CANADIAN THESES

THÈSES CANADIENNES

NOTICE

The quality of this microfiche is heavily dependent upon the quality of the original thesis submitted for microfilming. Every effort has been made to ensure the highest quality of reproduction possible.

If pages are missing, contact the university which granted the degree

Some pages may have indistinct print especially if the original pages were typed with a poor typewriter ribbon or if the university sent us an inferior photocopy.

Previously copyrighted materials (journal articles, published tests, etc.) are not filmed.

Reproduction in full or in part of this film is governed by the Canadian Copyright Act, R.S.C. 1970, c. C-30.

AVIS

La qualité de cette microfiche dépend grandement de la qualité de la thèse soumise au microfilmage. Nous avons tout fait pour assurer une qualité supérieure de reproduction

S'il manque des pages, veuillez communiquer avec l'université qui a conféré le grade

La qualité d'impression de certaines pages peut laisser à désirer, surtout si les pages originales ont été dactylographiées à l'aide d'un ruban usé ou si l'université nous a fait parvenir une photocopie de qualité inférieure

Les documents qui font déjà l'objet d'un droit d'auteur (articles de revue, examens publiés, etc.) ne sont pas microfilmés

La reproduction, même partielle, de ce microfilm est soumise à la Loi canadienne sur le droit d'auteur, SRC 1970, c. C-30.

THIS DISSERTATION HAS BEEN MICROFILMED EXACTLY AS RECEIVED

LA THÈSE A ÉTÉ MICROFILMÉE TELLE QUE NOUS L'AVONS REÇUE

Canada

Case Finding, Description and Outcome of a Selective Sample of Children Identified with and Treated for a Motor Delay of Non-specifiable Etiology in the First Four Years of Life

Linda Moxley-Haegert

A Thesis

in

The Department

of

Psychology

Presented in Partial Fulfillment of the Requirements
for the Degree of Doctor of Philosophy at
Concordia University
Montréal, Québec, Canada

July 1985

ISBN 0-315-30706-4

ABSTRACT

Case Finding, Description and Outcome of a Selective Sample of Children Identified with and Treated for a Motor Delay of Non-specifiable Etiology in the First Four Years of Life

Linda Moxley-Haegert, Ph.D
Concordia University, 1985

This study described developmentally delayed children with a motor delay of non-specifiable etiology (i.e., no established abnormal neurological signs or diagnosis). These children were identified and treated in the first 48 months of life. The answer to the first of the three major questions addressed in this study was that there is a definable and significant population of motor delayed children of non-specifiable etiology (1/3 of the total delayed population). These children differed from both non-delayed children and delayed children with a specifiable etiology. Although motor delay was the only variable that could distinguish these children from non-delayed children under 4 years of age; at follow-up when the children were 7 or 8 years old, delayed children performed significantly more poorly on motor, reading, and intelligence measures. These children responded more effectively to therapy with a decrease in motor delay than children with a specifiable etiology of delay. The second question evaluated the effectiveness of two forms of therapy in reducing motor delay. Intensive (in-centre) therapist mediated therapy resulted in a greater decrease in delay by discharge than infrequent (home programme) parent mediated therapy. This greater decrease was not maintained at follow-up 2 to 4 years later and these two treatment groups showed no differences in intelligence or

reading capabilities. Intensively treated children demonstrated more symptoms of hyperactivity than non-delayed children and performed more poorly in school than did infrequently treated and non-delayed children. These findings indicated that intensive therapy had no beneficial longer-term motor effects while there may be longer-term negative behavioural effects. The third question evaluated the effect of age at intervention initiation on longer-term outcome. Children initiating therapy before 2 years of age performed significantly better on motor and intelligence measures and better at school at follow-up than children initiating therapy after age 2. The findings supported Piaget's theory that motor abilities are of primary importance in the development of intelligence and the hypothesis derived from his theory that unremediated motor delay would result subsequently in lowered intellectual function. These findings suggest that evaluation of different therapy modalities should use as subjects children under age 2

Dedication

This work is dedicated to the many families with developmentally delayed children and to the children themselves who need so much more research than is being currently carried out in order to understand their problems better. Over the last 15 years the author has become increasingly interested and involved in the effort to understand better the delayed child and in particular the motor delayed child with a non-specifiable etiology for his/her delay. This study provides a beginning to a greater understanding of these children.

Having worked for a number of years with delayed children and their parents as an occupational therapist, I became increasingly aware that there was a sub-population of children who had a motor delay for which no etiology could be established. It appeared to me that this population was a significant one and that these children appeared to be even more responsive to therapy than children with an etiology which had been specified. It also appeared to me that these children were not being referred for therapy as early as were the children who had a delay with a specifiable etiology. Since these children might be more responsive to therapy and may be responsive to early therapy it seemed a shame that they were not being referred earlier for therapy. I felt that if it could be shown that these children were highly responsive to therapy and in particular to early therapy, this information might be of value in bringing about earlier referrals for therapy. These considerations led to the following research project at the Constance-Lethbridge Rehabilitation Centre and the Montreal Children's Hospital in

Montreal.

I would like to express my appreciation for the cooperation and support I received from the team members of the paediatric department at the Constance-Lethbridge Rehabilitation Centre and for the Centre's cooperation in allowing me to have time off from work in order to do this research. I would like to thank the occupational therapists at the Montreal Children's Hospital for their cooperation, as well. I wish also to thank the many caregivers and their children who agreed to participate in the study. Many of these children had to spend two to three half days with the psychometrician in order to complete the evaluation. Often this evaluation was difficult not only for the children but also for the parents who had to watch their children having problems during testing. I really appreciate, therefore, the parents' contribution to this research as they themselves received only a report of the findings of their children's evaluation.

I wish to express my great appreciation to Dr. H. Ladd, my thesis advisor, for his guidance and helpful suggestions and loyal support through my various trials as I attempted to finish my doctorate programme. I would also like to thank Dr. N. Taylor and Dr. B. Woodside for their helpful comments on an earlier draft of this thesis.

I received tremendous dedicated assistance from my psychometrician, without whom this thesis would not have been possible. André Bergeron went back to several homes a third and even a fourth time so that no data that were possible to collect would be lost. Several of the children assessed had severe behavioural problems and

André had even to bear with being bitten on one occasion. Thank you very much André! I would like to thank Katherine Laughton for her epidemiological assistance in helping me to pilot and standardize the data collection form and for her helpful comments on an earlier draft of this thesis. I would also like to thank Clara Bencsics who typed up 72, three and four page evaluation reports to be sent out to the parents and thanks go to Kay Brown and Frank Ellison who helped me learn how to use the word processor.

Finally, I would like to thank my family, David Haegert, and Kieran, Lawrence and Carlin for their continued patience and support throughout this whole 'going back to school' project. There have been times when their patience has been tried and at times they have shown this. I can remember Kieran when she was about two years old trying to think of the worst thing she could call me when she was mad at me and what she came up with was, "You studier, you". One time not too long ago my husband, David, was expressing his regret at not taking his Ph.D after his medical degree and Kieran remarked to him, "Well, its a good thing that you didn't, you would probably not be finished it yet!". However, on the whole I feel that they have been a pretty patient family and that we have all learned and grown closer together because at times the going has been hard.

TABLE OF CONTENTS

LIST OF TABLES

Chapter I

Introduction

This study was concerned with developmentally motor delayed children who had been identified at either the Montreal Children's Hospital or the Constance-Lethbridge Rehabilitation Centre as having manifested delayed motor development during the first 48 months of life. The particular category of delay which was of central concern to this study has been defined as motor delay of non-specifiable etiology. That is, there were not any major abnormal neurological signs (often referred to as hard signs as opposed to soft signs), and no specifiable diagnosis or etiology had been established. The empirical basis of such a definition had been established in terms of paediatric and/or neurological examination(s) documented in the children's medical records. There were two aspects as to the nature of this study. First, this study was a limited demographic study; second, it was an analytical study.

The demographic phase (medical chart review) of this study was limited only to those delayed children who were born between 1974-09-30 through 1976-09-30 and whose delay was identified between 0 and 48 months of age (472 children). Thus at the time of follow-up for the analytical phase of the study the children should have been in level two or three at school. The analytical phase of the study was based on a sample of 48 of these delayed children who had been considered, subsequent to the first diagnosis, to have either;

1) achieved a normal course of development on the basis of specified

medical, social and psychological variables, or

2) attained seven years of age with the continued label of non-specifiable etiology of motor delay or deficit.

Approximately 32% (137) of the original 472 children were to be included in these two categories by the time they were seven years of age.

The major independent variable of concern in the analytical phase of the study can be described as 'intervention therapy'. The aim of such therapy was to achieve, ultimately, a normal course of development. Such therapy was designed to enhance the children's development in physical, perceptual-cognitive, social and emotional spheres of living (Llorens, 1970). The intervention therapy exposed the children to, and encouraged the child in the participation of, as great a variety of stimulus situations as was possible within the time limits of the therapy programmes. These stimulus situations were chosen to be appropriate for each child's developmental level and changed appropriately in a developmental progression as the child developed (H. Bousefield, personal communication, Nov.1979; M.Walfish, personal communication, Oct.1979). This variable (intervention therapy) could be considered retrospectively at the Montreal Children's Hospital and Constance-Lethbridge Rehabilitation Centre in terms of the children either having received intensive in-centre therapy (6 or more hours per week for at least 12 months) or having received infrequent home programme therapy (less than 10 hours in 12 months).

Objectives

1. To establish a descriptive and demographic data base (derived from

medical record reviews) of the developmentally delayed children presenting in the first 48 months of life with motor delays which have no specifiable neurological or other biological cause(s).

2. To determine analytically the relationship between the intervention therapy programmes and functional status of the children both at discharge from therapy and at follow-up when the children are aged 7 to 8 years. Evaluations at discharge were based on the occupational therapists' functional motor scores from the discharge report. Evaluations at follow-up were based on measures of motor abilities, intelligence, behaviour, and self-concept for both the delayed children and an equivalent group of non-delayed children.

Present State of Knowledge

Contemporary research into developmental processes during the first several years of life has identified a sub-population of children with a motor delay of non-specifiable etiology. These children do not demonstrate major abnormal physical signs (these being associated usually with either prenatal or perinatal insult to the central nervous system (CNS)), and the cause(s) of the delay could not be determined clinically. Recent data have included children with a motor delay of non-specifiable etiology in studies of developmentally delayed children (Barna, Bidder, Gray, Clements, & Gardner, 1980; Gillberg, Rasmussen & Wahlstrom, 1982; Meier, 1976; Moxley-Haegert & Serbin, 1983). Children with a non-specifiable etiology of delay have been described variously as 'slow', 'floppy', 'irritable', 'clumsy' and so forth; however, these are descriptive labels and do not imply a specific etiology (Findlay,

1979; Henderson and Hall, 1982; Meier, 1976).

The child who has been identified during the first several years of life as having a non-specifiable etiology of motor delay could be expected to fall eventually into one of four rather broad, loosely defined categories. These are:

1. The child whose motor delay is associated eventually with mental retardation;
2. The child whose motor delay is ultimately described as cerebral palsy;
3. The child who has attained a normal course of development within the first several years of life; and
4. The child of normal intelligence who will continue to demonstrate a measurable motor delay or deficit with a non-specifiable etiology.

The child whose motor delay is associated with mental retardation. It is known that delay in motor development and competence often accompanies the cognitive and language deficits of mentally retarded children (Hogg, 1982). Molnar (1975) hypothesized that disordered maturation of autonomic postural control mechanisms could account for delay in gross motor development in retarded children and she showed that the emergence of the relevant autonomic postural control reliably preceded acquisition of the related gross motor skill. Thus a mild motor delay can only become obvious at the time at which an important skill (i.e. sitting, walking) should have developed. This may account for the general lateness in which a minor motor problem has often been referred for therapy (Fox,1979).

Motor differences have been found between even mildly mentally retarded individuals and those with normal intelligence. For example, it has been found that even mildly mentally retarded individuals have more difficulty with a motor response as the stimulus for the response becomes more complex (Multhern & Baumeister, 1971);they have been more able to respond to proprioceptive feedback than visual feedback cues unlike non-delayed controls (Anwar & Hermelin, 1979); and mildly mentally retarded individuals inspected a stimulus to be responded to longer than non-delayed individuals before making a response, even after they have understood what they must do (Brewer & Nettlebeck, 1977; Nettlebeck & Brewer, 1976; Wade, Newell & Wallace, 1978). This research explains why mildly retarded individuals have shown motor differences even when they appeared to be physically capable of the motor actions. It has been shown, as well, that difficulty in 'performability' of a motor task increases as the IQ decreases (Berkson, 1960a; 1960b; Wade et al., 1978).

Definite etiology has been established in about 85% of cases of severe mental retardation in several etiological studies (Czeizel, Lanyi-Engelmayer, Klugber, Metneki & Tusnady, 1980; Gustavson, Hagberg, Hagberg, & Sars, 1977; Mackey, 1982). Mental retardation of mild degree (IQ 50 - 70) has been long recognized as both a major problem and an etiological enigma (Costeff, Cohen, & Weller, 1983). The mildly retarded individual more often has retarded parents and/or siblings than does the more severely retarded individual (O'Reilly & Walentyrowicz, 1981; Penrose, 1963); the mildly retarded individual seems to be

disproportionately concentrated in the lower socioeconomic strata of the population (Drillien, Thomson & Burgoyne, 1980; Penrose, 1963); and medical investigations of people with mild retardation demonstrate clear-cut present or past neurological illness in only a small proportion (Penrose, 1963; 1972). Czeizel et al. (1980) felt they could establish etiology for 87.4% of mildly mentally retarded children when familiar-cultural factors were taken into account. Familial-cultural factors accounted for 49.4% of mildly retarded children. Subjects in their study were categorized as familial-culturally mentally retarded if their history revealed no specific significant etiological factors, no cerebral injuries attributed to drastic exogenic influence, no demonstrable genetic or somatic disorder, and at least one of the parents or one sibling had attended a special school or was unable to complete his/her studies in the first four grades of primary school. Costeff et al. (1983) presented evidence that the main cause of mild retardation may be brain damage as a result of biological disturbances during pregnancy, delivery or infancy, rather than polygenic heredity or non-biological social deprivation. This evidence was derived from a survey of the medical histories of a population having idiopathic retardation. After a very thorough investigation, 30% of this population which totaled 236 cases still had to be defined as having an idiopathic retardation. Broman, Nichols and Kennedy (1975) found that before birth the most reliable predictor of mental retardation at age 4 was maternal education. Such children would be defined as having a non-biological based retardation. Some of the children in the present study

may be of this idiopathic mild mental retardation classification category.

The child whose motor delay is described as cerebral palsy. Motor dysfunction has been a predictor of cerebral palsy. Cerebral palsy is by definition a motor disability resulting from a defect or lesion which may have a wide variety of causes (O'Reilly & Walentynowicz, 1981). O'Reilly and Walentynowicz (1981) found that 16% of cerebral palsy individuals had an idiopathic etiology.

Multiple variables have been found to be predictors of the motor dysfunction of cerebral palsy found in infants in a neonatal intensive care unit population by Ellison, Horn and Browning (1981). They found that very low birthweight (below 1750 grams), apnea, intracranial bleeding, neonatal seizures, treatment with O_2 or a chest tube, and race accounted for 91% of children with motor dysfunction. The etiology could not be identified for the other 9%. All of the children were identified as having a motor dysfunction at a very early age so would be classified as having a severe motor dysfunction. There have been few studies that have looked at the etiology of delay in children with a mild motor dysfunction and a diagnosis of cerebral palsy.

The child who has obtained a normal course of development. Czeizel et al.(1980), found in their study of mental retardation in Budapest, Hungary, that 6.5% of the children who had been diagnosed as being mentally retarded before the age of 7 turned out to be 'normal' when evaluated between the ages of 7 to 14 years. Many of these non-mentally retarded children had behavioural deviations at the time of assessment

and were presumably in the special schools because of these problems. There do not appear to be any prospective studies that have looked at school age children to determine sequela of motor delays identified when the child is very young. Thus an appropriate percentage of children who were motor delayed and are functioning normally by school age cannot be determined.

The child of normal intelligence who will continue to demonstrate a measurable motor delay or deficit with a non-specifiable etiology. In this group often either minimal 'soft' neurological signs (Hertzig, 1981), or transient abnormal neurological signs (e.g. transient abnormal electroencephalogram) early in life have been reported (Drillien et al., 1980). Some have manifested mild behavioural problems (Gillberg & Gillberg, 1983; Oberlaide, Dworkin, & Levine, 1979), while others have school achievement problems (Gillberg, Gillberg & Rasmussen, 1983; Younes, Rosner, & Webb, 1983). Henderson and Hall (1982) examined a group of children whose level of motor competence was significantly below normal but who showed no evidence of disease of the nervous system. They (Henderson & Hall, 1982) found that a large proportion of children of normal intelligence who experienced learning difficulties in aspects of schoolwork such as reading or number concepts also exhibited delayed motor behaviour. They and others have found that such children have more problems in preschool years, more significant medical events, have lower scores on neurodevelopmental examinations, tend to be more immature socially, and tend to have lower verbal intelligence quotient scores, and to be reading at a lower level than their non-motor delayed

controls (Brenner & Gillman, 1966; Gordon & McKinlay, 1980; Gubbay, 1975; Henderson & Hall, 1982; Orton, 1973).

Ruben and Barlow (1980) have demonstrated that non-optimal pregnancy factors, intrapartal and neonatal complications as well as minimal neurological abnormalities in infancy, may interface eventually with the children's normal development. These factors may cause (be factors in the etiology of) not only major neurological handicaps, but also learning disabilities, motor problems and behavioural difficulties, and such children often have been classified under the heading 'minimal brain dysfunction'. However, the contribution of other factors such as heredity and psycho-social conditions has been less well understood. Gillberg and Rasmussen (1982) found a population of motor delayed children who also had attentional deficits. They could not specify an etiology for these children, but these children tended to come from a lower social class than controls and there was a tendency for non-optimal hereditary, prenatal-perinatal and neonatal-postnatal factors to be found in this group. These etiology factors were significant, and Gillberg and Rasmussen (1982) concluded that hereditary, brain damaging and psycho-social conditions interact in a complex fashion with these children. Gillberg and Rasmussen (1982) also identified a population of children who were only motor delayed and had no attentional difficulties. The only variable that tended to differentiate these children from controls was non-optimal prenatal factors. Thus, they suggested that the etiology of this group's delay is more homogeneous. Although the differences were not

significant (numbers were small so significant differences would have been hard to obtain), their evidence suggested that the children who had an heredity for left-handedness were at greater risk of suffering from clinical sequela of non-optimal perinatal factors.

Why Researchers and Therapists Interested in Child Development Should be Interested in the Child with a Motor Delay of Non-specifiable Etiology

Although motor delay of non-specifiable etiology has not been a major concern historically to those interested in the development of young children, there are several reasons why it should be. First, in young children motor delay may be the first manifestation of a more pervasive disorder such as mental retardation or cerebral palsy. Second, delay may be a predictor of more subtle but important later problems, such as difficulties in learning to read or write or a behavioural problem. For example, Hewison (1982) demonstrated that adequate perceptual motor abilities were necessary in order to learn to read. Third, motor delay which is not treated or modified could be the cause of other problems in learning and behaviour. (For example, a child who does not walk will have to be carried by an adult who may feel resentful of the extra work load, thus difficulties in the relationship between the two begin, or a child who does not crawl will have a much smaller world to explore and learn manipulation skills than a child who is crawling). It is known that progression in one category of development can affect other categories, as in the case of learning to walk (gross motor skill) which in turn augments opportunities to move around and interact with a wider range of objects (fine motor skills)

(Hogg,1982).

