.061

Right=Difference Score of .13 to 1.0
Ambi=Difference Score of -.05 to .13
Left=Difference Score of -1.0 to -.05

TABLE 4

Chi Square for Dynamometer at Time of Referral

	Right	Ambilateral	Left
Group One	58.5% n=24	14.6% n=6	26.8% n=11
Group Two	54.8% n=23	21.4% n=9	23.8% n=10
Group Three	69.6% n=16	8.7% n=2	21.7% n=5

Total N=106
Raw Chi Square=2.31 df=4 p<.678

Right=Difference Score Greater than Zero
Ambi=Difference Score Equal to Zero
Left=Difference Score Less than Zero

Hypothesis Two

Hypothesis two predicted that: a) the same trend of increasing nondextrality with increasing likelihood of neuropathology would be present at adulthood; b) when compared to a group of normal controls, group 3 would be equally dextral, with groups 2 and 1, respectively, more sinistral. At time two then, group 1 would be most sinistral, group 2 less sinistral, and groups 3 and 4, equally, the least sinistral.

The first part of the hypothesis was tested with a multivariate analysis of variance, using those subjects with all test scores both at referral and at time two (n=83). With this population there were no significant group differences with the three tests at referral (F=.848, df=6, p<.564) or at time two (F=1.34, df=6/156, p<.240). There was no apparent trend of increasing dextrality at either time. As an additional post hoc analysis, a chi square was performed for each test and time of administration. Using the same criterion as in hypothesis one, there were no group differences for either the questionnaire or performance measures.

Part B of the hypothesis was also tested with a multivariate analysis of variance, using learning disabled (n=124) and control (n=47) subjects with all three tests at time two (total n=171). A multivariate analysis of variance for all four groups was significant (F=2.21, df 9/401, p<.020). Examination of univariate tests yielded a significant effect of the grip strength measure (F=3.04, df=3/167, p<.030). Subsets of groups whose means do not differ significantly were determined by the Student-Neuman-Keuls procedure. Group 1 and group 2 were placed in different groups, group 2 being significantly less dextral on this measure than group 1. For the other two measures, univariate analyses were not significant (table 5).

Post hoc analysis of frequencies by a chi square test proved nonsignificant, except for the dynamometer test (chi square=13.42, df 6, p<.036). Using the range of scores one standard deviation to either side of the mean of group 4 as a criterion for ambilaterality, group 3 is more ambilateral and less left-handed than any of the other groups, group 1 more right-handed and less ambilateral, and group 2 more left-handed and less right-handed (table 6). Using the criterion of difference scores greater than, equal to, or less than zero representing right-, mixed-, and left-hand performances respectively, the chi square (table 7) is no longer significant (chi square=7.75, df=6, p<.25).

Although the other univariate tests were not statistically significant, the results were meaningful. Using a less strict criterion for left- and right-handedness,[2] the per-

[2] Left defined as scores of 0 through 10, right defined as

TABLE 5

Multivariate Analysis of Variance For Adult Assessment

Effect	Test	Value	Approx. F	Hypoth. df	Error D.F.	Sig. of F
Group	Wilks	.888	2.217	9.0	401.72	.020

Univariate F-Tests from Multivariate Analysis

Variable	Hypoth. SS	Error SS	Hypoth. MS	Error MS	F	Sig. of F
Questionnaire	1339.79	67728.62	446.60	405.56	1.10	.350
Writing Time	.606	17.604	.202	.105	1.92	.128
Dynamometer	.065	1.206	.021	.077	3.04	.030

Dynamometer Measure
Student-Neuman-Keuls Procedure

Homogeneous Subsets

Subset 1

Group	2	4	3
Mean	.012	.043	.050

Subset 2

Group	4	3	1
Mean	.043	.050	.063

TABLE 6

Chi Square for Dynamometer at Adult Assessment

Criterion for Ambilaterality is Plus or Minus One Standard Deviation from the Mean of the Control Group

	Right	Ambilateral	Left
Group One	21.7% n=10	67.4% n=31	10.9% n=5
Group Two	3.7% n=2	83.3% n=45	13.0% n=7
Group Three	8.3% n=2	91.7% n=22	0.0% n=0
Group Four	8.5% n=4	85.1% n=40	6.4% n=3

Total N=171
Raw Chi Square=13.42 df=6 p<.036

Right=Difference Score of .13 to 1.0
Ambi=Difference Score of -.047 to .13
Left=Difference Score of -1.0 to -.047

TABLE 7

Chi Square for Dynamometer at Adult Assessment

	Right	Ambilateral	Left
Group One	69.6% n=32	8.7% n=4	21.7% n=10
Group Two	59.3% n=32	9.3% n=5	31.5% n=10
Group Three	82.3% n=20	4.2% n=1	12.5% n=3
Group Four	78.7% n=37	2.1% n=1	19.1% n=9

Total N=171
Raw Chi Square=7.75 df=6 p<.256

Right=Difference Score Greater Than Zero
Ambi=Difference Score Equal To Zero
Left=Difference Score Less Than Zero

centage of left-handers, as measured by the lateral dominanace questionnaire, resembled trends reported in the literature. Groups 1, 2 and 3 were equally left-handed, with 9% fewer left-handers in group 4 (see table 8).

In a separate analysis of writing hand preference, as determined by the lateral dominance questionnaire, the percentages of normal and learning disabled left-handers closely resembled the 8% and 17% figures noted by Satz (1972) for normal and pathological populations, respectively. Based on self-report of writing hand, all learning disabled groups are substantially more left-handed than controls (see table 9). Pooling all three learning disabled groups, there are 16.1% left-handers among the disabled learners and 6.4% among the controls. That the analysis was not significant could be because the effect of handedness was too small to be significant in a sample of this size.

This trend was also apparent in the writing time measure, with a chi square analysis, identifying left-handers as those with difference scores falling below one standard deviation of the mean for the control group on this measure. Use of the control group distribution as a criterion for handedness provided a built-in comparison between normal and pathological groups. The learning disabled groups were all substantially, although not significantly, more left-handed

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scores of 60 through 70

TABLE 8

Lateral Dominance Questionnaire at Adult Assessment

Percentages of Right-, Mixed-, and Left-Handers

	Right	Ambilateral	Left
Group One	82.6% n=38	6.5% n=3	10.9% n=5
Group Two	83.3% n=45	5.6% n=3	11.1% n=6
Group Three	79.2% n=19	8.3% n=2	12.5% n=3
Group Four	93.6% n=44	4.3% n=2	2.1% n=1

Total N=171
Raw Chi Square=4.36 df=6 p<.627

Pooling Groups One, Two and Three

	Right	Ambilateral	Left
Disabled Learners	82.3% n=102	6.5% n=8	11.3% n=14
Controls	93.6% n=44	4.3% n=2	2.1% n=1

Raw Chi Square=4.05 df=2 p<.131

TABLE 9

Self-Report of Writing Hand at Adult Assessment

Percentages of Right- and Left-Handers

	Right	Left
Group One	82.9% n=38	17.4% n=8
Group Two	85.2% n=46	14.8% n=8
Group Three	83.3% n=20	16.7% n=4
Group Four	93.6% n=44	6.4% n=3

Total N=171
Raw Chi Square=2.92 df=3 p<.402

Pooling Groups One, Two, and Three

	Right	Left
Disabled Learners	83.9% n=104	16.1% n=20
Controls	93.6% n=44	6.4% n=3

Corrected Chi Square=2.00 df=1 p<.156

on this measure (see table 10). Pooling all three learning disabled groups yielded marginal significance, with almost 15% more left-handed learning disabled subjects than controls.

Using the criterion of difference scores greater than, equal to, or less than zero as denoting right, mixed, and left-hand preferences respectively, the same pattern exists. All learning disabled groups contain two to three times as many left-handers as the control group at time two (see table 11). Pooling the learning disabled groups, the chi square is still not significant, but overall there are 10% more left-handers among the disabled learners.

Self-report of handedness was available from the questionnaire administered when the subjects were all approximately nineteen years old. Using that subgroup of subjects for whom both the self-report data and time two test data were available (n=157), a chi square analysis was performed. Subjects were asked which hand they preferred, responses coded according to the seven criteria noted earlier. No subjects reported having switched their handedness. The chi square was highly significant (see table 12) (chi square=27.1, df=12, p<.007). Examination of the chi square table revealed that more subjects in group 4 were strongly right-handed, and fewer predominantly right-handed, than in the other groups. In fact, collapsing these two right-handed

TABLE 10

Writing Time at Adult Assessment

Percentages of Right-, Mixed-, and Left-Handers

Criterion for Ambilaterality is Plus or Minus One Standard Deviation from the Control Group Mean

	Right	Ambilateral	Left
Group One	6.5% n=3	65.2% n=30	28.3% n=13
Group Two	9.3% n=5	75.9% n=41	14.8% n=8
Group Three	12.5% n=3	66.7% n=16	20.8% n=5
Group Four	8.5% n=4	85.1% n=40	6.4% n=3

Total N=171
Raw Chi Square=9.04 df=6 p<.171

Pooling Groups One, Two, and Three

	Right	Ambilateral	Left
Disabled Learners	8.9% n=11	70.2% n=87	21.0% n=26
Controls	8.5% n=4	85.1% n=40	6.4% n=3

Raw Chi Square=5.30 df=2 p<.070

Right=Difference Score of -1.0 to -.65
Ambi=Difference Score of -.65 to -.19
Left=Difference Score of -.19 to +1.0