Problems in motor behaviours develop because of a complex interaction between abnormalities in the processes underlying motor development and the impact of the environment on the person (Bruinicks, 1964; Hogg, 1982; Rarick, 1973). Hogg illustrated some of the factors that influence motor development and performance of delayed children without other specific physical handicaps (such children may be similar to the children with a delay of non-specifiable etiology in our study). He (Hogg, 1982) reviewed studies in which different phases of motor behaviour have been divided into those that indicate problems in processing information from the environment in order to execute a motor task (input studies) (Anwar & Harmelin, 1979; Brewer & Nettlebeck, 1977; Multhern & Baumeister, 1971; Nettlebeck & Brewer, 1976) and those concerned with the actual performance of an action (output studies) (Berkson, 1960a; 1960b; Wade et al., 1978). Hogg (1982) explained that from these studies we can realize that environmental influences will interact with emerging behaviour to impair or facilitate the development of motor capacities. It has been shown that motor development can be adversely affected by poor environment or by variations in environment (Ohwaki & Stayton, 1978; Dennis & Najarian, 1957; Francis, 1971; Piaget, 1959; Provence & Lipton, 1962). For example, Piaget (1959) found from personal observation that his own daughter was much more delayed in eye-hand co-ordination than her siblings, presumably because she was born in winter and had to wear mittens much of the time, because she soon caught up with other children of the same age when the warm weather came.

These findings have been complemented by studies of the impact of training on motor development. It has been shown that gross motor skills are responsive to planned interventions (Campbell, 1974; Harris, 1981; Morrison & Pothier, 1972); however, planned interventions to ameliorate visual-motor co-ordination have not been shown to be effective (Webb & Koller, 1979) (for a review of the studies on effectiveness of physical therapy and occupational therapy on fine and gross motor function see Henderson, 1981).

These findings have led this researcher to the final reason why motor delay of non-specifiable etiology should be of interest to researchers in the area of child development. There is evidence to suggest that the developmentally delayed child, without gross neurological impairment such as either the child identified as having a motor delay of non-specifiably etiology or the socially disadvantaged child has a 'better' overall prognosis than the child with a specifiable neurological impairment (Browder, 1981; Simeonsson, Cooper & Scheiner, 1982; Ramey & Smith, 1977; Wright & Nicholson, 1973). Fairly convincing evidence supports the efficacy of early intervention in successfully preventing developmental delays; in potentially normal children of poor parents (see Bronfenbrenner, 1975; Day & Parker, 1977; Field, Hallock, Ring, Demprey, Dabini & Shuman, 1979; Horowitz & Paden, 1973; Lazar & Darlington, 1982; and Northcott, 1973 for reviews of this research); in potentially normal children who were preterm and did not demonstrate neurological damage (Bromwich & Parmelee, 1979; Field, 1980; Masi, 1977; Scarr-Salapatek & Williams, 1973); and in potentially normal children

who were hearing impaired but demonstrated no neurological damage (Gallagher, 1973; Horton, 1976; Liff, 1976; Northcott, 1973). The efficacy of early intervention in preventing delays has been reported with such children for the following factors; benefit of treatment programmes (Barna et al., 1980; Kirk, 1969; Williams & Scarr, 1971), degree of success in early school functioning (Drillien et al., 1980) and degree of success in adult functioning (Milman, 1979).

The motor delayed children in this study may represent a population, then, which has the potential for normal development and which could be expected to respond favourably to therapy; and should possibly be given early therapy. There has been evidence which suggests that the earlier the intervention the better the effects of the intervention (Gilmer, Miller & Gray, 1975; Karnes, Teska, Hodges, & Badger, 1969; Levenstein, 1977). It has been speculated, however, that the infant with a delay who does not demonstrate clear physical abnormalities is often 'passed over', because of the belief that the infant will grow out of his/her difficulties (Denhoff & Hymen,1976). Others have shown that many attending physicians are reluctant to refer a child for treatment before a specific diagnosis is made (Fox, 1979). The obvious result is that the child with a motor delay of non-specifiable etiology may not have entered therapy in the early years of life; and, in the past, many of these children have not received any form of intervention until they entered school (Werner, Beinman, & French, 1971). There has been presented some empirical evidence to support the premise that many of these children will go on to have

problems in school (Gillberg et al., 1983; Henderson & Hall, 1982; Werner et al., 1971;). In conclusion, the evidence has suggested that the child with a motor delay of non-specifiable etiology should be given therapy early. Since there does not appear to be an adequate number of studies which have investigated, for this population specifically, the effects of intervention, many of these children continue to be untreated or very infrequently treated until they enter school and begin to have problems there.

Theoretical and Experimental Considerations Concerning the Effectiveness of Early Intervention for the Motor Delayed Child

It has been widely recognized since the 1930's and 1940's that the course of human development is not invariable but may be modified by environmental conditions. This recognition arose from observations of the development of children who have been reared in institutions (Goldfarb, 1945; Skeels & Dye, 1939; Spitz, 1946) and under conditions of isolation (Davis, 1940; 1947; Itard, 1932), and extreme poverty (Asher, 1939; Gordon, 1923; Sherman & Key, 1932; Wheeler, 1932). It was realized that such deprivation could result in delayed development, and the conclusion drawn by many was that intervention to provide an improved environment might prevent developmental delay, and thus subsequent school failure.

Programmes concerned with the prevention of developmental delay have received clear and direct impetus from Kirk's (1958) work with preschool children from low-income families and from several prospective studies which tried to change the environments of deprived children

(e.g., Dennis & Nazarian, 1957; Skodak & Skeels, 1945; 1949). Kirk's (1958) immediate post-intervention success in ameliorating the effects of poverty on the cognitive development of young children encouraged those concerned with environmental deprivation. Belief in the power of preschool intervention was diminished when Kirk (1969) reported very modest longer-term effects and when follow-up studies of subsequent preschool interventions showed no longer-term effects of raised intelligence scores (e.g. Blatt & Garfunkel, 1967; Bronfenbrenner, 1975; Cicirelli, 1969; Spicker, Hodges & McCandless, 1966; Weikart, 1967; White, 1973).

Partly as a result of these findings with preschoolers and partly as a result of studies which have demonstrated that the greatest gains were made with infants under two years of age (e.g. Gilmer et al., 1975; Karnes, Teska, Hodgens & Badger, 1969; Levenstein, 1970) psychologists had begun to argue that very early experience was critical to intellectual development and that intervention with infants (disadvantaged) was essential for permanent gains (for an alternate view see Clarke and Clarke, 1976). The importance of early versus late intervention has become one of the major controversies that has emerged in the early literature on intervention therapy. This controversy centers around the issue of 'critical', or 'sensitive' periods and the disputed importance of 'early experience'. The reader is referred to Rutter (1980) and Bronfenbrenner (1975) for reviews. Further support for earlier intervention has come from research indicating the beneficial effects of early stimulation on developing animals and

infants (see Bronfenbrenner, 1968; Field, Sostek, Goldberg, & Shuman, 1979 for reviews of this research). There has been a growing belief, more recently, in the relatively greater plasticity of the central nervous system during infancy (see Braun, 1978; Hecean & Albert, 1978; and Kertesz, 1979 for reviews of this research). Sandown, Clarke, Cox and Stewart (1981) showed that severely subnormal infants who made gains on tests of intelligence and social skills at a younger age during intervention were more likely to maintain that progress than children who made gains at a later age. Reviews on the effectiveness of early intervention with disadvantaged children have concluded that the optimal period for introducing intervention is prior to two years of age (Bronfenbrenner,1975; Gordon,1973). One study (Barna et al., 1980) included children with a delay of non-specifiable etiology. This study found that age at entry into their specific intervention programme did not affect differentially the rate of progress (mental age gains) during therapy for these children. Moxley-Haegert (1981) found a relationship between age of entry and motor gains made by the children with a non-specifiable etiology for their motor delay, but not between age and mental gains made. The younger children (1 - 18 months) made greater motor progress with intervention therapy than did the older children (19 to 36 months). The literature does not allow for a conclusive position on the 'critical' age for entry into an intervention programme for these children at this time but it does suggest that early intervention may be more effective than later intervention.

Certain theoretical considerations would indicate, as well, that

early versus late intervention would be important in relation to therapy for a child with a motor delay of non-specifiable etiology. These theoretical foundations were derived from the works of Harter (1978; 1981), Hebb (1947), Hunt (1961), Piaget (1951; 1952; 1954; 1959; 1970) and White (1959). Hebb's neuropsychological theory is primary to the idea that early intervention is important. His theory asserted that an organism's ability to learn in later life depends on the quality of its early, primary experience and learning. Hunt synthesized Hebb's neuropsychological theory and Piaget's theory of intelligence with his own research on central processes to argue that intelligence could be improved by early intervention.

Although there are many theories of development (see Langer, 1969 for a synthesis and critical review of the major contemporary theories of development), there is more in Piaget's theory of the development of intelligence which can be related to the motor delayed child and which would lead one to encourage therapeutic intervention for the young motor delayed child. For example, Piaget proposed that the manner in which a child learns is from active interaction with the environment, and that the greater the variety of circumstances to which a child is exposed, the greater is his/her capacity for coping (Piaget, 1952; 1959). Motor abilities and skills are obviously of primary importance if a child is to interact actively with the environment and thus to progress in development. The motor delayed child will not respond readily to the environment because it is difficult for him/her to respond physically in motor ways. A motor delay will reduce, as well, the variety of

experiences in which the child participates.

Piaget hypothesized that a reflexive schema will not persist or develop if it is not used continuously (Piaget, 1954; 1970). Motor delayed children could, thus, lose a reflexive schema through lack of use and could become more and more delayed due to the lack of experiences which demand an accommodative modification and which are necessary for progressive development. In addition, these accommodative modifications depend on a proper match between existing schemata and objects (Piaget, 1959;1970). Many motor delayed children may be provided with opportunities appropriate to their chronological age, but not to their developmental level. Therefore, if motor delayed children are provided with opportunities for experience related to their chronological age rather than developmental age, they will not be able to advance developmentally. These proposals of Piaget are relevant for the treatment of delayed children. Not only should motor delayed children be treated early and the therapy and stimulation be as varied as possible, but also the stimulation should be appropriate to the children's developmental age regardless of their chronological age.

Piaget states that subsequent and more complex forms of behaviour are based on and built from earlier and simpler forms (Piaget, 1952; 1959). If a child has not developed these early forms of behaviour the more complex forms cannot be built. The earlier the intervention to remediate a motor delay providing as great a variety of situations to which the child must accommodate his/her behavioural structures, the more differentiated and mobile the behavioural

structures should become. Thus, the rate of intellectual and psychological development should become more rapid with earlier intervention and the child should develop earlier in life an interest in the novel and the new. It could be predicted from this idea that the younger a child is when intervention is provided, the higher would be the child's level of functioning when he/she reaches school age. This level of functioning should be higher in terms of psychological development as well as intellectual and motor development according to Piaget's propositions (Piaget, 1951; 1954; 1959). Thus when evaluating the effectiveness of early intervention for motor delayed children both intellectual measures and psycho-social measures should be used as well as motor measures.

In order to explain the child's psychological development in relation to Piaget's theory one must consider the concept of initiative or motivation. Piaget states that as a wider variety of circumstances acquires capacity to evoke a child's interest, the child becomes curious about more things (Piaget, 1952). With this curiosity, according to Piaget, the child develops initiative. Other theorists such as Harter (1978;1981) and White (1959) would label 'initiative' as 'motivation'. Harter has postulated a model of intrinsic motivation affecting a child's concept of self which could have important implications for the development of the motor delayed child (Harter, 1978; 1981). It could be predicted from Piaget's theory that lack of intervention for the motor delayed child would have implications for the child's development of a sense of self by delaying the child's ability to differentiate self

from environment and thus would delay the child's development of an accurate perception of his/her own competence. One aspect of a child's sense of self, perceived competence, is central to Harter's model and she has developed a measure which evaluates what a child perceives in terms of his/her physical and cognitive competence and in terms of maternal and peer acceptance (Harter & Pike, 1984).

Perceived competence is considered to be an important correlate and mediator of a child's intrinsic motivation to be effective and to engage in mastery attempts (Harter, 1978;1981; White, 1958). Using Piaget's terminology, mastery attempts means to interact actively with the environment. In an earlier study by this researcher (Moxley-Haegert,1977) it was found that older, (7 to 8-year-olds), motor delayed children of non-specifiable etiology, needed more extrinsic reinforcers in order to persist at a difficult activity than did non-delayed control children. It could be inferred from these findings that there was a difference between delayed children and normally developing children in intrinsic motivation and thus in perceived competence. Based on Harter's model, Moxley-Haegert (1977) concluded that the children who had been motor delayed may not have had experienced a sufficient number of success experiences in order to keep on trying once they had found the task difficult. Harter hypothesized that success must occur early in the ontogenetic sequence and that it is especially critical in the first several years of life if the child is to develop and maintain the motivation to be effective (Harter, 1978). It might be suggested from these findings and theories that a therapy programme which is intensive

and tries to bring about change in the child's level of competence and number of success experiences may actually affect the rate in which the child can differentiate himself/herself from the environment and may thus affect the child's perception of his/her own competence when compared to a programme which is infrequent and is involved only minimally with the child. It could also be predicted that the younger a child is when therapy begins and when success experiences begin the more able the child will be to have a defined and correct perception of his/her own competence. Such effects of early intervention have been demonstrated for socially disadvantaged children when they are 13 and 14 years old (Miller & Bizzell,1984).

In conclusion, it has been these Piagetian theoretical propositions which have provided the underlying rationale for this research; i.e. early intervention therapy will have a positive effect on both the intellectual and psycho-social development of the child with a motor delay of non-specifiable etiology. The experience of early intervention (experience is defined by Piaget as the organism's encounters with his/her environment) would be continually building into the developing child a hierarchy of operations for processing information and for coping with different circumstances. These operations are defined as intelligence. If therapy helps a child's attempts at certain tasks to be a success rather than a failure, that is, if they result in competent performance, the child should experience feelings of efficacy. This, in turn, should maintain, if not increase, the child's motivation or initiative to continue to try to interact with the environment, to try

to perform new developmental tasks and thus to continue to learn and develop. In order for the motor delayed child to be prevented from becoming more and more delayed and to reach his/her potential level of intellectual capacity and psycho-social development an environmental intervention would have to be introduced as early as possible.

A variety of studies with developmentally delayed and non-delayed infants and children have supported the premise that primary sensorimotor development is highly sensitive to environmental factors (White, 1977; Rutter, 1980). There have been many studies directly concerned with the environmental factor of intervention therapy for several different categories of delayed children such as deprived children, biologically retarded children, deaf children and preterm children (see Bronfenbrenner, 1975; Caldwell, Bradley & Elardo, 1975; Day & Parker, 1977; Field et al.,1979; Horowitz & Paden, 1973; Mittler, 1977; Northcott, 1973; Piper, 1978; and Tzossen, 1976 for reviews of this research). The majority of studies have supported the general conclusion that there is an immediate positive effect of early therapeutic intervention. There are only a limited number of studies reported in the literature which are concerned with the effectiveness of early intervention for children with a motor delay of non-specifiable etiology identified in early childhood. Three studies involving such children have suggested that there is an immediate positive effect of therapy; that is, an increase in the child's rate of development when compared before and after therapy (Barna et al., 1980; Findlay, 1979; Moxley-Haegert and Serbin,1983).

An important question yet to be determined would be whether or not treatment of children with a motor delay or deficit of non-specifiable etiology has a longer-term (months to years) impact. Bronfenbrenner (1975) states that there has been, in general, a lack of studies concerned with extended follow-up for the disadvantaged child. He and others concluded that the effects of early educational intervention often do not persist once the intervention ceases (Blatt & Garfunkel, 1967; Bronfenbrenner, 1975; Cicirelli, 1969; Kirk, 1969; Klaus & Gray, 1967; Spicker et al., 1966; Weikart, 1967; White, 1973). Longer-term effects have emerged, however, when intervention has been extensive (Gordon, 1973; Heber, 1978). Later studies have suggested that the longer-term effects of early intervention could be considered in terms of retention in grade, or being placed in special classes (Lazar & Darlington, 1982) rather than in terms of an intelligence quotient change.

Gordon's (1973) and Heber's (1978) findings raise the question of the importance of duration of intervention therapy for the child with a delay of non-specifiable etiology. Both the Findlay (1979) and Moxley-Haegert and Serbin (1983) studies were of short duration (one month), thus any differential effect of duration could not be evaluated. In the study of Barna et al. (1980) the duration of therapy varied from child to child (average 12 months). Their data (Barna et al., 1980) show that there was a positive relationship between duration of intervention therapy and mental age gains. The longer the duration the greater the average monthly gains made in mental age by the children with a non-

specifiable etiology for their delay. It could be stated, thus, that the factor of duration of therapy is critical in attempting to study the association between institutional intervention therapy and both immediate and longer-term outcome of children with a motor delay or deficit of non-specifiable etiology.

A review of a number of early intervention programmes for handicapped and mentally retarded children, and for children who were considered at risk for developmental delay, revealed certain parameters that set effective programmes apart from others. It appears that one of the most important criteria for success of an intervention programme with infants is that there be a strong parental involvement component to the intervention (Bronfenbrenner, 1975; Goodson & Hess, 1983; Gray & Wandersman, 1980; Rosenburg, 1977; Schaefer & Moersch, 1977). When parents are highly motivated and give high priority to the intervention, intervention is most likely to be successful. Thus successful programmes are those which can motivate parents and which allow parents to feel a certain amount of responsibility for the treatment process. It has been shown in a previous study that infrequent home programme therapy for children with a non-specifiable etiology for their motor delay gives most of the responsibility for the intervention to the parents without providing a way to motivate the parents' involvement in the intervention (Moxley-Haegert & Serbin, 1983). However, it was also shown in this study that it was possible to motivate these parents, and when parents were more motivated, the children made more progress (Moxley-Haegert & Serbin, 1983).

Other important factors include a well defined instructional model for structuring the programme (Levenstein, 1977; Schaefer & Moersch, 1977), attention to the child's total development, appropriate instructional materials (Karnes, 1969), and an intensive programme (5 days a week) maintained for several years (Garber & Heber, 1977; Gordon, Guinagh, & Jester, 1970; Heber, 1978). A combination of such parameters seems to be requisite for all successful programmes regardless of the etiology of the child's delay. Moreover, findings have shown that when nutritional supplements and good medical care are provided as well as developmental intervention to poor people's children, the children demonstrate improved development (Ramey & Campbell, 1979; Jensen, 1981). However, the benefits of good intervention programmes are over and above any benefits such as health screening, nutritional supplementation and family services that the individual programmes may also provide (Ramey & Campbell, 1979). It could be suggested from the research that an infrequent home programme which does not encorporate ways of motivating parents would not be as effective in ameliorating children's delays as an intensive structured programme of therapy.

At present there is a paucity of information concerning the stability and significance of a motor delay of non-specifiable etiology diagnosed at a very young age. For example, are there certain demographic variables such as maternal age at birth, as suggested by several researchers (Gillberg,1980; Jayaxkara & Street,1978; Tizard & Grad, 1971), or abnormal head circumference (Smith,1981), which might separate children with motor delays of non-specifiable etiology from

normally developing children? It is not known in addition, what problems these particular children (children with a motor delay of non-specifiable etiology) have later in life; and more importantly how many of these motor delayed children have transient versus permanent delays. It has been hypothesized that over-stimulation could be harmful for a child who does not need the extra stimulation in order to progress (i.e. a child who has a transient motor delay) (Bromwich, 1977). It is known as well, that parents of even minimally delayed infants experience stress (Field et al., 1978; Poznanski, 1973) and therefore it may be important to identify those children with transient delays from those with permanent delays.

In conclusion, this research has been designed to broaden the knowledge about the population of children with a diagnosed motor delay of non-specifiable etiology. Theory suggests that motor delays in children make them susceptible to both intellectual and psychological negative effects if these delays are not treated. Children with motor delays of non-specifiable etiology would probably be responsive to early intervention but at present little is known as to the effectiveness of treating them. The complete study was designed to evaluate longer-term effectiveness of therapy as well as shorter-term effectiveness of therapy for these children. It was realized that not all these children might need intervention. It was thus important to try to characterize the particular children with delays of non-specifiable etiology who were in need of treatment.