TABLE 11

Writing Time at Adult Assessment

Percentages of Right- and Left-Handers

	Right	Left	(No Ambilaterals Reported)
Group One	78.3% n=36	21.7% n=10	
Group Two	88.9% n=48	11.1% n=6	
Group Three	83.3% n=20	16.7% n=4	
Group Four	93.6% n=44	6.4% n=3	

Total N=171
Raw Chi Square=5.19 df=3 p<.157

Pooling Groups One, Two, and Three

	Right	Left
Disabled Learners	83.9% n=104	16.1% n=20
Controls	93.6% n=44	6.4% n=3

Corrected Chi Square=2.00 df=1 p<.156

Right=Difference Score Less Than One
Ambi=Difference Score Equal To One
Left=Difference Score Greater Than One

classifications yields a nonsignificant chi square (chi square=13.6, df=9, p<.135). It seems that the frequency differences apparent in the chi square are due more to a greater number of right-handers with strong, rather than weak, preferences among group 4, than to fewer left handers in this group.

With the interview data, as with the other measures, differences between the clinical and control populations are meaningful, if not significant. Pooling subjects with strong and weak right-hand preferences, and those with strong and weak left-hand preferences, the chi square is still nonsignificant (chi square=4.83, df=3, p<.184), but the relative proportions are meaningful. All three clinic groups were two to three times as left handed as the control group (see table 13). Comparing the disabled learners as a whole to control subjects, there are 9% more left-handers among the disabled learners, a proportion comparable to that found with the other measures.

Although there is no control group for subjects at time of referral, percentages of left-handers on the lateral dominance questionnaire can be compared with the percentage of left-handed control subjects. This entails comparing adult scores with child scores, but since a post-hoc repeated measures analysis revealed no effect of time for this measure, the comparison can be considered, albeit with caution. Us-

TABLE 12

Chi Square of Self-Reported Handedness

	Strong Right	Predom. Right	Ambi.	Predom. Left	Strong Left
Group One	54.1% n=20	27.0% n=10	5.4% n=2	5.4% n=2	8.1% n=3
Group Two	67.9% n=36	17.0% n=9	1.9% n=1	3.8% n=2	9.4% n=5
Group Three	65.0% n=13	10.0% n=2	0.0% n=0	0.0% n=0	25.0% n=5
Group Four	91.5% n=43	2.1% n=1	0.0% n=0	4.3% n=2	2.1% n=1

Raw Chi Square=27.146 df=12 $p<.007$

TABLE 13

Chi Square of Self-Reported Handedness

Pooling Strong Right with Predom. Right,
Strong Left with Predom. Left

	Right	Ambilateral	Left
Group One	81.1% n=30	5.4% n=2	13.5% n=5
Group Two	84.9% n=45	1.9% n=1	13.2% n=7
Group Three	75.0% n=15	0.0% n=0	25.0% n=5
Group Four	93.6% n=44	0.0% n=0	6.4% n=3

Raw Chi Square=8.229 df=6 p<.221

Pooling Groups One, Two, and Three

	Right	Ambilateral	Left
Disabled Learners	81.8% n=90	2.7% n=3	15.5% n=17
Controls	93.6% n=44	0.0% n=0	6.4% n=3

Raw Chi Square=3.94 df=2 p<.139

ing both a strict and a loose criterion for left- and right-handers, there are, respectively, seven and twelve percent more left-handers overall, among disabled learners. (see table 14 and table 15).

TABLE 14

Lateral Dominance Questionnaire

LD Subjects at Time of Referral Compared with Adult Controls
(Strict Criterion for Questionnaire)

	Right	Ambilateral	Left
Group One	75.6% n=31	14.6% n=6	9.8% n=4
Group Two	73.8% n=31	16.7% n=7	9.5% n=4
Group Three	78.3% n=18	13.0% n=3	8.7% n=2
Group Four	80.9% n=38	17.0% n=8	2.1% n=1

Total N=153
Raw Chi Square=2.81 df=6 p<.832

Pooling Groups One, Two, and Three

	Right	Ambilateral	Left
Disabled Learners	75.5% n=80	15.1% n=16	9.4% n=10
Controls	80.9% n=38	17.0% n=8	2.1% n=1

Raw Chi Square=2.61 df=2 p<.270

Right=70 Ambilateral=5 to 65 Left=0

TABLE 15

Lateral Dominance Questionnaire

LD Subjects at Time of Referral Compared with Adult Controls
(Loose Criterion for Questionnaire)

	Right	Ambilateral	Left
Group One	78.0% n=32	12.2% n=5	9.8% n=4
Group Two	73.8% n=31	7.1% n=3	19.0% n=8
Group Three	87.0% n=20	0.0% n=0	13.0% n=3
Group Four	93.6% n=44	4.3% n=2	2.1% n=1