Hypotheses

The general hypothesis was that there would be a difference in terms of developmental level among the three groups (motor delayed infrequent home programme therapy, motor delayed intensive in-centre therapy and non-delayed no therapy). The primary prediction relating to groups differences was that children who were not delayed would be significantly different from motor delayed children in all spheres of development when the children were 7 ot 8 years of age.

The specific hypothesis related to therapy effectiveness. This was that there would be a decrease in percentage motor delay at the time of discharge from therapy for all motor delayed children receiving therapy regardless of the intensity of therapy.

The first prediction relating to therapy effectiveness was that the therapy as specified would be more effective in reducing motor delay for the children with a non-specifiable etiology than for the children with a specifiable etiology for their delay. A specific related prediction which would verifiy the effectiveness of therapy was that children with a non-specifiable etiology for their delay:

1. would demonstrate a greater decrease in fine, gross,

and average motor percentage delay at the time of discharge.

The second prediction relating to therapy effectiveness was that children receiving an intensive intervention therapy (no less than 6 hours per week for at least 12 months) would demonstrate a higher level of development when compared to children receiving an infrequent home programme therapy (10 hours or less of in-centre therapy for 12 months).

A third prediction relating to therapy effectiveness was that children who received therapy before 2 years of age would demonstrate a higher level of development than children treated between 2 and 4 years.

Specific related predictions which would verify the greater effectiveness of intensive therapy and of early therapy were that children receiving intensive or early therapy:

1. would demonstrate a greater decrease in fine, gross, and average motor percentage delay at the time of discharge from therapy;

2. would demonstrate a lower average motor percentage delay at follow-up 2 to 4 years after discharge from therapy;

3. would demonstrate an higher intellectual level at follow-up 2 to 4 years after discharge from therapy;

4. would demonstrate more accurate perceptions of their

own competence at follow-up 2 to 4 years after discharge from therapy; and

5. would demonstrate fewer behavioural problems at follow-up 2 to 4 years after discharge from therapy.

Methodological Considerations

One of the major methodological issues that has emerged from the literature is how to determine and evaluate effectiveness of intervention. It has been usual to evaluate cognitive function using the Intelligence Quotient (IQ). It should be emphasized that although the major factor used to evaluate intervention effectiveness has been the child's progression in various developmental categories or intelligence measures such as the standardized intelligence quotient (IQ), this is only one way of evaluating an intervention programme. While IQ or developmental quotient (DQ) can be an appropriate measure of effectiveness in relation to immediate gain, it may not be an appropriate measure in relation to longer-term gain. In fact, when using IQ as a measure of treatment effectiveness some children who have demonstrated an increase initially, have returned to a pre-intervention level (Bronfenbrenner, 1975). In contrast, recent longer-term evaluations (Lazar & Darlington,1982; Schweinhart & Weikart, 1980) have demonstrated that the important and longer-lasting effect of intervention for disadvantaged children was that fewer of such children receiving preschool intervention either have been retained in grade or have been placed in special classes. It would be possible, thus, that

measures of school achievement and/or functional and behavioural indices may be meaningful in evaluating treatment effectiveness, particularly in reference to longer-term effect. Zigler and Trickett (1978) have been among the main proponents of broadening the evaluation criteria. They (Zigler & Trickett, 1978) argued that gains in social competence rather than gains in intelligence scores should be the ultimate criteria of programme effectiveness and other factors of a social psychological nature would be important criteria. In this study it was felt that evaluation of motor gains was an important measure of treatment effectiveness since it was a motor delay the intervention programme specifically was trying to ameliorate. Based on Piaget's theory that subsequent and more complex forms of behaviour such as intellectual and psychological development are based on and built from earlier and simpler forms of behaviour such as motor behaviour, it was felt that intellectual and psychological measures were also important. As well, since the intensive intervention programme therapy also encorporated behavioural treatment into its programme, measures of behaviour were also implemented to evaluate treatment effectiveness.

This was both a descriptive and analytic study. There was a larger group of children referred for assessment to the occupational therapy department for a possible motor delay before 48 months of age who were evaluated and described using data obtained only from the medical records. There was a smaller group of children who were intensively assessed at follow-up when the children were at least seven years of age. While there has been an overlap in methodology between the

descriptive and analytic phase of this study, for the purpose of clarity the methods will be presented in two distinct chapters. There will be one chapter for the methodology of the descriptive phase and one for the methodology of the analytic phase. This procedure has been repeated for the results. That is, chapters II and III are concerned with the descriptive phase and chapters IV and V are concerned with the analytic phase.

The study cohort was based upon case finding from medical records of children born from September 30, 1974 through September 30, 1976. All children who had been born in these years, and evaluated at either the Montreal Children Hospital or the Constance-Lethbridge Rehabilitation Centre were considered for the descriptive phase of the study. Seventy-two of these children were selected subsequently for the analytic phase.

Chapter II

Method

Descriptive phase

Subjects. The total sample of children referred for a motor assessment and considered for the descriptive phase of the study consisted of 472 children. The criteria for admission into the descriptive phase of the study were: that the children had been referred to the Occupational Therapy Department at the Montreal Children's Hospital (MCH) or to the Paediatric Service at the Constance-Lethbridge Rehabilitation Centre (CLRC) and accepted for evaluation before the age of 48 months, and had been born between September 30, 1974 and September 30, 1976. These children were divided into three groups. One group consisted of children who were found to have no delay. That is, they were found not to have a delay or had a delay between 1% and 10% in only one of the three categories of motor development (gross motor, fine motor, and perceptual-motor development). The percentage delay was calculated by the researcher at the time of the review of the medical records by comparing developmental age with chronological age. A percentage delay was calculated rather than making use of the actual delay (e.g. 2 months delay) to take into account the different ages of the children at the time of assessment. For example, a 2-month delay is a much more significant delay for a 6 month old than for an 18 month old. Children who had a minimum motor delay of 10% in at least two categories of motor development (gross motor, fine motor, or perceptual-motor) or had a minimum motor delay of 20% in one of the three

categories were considered to be delayed.

These delayed children were divided into two groups based on etiology. The first group of delayed children had a specifiable etiology for their delay according to data documented in their medical records at the time of initial assessment. The second group of children had a non-specifiable etiology for their motor delay.

In accounting for the first group, children with a specifiable etiology of delay, etiological factors have been grouped into prenatal, natal and postnatal categories. Prenatal factors comprised those operating from the time of conception to time of labour, such as hereditary or genetic conditions, prenatal infections, prenatal anoxia, haemorrhage, Rh problems and metabolic disturbance. Natal factors included various causes of anoxia and constitutional factors such as prematurity (less than 37 weeks gestational age), postmaturity (over 43 weeks gestational age with recorded clinical postmaturity features), caesarean section due to fetal distress (i.e.not due to repeat caesarean or elective caesarian), dystocia, placenta praevia, or placental abruptio. Postnatal factors included trauma, infections, vascular accidents, anoxia, tumors, convulsions other than febrile convulsions, encephalitis, and infection. All 247 children with specifiable etiologies at the time of assessment in occupational therapy were excluded from the study after identification, because the researcher was interested specifically in those children with non-specifiable etiology of delay.

The second group of delayed children was defined as having a non-

specifiable etiology of delay." That is, there were no major abnormal neurological signs and no specifiable etiology had been identified in each child's medical record by either the neurologist or paediatrician at the time of evaluation by the treating occupational therapist (or by the time the child was 48 months of age).

Procedures. The first step was the search of the medical records for the total sample of motor delayed children, which was 472 children. The second step was to identify the appropriate children for this study. One hundred and seventy-nine children were defined from this medical record search as having a non-specifiable etiology for their motor delay at 48 months of age. There were five goals in this medical record search: 1) To extract the medical history for each child; 2) to determine diagnostic eligibility for inclusion in the study; 3) to classify each child according to the two levels of therapy defined for the analytic (follow-up) phase of the study; 4) to establish the functional motor and chronological ages and thus percentage delay of each child at the time of his/her initial and discharge occupational therapy assessment; and 5) to obtain basic demographic information on each child and family. The data extracted from medical records and used subsequently as part of the basis for describing motor delayed children of non-specifiable etiology and the evolution of these children has been standardized for all children (Appendix A). A methodological issue does emerge in relation to the accuracy and reliability of these data since the demographic data were extracted from medical records and previous functional evaluations. Since there would be an explicit dependency

upon the accuracy of these records, every attempt has been made to minimize the negative effect that this could have had on the reliability of the data. The data form was piloted and standardized in consultation with an epidemiologist. One person collected all these data so that data in the medical records were all interpreted in the same manner. The correctness of this interpretation was verified on five cases with two people collecting data on the same subject and each collection was compared with the other. The data collected by the two persons were very similar. The accuracy of data collection from day to day was verified by the data collector who gathered data twice on five medical charts on two different days. The data collected were very similar from day to day.

A factor to be considered in evaluating the effectiveness of therapy was functional motor age of each child versus chronological age during the course of therapy. Functional motor age versus chronological age was not manipulated in this research, but was taken into consideration retrospectively. The functional age was determined for each child in the initial and subsequent assessments by the therapist and recorded in the patient's file. This has been done by the evaluative procedure standardized by Talbot (1974, 1977).

Chapter III

Results

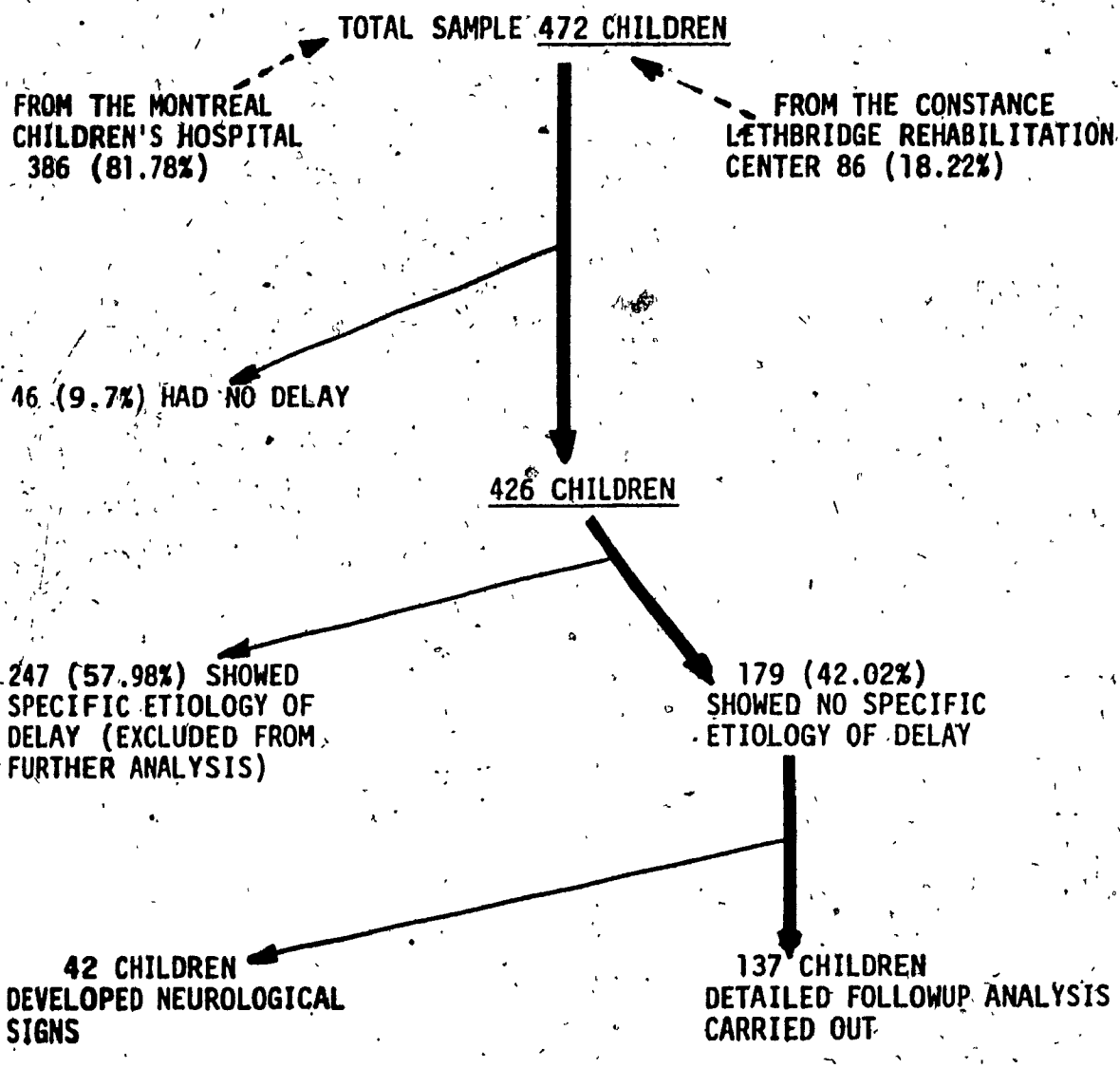
The results of the descriptive phase are presented in four major sections. These include: first, a description of the complete population referred for an assessment of a motor delay; and second, a description of the children with a non-specifiable etiology of delay; third, comparisons of the children with a non-specifiable etiology for their delay and children with a specifiable etiology for their delay; and fourth, comparisons between the two genders (male and female) and the three treatment groups (infrequent home programme therapy, intensive in-centre therapy, and no therapy).

Description of the Complete Population Referred for Assessment of Motor Delay by 48 Months of Age

A total number of 472 children born between 1974-09-30 through 1976-09-30 were referred before the age of 48 months to either the Montreal Children's Hospital (MCH) occupational therapy department or the Constance-Lethbridge Rehabilitation Centre (CLRC) paediatric service for assessment of and/or treatment for a developmental motor delay. (Three hundred and eighty-six (81.78%) were referred to the MCH and 86 (18.22%) were referred to the CLRC). As can be seen from Table 1, 46 of all the children (9.7%) who were assessed were found either not to have a delay, or to have no more than a 10% delay in only one of three categories of motor development (gross motor, fine motor, and perceptual-motor). Thirty-four of these 46 non-delayed children were

TABLE 1

DISTRIBUTION OF TOTAL POPULATION AT AGE 4 BY MOTOR DIAGNOSTITIC CLASSIFICATION



males and 12 were females. The other 426 children either had a minimum motor delay of 10% in at least two of the three areas of motor development, or they had a minimum motor delay of 20% in one of the three areas. Of the 426 motor delayed children, 247 (57.98%) had been assigned a specifiable etiology for their delay at the time of assessment, by neurological or paediatric evaluation; and 179 (42.02%) could not be assigned a specifiable etiology for their delay at the time of their assessment. All 247 children with specifiable etiologies within the first 48 months of life were excluded from the descriptive phase of the study. This was done because of the specific interest in those children with non-specifiable etiology of delay.

Comparisons of Children Who by the Age of 7 and 8 Years had a Non-specifiable Etiology for Their Delay With Those Who at the Same Age had a Specifiable Etiology for Their Delay

One hundred and seventy-nine children (42.02%) of the delayed population could be defined according to their medical diagnosis as having a non-specifiable etiology of delay at initial assessment. That is, there were no major abnormal neurological signs and no specifiable etiology had been established or identified by the neurologist or paediatrician at the time of evaluation by the occupational therapist. Table 2 provides a description of the 179 delayed children at age 7 and 8 years and the 46 non-delayed children by sex and treatment received. From Table 2 it can be seen that by the time these children were aged 7 to 8 years of age, 42 of the original 179 children who had had a non-specifiable etiology of delay (23.55%) now had, according to their

Table 2

Distribution of Population of Children, Who at Initial Assessment Had Non-specifiable Etiology for Their Delay, at Age 7 or 8 by Motor Diagnostic Classification, Gender, and Treatment Category

No Delay

	Male		Female		Total	
	Number	Percent	Number	Percent	Number	Percent
No treatment	32	94.12	12	100.00	44	95.65
Home treatment	01	02.94	00	00.00	01	02.17
Intensive treatment	01	02.94	00	00.00	01	02.17
Total	34	100.00	12	100.00	46	100.00

Specifiable Etiology of Delay

	Male		Female		Total	
	Number	Percent	Number	Percent	Number	Percent
No Treatment	05	22.7	07	35.0	12	28.6
Home Treatment	11	50.0	10	50.0	21	50.0
Intensive Treatment	06	27.3	03	15.0	09	21.4
Total	22	100.0	20	100.0	42	100.0

Table 2 (cont)

Non-specifiable Etiology of Delay

	Male		Female		Total	
	Number	Percent	Number	Percent	Number	Percent
No Treatment	15	17.4	10	19.6	25	19.0
Home Treatment	37	43.0	32	62.7	69	50.0
Intensive Treatment	34	39.6	09	17.7	43	31.0
Total	86	100.0	51	100.0	137	100.0

charts, been given a specifiable etiology. In general, these children with specifiable etiology had developed abnormal hard neurological signs (21 had abnormal electroencephalograms, 6 had abnormal computerized axial tomographic scans (C.T.scans), 2 had abnormal echoencephalograms and for 3 the skull x-ray showed a definite abnormality). For 5 children genetic abnormalities had been diagnosed, and for 6 children electromyelographic abnormalities were reported and 11 children had been identified with auditory abnormalities that were considered severe enough to interfere with the development of language. Two children were identified as being legally blind. There was some overlap in categories (i.e. some children demonstrated both abnormal EEGs and C.T. scans).

Method of analysis. Initially the χ^2 test for examining differences among the distributions was performed on the etiology distribution (specifiable versus non-specifiable etiology of delay). Subsequent to significant findings or to indicated differences a multivariant analysis of covariance (MANCOVA) was performed to examine differences among means. A MANCOVA was used as opposed to an ANCOVA when related variables were examined. In the MANCOVA subsequent univariate results were interpreted only when a significant overall multivariate F was obtained.

Etiology (specifiable versus non-specifiable etiology of delay) did not make a difference in terms of whether the delayed children received physiotherapy, $\chi^2(2, N = 179) = .24, p < .62$, occupational therapy, $\chi^2(2, N = 179) = 1.2, p < .25$, or speech therapy, $\chi^2(2, N = 179) = 1.1, p < .28$. As well, etiology made no difference in terms of whether a child received

no treatment, infrequent home programme treatment, or intensive in-centre treatment, $\chi^2(2, N = 179) = 3.4, p<.18$. The number and percentages in each category are shown in Table 3.

It was noted that there appeared to be a difference in response to therapy between children with a specifiable etiology and children with a non-specifiable etiology of delay. The mean values for initial assessment and discharge assessment can be seen in Table 4. The average percentage motor delay for the children with a non-specifiable etiology of delay at initial assessment was 30.62% while the average delay for the children who developed a specifiable etiology was 35.69%. At final assessment the average delay for the children with a non-specifiable etiology of delay had decreased to 21.73%, while for children with specifiable etiology of delay the average delay had changed minimally with an average delay of 33.00%.

It was hypothesized that therapy would have a differential effect on children with a non-specifiable etiology for their delay as compared to children with a specifiable etiology for their delay. In order to evaluate this hypothesis, a multivariate analysis of covariance (MANCOVA) in which the initial assessment scores served as covariates was used to evaluate treatment effectiveness for these two groups. Change in fine, gross, and average motor percentage delay from initial assessment to discharge was compared for the children with specifiable and non-specifiable etiology for their delay. This strategy was chosen in preference to the repeated measures design since comparisons of the two approaches have suggested that the former model more accurately reflects the data.