Total N=153
Raw Chi Square=11.38 df=6 $p<.077$

Pooling Groups One, Two, and Three

	Right	Ambilateral	Left
Disabled Learners	78.3% n=83	7.5% n=8	14.2% n=15
Controls	93.6% n=44	4.3% n=2	2.1% n=1

Raw Chi Square=5.96 df=2 $p<.050$

Right=60 to 70 Ambilateral=15 to 55 Left=0 to 10

Hypothesis Three

Hypothesis three predicted that over time individuals would become more dextral as an inverse function of the probability of neuropathology. Subjects with hard signs (group 1) would show the least change to dextrality, those with soft signs (group 2) and no signs (group 3) showing, respectively, increasing amounts of dextrality. The following results show that this hypothesis was not supported.

This hypothesis was explored in two different ways: examining both magnitude and incidence of dextral shift among the three groups. For each subject a difference score was calculated for each test, which represented the difference between test performance at referral and time two.

To determine if differences between the groups exist in the magnitude of change, a multivariate analysis of variance was performed on the difference scores of subjects receiving all tests at both times (n=83). The multivariate analysis was not significant ($F=1.23$, $df=6/156$, $p<.291$). A second multivariate analysis using only those subjects for whom change scores on all three tests were in the direction of dextrality, could not be performed, due to the small number of subjects.

To determine if differences between the groups exist in frequency of change towards dextrality or sinistrality, irrespective of degree, a chi square was performed for each test, using only those subjects with difference scores for all three tests. Dividing subjects according to direction of change (towards dextrality, no change, towards sinistrality) did not yield significant differences. Chi square tests for the questionnaire (chi square= 3.42, df=4, p<.489), writing time measure (chi square=5.31, df=4, p<.256) and dynamometer (chi square= 1.95, df=4, p<.743) were all nonsignificant.

Since the expected trend was not apparent, a post hoc repeated measures analysis of variance was performed on each test to see whether subjects actually did become more dextral on any of the tests over time, irrespective of group differences. Those subjects with scores on all three tests at both referral and time two were used for the analysis (n=83), so that direct comparisons with the multivariate change score analysis could be made.

For the writing time measure the time effect was highly significant (F=20.77, df=1/80, p<.000), but neither the effect of group (F=1.15, df=1/80, p<.320) or the group by time interaction (F=.351, df=1/80, p<.750) were significant. Examination of group means indicates that all groups become increasingly dextral over time on the writing time measure (see table 16).

TABLE 16

Repeated Measures Analysis for Writing Time

Source	SS	DF	MS	F	Sig. of F
Group	.094	2	.047	.23	.794
Time	.653	1	.653	20.77	.000
Group and Time	.049	2	.024	.79	.456

Group Means
(larger negative value
indicates more dextrality)

	Time of Referral		Adult Assessment	
Group One	-.135	s.d.=.31 n=33	-.303	s.d.=.37 n=33
Group Two	-.210	s.d.=.32 n=34	-.313	s.d.=.35 n=34
Group Three	-.234	s.d.=.29 n=16	-.320	s.d.=.38 n=16

For the dynamometer measure, the group (F=1.15, df=1/80, p<.320), time (F=.004, df=1/80, p<.950) and group by time interaction comparisons (F=.351, df=2/80, p<.705) were all nonsignificant. This was also the case with the questionnaire measure, where the effects of group (F=.385, df=1/80, p<.68), time (F=.349, df=1/80, p<.556), and the group by time interaction (F=2.14, df=1/80, p<.124) were, again, nonsignificant.

Hypothesis Four

Hypothesis four is an exploratory hypothesis, postulating that subjects with soft signs (groups 1 and 2) might actually become more dextral, if nondextrality can be considered as a developmental soft sign which is eventually outgrown. Since only five out of twenty-four of the possible soft signs can be considered "developmental," not every child in group 1 or 2 exhibits developmental soft signs. Likewise, a subject in group 1 may, but does not invariably, have some soft neurological signs. Hence, this is not a pure test of the hypothesis and as such is considered exploratory. Since previous analyses, noted earlier, indicate that no differences exist between younger and older children (ages 7-0 to 9-11, and 10-0 to 13-11) the pooled scores at time of referral were used.

Examining the change score MANOVA for hypothesis 3, it would be anticipated that the change scores would have shown a different pattern. Groups 1 and 2 should actually show more change to dextrality than group 3. However, as noted earlier, both multivariate and chi square analyses were non-significant. The hypothesis was not supported.

DISCUSSION

The expectation (Hypothesis 1) that incidence of nondextrality in learning disabled children would increase as a function of degree of neuropathology was not confirmed; children with hard, soft, or no neurological signs were equally nondextral. Nor were there group differences at young adulthood, fifteen years later (Hypothesis 2a). The dynamometer did produce group differences, but not in the expected direction. Although there is some question as to the validity of the two performance measures--the dynamometer as a handedness performance measure in general and writing time as a measure for learning disabled populations---failure to find group differences in the lateral dominance questionnaire confirms the negative findings with the performance measures.

The comparison of learning disabled subjects at time two with controls, however, yielded a meaningful, if not statistically significant, result. Learning disabled subjects as a whole were more sinstral than controls. Sinistrality is greater among clinic-referred learning disabled subjects irrespective of degree of neuropathology.

It was anticipated that from middle childhood to adulthood subjects would become more dextral as an inverse function of neuropathology (Hypothesis 3). Pathological left-handers should be less likely to show the usual developmental trend towards dextrality. There were no group differences in either direction or magnitude of manual shift. Only on the writing time measure did subjects, irrespective of group membership, show the anticipated shift towards dextrality. Because there was no control group for this analysis, it is not known whether the dextral shift would even be apparent in a normal population over this time span, or if possible confounds in the scoring method masked the effect. However, failure to find group differences is corroborated by the lack of group differences at either time of referral or at the adult assessment.

Examination of change scores (discussed in Hypothesis 3) revealed that subjects with developmental soft signs (groups one and two), who might be expected to go through a developmental "catch up" phase, did not show more change towards dextrality (Hypothesis 4) than group 3. Since soft signs as a whole did not show any improvement, it is not surprising that these subjects failed to develop more mature, that is, more dextral, handedness.

Learning Disabled versus Control Subjects

The comparison of learning disabled subjects at time two with normal controls, was meaningful, if not statistically significant. The relative proportions of learning disabled and control subjects identified as left-handers on a number of measures, closely paralleled the figures of seventeen and eight percent, for pathological and normal populations respectively, noted by Satz (1972).

Satz's figures were based on data from epileptic and retarded populations with known neuropathology. In the current study, learning disabled subjects were more sinistral than controls irrespective of neuropathology. Studies which have found more sinistrality among learning disabled populations vary in the exact percentages of sinistrality reported because of varying assessment procedures. Nonetheless, the current study found about ten percent more sinistrals among the learning disabled subjects, an average difference similar to most studies.

The exact percentages of left-handers varied from test to test, but the relative surplus among the learning disabled group remained consistent, with about ten percent more left-handers among the learning disabled subjects. Even with a moderately large sample size (n=171), the effect does not appear to be strong enough to reach statistical significance. However, since the relative percentages of learning disabled and control subjects so closely parallel previously reported figures, this finding amounts to a replication of previous work. Learning disabled subjects in a clinic sample, as young adults, are less dextral than their nondisabled counterparts, irrespective of degree of neuropathology.

The analysis of the self-report measure of handedness was statistically significant. Group differences between learning disabled and control subjects in degree of right-handedness were found; control subjects were much less likely to be weak right handers. Given a greater number of subjects in the two right-handed cells of the chi square, it is not surprising that significance was achieved on the basis of these two classifications, rather than on the basis of a right-hand/left-hand comparison. This finding indicates that if there had been more subjects, a right-hand/left-hand comparison might also have been significant. Theoretically, it is not surprising that right-handed learning disabled subjects are less strongly right-handed than the control subjects. Using a strict criterion for right-handedness, these weak right-handers would be considered ambilaterals. This result fits in with previous findings of more mixed-handedness among the learning disabled (i.e., Harris, 1957).

It is unfortunate that there was no control group at time of referral, so that relative percentages of sinistral learning disabled and nondisabled children could be discussed. However, comparing questionniare scores cf the learning disabled children at time of referral with time two scores of adult controls, there are an average of seven to thirteen percent more left-handers among the learning disabled sample, depending upon the criterion used. Since it would be preferable to use scores of normal children for comparison, only tentative conclusions can be made, but the trend supports the adult findings.

Performance Measures

Returning to the first hypothesis, although the overall multivariate analysis of subjects at time of referral was not significant, the dynamometer measure considered independently in a chi square analysis does produce marginally significant group differences. During the adult assessment, the effect was even stronger, yielding a significant multivariate analysis due exclusively to the dynamometer measure. Examination of means indicates that group 2 was significantly less dextral on this measure than group 1 at both assessments.