Table 3

Number and Percentages Treated (No Therapy, Infrequent Home Programme Therapy, and Intensive In-centre Therapy) in Occupational Therapy, in Physiotherapy, and in Speech Therapy by Diagnostic Category (Specifiable and Non-specifiable)

	General Treatment		
	Specifiable	Non-specifiable	Total
Not treated	12	25	37
	32.4%	67.6%	20.7%
Infrequent Home treatment	22	69	91
	24.2%	75.8%	50.8%
Intensive In-Centre treatment	8	43	51
	15.7%	84.3%	28.5%
			179
			100.0%

	Occupational Therapy		
	Specifiable	Non-specifiable	Total
No treatment	11	23	34
	32.4%	67.6%	19%
Treatment	31	114	145
	21.4	78.6%	81.0%
			179
			100.0%

Table 3 (cont)

	Physical Therapy		
	Specifiable	Non-specifiable	Total
No Treatment	29	102	131
	22.1%	77.9%	73.2%
Treatment	13	35	48
	27.1%	72.9%	26.8%
			179
			100.0%

	Speech Therapy		
	Specifiable	Non-specifiable	Total
No treatment	35	100	136
	25.7	74.3%	76.0%
Treatment	7	36	43
	16.3%	83.7%	24.0%
			179
			100.0%

Table 4

Mean Initial and Discharge Percentage Delay Scores for Fine Motor, Gross Motor and Average Motor Percentage Delay by Diagnostic Category (Specifiable and Non-specifiable)

Group	Fine Motor Delay N=82	Gross Motor Delay N=152	Average Motor Delay N=179
Non-Specifiable			
Initial	30.39	26.60	30.62
Discharge	21.48	19.34	21.73
Specifiable			
Initial	34.24	37.93	35.69
Discharge	36.14	36.09	33.00

in this study (Huck & McLean, 1975). Perceptual-motor percentage delay scores were not used in the analysis because more than 50% of the population did not have data recorded for the initial assessment.

The overall MANCOVA was significant, $F(3,73) = 8.16, p<.001$. As can be seen in Table 5, the univariate analysis of covariance demonstrated a significant difference between the groups for gross motor percentage delay, $F(1,75) = 8.88$, $p<.01$, fine motor percentage delay, $F(1,75) = 7.57$, $p<.01$, and average percentage motor delay, $F(1,75) = 7.31, p<.01$. The initial assessment measures used as covariates did not demonstrate significant differences for any of the three variables, fine motor percentage delay, $F(1,75) = .35$, $p<.55$, gross motor percentage delay, $F(1,75) = .08$, $p<.78$, or average motor percentage delay, $F(1,75) = .178$, $p<.67$. Thus the groups did not differ before therapy began. Thirty-eight percent of the cases were rejected because of missing data. The results of the MANCOVA supports the prediction that the children with a non-specifiable etiology of delay would make more gains by the time of discharge as a result of therapy than would the children who later were diagnosed with a specifiable etiology of delay.

Several variables were thought to be of potential importance in determining retrospectively whether a child who originally had a non-specifiable etiology of delay would be rediagnosed with a specifiable etiology of delay, or would remain with a non-specifiable etiology of delay at school age. The basis upon which these variables were considered was the fact that they were variables for which reliable information was available. Their means or ratios can be seen in Table 6. These

Table 5

Multivariate Analysis of Covariance Summary Table

Effects of Diagnostic Category (Specifiable and Non-specifiable)

on Change in Percentage Delay After Treatment

	df	MS	F	p
MANCOVA F - Pillais	3,73		8.16	<.001*
ANCOVA F's				
Gross Motor	1	2614.87	8.88	<.004*
Error	75	294.39		
Fine Motor	1	2535.61	7.57	<.007*
Error	75	334.78		
Average Motor	1	2060.07	7.31	<.009*
Error	75	281.00		

*$p<.01$

Table 6

Means or Ratios for Variables (Mother's Age at Birth of Child, Number of Prenatal and Postnatal Problems, Gestational Age, Birthweight, Total Number of Major Problems, Large or Small Head Circumference, and Socioeconomic Level) for each Diagnostic Category (Specifiable and Non-specifiable)

	Specifiable	Non-specifiable
Mother's age at child's birth (years)	25.30	27.80
Number of prenatal problems	.68	.80
Number of postnatal problems	1.00	.92
Gestational age (weeks)	39.60	39.50
Birthweight (grams)	3339.64	3114.45
Total number of major problems	1.60	1.00
Large head circumference/ normal head circumference	.13/1	.15/1
Small head circumference/ normal head circumference	.32/1	.12/1
Socioeconomic level-parental vocational status (scale 1-7)	4.95	5.20

variables (mother's age at the birth of the child, number of prenatal problems, gestational age, birth weight, number of postnatal problems, total number of major problems, ratio of large or small head circumference, and socioeconomic level) were entered into a discriminant analysis (Table 7). The stepdown discriminant analysis entered each of the variables in order of their discriminating values. The most discriminating variable was the total number of major problems, Wilks lambda = .95, $p<.03$. Mother's age at the birth of the child was entered next, Wilks lambda = .94, $p<.03$. Small head circumference was entered next, Wilks lambda = .93, $p<.05$. The F level of number of prenatal problems, gestational age, birthweight, number of postnatal problems, large head circumference and socioeconomic level were insuffient to enter into the equation. These variables did not, therefore, add significantly to the discriminating value of the other measures. It would appear that children with younger mothers (M specifiable = 25.3, M non-specifiable = 27.8), more major problems which included febrile seizures, minor structural abnormalities, and failure to thrive, (M specifiable = 1.6, M non-specifiable = 1.0) and more often having a head circumference in the lower 25 percentile (ratio small head circumferance versus normal head circumference specifiable = .32/1, ratio small head circumference versus normal head circumference non-specifiable =.13/1) were more likely to develop a specifiable etiology of delay than children with older mothers, fewer major problems and head circumference above the lower 25th percentile. These three variables correctly classified 71.21% of the children into the appropriate groups

Table 7

Discriminant Analysis Summary Table

Mother's Age, Number of Prenatal and Postnatal Problems, Gestational Age, Birthweight, Large or Small Head Circumference and Socioeconomic Level by Diagnostic Category (Specifiable or Non-specifiable)

Variables Entered	Wilk's Lambda[a]	p[b]	B[c]
Total number of major illnesses	.953	.03*	.58
Mother's age at child's birth	.938	.03*	-.44
Small Head circumference	.929	.05*	.43

*$p < .05$

Note: the Eigenvalue and its accompanying canonical correlation are .07 and .26 respectively.

[a]A measure of group homogeneity.

[b]Only those variables that significantly increase the distance between group centroids were entered.

[c]Standardized linear weights that optimize the distance between the group centroids (for the group with non-specifiable etiology of delay the centroid was -.139, and for the group with specifiable etiology the group centroid was .537 ($F = 2.61, 3/129df, p < .005$).

(specifiable or non-specifiable etiology of delay). This is greater than the chance level of 50%. The F value for the discriminant analysis equalled 2.61,3/129 df, and was significant at the $p < .01$ level.

Summary of Comparisons of 7 and 8 Year Old Children Who Had Non-specifiable Etiology of Delay with Children Who Had Specifiable Etiology Of Delay

No differences were found in the type or amount of therapy received by the children with a specifiable and non-specifiable etiology of delay. As predicted in the hypotheses the children with a non-specifiable etiology of delay benefited more from therapy than did the children with a specifiable etiology of delay. Variables such as number of major problems (included such factors as febrile seizures, failure-to-thrive, minor structural abnormalities), mother's age, and head circumference below the 25th percentile were able to discriminate between the specifiable and non-specifiable etiology of delay groups with a 71.2% accuracy.

Comparisons of Delayed Children with Non-specifiable Etiology of Delay and Children with No Delay

As can be seen in Table 8 only 20% of the mothers of the children with a non-specifiable etiology for their delay had more than one pregnancy difficulty (i.e. bleeding, needing to take medications, sickness). As well, only 34% had more than one minor birth problem (i.e. long labour, repeat caesarean birth, induced birth). Only 15% had more than one postnatal problem (i.e. jaundice, swallowing amniotic fluid, low APGAR). These children tended to have no history of seizure,

Table 8

Number of Prenatal, Perinatal, and Postnatal Problems, Seizures, Small or Large Head Circumference, Structural Abnormalities, Failure-to-thrive, Ear Infections, and Behavioural Problems for All Children

	No delay	Specifiable	Non-specifiable
Prenatal Problems (bleeding, sickness drugs taken, other)	0=54.8% 1=33.3% 2= 9.5% 3= 2.4%	0=50.0% 1=35.7% 2= 9.5% 3 or more=4.8	0=56.2% 1=24.1% 2= 9.5% 3 or more=10.2
Perinatal Problems (labour over 12 hours, drugs, caesarean, induced)	0=39.1% 1=26.1% 2=15.2% 3 to 4=10.9%	0=23.8% 1=23.8% 2=31.0% 3 to 5=21.4%	0=32.1% 1=29.9% 2=21.9% 3=12.5%
Immediate Postnatal Problems	0=63% 1=26.1% 2= 4.3% 3 to 5=6.6%	0=35.7% 1=31.0% 2=23.8% 3= 7.2%	0=47.4% 1=37.2% 2= 7.3% 3= 8.0%
Seizures	no=71.7% yes=28.3%	no=42.9% yes=57.1%	no=85.0% yes=15.0%
Large Head Circumference	no=89.1% yes=10.9%	no=85.7% yes=14.3%	no=85.0% yes=15.0%
Small Head Circumference	no=91.3% yes= 8.7%	no=76.2% yes=23.8%	no=85.0% yes=15.0%
Structural Abnormalities	no=89.1% yes=10.9%	no=66.7% yes=33.3%	no=79.6% yes=19.7%
Failure-to-thrive	no=87.0% yes=13.0%	no=69.0% yes=31.0%	no=79.6% yes=19.7%
Ear Infections	no=69.6% yes=30.4%	no=83.3% yes=16.7%	no=66.4% yes=33.6%
Behavioural Problems	no=80.4% yes=19.6%	no=76.3% yes=23.8%	no=80.3% yes=19.7%
Social Problems	no=81.3% yes=18.7%	no=69.4% yes=30.6%	no=74.3% yes=25.7%

measurable head circumference in the lower or upper 25th percentile and no structural abnormality. Thirty-four percent had had ear infections and 20% were hospitalized for failure-to-thrive.

As can be seen from Table 9 the average age of the mothers and fathers at the birth of these children was 27.8 (range 14 to 47) and 30.76 (range 18 to 55) respectively. Mother's average age is slightly higher than the mean age of 25.9 for mothers in the years 1974 to 1976 (Canada Central Bureau of Statistics, 1980), but it was no higher than for the non-delayed children. Forty-one percent of the children were first born, and 33 were second born, and the rest (26%) were later born. The average education level of these children's mother was 12.3 years (range 3 to 20 years) and of the fathers was 13.07 years (range 2 to 20 years). The average socioeconomic level on a scale of 1 to 7 was 5.22

The average age at which delays were identified was 27.7 months with a range from 6 to 47 months of age. The majority of delays were in the gross motor area (N=98,71.5%) with the average gross motor delay being 29% (range 0-96%). Twenty-seven of these children with a gross motor delay (19.7%) also had a delay in another area of motor development. Fifty-seven (41.6%) of the children had a language delay as well as a motor delay.

Several variables (prenatal, postnatal, family and child variables) were entered into a discriminant analysis to determine what combination of variables, if any, could be found to discriminate between children with a motor delay of non-specifiable etiology and children with no

Table 9

Means and Ranges of Mother's and Father's Age and Educational Level at Child's Birth, Socioeconomic Level and Language by Etiology (No Delay, Specifiable, and Non-specifiable)

	No Delay	Specifiable	Non-specifiable
Mother's Age (years)	27.80 (19-39)	25.32 (18-37)	27.82 (14-47)
Father's Age (years)	33.06 (19-41)	29.10 (19-43)	30.76 (18-55)
Mother's Education (years)	12.59 (6-16)	10.30 (4-18)	12.30 (3-20)
Father's Education (years)	14.00 (10-18)	12.17 (3-18)	13.07 (4-20)
Socio-Economic Level-Parental Vocational Status (scale 1-7)	4.79 (1-7)	4.95 (2-7)	5.20 (1-7)

Note: The numbers in parentheses are ranges.

motor delay. The means for these variables can be seen in Tables 8 and 9. These variables (mother's and father's age at the birth of the children, number of prenatal, perinatal, and postnatal problems, total number of major problems, large or small head circumference, febrile seizures, structural abnormalities, ear infections, and socioeconomic level) were entered into a discriminant analysis (Table 10).

The stepdown discriminant analysis entered the variables in order of their discriminating values. The most discriminating variable was chronic ear infections, Wilks lambda = .98, $p<.1$. Structural abnormalities was entered next, Wilks lambda = .96, $p<.1$. Number of perinatal problems, Wilks lambda = .94, $p<.088$ and number of social problems, Wilks lambda = .93, $p<.1$, were entered last. All other variables had an F level of insufficient value to enter into the equation.

It appeared that there was a tendency for children manifesting chronic ear infections, more minor structural abnormalities, more perinatal problems and more social problems than other children to have a delay of non-specifiable etiology. These four variables, however, could only correctly differentiate the children into the two groups at a rate of slightly greater than chance (56%). However, when average motor delay was entered into the discriminant analysis, Wilks lambda = .53, $p<001$, the correct discriminatory rate increased to 89.33%. Thus, it would appear that the only variable which is able to correctly discriminate the children with a delay of non-specifiable etiology from non-delayed children is the motor delay.

Table 10

Discriminant Analysis Summary Table

Parent's Age at Child's Birth, Number of Prenatal, Perinatal and Postnatal Problems, Large or Small Head Circumference, Febrile Seizures, Structural Abnormalities, Failure-to-thrive, Chronic Ear Infections, Number of Social Problems, and Socioeconomic Level by Delay (No Delay or Delay with Non-specifiable Etiology)

Variables Entered	Wilks lambda [a]	p [b]	B [c]
Chronic Ear Infections	.975	.1	.57
Structural Abnormalities	.957	.1	.55
Number of Perinatal Problems	.938	.088	.49
Number of Social Problems	.929	.1	.37

Note: The Eigenvalues and their accompanying canonical correlations are respectively .075 and .27 respectively.

[a] A measure of group homogeneity.

[b] Only those variables that significantly increase the distance between group centroids were entered.

[c] Standardized linear weights that optimize the distance between group centroids (for the group with non-specifiable etiology of delay the centroid was .15 and for the non-delayed group the centroid was -.48 $F=.16,4/102$ df, $p<.66$).

Description of the Children Who at Age 7 Still Had a Non-specifiable Etiology for Their Delay

When the delayed children were 7 and 8 years of age, 137 (32.16% of the original 472 children in the complete population) of the 179 children who had had a non-specifiable etiology at initial assessment continued to have a non-specifiable etiology for their delay. This is the particular population of interest in this study.

Tables 8 and 9 provide a description of the 137 children with a non-specifiable etiology of delay as well as those with a specifiable etiology for their delay and those who had no delay when they were assessed in occupational therapy. For the children with a non-specifiable etiology of delay the pregnancy and delivery were normal and no abnormality had been detected at birth.

Gender Distribution. Method of analysis. The female to male ratio was .6/1 (n female = 51, n male = 86) for all the children with a non-specifiable etiology of delay. The female to male ratio for the general population is .8/1 at birth. Thus the ratio of males to females was slightly higher in the study population than was present in the general population at birth.

The chi square analysis was used for examining differences in the distribution of gender. Subsequent to significant findings or to indicated differences a multivariate analysis of covariance (MANCOVA) to examine differences between the means was performed. The chi square analysis which was performed showed that there was no significant difference in the female/male ratio in each diagnostic category

(specifiable versus non-specifiable etiology of delay), $X^2(2, N = 179) = .77$, $p<.67$.

It was noted that although males accounted for 60.4% of the delayed population and females accounted for 39.6% of this population, almost four times as many males as females were intensively treated. When a chi square analysis was performed (gender by treatment) the chi square X^2 (2, $N = 137$) = 11.91) was significant at the $p<.001$ level. That is, there was a significant correlation for gender and treatment in the direction of more males receiving intensive treatment than females (Table 11). It was important to determine whether this difference in administration of intensive therapy was due to females being less responsive to therapy than males. Mean values for initial assessment and discharge assessments can be seen in Table 12. The average percentage motor delay for males and females at initial assessment was 23.97 and was 28.37 respectively. At discharge assessment the average delay for males was 21.13 and for females was 22.48. A multivariate analysis of covariance was used to compare males and females in terms of change in fine, gross, and average motor percentage delay from initial to discharge assessment. The overall MANCOVA was not significant, $F(3,90) = .267$, $p < .85$. This indicates that neither males nor females made more gains from initial assessment to discharge assessment in fine motor, gross motor or average motor development.

Treatment Distribution Method of analysis. Similar methods of analysis were used for treatment distribution as for examination of the etiology distribution. In general, the children with a non-specifiable

Table 11

Numbers and Percentage of Children by Gender and Treatment

	No treatment	Home Treatment	Intensive Treatment	
Male	15	37	34	86
	10.9%	27.0%	24.8%	62.7%
Female	10	32	09	51
	7.3%	23.4%	6.6%	37.3%
Total	25	69	43	137
	18.2%	50.4%	31.4%	100%

Table 12

Mean Initial and Discharge Percentage Delay for Fine Motor, Gross Motor and Average Motor Percentage Delay by Gender

Group	Fine Motor Delay	Gross Motor Delay	Average Motor Delay
Male			
Initial Assessment	23.50	23.41	23.97
Discharge Assessment	20.60	19.00	21.13
Female			
Initial Assessment	27.36	28.53	28.37
Discharge Assessment	24.26	22.14	22.48

etiology of delay were treated either by a home programme (n = 69, 50.37%) or by a more intensive in-patient treatment programme (n = 42, 31.39%). Twenty-five children (18.25%), however, were given no treatment in either type of programme. One cannot evaluate the difference among all three types of treatment on the change in motor delay because a final evaluation was missing for 78.4% of the children who were not treated. In order to evaluate the hypothesis that treatment would be effective in decreasing the percentage motor delay from initial to discharge assessment assessment a t-test was performed. As had been predicted there was a significant difference between average percentage motor delay at initial and discharge assessment for all delayed children with a non-specifiable etiology. The mean initial delay was 30.29 and at discharge the mean delay was 21.73. This difference was significant according to the t-test (75) = 4.8, $p<.01$. This supports the hypothesis that the average percentage motor delay would be significantly less at discharge assessment than at initial assessment.

It had been predicted that intensive therapy would be more effective than infrequent therapy in decreasing the percentage motor delay from initial to discharge assessment. The differential effect of treatment (infrequent and intensive) on the change in average percentage motor delay was analyzed by a multivariate analysis of covariance. This analysis demonstrated a significant $F(3,53) = 5.08$, $p<.01$. The mean values are presented in Table 13. As can be seen in Table 14 the univariate analysis of covariance demonstrated a significant difference between the groups for gross motor percentage delay, $F(2,51) = 7.7$,

Table 13

Mean Initial Assessment and Final Assessment Scores for Fine Motor, Gross Motor, and Average Motor Percentage Delays by Treatment (Infrequent Home Programme and Intensive In-Centre)

Group	Fine Motor Delay	Gross Motor Delay	Average All Motor Delay
Infrequent Home Programme			
Initial	31.20	31.87	31.54
Discharge	26.60	24.43	27.09
Intensive			
Initial	31.60	32.10	32.57
Discharge	23.05	21.42	22.40

Table 14

Multivariate Analysis of Covariance Summary Table

Effects of Treatment Condition (Infrequent Therapy and Intensive Therapy) on Fine Motor, Gross Motor and Average Motor Percentage Delay for Children with Motor Delay of Non-specifiable Etiology

	df	MS	F	p
MANCOVA F - Pillais	3,53		5.08	.006**
ANCOVA F's				
Gross Motor	2	1893.26	7.67	.004**
Error	51	246.61		
Fine Motor	2	1050.90	3.33	.03*
Error	51	315.54		
Average Motor	2	1142.49	4.41	.008**
Error	51	252.70		

*p<.05

**p<.01

p,.01, for fine motor percentage delay, $F(2,51) = 3.3$, $p<.05$, and for average percentage motor delay for all motor delays, $F(2,51) = 4.4$, $p<.01$. The pretest scores used as covariates did not demonstrate significant differences between the groups prior to the intervention for gross motor initial assessment scores, $F(2,51) = .65$, $p<.42$, for fine motor initial assessment scores, $F(2,51) = .25$, $p<.61$, or for average motor initial assessment scores, $F(2,51) = .75$, $p<.39$. As had been predicted in the hypotheses, intensive therapy caused a greater decrease in motor delay than did infrequent home therapy.