Although significant, within the context of manual preferences this result is not very meaningful. From the hypothesis, group 1 would be expected to be less dextral than group 2. These findings are not surprising, considering the dynamometer's poor record as a measure of lateral preference. Reliability over fifteen years, in a separate study of this same population, was only .365 (right hand) and .362 (left hand), two of the poorest reliability coefficients of any test in this population (Sarazin and Spreen, 1983). In a factor analytic study of handedness measures, Barnsley and Rabinovitch (1970) found that the dynamometer has one of the lowest factor loadings (.38) of any test considered. The dynamometer is generally used as a measure of gross motor strength. These unexpected group differences could be due to a number of factors unrelated to lateralization of manual performance per se. That the writing time measure, with a much higher (.83) factor loading (Barnsley and Rabinovitch, 1970) does not produce such group differences, lends credibility to the belief that the dynamometer may not be appropriate as a manual performance measure.

Although the writing time measure may be a better performance handedness measure, a note of caution is necessary. Writing speed may be confounded with the nature of the learning disability; those with developmental agraphia may show a less lateralized performance than those for whom writing one's name is a well-learned skill. With a learning disabled population, a non-academic dexterity measure, such as a peg-moving task, may be more valid. Although the writing time measure should be considered with reservations, the trends observed were confirmed by the lateral dominance questionnaire.

Performance Criteria

Handedness is a continuum, but for purposes of discussion it is often convenient to trichotomize scores into left-, right-, and mixed-hand performances. As noted earlier, the choice of critera should be based on the theoretical question posed. In this study it was not important to define exact percentages of left- right- and mixed-handers, but rather to examine the learning disabled groups relative to each other and to the nondisabled population. Two different criteria for handedness were used. The first utilized the distribution of control subjects to define cutoff scores, while the second used an absolute criterion, considering only the direction of the difference scores. The first criterion, although forcing the proportion of right- and left-handers in the control group to be approximately equal, did allow for examination of group frequency differences relative to the distribution in a nondisabled sample. Using this criterion, the chi square for the dynamometer measure at time two corroborated the univariate test findings.

Although the second criterion is a purer reflection of actual performance differences, it was not sensitive to group differences on the dynamometer measure. That self-proclaimed ambilaterals often demonstrate a right-hand advantage, so that group means are skewed to the right, is not considered. The first criterion, although arbitrary, seems both conceptually appropriate and adequate for detecting group frequency differences.

Sex Differences

Ideally, in a follow up study all subjects should have scores for all measures at both times to protect against biases produced by subject loss. In the current study this was not possible; a subgroup of subjects with scores on all tests at both times was needed to examine group trends over time. A number of biases could be present in this subgroup. Previous studies suggest that males may be more sinistral than females (ie, Smart, Jeffrey and Richards, 1980). Although the ratio of males to females in the two larger groups of subjects, with all three scores at either time of referral or at time two, was the same across groups, this was not true for the smaller subgroup, with all three scores at both assessments. In this group there were a disproportionatly large number of females in group three.[3] Fortunately, this bias in the data should serve to strengthen the anticipated trend. Group three should be comparatively more dextral than the other groups because of either the predominance of females or the paucity of pathological left-handers. Since this trend was not noted, the possible confound can be dis-

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[3] Group three was fifty percent female, whereas groups one and two were each twenty-five percent female.

missed.

Subject Loss

Selective loss of subjects can arise from external factors, such as equipment failure or experimental error, or from subject factors, such as refusal to participate or an inability to complete a test. Inability to complete a test is most likely to introduce a bias into the data, excluding those subjects who are more impaired. Four subjects at time of referral and six subjects at time two were excluded because they were unable to write with their nondominant hand and could not complete the writing time test. The restricted sample of subjects with all tests at both times excludes all of these subjects, of which only one was excluded at both times. According to Bishop (1980) these subjects, with a hypofunction of the nondominant hand, could likely be pathological left-handers. It is unfortunate that writing time, a difficult test for some, was selected as a performance measure. A better measure would be one that could be performed even with substantially reduced functioning of the nondominant hand.

Change Scores

Analysis of change scores revealed no group differences in either magnitude or frequency of dextral shift. Given the failure to find group differences at both time of referral and young adulthood, these results are not surprising. However, change scores may have been biased due to the nature of the difference scores. Difference scores (r-l/r+l), calculated so that the difference between hands was divided by the sum of the two hands, attempted to compensate for individual differences in overall ability. However, when considering a single subject over time, increases in ability with either hand (such as grip strength) may not increase in proportion to right- minus left-hand performance differences. Over time, increased muscular development results in a higher performance baseline, irrespective of lateral performance differences. Since the difference score is computed by dividing by the total performance, a larger value in the denominator results in a smaller difference score; with a right-hand advantage this means a score closer to sinistrality. Since most subjects have a right-hand advantage on performance measures, overall change scores may be biased by a possible tendency for adult scores to be more in the direction of sinistrality than childhood scores.