It was of interest to try to identify what variables, if any, influenced whether each child was treated by home treatment, by intensive treatment or not at all. A stepdown discriminant analysis was performed: the variables included age of the child at initial assessment, gender of the child, socioeconomic level and average percentage motor delay at initial assessment. These variables were chosen because it was thought that they might influence the treatment received. The mean values for these variables are shown on Table 15.

The stepdown discriminant analysis entered each of the variables in order of their discriminating values. The most discriminating variable, socioeconomic level was entered first, Wilks lambda = .84, $p<.005$ and gender was entered next, Wilks lambda = .76, $p<.001$. The F level for age and average percentage motor delay at intake was insufficient to enter into the equation. These two variablesdid not, therefore, add significantly to the discriminating value of the other two variables. The two variables, gender and socioeconomic level however, could only

Table 15

Mean Age, Gender, Average Delay on Initial Assessment and Socioeconomic Level for each Treatment Level (No Therapy, Infrequent Home Programme Therapy and Intensive Therapy) for the Children with a Motor Delay of Non-specifiable Etiology

	Age (months)	Gender Male/Female	Average Delay (Percentage)	SES (Parental Vocational Status - scale 1-7)
No Therapy	22.0	15/10	29.5	6.0
Infrequent Therapy	25.2	37/32	29.7	5.6
Intensive Therapy	27.6	34/9	31.9	4.5

classify the children correctly into the three groups at the level of chance (32.46%)(Table 16). Upon closer look it was determined that 65% of the no treatment group and 60% of the intensive treatment group were correctly classified while only 1% of the home treatment group was correctly classified. It would appear that children of any gender or socioeconomic level were treated by home treatment, while families from a higher socioeconomic level tended to have their delayed sons treated by intensive treatment, and families from lower socio-economic levels tended to have proportionately more delayed daughters not treated.

Summary of Description of the Children Who at Age 7 still Had a Non-specifiable Etiology for Their Delay

Thirty-two percent (n=137) of the original delayed population (N=426) still had a delay of non-specifiable etiology at age 7 and 8. No variables other than motor delay could be found which differentiated the children with a motor delay of non-specifiable etiology from the non delayed children.

Significantly more males were intensively treated than were females and more males were referred for evaluation of a potential delay when there was no delay. Gender did not influence the effectiveness of the treatment on the children.

No variables could be found that could discriminate whether a child would receive no therapy, infrequent therapy, or intensive therapy. However, when the data were examined more closely it could be shown that gender and socioeconomic level correctly placed the children in either the intensive treatment or no treatment group with a 63% accuracy.

Table 16

Discriminant Analysis Summary Table
Gender of Child, Age of Child and Average Percentage Motor Delay at Initial Assessment and Socioeconomic Level for Children with a Motor Delay of Non-specifiable by Treatment

	Wilks Lambda[a]	p[b]	B[c]	
Variables Entered				
			Functions 1	2
Socioeconomic level	.84	.005*	.64	.76
Gender	.76	.002*	.72	-.09

*p<.01

Note: The Eigenvalues are respectively .31 and .0001 for function 1 and 2 and their accompanying canonical correlations are .48 and .01 for function 1 and 2.

[a]A measure of group homogeneity.

[b]Only those variables that significantly increase the distance between group centroids were entered.

[c]Standardized linear weights that optimize the distance between group centroids (for the group with no treatment the function 1 centroid was .53 and function 2 was -.005, and for the group with intensive treatment function 1 was -.55 and function 2 was .000. (No treatment and home treatment, $F(1,62)=74, p<.7$; no treatment and intensive treatment $F(1,62)=1.3, p<.24$; home treatment and intensive treatment, $F(1,62)=11.25, p<.001$.

As hypothesized, it was shown that the average percentage motor delay was significantly less at discharge than upon initial assessment for all children with a delay of non-specifiable etiology who received therapy. It was shown, as well, that for all children with a delay of non-specifiable etiology, the intensive in-centre therapy was more effective than infrequent therapy in reducing the percentage delay of the children. Thus the hypothesis that intensive therapy would be more effective in reducing delay by the time of discharge from therapy was supported.

Chapter IV

Method

Analytic (follow-up) Phase

Subjects. The sample selected from the original 472 children for the analytic (follow-up) phase consisted of 72 children. The criteria for admission depended on the group assignment of each child. The control group consisted of 24 children born after 1974-09, who were referred for an evaluation at either the Montreal Children's Hospital (MCH) or the Constance-Lethbridge Rehabilitation Centre (CLRC), were assessed and found to have no motor delay (that is, they were found either not to have a delay or to have a delay between 1% and 10% in only one of the three categories of motor development - gross motor, fine motor, and perceptual-motor), and who should have completed level one in school but not level three at the time of follow-up. The non-delayed group of children were equated with the two groups of delayed children for chronological age and socioeconomic status at the time of evaluation as closely as possible. Socioeconomic level was based on the Hollingshead scale (Hollingshead, 1957). The samples used to standardize the scale values for each measurement instrument provided another comparison sample which would form a data base against which the clinical results of the delayed groups could be evaluated comparitively.

The two treatment groups consisted of 48 children who were born after 1974-09, were referred for an evaluation at one of the two treatment centres, and were assessed and found to have a motor delay (that is, the child had a minimum motor delay of 10% in at least two

categories of motor development, gross motor, fine motor, or perceptual-motor, or had a minimum motor delay of 20% in one of the three categories). The etiology for the motor delay of these 48 children was non-specifiable, and all 48 children should have completed in school level one and not level three at the time of follow-up.

The delayed sample was further divided into two groups (24 in each) on the basis of the major independent variable,'intervention therapy'. There were two categories of this variable which can be differentiated as either 'infrequent home programme therapy' or as 'intensive in-centre therapy'. The children in these two groups were ranked for chronological age, initial functional motor age and duration of therapy. These delayed children were then matched on these variables as closely as possible.

The children receiving infrequent home programme therapy were treated by an occupational therapist for no more than 10 hours within the year following the assessment. The children who received intensive in-centre therapy were treated for at least an average of 6 hours per week and for a minimum of 12 months.

The major determinant as to whether children received home programme therapy or in-centre therapy was geographic , that is, all the children who had received home programme therapy in the follow-up phase of the study lived in suburban parts of the Greater Montreal area. The decision to use as subjects, children who lived outside the Greater Montreal area in the infrequent home programme group, was made in order to try to control for the possible effect of parental motivation on outcome of the children's motor development. Thus it could not be said

that a child did not receive intensive therapy because the parents were not motivated to bring him/her for such intensive therapy. A methodological issue does emerge in relationship to the potential confounding variable produced by choosing children in the two groups who lived in different areas. However, most of the children who lived outside of the Greater Montreal area did not live in rural areas but still lived in highly populated areas with good schools and other facilities. The major difference was that the large hospitals or the rehabilitation centres which offered treatment services for young delayed children were not easily accessible. Therefore, it is not felt that these modest differences in environment would cause a major confound to the reliability of the results.

Children were excluded from the delayed groups in the follow-up study if a diagnosis had been made at any time suggesting a specific disorder such as cerebral palsy or mental retardation or a severe emotional disorder. A child was also excluded if there had been a major traumatic injury or illness after initial evaluation and diagnosis of non-specifiable etiology, and if the motor delay was determined to be the result of impaired hearing, sight or other such physical abnormality.

In the intensively treated group there were 13 children who had an average percentage motor delay between 15% and 35% and 11 who had a delay from 36% and 55% at initial assessment. In the home treated group there were 14 children who had an average motor delay from 13% and 35% and 10 who had a delay from 36% and 55% at initial assessment. A

description of the subjects in the analytic phase of the study is provided in Table 17.

Institutional Intervention Therapy

The occupational therapy department at the Montreal Children's Hospital and the Paediatric Service of the Constance-Lethbridge Centre are responsible for assessment and treatment of children between zero and 48 months of age who present with motor delays including those children diagnosed as having clearly defined neurological symptoms as well as those without such symptoms. The departments have had the same head-of-department, minimal staff turnover, and no major methodological changes in the treatment programme during the time period used to determine the total patient sample.

The therapy programmes per se have been based on Llorens' (1970) definition of intervention therapy - it is "a facilitation process towards mastery of life tasks". The direct implication is that the therapy has been concerned with enhancing each child's development in spheres of living (physical, perceptual-cognitive, social and emotional). The method of treatment which has been used in both the infrequent home programme therapy and intensive in-centre therapy is a combination of developmental remedial motor exercises and the facilitation approach. The exercises have been aimed initially towards sequential normalization of postural tone and gross motor co-ordination so as to develop and increase sensory-motor interaction with the environment as described for example, by Bobath and Bobath (1972). The subsequent exercises have been directed towards improving a child's abilities in fine motor, and

Table 17

Group Mean and Ranges of Age and Average Percentage Motor Delay at Initial Assessment and Group Distribution by Gender and Handedness and Socioeconomic Status

	Infrequent Home Therapy	Intensive In-centre Therapy	No Therapy
Variables			
Age in Months at Initial Evaluation	26.01 (06-47)	29.90 (07-47)	28.62 (08-47)
Age in Months at Follow-up	96.71 (84-108)	99.08 (84-114)	96.71 (84-112)
Average Percentage Delay At Initial Evaluation	29.95 (13-55)	35.37 (15-55)	1.45 (0-7)
Sex Male	12.00	20.00	16.00
Female	12.00	4.00	8.00
Hand Dominance			
Right	16.00	18.00	20.00
Left	5.00	3.00	3.00
Mixed	3.00	3.00	1.00
Socioeconomic Status	5.50 (1-7)	4.50 (1-7)	4.90 (1-7)

Note: Numbers in parentheses are ranges.

adaptive and self-care skills. The latter are skills upon which the more cognitive and social skills are developed subsequently.

The infrequent home programme therapy was based on instruction by the occupational therapist to the parent in assisting each child in the appropriate motor exercise regime. This form of therapy was generally provided by the occupational therapy department at the Montreal Children's Hospital.

The intensive in-centre therapy programme at the Constance-Lethbridge Centre included a co-ordinated multidiscipline programme planned to the individual needs of child and family and was based on developmental approach. This service was offered to families of infants, toddlers, and preschoolers (0-5 years) with developmental delays and management difficulties. The team for the intensive programme included a physiotherapist, a nursery therapist, a psychologist, a social worker, a speech pathologist, a paediatrician, a dietician, and a nurse as well as the occupational therapist who would work with the child and family. The occupational therapist and physiotherapist work together to assess and treat developmental delays in the area of fine and gross motor development, perceptual and sensori-motor planning. The occupational therapists assess and treat developmental delays in the areas of behavioural and personal social skills. These therapists also consult and assist in programmes within the family unit, preschool class or community group. In contrast to the children in the home programme therapy, children who were intensively treated generally received in addition to occupational therapy and

physiotherapy, therapy from the speech pathologist. The speech pathologist provided individual or small group treatment to help develop vocabulary, well formulated sentences, correct sound and pronunciation, and appropriate use of communication skills. The speech pathologist also offered parent training courses to encourage language stimulation and basic teaching strategies for developing pre-language and early language skills. The intensively treated child would always spend some time between therapies or before and after therapy in the nursery. The nursery therapist was responsible for supervision and facilitation of the child's play and social interactions, and was particularly interested in the child's ability to function in a group, and in the child's exploratory and creative drives. A social worker and psychologist were available for the family and the child. The psychologist assessed and treated the child's behavioural and emotional problems within the child's family whenever it was considered necessary. The social worker offered counselling or practical help with knowledge of community resources. A paediatrician was responsible for the medical and neurological evaluation of the children enrolled in the intensive in-centre programme. Medical intervention was adjusted according to the specific needs of each child. A dietician and nurse were available for consultation with the team. The nurse was responsible for any acute medical problems and was available to parents for support counselling. The dietitian was responsible for nutrition programmes and was available to parents for nutritional assessment and counselling for the children. When a child entered the intensive in-centre programme, a treatment

schedule was formulated which included individual, group, and nursery school programmes. The parents were expected to be actively involved by observing and participating in therapy sessions and by attending periodic parent conferences for information and discussion.

Pre andPosttest and Follow-up Measures

Functional measures - Motor measures. Each occupational therapist who would perform both initial and discharge evaluation of each child could derive a functional age, using the Talbot (1974; 1977) scales. The Talbot scales were standardized on a Montreal sample of children, aged 0 to 6 years, obtained from general clinics at Ste. Justine Hospital. This scale consists of separate motor scales for fine motor co-ordination, gross motor co-ordination and perceptual motor co-ordination. A functional age in each area of motor development can be derived from this scale. An average percentage delay was obtained by dividing the average functional age of the three motor areas by the chronological age and multiplying by 100. This percentage would be subtracted in turn from 100 to establish the percentage delay.

The follow-up motor measure was the Bruinicks-Oseretsky Test of Motor Proficiency (Bruinicks, 1978), a test of evaluation of motor functioning appropriate for 7-to 8-years-old. For the purpose of this study the short form was used. The short form which has eight subtests and scores, relates well to the long form for functional motor age. The short form can be used for screening purpposes when a specific diagnosis is not necessary. The eight subtest scores include evalua tion of running speed and agility, balance, bilateral co-ordination, strength,

upper limb co-ordination, response speed, visual-motor control, and upper-limb speed and dexterity. This test has been standardized on 259 children aged 7, 8, and 9. This particular motor test was chosen because it is used frequently by occupational and physiotherapists to evaluate motor function, and because a developmental motor age and thus percentage motor delay could be obtained. This follow-up percentage motor delay could then be compared with initial and discharge occupational therapy assessment scores. Studies have been carried out comparing mentally retarded children and learning disabled children to normally developing children using this test (Bruinicks,1978). Seventy-two mildly retarded children (IQ 61-75) were compared to 72 normal subjects matched for chronological age. It was shown that subjects with normal IQ's performed significantly better than mildly retarded subjects of the same chronological age on all parts of the Bruinicks-Oseretsky Test (Bruinicks,1978). Fifty-five learning disabled children (who were two years below grade level in reading and were not retarded) were also compared to children matched for chronological age who were neither learning disabled and nor retarded. The latter performed significantly better than the learning disabled children of the same chronological age on the Bruinicks-Oseretsky Test of Motor Proficiency (Bruinicks, 1978).

Functional measures - Intelligence measure. Intelligence was measured using the Wechsler Intelligence Scale for Children - Revised (Wechsler, 1974). The current edition of the Wechsler Intelligence Scale for Children - Revised (WISC-R) has 12 subtests. For the purposes

of this study all the requisite performance subtests and the four verbal subtests, arithmetic, vocabulary, comprehension and digit span were used. Information and similarities were not used because they show strong correlation with vocabulary for the age group in this study and thus would provide a duplication of information. This test was standardized on a total population of 2200 American children aged 6 1/2 to 16 1/2. This assessment tool was chosen because it has been used frequently in past research, it correlates highly with other intelligence tests (i.e.full scale IQ correlates at .82 level with Stanford Binet IQ and .95 with Wechsler Adult Intelligence Scale) and it is used frequently in the identification of a learning disability (M. Gollick, personal communication, September, 1983).

Functional measures - Reading measure. Reading level was measured using the Stanford Diagnostic Reading Test (Karlsen, Madden, & Gardner,1976). This test measures the major components of the reading process. The word reading and reading comprehension sections of this test were used. The 'red' level, which is designed for children to use at the end of level one, in level two and for low-achieving children in level three, was selected because most of the children in this study should have been in level two at the time of follow-up.

The Stanford Diagnostic Reading Test differs from most reading survey or achievement tests in two important ways. First, since its primary purpose as a diagnostic instrument is to identify a pupil's strengths and weaknesses more detailed coverage of reading skills is provided. Second, because it places more emphasis on the low achiever

than on the high achiever, the test contains more easy questions than do most reading tests. Since it was felt that many of the motor delayed children could potentially have reading difficulties (Henderson & Hall, 1982), a reading test with more easy items was chosen.

Behavioural and psychological measures - Perceived competence and social acceptance measure. Perceived competence and social acceptance was measured by the Pictorial Scale of Perceived Competence and Social Acceptance for Young Children (Harter & Pike, 1984). It was felt that measures other than functional measures would be important in the evaluation of the effectiveness of therapy (Zigler & Trickett,1978). Four subscales are included in this measure providing a profile of scores across the following domains: cognitive competence, physical competence, peer acceptance and maternal acceptance. The version for the children in the first and second levels at school was chosen on the basis of S. Harter's (personal communication, September, 1983) suggestion that this would be the most appropriate scale if there were to be any delayed children in the sample. Harter's test was chosen over other tests (i.e. Coopersmith, 1967, Piers & Harris, 1969) because instead of calculating a single score Harter tries to assess self judgement separately within the specific domains identified above.

Behavioural and psychological measures - Behavioural measure. The Child Behaviour Check List (CBCL) (Achenbach,1982) was used as a measure of behaviour. The CBCL was designed to assess a wide variety of behaviours that are of clinical concern. Results can be scored on a Child Behaviour Profile which is a standardized profile for portraying and

categorizing the behavioural disorders and competencies of clinically referred children (referred for behavioural problems) (see Achenbach 1978; 1979; Achenbach & Edelbrock, 1979; Edelbrock & Achenbach, 1980 for more information about this test). Profiles have been standardized separately for each sex at ages 4 to 6, 6 to 11, and 12 to 16 years. Those subscales where Achenbach found gender differences (externalization and internalization) were eliminated from analysis since the subjects in this study could not be matched for gender. The scale for age 6 to 11 was used. These scales were normalized on 50 cases from each gender for each age (1300 in total), in such a way as to maintain a roughly normal distribution with respect to socioeconomic status.

The Child Behavior Check List consists of 20 social competence items and 118 behaviour problem items. Parents fill out this form and it has been found that there is high inter-parent reliability, $r = .985$ for behaviour problems and $r = .978$ for social competence.

Achenbach and Edelbrock (1981) have standardized their scale on normal children (children never referred to a psychological or psychiatric centre for treatment) and on a clinical sample of children who had been referred within the last year and accepted for treatment for a psychological or psychiatric problem. It was felt that this could be an appropriate evaluation for the children in the present study because motor delayed children have been identified as having behavioural problems (Gillberg & Gillberg, 1983; Oberlaide et al., 1979).

Socioeconomic status measure. Socioeconomic status (SES) was scored on Hollingshead's (1957) 7-step scale of occupation as reported in the child's medical record (all medical records had the occupation of the parent on the intake form). If both parents worked, the higher-status occupation was used to score SES. Occupation was chosen as the single index of SES because this information could be obtained on significantly more children than parents' educational level plus occupation. Hollingshead and Redlick (1958) found occupation to be the best single index of their highly detailed social class stratification. In addition, occupation is more likely to be reported in a uniformly scalable manner and occupational level is more likely to have a stable meaning in terms of SES than are either income or education, for which levels have been changing radically over time and from region to region (Hollingshead, 1957).