For the writing time measure, at least, this was not a problem. Repeated measures analysis of variance demonstrated a highly significant dextral shift over time. Considering the differential amount of writing practice acquired by the dominant hand, generally right, this is not a surprising result. However, for the dynamometer measure, this may have been a confound, for, as noted earlier, there were not enough subjects switching towards dextrality on all three measures to perform a multivariate analysis.

Ceiling effects of the questionnaire measure may also help explain why so few subjects became more dextral; with only seven handedness questions, many manual tasks were excluded. Even if an individual receiving a score of 70 on the questionnaire developed an additional right-hand preference, if this task was not included in the questionnaire, the subject's score will not reflect this increase in dextrality.

Although the failure to find group differences at either childhood or adulthood indicates that this possible built-in sinistral shift probably did not affect the results, it is important to note its biasing effect in developmental studies, in light of the popularity of this method of calculating difference scores.

No dextral shift was noted for any of the groups, except on the writing time measure which was probably contaminated by practice effects. It is unfortunate that change scores for the control population were not available. Without a control population it is impossible to say whether the dextral shift was just not apparent over a moderately short time interval, or if the possible sinistral bias in computing difference scores, and possible ceiling effect of the questionnaire, disguised any dextral change.

Maturational Lag

Change scores were analyzed, not only to see if pathological left-handers would remain left-handed, but to determine if those with developmental soft signs would "catch up" and become more dextral. Although mixed-handers have been the focus of most maturational delay studies, the current method of analysis looks at relative shifts towards dextrality among all subjects. A separate analysis of mixed-handers would seriously reduce the number of subjects. Although a maturing mixed-hander can become more sinistral, since most individuals are right-handed, it was assumed that for the majority the shift would be towards dextrality, hence the focus of this analysis. The hypothesis was not supported for a number of possible reasons.

As Satz and Sparrow (1970) noted, mixed laterality may be greater in groups who are younger, or in populations with neurological damage. The first part of this hypothesis was not examined. Although analyses of subjects under and over age ten at time of referral showed no differences in degree of nondextrality, Satz and Sparrow were referring to children under five, who were not the focus of this study. Group differences may exist in much younger subjects.

Alternatively, a maturational lag may not precede a catch up phase. Soft signs in general, and developmental soft signs in particular, may not indicate a neurological status which eventually matures. In a separate analysis of the same learning disabled population, Hern, Simpson and Spreen (1983) found that soft signs do not necessarily disappear over time; in fact, occurence of soft signs increased. Of the five developmental soft signs,* only one showed any improvement after fifteen years. It is not surprising then, that groups one and two did not become significantly less dextral. If nondextrality arises from a state of neurological immaturity, it is not likely to improve over time if other soft neurological signs persist. That at adulthood such large differences between the percentage of sinistrals in the learning disabled group as a whole and the control group were noted, is a further indication that no catch up phase occurs.

The maturational lag concept may still be viable if, as some researchers (ie, Thompson, 1971) suggest, the lag is not due to neurological factors. In this case, dividing sub-

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* Synkenesia, Incoordination, Disdidcdochckinesia, Simultagnosia, Graphaesthesia

jects according to neurological status will not produce group differences. Perhaps some other variable can better distinguish maturationally lagging subjects from those in whom no delay exists.

Conclusions

There were proportionately fewer sinistrals among the controls than among clinic-referred learning disabled subjects. Although this finding is not unexpected, the reasons for it are unclear. It was anticipated that neuropathology, being more common in clinic than general populations, could account for reports of more learning disabled sinistrals in the former population, but not in the latter.

The concept of pathological left-handedness is well established for populations with hard neurological damage (ie, Satz, 1972b, Satz et al, 1979) and has been demonstrated among those with soft neurological signs (Nichols and Chen, 1981). Although it was expected that nondextrality would increase as a function of degree of neuropathology, this was not confirmed. Even the subjects in group 3, with no neurological signs, were as nondextral as their counterparts with neurological signs.

There are a number of possible explanations. As stated earlier, since this is a clinical population, perhaps nondextrality served as a "warning sign" for teachers and physicians, possibly leading to more frequent referrals. Although in the past ten years this practice seems to have become less popular, these subjects were seen an average of fifteen years ago, when this bias may have affected the subject selection. A replication on a similar population referred more recently might answer this question.

Alternatively, the possibility exists that neurological damage, irrespective of degree, might be a contributing factor. Although subjects in group 3 had no soft or hard neurological signs at the time of referral, this does not preclude the possibility that they could have incurred some mild neurological damage. An analysis of the persistence of neurological signs, performed by Hern, Simpson and Spreen (1983), indicated that subjects in group 3 did not remain without neurological signs. When they reached young adulthood, 72.5 percent of these subjects had soft signs and 11.8 percent had hard signs of neurological damage. Although neurological status changes, the percentage of sinistrals, as measured by the questionnaire, does not increase, remaining as high as in the other two groups at both times.[5] It is possible that subjects in this group suffered from some mild neurological damage which went undetected at the time of the first examination, reflected in the high incidence of sinis-

[5] Using a strict criterion there are 12% sinistrals at both times. With a more relaxed criterion, there are 18% at the time of referral and 12% at time two.

trality at both times.

That sinistrality increases as a function of decreasing cortical integrity has been demonstrated in retarded populations, with a much greater amount of neuropathology. Grouping severely retarded with profoundly retarded, and mildly retarded with moderately retarded subjects, Hicks and Barton (1975) found less dextrality in the more severely disabled. Differences in dextrality between groups one and two of the current study may not be detectable, because differences in the amount of neuropathology is not as extreme as in the Hicks and Barton study.

Subjects in group 3 may have suffered some mild damage which was not severe enough to be detected until adulthood. They too, may have suffered just enough damage to be prone to a pathological switching of handedness. In a normal population sinistrality does not seem to increase as a function of possible mild perinatal damage (Mc Manus, 1981). With a learning disabled group, including those with no overt neurologial signs, the possibility of subtle neurological damage may be greater, especially since a substantial portion of these subjects later manifest neurological signs. It is still not known exactly how much damage is needed to produce pathological left-handers, but the amount may be quite small.

In conclusion, these results must be interpreted with caution. Although the percentages are similar to those found in other studies, there were no significant differences between disabled and normal learners in incidence of sinistrality on any of the tests. The exact percentages vary from test to test, but on all but the dynamometer measure the difference of about ten percent between learning disabled and control subjects is maintained. Overall, the results suggest that there are fewer dextrals among learning disabled subjects in a clinic population, irrespective of degree of neuropathology, and that these differences are maintained over time.

That clinic-referred learning disabled subjects are more sinistral, regardless of overt neuropathology, must be confirmed in another population. A larger sample size is needed; the base rate of sinistrality in the general population is so low that many subjects are needed to detect significance. The clinic population should consist of children referred recently, by physicians and teachers who do not consider nondextrality to be a "warning sign" of pathology. A valid, non-academic performance measure of handedness, which can be performed even by those with substantially reduced functioning, such as a peg moving task, should be used so that those with possible hypofunctioning of the nondominant hand can be detected. If the previous findings are confirmed and sinistrality is higher among all three learning disabled groups, then it can be postulated that small amounts of neuropathology are needed to cause pathological left-handedness, but that large amounts are needed before pathological left-handedness increases significantly. To study the persistence of nondextrality into adulthood, to see if pathological left-handedness impairs normal maturation, it would be necessary to first confirm the dextral shift in a normal population. Given an adequate difference score formula, or, ideally, a performance measure unaffected by developmental trends, the persistence of pathological left-handedness can be established.

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APPENDIX 1

Hard and Soft Neurological Signs

		Hard Signs	Soft Signs
1.	Ataxia	Marked	Slight
2.	Asymmetry of Skull or Limbs	soft sign only	
3.	Anosmia	Marked	Slight
4.	Visual Field Defect	hard sign only	
5.	Diplopia	hard sign only	
6.	Strabismus	soft sign only	
7.	Saccadic Movements	soft sign only	
8.	Nystagmus	Definite, bilateral	Unsustained, unilateral
9.	Dysarthria	Marked	Slight
10.	Dyspraxia of Tongue Movements	soft sign only	
11.	Choreiform, Athetoid Movements	Marked	Slight
12.	Resting Tremor	Marked	Slight
13.	Resting Muscle Tone	Marked spasticity	Mild spasticity
14.	Paresis	Asymmetrical	Mild bilateral weakness

	Hard Signs	Soft Signs
15. Diminished or Hyperactive Tendon Reflexes	Grade 2 unilateral Grade 3 uni or bilateral	Grade 1 uni or bilateral, Grade 2 bilateral
16. Ankle Clonus	Sustained	Unsustained
17. Babinski Sign	Right, left, bilateral	
18. Synkinesia (developmental soft sign)	soft sign only	
19. Incoordination (developmental soft sign)	Marked	Slight
20. Heel/Knee testing	Marked	Slight
21. Intention Tremor	Marked	Slight
22. Disdiodocho-kinesia (developmental soft sign)	Marked	Slight
23. Anesthesia	Very Marked	Moderate or Slight
24. Simultagnosia (developmental soft sign)	Unilateral	Bilateral
25. Position Sense	Marked	Slight
26. Graphaesthesia (developmental soft sign)	soft sign only	

APPENDIX 2

Lateral Dominance Examination

1. Show me how you throw a ball.
2. Show me how you hammer a nail.
3. Show me how you cut with a knife.
4. Show me how you turn a door knob.
5. Show me how you use scissors.
6. Show me how you use an eraser.
7. Show me how you write your name.

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