Procedures. The second phase of the study was the follow-up. The first step was to locate the families of the 48 delayed children and the 24 non-delayed children by telephone and to ask them if they were willing to receive a letter of request soliciting permission for and co-operation in two home visits (see Appendix B). The details of the letter centered around what would be accomplished in the home visits. The second step was a telephone call follow-up to the letter. The purpose of the second telephone call was to arrange for the first of the two home visits with both the child and family in order to evaluate the child. If contact was not made by mail and/or telephone then attempts were made to locate the child by communicating with the original

referring physician and by reviewing current telephone directories. If the selected child could not be located, a back-up match was located. The third step was to make the individual home visits for the children in the three groups. Two visits of approximately 2 to 3 hours each were planned. The aims of the first visit was to acquaint the child and parents with the interviewer, to have a consent form signed (see Appendix C), to complete a status up-date questionnaire (as in Appendix A), and to begin the assessment of the child. The second visit was to complete the assessment. The psychometrician (B.A. psychology) who administered this assessment was trained by the researcher in the administration of the five standardized measurement instruments. The psychometrician performed all 72 evaluations and was blind as to the diagnostic or treatment category for all children in the three groups. The parents were asked to complete the Achenbach Behavior Checklist (1982). This is a measure to identify major behavioural problems within the child. The psychometrician tested the child for level of motor functioning using the Bruinicks-Oseretsky Test of Motor Proficiency (1978) and administered WISC-R (Wechsler, 1974) subtests. The test of reading proficiency (Stanford Reading Test, Karlsen, Madden & Gardner, 1976) was administered. The Pictorial Scale of Perceived Competence and Social Acceptance for Young Children (Harter & Pike,1984) was also administered. Information directly relating to retention-in-level (grade) or special class or special school was obtained from the parents for all children.

As soon as all the assessments were finished, the parents were sent

a thank-you letter (Appendix D) and the data were recorded by the investigator. After all data were entered, a complete report was written based on the evaluation. This report gave parents detailed information of the results and suggestions as to how any persisting difficulties might be ameliorated (Appendix E). These reports were written according to the protocol for a written assessment at the McGill-Montreal Children's Hospital Learning Centre, Montreal, Quebec, since the investigator is a clinical psychologist who received part of her clinical training at this centre.

Chapter V

Results

The results of the analytic phase (follow-up study) are presented in three major sections. These include: first, description and comparisons among the three treatment groups (delayed children receiving infrequent home programme therapy, delayed children receiving intensive in-centre therapy and non-delayed children receiving no therapy); second, description and comparisions of delayed children between the two age groups at which therapy began (assessed and treated before age 2 or between age 2 and 4) and third, comparisons between the motor delayed children who were retarded and non-retarded at follow-up.

Treatment Populations Prior to the Intervention

Method of analysis. Initially, univariate analyses of variance (ANOVA) were performed in order to determine whether the control variables of chronological age and socioeconomic level resulted in equivalent groups when the non-delayed children were included; and whether the control variables of initial functional motor age and duration of therapy actually insured a statistically acceptable degree of homogeneity between the infrequently home programme treated children and the intensively in-centre treated children. These analyses used treatment group as the independent variable (delayed children receiving infrequent home programme therapy, delayed children receiving intensive in-centre therapy, and non-delayed children receiving no therapy). Age (in months), duration of therapy (in months), severity of delay as reflected in the average percentage motor delay when the children were

initially assessed by the occupational therapist, and socioeconomic level as measured by the Hollingshead scale (1957) were the dependent variables.

The univariate analysis revealed that there were no initial differences among the three groups (delayed children receiving infrequent home programme therapy, delayed children receiving intensive in-centre therapy, and non-delayed children receiving no therapy) for the control variables of chronological age and socioeconomic level or between the two delayed groups in average motor delay and duration of therapy (Appendix F).

Differences Among the Treatment Groups

Method of analysis. Initially a multivariate analysis of variance (MANOVA) was performed to examine differences among means. A MANOVA was used as opposed to an ANOVA because of the theoretical prediction that the variables would be related and because correlations between variables were in the predicted direction (Turner,1978). In this analysis treatment groups (delayed children receiving infrequent home programme therapy, delayed children receiving intensive in-centre therapy, and non-delayed children receiving no therapy) was the independent variable and the motor measure (Bruinicks-Oseretsky), reading measure (Stanford Diagnostic Reading Test),intelligence scores (Wechsler Intelligence Scale for Children - Revised), perceived competence and social acceptance measures (Pictorial Scale of Perceived Competence and Social Acceptance) and behavioural measures (Achenbach Behavioural Checklist) were the dependent variables. Since the MANOVA

was found to be significant, $F(30,60) = 1.51$, $p<.05$ all subsequent univariate results were interpreted.

Functional variables - Motor scores. Method of analysis. In order to evaluate the hypothesis that non-delayed children would perform better at follow-up on motor measures and that intensive in-centre treated delayed children would perform better than infrequent home treated delayed children on motor measures as measured by the Bruinicks-Oseretsky Test of Motor Proficiency, the univariate results following the significant MANOVA were interpreted. The means are shown in Table 18. As can be seen in Table 19, the univariate analysis of variance (ANOVA) demonstrated a significant difference among the groups for the motor measure, $F(2,67) = 18.9$, $p<.001$.

When Least Significant Differences (LSD) tests were performed the non-delayed no therapy group had scored significantly higher for motor skills than both the delayed home programme therapy group, $p<.05$, and the delayed intensively in-centre treated group, $p<.05$. This supports the hypothesis that the motor performance scores for both delayed groups of children would be significantly lower than scores of the non-delayed group.

There was no difference in motor function at follow-up between the delayed home programme therapy group and the delayed intensive in-centre therapy group. A multivariate analysis of covariance (MANCOVA) was performed in order to examine more closely the differential effect of infrequent home programme therapy and intensive in-centre therapy on motor function of delayed children at follow-up. Initial assessment

Table 18

Functional Variables at Follow-up

Mean Motor Scores, Percentile Ranks and Grade Equivalents for Reading, for Intelligence Scores for each Treatment Group (Delayed Infrequent Home Programme Therapy, Delayed Intensive In-centre Therapy and Non Delayed No Therapy)

	N=22 Delayed Infrequent Home Therapy	N=20 Delayed In-centre Therapy	N=24 Non-Delayed No Therapy
Motor Scores (Bruinicks-Oseretsky)	12.08	4.08	40.50
Reading Scores Percentile Ranks	30.00	25.67	59.20
Grade Equivalent (Stanford)	1.70	1.60	2.50
Intelligence Scores (WISC-R)			
Arithmetic Scaled Score	7.05	7.00	11.12
Vocabulary Scaled Score	7.64	8.45	12.21
Comprehension Scaled Score	7.04	8.10	11.33
Digit Span Scaled Score	6.80	6.10	9.12
Picture Completion Scaled Score	8.50	9.40	11.58

Table 18 (cont)

	N=22 Delayed Infrequent Home Therapy	N=20 Delayed In-centre Therapy	N=24 Non-delayed No Therapy
Picture Arrangement Scaled Score	6.10	7.43	11.30
Block Design Scaled Score	7.40	7.75	11.70
Object Assembly Scaled Score	7.40	7.90	10.30
Coding Scaled Score	6.50	5.50	9.70
Verbal Subscale Score	81.64	84.15	105.00
Verbal above 79	N=13 96.69	N=12 95.25	N=24 105.00
Verbal below 80	N=9 60.25	N=8 67.25	N=0
Performance Subscale Score	81.5	84.3	106.29
Performance Above 79	N=12 100.83	N=13 95.7	N=24 106.29
Performance Below 80	N=10 58.30	N=7 63.71	N=0
Full Intelligence	80.04	83.05	106.00
Full Intelligence Above 79	N=12 98.16	N=13 94.00	N=24 106.00
Full Intelligence Below 80	N=10 58.30	N=7 62.71	N=0
Untestable	N=2	N=4	N=0

Table 19

Multivariate Analysis of Variance Summary Table of Functional Variables Effects of Treatment Condition (Delayed Infrequent Home Programme Therapy, Delayed Intensive In-centre Therapy, and Non-delayed No Therapy) on Motor Scores, Intelligence Scores and Reading Scores

	df	MS	F	p
MANOVA F - Pillais	30,69		1.51	<.05*
ANOVA F's				
Motor Scores	2	8652.77	18.90	<.001**
Error	67	457.38		
Reading Scores	2	5310.26	4.13	<.02*
Error	67	1283.10		
Intelligence Scores				
Arithmetic	2	116.42	10.24	<.004**
Error	67	11.36		
Vocabulary Scores	2	144.14	13.13	<.005**
Error	67	10.97		
Comprehension	2	125.02	12.04	<.001**
Error	67	10.38		
Digit Span	2	56.75	4.78	<.02*
Error	67	11.84		
Picture Completion	2	61.16	5.40	<.007**
Error	67	11.28		

Table 19 (cont)

	df	MS	F	p
ANOVA F's				
Picture Arrangement	2	164.05	11.45	<.005**
Error	67	14.32		
Block Design	2	129.48	8.46	<.006**
Error	67	15.29		
Object Assembly	2	61.76	6.49	<.007**
Error	67	9.57		
Coding	2	103.46	8.49	<.006**
Error	67	12.17		
IQ Verbal	2	3865.05	12.00	<.005**
Error	67	322.06		
IQ Performance	2	4358.72	12.28	<.005**
Error	67	354.82		
IQ Full	2	4758.50	13.66	<.02*
Error	67	348.11		

*$p<.05$
**$p<.01$

motor scores were compared to discharge assessment and to follow-up assessment scores. This strategy was chosen in preference to the repeated measures design since variations in initial scores which could affect outcome results would be controlled by the covariance approach (Huck & McLean, 1975). Mean values are presented in Table 20. At follow-up, the intensively treated children again appeared to be somewhat more delayed. The overall MANCOVA statistic was significant, $F(2,30) = 7.0$, $p<.01$.

As can be seen in Table 21, there was a difference when comparing the initial assessment to discharge assessment $F(1,31) = 9.2$, $p<.01$ and a difference when comparing initial assessment to follow-up assessment, $F(1,31) = 10.5$, $p<.001$. The initial scores used as covariates were not significant for average motor delay at initial assessment when compared by treatment group for discharge assessment $F(1,31) = .56$, $p<.58$, and follow-up assessment $F(1,31) = 1.93$, $p<.16$.

These results indicated that the decrease in average percentage motor delay was larger at the termination of therapy for the children who were given in-centre intensive therapy than for the children who were given infrequent home programme therapy. This finding supports the hypothesis that intensive therapy would have a greater effect on decreasing the children's motor delay in the shorter term than would infrequent therapy. This decrease in motor delay for the intensively treated group, however, was not maintained at follow-up. The children receiving infrequent home programme treatment maintained their decrease in delay and at follow-up their delay was significantly lower than the

Table 20

Mean Values for Average Percentage Motor Delay at Initial and Discharge Assessment and at Follow-up Assessment by Treatment (Infrequent Home Programme Therapy and Intensive In-centre Therapy)

	Initial Assessment	Discharge Assessment	Follow-up Assessment
Infrequent Home Programme Therapy	29.95	25.13	25.36
Intensive In-centre	35.37	20.65	31.13

Table 21

Multivariate Analysis of Covariance Summary Table

Effects of Treatment Condition (Infrequent Home Programme Therapy and Intensive In-centre Therapy) on Average Percentage Motor Delay at Dischage Assessment and at Follow-up Assessment with Initial Assessment Scores Used as Covariates

	df	F	p
MANCOVA F - Pillais	2,30	7.01	<.003*
Contrasts			
Average Percentage Motor Delay at Discharge Assessment	1 31	9.17	<.005*
Average Percentage Motor Delay at Follow-up Assessment	1 31	10.48	<.003*

*p<.01

intensively treated children. Thus, the hypothesis that intensive therapy would have greater longer-term effectiveness than infrequent therapy on motor delay was not supported.

Functional variables - Intelligence scores at follow-up. Method of analysis. The same method of analysis as for the motor scores was used to determine potential differences between motor delayed children and non-delayed children and the effect on motor delayed children of infrequent home programme therapy or intensive in-centre therapy on intellectual performance as measured by the Wechsler Intelligence scale for Children-Revised (Wechsler,1974).

Mean values can be seen in Table 18. As can be seen in Table 19, the ANOVAs were significant for each intelligence score. LSD tests demonstrated that the non-delayed no therapy group was higher for all intelligence measures than either the delayed infrequent home programme therapy group or the delayed intensive in-centre therapy group. Thus, the hypothesis that the non-delayed children would be functioning at a higher intellectual level than the motor delayed children when the children were 7 or 8 years of age was supported. There was no difference between the two delayed treatment groups. Therefore, the hypothesis that the intensively treated children would be functioning at follow-up at a higher intellectual level than the infrequently treated children was not supported.

Functional variables - Reading scores at follow-up. Method of analysis. Following the significant MANOVA the univariate analyses were interpreted in order to evaluate the effect of infrequent home programme

therapy and intensive in-centre therapy on delayed children's reading capabilities, at follow-up, and differences from non-delayed children on reading scores as measured by the Stanford Diagnostic Reading test. Mean values can be seen in Table 18. As can be seen in Table 19, the ANOVA was significant for reading percentile rank, $F(2,67) = 4.1$, $p<.05$. The LSD test demonstrated that the non-delayed no treatment group was higher for percentile rank in reading than both the delayed groups, thus supporting the hypothesis that non-delayed children would function at a higher level at age 7 and 8 than delayed children. There was no difference between the two delayed groups so the hypothesis of greater longer-term effectiveness of intensive therapy was not supported.

Psychological variables - Perceived competence and social acceptance at follow-up. Method of analysis. In order to determine the effect of infrequent therapy and intensive therapy on delayed children and the potential differences of these children from the non-delayed children on perceived competence and social acceptance (normal versus abnormal) as measured by the Pictorial Scale of Perceived Competence and Social Acceptance (Harter & Pike,1984) the univariate analyses following the significant MANOVA were interpreted. Mean values for the treatment scores can be seen in Table 22. As can be seen in Table 23 the ANOVA demonstrated no significant differences among the three groups for feelings of cognitive competence, $F(2,67)=.68$, $p<.50$, for feelings of peer acceptance, $F(2,67)=.76$, $p<.46$, or for feelings of maternal acceptance, $F(2,67)=.93$, $p<.40$. However, there was a trend towards a difference for feelings of physical competence, $F(2,67)=2.75$, $p<.07$.

Table 22

Psychological Variables at Follow-up

Mean Percentage Scores on the Harter Pictorial Scale of Perceived Competence and Social Acceptance for Young Children and Percentile Scores for the Achenbach Child Behaviour Checklist for Each Treatment Group (Delayed Infrequent Home Programme Therapy, Delayed Intensive In-centre Therapy, and Non-delayed No Therapy

	Infrequent Home Therapy	Intensive In-centre Therapy	Non-delayed No Therapy	Harter[a] Ranges
Self Concept				
Cognitive Competence	86.10	80.4	83.3	76-94
Peer Acceptance	78.26	74.3	80.45	78-97
Physical Competence	82.84	74.2	82.91	64-91
Maternal Acceptance	65.94	70.10	64.41	62-89
General Competence (combines cognitive and physical competence)	83.10	77.5	83.33	
Social Acceptance (combines peer and maternal acceptance)	74.10	72.40	71.70	
Behavioural Scores				
Physical Activities[b]	40.54	38.37	46.54	
Social Activities[b]	35.00	33.33	40.50	
School Performance[b]	25.12	17.87	34.12	
Schizoid[c]	60.46	62.04	60.46	
Depression[c]	54.29	58.46	59.70	

Table 22 (cont)

	Infrequent Home Therapy	Intensive In-centre Therapy	Non-delayed No therapy
Somaticism[c]	58.50	57.50	57.00
Withdrawal[c]	61.50	65.60	59.20
Hyperactivity[c]	65.12	69.00	60.90
Aggression[c]	57.10	57.80	56.70
Delinquency[c]	58.04	60.70	57.75

[a]Harter ranges are available only for the four individual measures and not for the combined measures.

[b]For the three performances on the Achenbach physical activities, social activities and school performance the lower the score the more abnormal, <20=abnormal.

[c]For all the other Achenbach scores the higher the score the closer to abnormal, >70=abnormal.

Table 23

Multivariate Analysis of Variance Summary Table of Psychological Variables at Follow-up

Effects of Treatment Condition (Delayed Infrequent Home Programme Therapy, Delayed Intensive In-centre Therapy and Non-delayed No Therapy) on Self Concept Measures and Behavioural Measures

	df	MS	F	p
MANOVA F - Pillais	30,69		1.51	<.05**
ANOVA F's				
Cognitive Competence	2	158.75	.68	<.50
Error	67	231.79		
Peer Acceptance	2	209.28	.76	<.46
Error	67	272.86		
Physical Competence	2	514.70	2.75	<.07*
Error	67	187.30		
Maternal Acceptance	2	183.50	.93	<.40
Error	67	197.10		
General Competence	2	224.57	1.50	<.21
Error	67	142.70		
Social Acceptance	2	32.60	.19	<.80
Error	67	171.00		
Physical Activities	2	431.20	2.70	<.07*
Error	67	157.70		
Social Activities	2	206.20	1.05	<.35
Error	67	194.70		
School Performance	2	1114.29	5.90	<.004***
Error	67	186.01		

Table 23 (cont)

	df	MS	F	p
Schizoid	2	36.62	.32	<.72
Error	67	111.35		
Depression	2	195.29	2.30	<.10*
Error	67	84.51		
Somaticism	2	22.14	.24	<.78
Error	67	90.14		
Withdrawal	2	235.50	2.30	<.10*
Error	67	98.98		
Hyperactivity	2	309.90	3.60	<.03**
Error	67	85.01		
Aggression	2	21.95	.21	<.81
Error	67	104.98		
Delinquency	2	103.76	1.99	<.14
Error	67	51.76		

*p<.1
**p<.05
***p<.01

Although there was no significant difference among the groups for feelings of peer acceptance, it was noted that feelings of peer acceptance for the children who received intensive in-centre therapy was below the lowest range found by Harter for her standardization subjects.

Behavioural variables - Behavioural Scores at follow-up. Method of analysis. A similar method of analysis as for the perceived competence and social acceptance scores was performed to determine the effect of treatment (infrequent therapy and intensive therapy) on delayed children and the potential behavioural difference of these delayed children from non-delayed children on behaviour as evaluated by the parents when the Achenbach Child Behavior Checklist was employed.

Means for the behavioural scores are presented in Table 22. As can be seen in Table 23, the ANOVA demonstrated a significant difference among groups for school performance, $F(2,67) = 5.9$, $p<.01$, and hyperactivity, $F(2,67) = 3.6$, $p<.05$, and a trend for the number of physical peer related activities participated in, $F(2,67)=2.7$, $p<.07$, for withdrawal, $F(2,67) = 2.3$, $p<.10$, and for depression, $F(2,67)=2.3$, $p<.10$.

When Least Significant Differences (LSD) were performed on school performance scores each treatment group was significantly different from each other group. This indicates that the intensively in-centre treated children were reported by their parents as performing more poorly in school than both the infrequent home programme treated children and the non-delayed children but the non-delayed children were reported as performing significantly better in school than both the delayed groups.

The infrequent home programme treated group was in the middle but significantly different from each other group.

When LSD tests were performed on hyperactivity scores the intensively in-centre treated group was significantly different from the non-delayed group. The infrequent home programme treated group did not differ from either of the other two groups. This indicates that the intensively treated children were reported to have more symptoms of hyperactivity than the non-delayed group.

Summary of Differences Between the Motor Delayed Children and the Non-delayed Children

There was no difference among the three treatment groups (delayed infrequent home programme therapy, delayed intensive in-centre therapy and non-delayed no therapy) for age at follow-up or for socioeconomic level. There was a difference between the two delayed groups and the non-delayed group for all functional measures, namely motor, reading, and intelligence measures. The non-delayed group was significantly higher than both delayed groups for all these measures.

The children in the intensively treated group appeared to feel less accepted by their peers than the children Harter used for her standardization (Harter & Pike,1984). The non-delayed group demonstrated significantly better school performance than the two delayed groups and parents of non-delayed children reported significantly fewer symptoms of hyperactivity than did parents of the intensively treated children.

Summary of Differences Between the Delayed Groups

There was no significant difference between the two therapy groups

(infrequent home programme therapy and intensive in-centre therapy) for duration of therapy, average percentage motor delay at initial assessment, age at follow-up and socioeconomic level at follow-up. Therapy was shown to produce a greater decrease in the percentage motor delay for intensively treated delayed children when initial assessment was compared to the discharge assessment than it did for infrequently home treated children. There was no maintenance of this decrease at follow-up, however, when the children were 7 and 8 years of age. Intensively treated children were significantly more delayed than infrequently treated children when initial delay scores were covaried out. This supports the hypothesis that the children who received intensive in-centre therapy would show a greater decrease in motor delay from initial assessment to discharge assessment but since this effect was not maintained at follow-up the hypothesis that there would be greater longer-term effectiveness of intensive therapy was not supported. It appears that, although the decrease in delay was not as great for the infrequently treated children, this decrease was maintained at follow-up.

There was no difference between the two therapy groups in intelligence scores and reading scores at follow-up. Delayed children with intensive in-centre therapy were reported by their parents as performing significantly more poorly in school than the delayed children who received infrequent home programme therapy. There was no difference between the two delayed treatment groups on all other follow-up measures.

Difference Between Children Treated at a Younger Age as Opposed to an Older Age

Populations prior to the intervention. Method of analysis. In order to determine whether there were differences in average percentage motor delay, socioeconomic level, type of therapy received, and age of the children at follow-up between children who received therapy between birth and 2 years of age and children who received therapy between 2 and 4 years of age an analysis of variance (ANOVA) was performed. The ANOVA revealed that there were no differences between the groups for average percentage motor delay, $F(1,46) = .22$, $p<.64$, socioeconomic level, $F(1,48) = .002$, $p<.96$, treatment given $F(1,46) = .11$, $p<.72$, age at follow-up, $F(1,48) = .14$, $p<.71$, and duration of therapy, $F(1,46) = .06$, $p<.80$ (Appendix G).

Differences between the two treatment age groups. Method of analysis. Initially a MANOVA was used to examine differences between means of children assessed and treated between birth and 2 years of age and those assessed and treated between 2 and 4 years of age. A MANOVA was used for the same reason as when treatment groups were compared. Since the MANOVA was significant, $F(9,28) = 4.47$, $p<.01$, all the subsequent univariate results were interpreted. In this analysis age at treatment (at or before 2 years and after 2 years) was the independent variable and the motor measures (follow-up motor scores on the Bruinicks-Oseretsky test), reading measures (Stanford Diagnostic Reading Test), intelligence measures (Wechsler Intelligence Scale for Children - Revised), the self concept measure of maternal acceptance

(Pictorial Scale of Perceived Competence and Social Acceptance) and behavioural measures of social activities, school performance, and somaticism (Achenbach Behavioural Checklist) were the dependent variables. These measures were chosen because examination of the means indicated that there might be differences.

Differences between the two treatment age groups with respect to the functional variables - Motor scores at follow-up. Method of analysis. Following the significant MANOVA, ANOVA results were interpreted in order to determine the effect of age when treatment was initiated on motor development of children with a delay of non-specifiable etiology. The means are presented in Table 24. As can be seen in Table 25 this difference was significant ($p,<.01$).

In order to determine the effects of early therapy versus late therapy on the decrease in motor delay of children with a delay of non-specifiable etiology from initial assessment to discharge assessment and from initial assessment to follow-up assessment a multivariate analysis of covariance (MANCOVA) was performed, using the initial assessment scores as covariate scores. The MANCOVA was significant, $F(2,30) = 9.22$, $p<.001$. The mean values are shown in Table 26. ANOVA's were significant when comparing the initial assessment to discharge assessment scores, $F(1,31) = 7.2$, $p<.01$, and to follow-up scores, $F(1,31) = 15.6$, $p>001$ (Table 27). The initial assessment scores used as covariates were not significant, $F(1,31) = .59$; $p<.56$. These results support the hypothesis that the decrease in average percentage motor delay would be greater for children who were treated when they were younger as opposed to older and

Table 24

Mean Scores for Functional Variables (Percentile for Motor Scores, for Word Reading Scores, for Total Reading, and Verbal, Performance and Full Scale Intelligence Quotients) by Age at Treatment (Between Birth and 2 Years and Between 2 and 4 Years)

	Younger Age	Older Age
Percentile Motor Scores	21.36	4.77
Percentile Rank Word Reading	42.00	31.00
Percentile Rank Total Reading	38.09	30.59
Verbal Scores	94.23	77.66
Performance Scores	91.38	79.17
Full Scale Intelligence	93.63	78.74

Table 25

Multivariate Analysis of Variance Summary Table of Functional Variables Effects of Age at Treatment (Between Birth and 2 Years and Between 2 and 4 Years) on Motor, Reading and Intelligence Measures

	df	MS	F	p
MANOVA F - Pillais	9,28		4.47	<.002***
ANOVA F's				
Motor Scores	1	2150.05	8.87	<.005***
Error	36	242.37		
Word Reading	1	858.65	.57	<.45
Error	36	1498.63		
Verbal IQ	1	2123.94	6.77	<.01***
Error	36	313.48		
Performance IQ	1	1038.18	2.69	<.1*
Error	36	385.31		
Full Scale IQ	1	1734.16	4.86	<.03**
Error	36	356.93		

*$p<.1$
**$p<.05$
***$p<.01$

Table 26

Mean Average Motor Delay at Initial Assessment, Discharge Assessment, and Follow-up Assessment by Age at Treatment (Between Birth and 2 Years and Between 2 and 4 Years)

	Initial Assessment	Discharge Assessment	Follow-up Assessment
	N=48	N=48	N=46
Treated Before 2 Years	24.00	13.21	16.29
Treated After 2 Years	32.06	20.47	26.53

Table 27

Multivariate Analysis of Covariance Summary Table

Effects of Age at Treatment (Between Birth to 2 Years and Between 2 and 4 Years) on Average Percentage Motor Delay at Dischange Assessment and Follow-up Assessment

	df	MS	F	p
MANCOVA F - Pillais	2,30		9.2	<.001*
ANCOVA F's				
Average Percentage Motor Delay at Dischange	1	1613.17	7.2	<.01*
Error	31	221.81		
Average Percentage Motor Delay at Follow-up	1	2135.85	15.6	<.004*
Error	31	136.37		

*p<.01

that this effect would be maintained even after discharge at the follow-up assessment when the children were 7 or 8 years of age.

Differences between the two treatment age groups with respect to the functional scores - Intelligence scores at follow-up. Method of analysis. Following the significant MANOVA, ANOVA results were interpreted in order to determine the effect of earlier treatment versus later treatment on follow-up intelligence scores.

Mean values can be seen in Table 24. Examination of the means indicated that the children who were treated at a younger age appeared to have higher verbal scale, performance scale, and full scale intelligence quotients (IQ) than children who were treated at an older age.

As can be seen in Table 25, the ANOVAs were significant for verbal scale intelligence quotient, $F(1,36) = 6.8, p<.01$ and for full scale intelligence quotient, $F(1,36) = 4.9, p<.05$, and demonstrated a trend towards significance for performance scale intelligence quotient, $F(1,36) = 2.7, p<.1$. This supports the hypothesis that children who received therapy at a younger age would obtain higher IQ scores when they were 7 or 8 years of age than children who were treated at an older age.

Differences between the two treatment age groups with respect to the functional variables - Reading scores at follow-up. Method of analysis. Following the significant MANOVA, ANOVA results were interpreted in order to determine the effect of early treatment versus later treatment on reading scores. As can be seen in Table 25 the ANOVA

was not significant, $F(1,36) = .57$, $p<.45$.

Difference between the two treatment age groups with respect to psychological and behavioural variables - Perceived competence and social acceptance at follow-up. Method of analysis. Following the significant MANOVA, ANOVA results were interpreted in order to determine the effect of age at treatment for children with a delay of non-specifiable etiology on perceived competence and social acceptance. Mean values for the treatment scores can be seen on Table 28. As can be seen in Table 29 there was no significant difference between the two age groups $F(1,36)=1.86$, $p<.18$.

Differences between the two treatment age groups with respect to the psychological and behavioural variables - Behavioural scores at follow-up. Method of analysis. The same method of analysis as for the perceived competence and social acceptance scores was performed to determine the effect of age at treatment for delayed children of non-specifiable etiology on behavioural measures as determined by the Achenbach Behavioral Checklist which was filled out by the parents. Means for behavioural scores are presented in Table 28.

As can be seen in Table 29, the ANOVA demonstrated a significant difference for social activities, $F(1,36) = 19.26$, $p<.01$, for school performance, $F(1,36) = 14.07$, $p<.01$, and demonstrated a trend for somaticism, $F(1,36) = 3.55$, $p<.07$. These results support the hypothesis that that children who were treated at a younger age would perform better in school at age 7 and 8 and would participate more frequently in social activities than the children who were treated at a later age.

Table 28

Mean Scores for Behavioural and Psychological Variables for which there Appeared to be a Difference (Maternal Acceptance, Social Activities School Performance, Somaticism) by Age at Treatment (Between Birth and 2 Years and Between 2 and 4 Years)

	Age (at or before 24 months)	Age (after 24 months)
Maternal Acceptance	62.63	70.29
Social Activities	43.80	29.84
School Performance	32.36	16.60
Somaticism	61.13	56.78

Table 29

Multivariate Analysis of Variance Summary Table

Effects of Age at Treatment (Between Birth and 2 Years and Between 2 and 4 Years) on Behavioural and Psychological Scores at Follow-up

	df	MS	F	p
MANOVA F - Pillais	9,28		4.47	<.002**
ANOVA F's				
Maternal Acceptance	1	458.58	1.86	<.18
Error	36	245,58		
Social Activities	1	2676.41	19.26	<.004**
Error	36	138.91		
School Performance	1	2811.50	14.07	<.001**
Error	36	199.86		
Somaticism	1	280.73	3.55	<.07*
Error	36	79.03		

*p<.1

**p<.01

Summary of the Effects of the Age of the Child at the Time They Received Therapy

There were no differences between the younger treated children (therapy initiated between birth and 2 years of age) and the older treated children (therapy initiated between 2 and 4 years of age) in the type of therapy received, the duration of therapy, the average percentage motor delay at initial assessment, the age at follow-up, or their socioeconomic level. The children who received therapy from birth to 2 years of age made greater motor gains from initial assessment to discharge assessment than the children who received therapy between 2 and 4 years of age and these increased gains were maintained at follow-up. The younger treated children performed significantly better on the motor test (Bruinicks-Oseretsky) at follow-up. The younger treated children had significantly higher IQs at follow-up and according to their parents' report they performed better at school, and participated in more social activities.

Differences Between Children Functioning at Follow-up in the Retarded-Borderline Intellectual Range as Opposed to Children Functioning in at Least the Low-average Range

There appeared to be two separate populations in terms of intelligence within the two delayed groups, as can be seen in Table 30. There was a group of 23 children who had intelligence quotients below normal ($n = 17$) or who were untestable due to severe delays ($n = 3$) and severe emotional problems ($n = 3$). Of the 17 testable children the average verbal IQ was 63.35, the average performance IQ was 60.52, and

the average full scale IQ was 60.51. This was in contrast to 25 children who received scores in the average or above average range. These children received an average verbal IQ of 96, an average performance IQ of 98.2 and an average full scale IQ of 96.98. Mean full scale IQs are present in Table 30. The IQs for these two groups were statistically different, $F(2,42) = 4.9$, $p<.01$. It can be seen in Table 31 that when these 17 retarded or borderline children were deleted from the statistical analysis the remaining 25 children still differ significantly from children in the non-delayed group, $F(2,47)=5.2$, $p<.01$. According to the LSD test the two delayed treatment groups did not differ from each other when the retarded and borderline children were removed from the analysis and they both continued to remain significantly lower in intelligence scores than the non-delayed group.

It was also of interest to determine if there was a combination of variables which would be successful in discriminating among the children who were originally delayed and who at follow-up were assessed as having either borderline-mentally retarded intelligence scores or at least low average intelligence scores. Several variables (number of pre or post natal problems, total number of major problems, mother's age at the child's birth, gestational age of the child, gender, socioeconomic level, age of child at initial assessment, handedness, seizures, treatment received, number of social problems at the time of assessment and average percentage initial motor delay at assessment) were entered into a step-down discriminant analysis. Mean values for these variables can be seen in Table 32. These variables were chosen from the important

Table 30

Means of Full Scale Intelligence Quotients with Retarded and Borderline Children Included and with These Children Excluded for Delayed Children

	Delayed Children
Retarded and Borderline Children Included	N=42 81.55
Retarded and Borderline Children Not Included	N=25 96.98
Only Retarded and Borderline Children	N=17 60.51
Untestable Children	N=6

Table 31

Analysis of Variance Summary Table

Differences Among the Groups (Delayed Infrequent Home Programme Therapy, Delayed Intensive Therapy, and Non-delayed No Therapy) in Intelligence Quotients (Children with Retarded and Borderline Intelligence Included and Children with Retarded and Borderline Intelligence Excluded)

	df	MS	F	p
ANOVA F				
Children with Retarded and Borderline Scores Included	2	4758.50	13.66	<.005*
Error	61	348.11		
Children with Retarded and Borderline Scores Excluded	2	807.83	5.20	<.01*
Error	47	155.19		

*p<.01

Table 32

Means of Variables Entered into the Discriminant Analysis Discriminating Retarded-borderline Children from Low Average-average Children at Follow-up

	Borderline-Retarded N=23	Low Average-average N=25
Number of Prenatal Problems	.91	.76
Number of Postnatal Problems	1.41	.95
Number of Major Problems	1.10	.90
Mother's Age at Child's Birth(years)	28.08	29.04
Gestational Age(weeks)	39.31	39.47
Gender (ratio-male/female)	1/1.1	1/.2
Age at Initial Assessment(months)	31.35	26.96
Average Percentage Motor Delay at Initial Assessment	37.05	27.16
Socioeconomic level	4.17	5.08
Handedness (ratio-right/left)	1/.63	1/.23
Seizures (ratio-no/yes)	1/.54	1/.17
Small Head Circumference (ratio-no/yes)	1/.18	1/.12
Number of Others Delayed in Family	.27	.20
Number of Social Problems	1.21	.51
Treatment (ratio-infrequent/intensive)	1/1.2	1.5/1
Hyperactivity(percentile rank)	69.00	64.40
Weight (in grams)	3266.00	3432.97

variables found by Gillberg and Rasmussen and their collegues (Gillberg & Rasmussen, 1982; Gillberg et al., 1983) based on a multivariate analysis of variance comparing the two groups which found significant differences or trends between the groups in gender, average motor delay, handedness, febrile seizures, number of social problems, and socioeconomic level.

The discriminant analysis entered the variables in order of their discriminating values. Eleven variables were entered into the analysis. These variables, average motor delay at initial assessment, febrile seizures, gender, socioeconomic level, number of social problems at the time of initial assessment, gestational age in weeks and weight in grams, other delays in the family, small head circumference, age at initial assessment, and percentage hyperactivity could correctly classify 89% of the 64 cases into either the retarded-borderline or at least low average intelligence group (see Appendix H). The F ratio level for all other variables was insufficient to enter into the equation. However, one useful function was found which included only three variables, the child's gender, the age of the child at initial assessment, and the average motor delay at initial assessment (see the discriminant functions in Table 33) and which achieved nearly as high a percentage as the 11 variables. Eight-three per cent of the cases as opposed to a chance level of 50% were correctly classified into the two groups. This is significant.

This finding indicates that there was a significant difference between the intellectually borderline-mentally retarded group of

Table 33

Discriminant Analysis Summary Table

Gestational Age, Gender, Age and Average Percentage Motor Delay at Initial Assessment by Intellectual Level at Follow-up (Retarded-borderline or Low Average-average)

Variables Entered	Wilks Lambda[a]	p[b]	B[c]
Gender	.84	.01*	.76
Average Percentage Motor Delay at Initial Assessment	.70	.001*	.89
Age at Initial Assessment	.56	.0001*	.83

*$p < .01$

Note: The Eigenvalue and its accompanying canonical correlation are .75 and .66 respectively.

[a]A measure of group homogeneity.

[b]Only those variables that significantly increase the distance between group centroids were entered.

[c]Standardized linear weights that optimize the distance between the group centroids (for the group with borderline-mentally retarded intelligence scores the centroid was 1.0 and for the group with low average-average intelligence scores the centroid was -.7($F = 9.6, 3/38$ df, $p < .001$).

children at follow-up and the intellectually low average-average group of children at follow-up for gender, age at initial assessment and average percentage motor delay at intake. The intellectually borderline-mentally retarded group consisted of children who were initially assessed at an older age, had a more severe motor delay and were both boys and girls, while the intellectually low average-average children were mostly boys who were assessed and treated at a younger age and had a less severe motor delay.

It was of interest to determine if there was a difference in the effectiveness of therapy between the children who at follow-up demonstrated borderline-retarded IQs and those who demonstrated at least low average IQs at follow-up. Mean values for initial assessment, discharge, and follow-up assessments can be seen in Table 34. The average percentage motor delay at initial assessment for children with a borderline-retarded intelligence score was 37.00% and for the children with at least a low average intelligence was 27.16%. At final assessment the average motor delay for children who were borderline-retarded at follow-up had decreased to 32.37% while the average motor delay for children who were at least of low average intelligence at follow-up had decreased to 19.81%.

Change in fine motor percentage delay, gross motor percentage delay and average motor percentage delay from initial assessment to discharge assessment, comparing children with a follow-up intelligence in the borderline-retarded range to children with a follow-up intelligence in the low average-average range was examined by a MANCOVA in which initial

Table 34

Mean Initial and Discharge Percentage Delay for Fine Motor, Gross Motor and Average Motor Percentage Delay by Intelligence (Retarded-borderline and Low Average-average)

	Fine Motor Delay	Gross Motor Delay	Average Motor Delay
	N=48	N=48	N=46
Group			
Retarded -Borderline			
Initial Assessment	36.64	36.72	37.00
Discharge Assessment	36.38	23.00	32.37
Follow-up Assessment			26.71
Low Average -average			
Initial Assessment	25.65	29.45	27.16
Discharge Assessment	20.00	18.44	19.81
Follow-up Assessment			16.27

assessment scores served as covariates. The overall MANCOVA was significant, $F(4,42)=4.05, p<.01$. As can be seen in Table 35, the univariate analysis of covariance demonstrated a significant difference between the groups at discharge for gross motor delay, $F(1,45) = 5.79$, $p<.01$, for average motor delay, $F(1,45) = 6.01$, $p<.01$, but not for fine motor delay, $F(1,45) = 1.77$, $p<.17$. At follow-up, there was a significant difference in motor scores, $F(1,45) = 14.1$, $p<.001$, with the children with at least a low average intelligence score performing significantly better in the Bruinicks-Oseretsky Test of Motor Proficiency than the children with retarded-borderline intelligence (Table 35). The initial assessment scores did not differ for gross motor, $F(1,45) = 1.08$, $p<.3$, or fine motor delay, $F(1,45) = 2.59$, $p<.12$, for average motor delay, $F(1,45) = 2.86$, $p<.1$, or for follow-up delay, $F(1,45) = .177$, $p<.67$. This indicates that children with at least a low average intelligence at 7 and 8 years of age had made more gross motor and average motor gains as a result of therapy than children with a retarded or borderline to retarded intelligence score at follow-up.

Summary of Differences Between the Children who were of Retarded-borderline Intelligence and Children Who were of at Least Low Average Intelligence at Follow-up

When combining the two delayed groups resulting in 48 children, there appeared to be two different groups in terms of intelligence. There was a group of children who were of retarded or borderline intelligence ($n=23$) and there was a group of children who were of low average or average intelligence ($n=25$). These two groups of children

Table 35

Multivariate Analysis of Covariance Summary Table

Effects of Treatment (Change in Delay after Therapy) on Children who at Follow-up either were Retarded-borderline or Low average-average in Intelligence

	df	MS	F	p
MANCOVA F - Pillais	4,42		4.05	.004*
ANOVA F's				
Gross Motor	1	1090.62	5.79	.003*
Error	45	188.20		
Fine Motor	1	428.40	1.77	.174
Error	45	241.18		
Average Motor	1	984.38	6.01	.003*
Error	45	163.67		
Follow-up Motor	1	2428.52	14.14	.001*
Error	45	171.72		

*$p < .01$

differed significantly, in the age at which they began treatment, the average delay at initial assessment, and their gender. The children of lower intelligence consisted of both boys and girls who began their treatment at an older age and had a greater delay. The children with higher intelligence scores were boys who were treated at an earlier age and who were less delayed at initial assessment.

Chapter VI

Discussion

The discussion is presented, firstly, in three major sections. These include: first, a summary of the findings; second, a discussion of the significance of the findings; and third, conclusions and research suggestions. The summary of the findings and the discussion of the significance of the findings are presented, secondly, in relationship to the three major questions that were addressed in this study. These questions were:

First, is there a definable population of motor delayed children with non-specifiable etiology of delay that can be described and categorized when these children have reached school age? In particular, was there support for the two hypotheses which related to the way this population of children would be different from other populations of children. The first hypothesis was that these children would be different from non-delayed children, and the second hypothesis was that these children would be more responsive to therapy than children with a specifiable etiology for their delay;

Second, what were the main effects of an early intervention therapy which was concerned with enhancing the development in physical, perceptual-cognitive, social and emotional spheres of living on children with a non-

specifiable etiology for the motor delay? In particular, was there support for the two hypotheses relating to therapy effectiveness? The first of these hypotheses was that intervention would be effective in decreasing motor delay. The second hypothesis was that intensive therapy would be more effective than infrequent therapy, both in the shorter-term (at discharge from therapy) and in the longer-term (2 to 4 years after discharge from therapy);

Third, were there any differential effects for various subgroups of delayed children? In particular, was there support for the hypothesis that early intervention (before 2 years of age) would be more effective than late intervention (between 2 and 4 years of age).

The children of special interest in this study were children who had a motor delay in which no specifiable etiology could be established. That is, these children did not have any major abnormal neurological signs, and no specifiable diagnosis or etiology had been established according to paediatric and/or neurological examination(s) documented in the children's medical record. These children had manifested delayed motor development during the first 48 months of life. Their motor delay had been identified either at the Montreal Children's Hospital (MCH) or the Constance-Lethbridge Rehabilitation Centre (CLRC), in Montreal, Quebec. These children were 7 and 8 years of age at the time of follow-up.

Summary of Findings

The population of children with a motor delayof non-specifiable etiology. A population of children with a motor delay of non-specifiable etiology was identified in this study. At the age of 7 and 8 these children consisted of nearly one-third (n = 137) of all delayed children (N = 426) born between 1974-09-30 and 1976-09-30 who were referred for assessment before 48 months of age to either the MCH or the CLRC. The only variables (prenatal, perinatal, postnatal, family and child) which could significantly distinguish this group of children with a motor delay of non-specifiable etiology from a non-delayed group of children was a motor delay which had been identified before 48 months of age. The non-delayed children were children referred for evaluation of a possible motor delay and identified by occupational therapists as having no delay. The differences between non-delayed and delayed children of non-specifiable etiology were evaluated when the children were 7 and 8 years old. The evaluations were functional, behavioural and psychological. The functional evaluations given at follow-up (when the children were 7 and 8 years of age) included a test of motor capabilities (Bruinicks-Oseretsky Test of Motor Proficiency), a test of intellectual abilities (Wechsler Intelligence Scale for Children - Revised), and a test of reading capabilities (Stanford Diagnostic Reading Test). The behavioural and psychological evaluations included a measure of perceived competence and social acceptance (Harter Pictorial Scale of Perceived Competence and Social Acceptance) and a measure of behaviour (Achenbach's Child Behavior Checklist). As hypothesized, the

non-delayed group performed at a higher level on all functional measures at follow-up. That is, they had significantly better motor skills, significantly higher intelligence scores and significantly higher reading levels. They also performed better in school according to parents' reports and demonstrated significantly fewer symptoms of hyperactivity than delayed children who had been intensively treated.

Three variables were found which could distinguish the motor children with a motor delay of non-specifiable etiology from the children with a motor delay of specifiable etiology according to paediatric or neurological examination. The delayed children with a motor delay of specifiable etiology tended more often to have a head circumference below the 25th percentile, to have had more major illnesses and to have younger mothers than did the delayed children with a non-specifiable etiology. As was hypothesized, children with a non-specifiable etiology for their motor delay appeared to be more responsive to therapy and to show a greater decrease in their motor delay by the time of discharge than did children for whom an etiology of motor delay could be specified.

Main effects of early intervention. There were several major findings in this study which related to the effectiveness of therapy on developed abilities, and on children's and parents' attitudes for children with a motor delay of non-specifiable etiology. Based on Piaget's theory of the development of intelligence (Piaget, 1952; 1959), it had been hypothesized that early intervention would have a positive effect on both the intellectual and psycho-social development of the

motor delayed child with a non-specifiable etiology. In support of this hypothesis it was found that all motor delayed children who received therapy, either infrequent home programme therapy or intensive in-centre therapy, demonstrated a significant decrease in their average motor delay from initial assessment to the time of discharge from therapy. Both therapy programmes were considered to be "a facilitation process towards mastery of life tasks" (Llorens, 1970).

The infrequent home programme therapy was based on instruction by theoccupational therapists to the parents in assisting the children in the appropriate motor exercise regimes. These children received in-centre treatment by the occupational therapist for no more than 10 hours during the year following their assessments. The intensive in-centre therapy was provided in a co-ordinated multidiscipline programme which was planned to meet the individual needs of each child and family (Kord, 1982) and used a developmental approach (Lloren, 1970). Children receiving intensive in-centre therapy were given individual therapy in the treatment centre for at least an average of 6 hours per week and for a minimum of 1 year. The effects of the two intervention programmes were evaluated both at discharge from therapy when the children were 2 to 5 years old and at follow-up when the children were 7 and 8 years old. At discharge each evaluation was based on the occupational therapist's functional motor scores from the discharge report. At follow-up the evaluations were the same as in the comparisons of the non-delayed and delayed groups.

As hypothesized, children participating in the follow-up phase of

this study who were treated intensively, demonstrated a greater decrease in their average motor delay at discharge from therapy when they were from 2 to 5 years of age than did infrequently home programme treated children. This decrease in motor delay demonstrated at discharge from therapy was not maintained for the intensively treated children after the therapy stopped. This was demonstrated by the findings at follow-up when the children were 7 and 8 years of age. There were no differences between the results of the intensively treated and infrequently treated children on the functional evaluations provided at follow-up, although when initial motor scores were used as covariants there wasa difference in motor capabilities at follow-up. The infrequently treated group had less of a motor delay than the intensively treated group at follow-up. These findings indicate that the early effects of intensive intervention on motor capabilities were probably not permanent and the hypothesis that there would be a positive longer-term effect of intensive therapy was not supported.

There were, however, differences in behavioural and psychological evaluations between the intensively treated and infrequently treated children at follow-up. The intensively treated children felt less accepted by their peers at follow-up than did the children in the standardized sample of Harter and Pike (1984). Parents of intensively treated children reported that their children performed more poorly in school than did parents of infrequently home programme treated children although there was no difference in reading capabilities according to the follow-up test measure.

Hypothesized differential effects of early intervention. Certain populations of children appeared to be differentially affected by therapy. As hypothesized, children with a motor delay of non-specifiable etiology who were treated under the age of 2 years were functioning at a significantly higher level at age 7 and 8 than were children with a motor delay of non-specifiable etiology who were treated after 2 years of age.

Non-hypothesized findings. It was observed at follow-up that there appeared to be two completely different sub-populations in the groups of children with a motor delay of non-specifiable etiology. Both of these sub-populations were different from the non-delayed children. One delayed sub-population consisted of children who were mentally retarded or of borderline intelligence and these children tended to be in special schools. The second sub-population consisted of children who were of average intelligence, but continued to have motor problems and were having difficulty coping with the regular school programme although they were still in regular schools. Children with motor delays of non-specifiable etiology who were functioning at the ages of 7 and 8 in the retarded or borderline retarded intelligence range had not responded as readily to therapy by demonstrating a decrease in their delay as had children with a motor delay of non-specifiable etiology who at the same age were functioning in at least the low average range of intelligence. These two sub-populations of children (children with retarded-borderline intelligence and children with at least low average intelligence at follow-up) differed in the age at which therapy began, in gender, and in

their percentage of delay at the time at which therapy began. The lower IQ children were older and were more delayed by the time therapy began and included a nearly equal number of boys and girls, while higher IQ children were younger and less delayed at the time therapy began and were mostly boys.

There were no differential effects of therapy according to the gender of the children. That is, both boys and girls responded equally well to therapy. In terms of those receiving therapy there was, however, a meaningful bias in gender for the children who received intensive therapy and for those who were treated early. Only slightly fewer females than males were assessed as being delayed (.6/1). There was only one female treated intensively for every four intensively treated males, however, and many more males than females were treated before they were 2 years of age.

Significance of Findings About the Population of Children with Non-specifiable Etiology for Their Delay

There does appear to be a significant population of children with a diagnosis of non-specifiable etiology for their delay which should be recognized. This population consisted of 32% (n = 137) of the total delayed population of children referred to the two centres (N = 426) for assessment and treatment of a motor delay before 48 months of age; and who were born between 1974-09-30 through 1976-09-30. At the time of follow-up, children with a delay of non-specifiable etiology differed from children with a delay of specifiable etiology in terms of maternal age at birth, total number of major problems, and number of children

with abnormally small head circumference. They were not distinguishable, however, from the non-delayed children by any variable other than motor delay at the time of initial assessment. When these children were 7 and 8 years of age, however, they were different in motor function, intellectual function, reading capabilities, and success in school. Attention needs to be directed towards the children with a motor delay of non-specifiable etiology in terms of therapeutic intervention, descriptive characteristics and research. It would appear from the literature that these children form a population which has been previously neglected. Three studies on the effects of therapy were found which appeared to identify this population specifically (Barna et. al., 1980; Findlay, 1981; Moxley-Haegert & Serbin, 1983). Not all of these studies examined delayed children of non-specifiable etiology exclusively. These three studies did identify, however, the children of non-specifiable etiology as a distinct population. Other researchers have identified school age children who were motor delayed and had school difficulties (Gillberg & Rasmussen, 1982; Gillberg et. al., 1983; Younes et. al., 1983). Such children are similar to the sub-population of children with a motor delay of non-specifiable etiology identified in this study who were not retarded at follow-up.

There are several reasons, as discussed in the introduction, why children with a delay of non-specifiable etiology could form an independent population of concern to those interested in child development. The first reason suggested, was that the motor delay in young children may be the first manifestation of a more pervasive

disorder which would be evident at a later stage(s) of development. Various researchers have suggested, for example, that motor delay would be an early manifestation of mental retardation (Hogg, 1982; Molnar, 1975). The ramifications of this suggestion will be discussed in the section of "Significance of Non-hypothesized Findings".

The second reason that researchers should be interested in the population of children with a non-specifiable etiology for their motor delay was that motor delay may be a predictor of more subtle but important problems occuring at later developmental stage(s), such as difficulties learning to read or write or behavioural problems. It has been shown by several researchers that motor delayed children who are not mentally retarded do have school achievement problems (Gillberg et al., 1983; Henderson and Hall, 1982; Younes et al., 1983). It has been shown that many of the children with motor delays have behavioural problems as well as school achievement problems (Gillberg & Rasmussen, 1982).

Almost all the motor delayed children evaluated at follow-up (48 children) were having problems in school and continued to have a motor delay. Even children who were not retarded at follow-up were having difficulties in school. In fact, only five of the 48 children were functioning at an average level in all functional variables of the evaluation provided at follow-up and only six children were in regular classes and not receiving extra help at school. The children who were not retarded appear to be very similar to the population described by Gillberg, Gillberg, Rasmussen, and Wahlstrom (Gillberg & Rasmussen,

1982; Gillberg, et al., 1982; Gillberg and Gillberg, 1983; Gillberg et al., 1983). They have labeled this population as children suffering from 'minimal brain dysfunction'.

The third reason for an interest in children with motor delays, was that motor delay could be the cause at a later developmental stage(s) of problems in learning and behaviour. Whether a motor delay was the cause of later problems, parents of these children did perceive them as problem children. These problems were reported by parents, most particularly, on the hyperactivity scale of the Achenbach Behavioral Checklist. Twenty-one of the 48 delayed children were scored by their parents as functioning more than two standard deviations above the mean for hyperactivity. Thirty-six of the 48 children were perceived as having school problems and 21 of the 48 children were considered to have social problems (scored by their parents as functioning more than two standard deviations from the mean). These results were significant when they were compared to the reports of the parents of normally developing children.

Diagnostic distribution. In this study the etiology category (specifiable versus non-specifiable) did not influence whether a child received therapy or whether he/she received infrequent home programme therapy or intensive in-centre therapy. It was shown in this study, however, that systematic early intervention therapy (both infrequent and intensive) effectively decreased the motor delay of children with a non-specifiable etiology for their motor delay while it was relatively ineffective for children with a specifiable etiology for their delay.

These findings support the research evidence that systematic early intervention which uses a developmental approach is relatively unsuccessful in improving the development of children who demonstrate neurological impairment (including those with cerebral palsy and visual impairment, and multiple handicaps) (Barna et al., 1980; Drillien et al., 1980; Kirk, 1969; Williams & Scarr, 1971).

Nearly every therapy programme has a waiting list. Therefore, most programmes would like to be able to predict which children would most likely be affected favourably by the specific treatment programme offered. It appears from the present findings that children with a non-specifiable etiology for their motor delay are likely to benefit from the specific therapy given in the present study. However, many children who at the time of treatment had a delay of non-specifiable etiology later developed abnormal neurological signs. These children, who were reclassified as having a delay of specificable etiology, did not respond as readily to the therapy provided in this study as the children who remained with a delay of non-specifiable etiology. It would be important, if possible, to be able to predict which children are likely to be reclassified as having a delay of specifiable etiology and therefore are not likely to benefit from the particular therapy provided in the two treatment centres participating in this study. The findings of this study showed that children with more major problems (including such problems as febrile seizures, minor structural abnormalities and failure to thrive), with head circumferences in the lower 25th percentile range and with younger mothers tended to be reclassified as

having a delay of specifiable etiology. These variables may therefore be predictors of the type of child for which different therapy methods should be developed.

Although the current therapy approach may be effective for children having a delay of a non-specifiable etiology, research is clearly needed in order to develop appropriate therapy for children with neurological damage and for those who subsequently develop neurological signs. More specific treatment approaches could be developed for these children by combining individual observation and measurement and developing individual curricula based on current knowledge of growth and development, on current treatment techniques and on the idiosyncrasies of individual children. By constantly monitoring target behaviours with appropriate programme changes, treatment programmes may be developed that objectively produce positive results.

Significance of the Follow-up Findings in Effects of Early Intervention Therapy

There are theoretical and reasearch indications that this population with a motor delay of non-specifiable etiology may be responsive to therapy and perhaps be even more responsive to early intervention therapy than other populations of delayed children (Browder, 1981; Barna et al., 1980; Harter, 1981; Hunt, 1961; Piaget, 1951; 1952; 1954; 1959; 1970; Ramey & Smith, 1977; Simeonsson et al., 1982). These indications provide another reason why this population should be considered as important for those interested in child development. As hypothesized, it was demonstrated in this study that

all children with a motor delay of non-specifiable etiology responded favourably to early intervention therapy regardless of whether it was intensive or infrequent. It is not known whether delayed children who were not treated would have had a similar decrease in delay during the same period of time. The decrease in delay, however, was significant for treated children when initial assessment was compared to discharge assessment.

This study reports similar findings for motor gains of the intensively treated children with a delay of non-specifiable etiology as were reported for the cognitive gains of disadvantaged children in the early results from evaluations of Head Start and experimental preschool intervention programmes (see Bronfenbrenner, 1975; Day & Parker, 1977; Field et al., 1979; Horowitz & Paden, 1973 for reviews of this research). That is, the initial motor gains resulting from the intensive intervention were not sustained when therapy stopped just as the initial cognitive gains resulting from the preschool intervention programmes were not sustained. In the present study the infrequently treated home programme or control group had the same level of motor delay at follow-up as they had had at the time of discharge from therapy. The intensively treated in-centre programme children, in contrast, demonstrated an increase in their motor delay from their discharge assessment to their follow-up assessment. This increase in delay ,however, did not drop them back to the original level of delay present at initial assessment.

The immediate response to these findings should be to examine more

closely the primary instrument for intervention, i.e. the in-centre intensive therapy programme, in order to determine whether anything could be added to the programme to aid in the maintenance of the children's gains. In the intensive programme, the government mandate is to treat children until school age is reached. The children are then considered to be the schools' responsibility. Unfortunately most teachers are not trained to do motor interventions and thus when children enter the regular school system any concentrated work on motor control usually stops. The only mandate the intervention programme has past school age is consultation with the schools (if requested by the school) and follow-up reassessment (if requested by the parents). For this reason many of these delayed children are referred to special schools which have their own physical therapists and occupational therapists or are referred to special classes which have specially trained teachers for children with such problems. There may be, however, negative side effects to special schools. Even though these schools have their own therapists and the children receive more specialized help, the children do not have as much opportunity to play with their non-delayed peers of the same age as do children in regular classes. The children may feel, thus, different from their age mates and become stigmatized both by themselves and their parents as children with a problem. It would appear that a better plan may be for intervention therapists to provide more community education in order to alert the regular schools to potential problems. This should promote early requests for child assessment. Perhaps work needs to be done to

alert schools to the possible need of a permanent occupational therapist or physical therapist consultant within the regular school system.

When Bronfenbrenner (1975) studied treatment programmes to identify features which made them effective, he found that neither the programme nor the teacher were important in fostering and sustaining child development. Instead the single most important factor was the family. More specifically the most effective programme was one in which therapists came into the home at least once a week and taught parents how to treat their own children. In a previous study by the present researcher a programme in which parents provided the therapy was shown to be effective for children with a motor delay of non-specifiable etiology (Moxley-Haegert & Serbin, 1983). This finding could explain some of the results in the present study in which parents of the intensively treated children were not taught to carry out the programme. Perhaps if therapists came into the home and taught parents to carry out the treatment, the reduction of the children's motor delay by the intervention might have continued into the school years as did the milder effects of the infrequent home programme therapy. However, there would probably need to be periodic reassessments and programme changes for the children and refresher courses provided for the parents if programme effects were to be maintained.

Intensity and duration were other factors involved in effective treatment programmes. The most effective in-centre programmes were carried out 5 days a week for more than 4 years (Heber, 1978; Ramey & Haskins, 1981a; 1981b). Perhaps the intensive in-centre programme

needed to be more intensive (the average was 6 hours a week) or of longer duration (the average was 18 months) in order to maintain motor gains.

There are, thus, two possible alternatives to consider in order to try to maintain the shorter-term positive effects of the intensive programme. The first is to make parents an integral part of the therapy programme by teaching them to be the child's therapist. The second is to increase the intensity and duration of the in-centre programme. Obviously the most time-efficient alternative for the therapists would be to train the parents if this approach could be shown by research to be as effective as an intensive, prolonged in-centre programme. In this regard it has been shown that the intensity and duration of parent mediated therapy can be increased by appropriate parent education (Moxley-Haegert & Serbin, 1983). Thus, it is theoretically possible that the children's gains could be maintained and even increased with the home programme approach. When the negative psychological and behavioural side effects which appear to be the results of the intensive programme have been considered, one would certainly choose parent training and a home programme over the alternative.

Parents of the children in the intensively treated group reported poorer school performance than did parents of the other two groups. These results are noteworthy since the intensively in-centre treated children did not perform differently on reading or intelligence scores from children in the infrequent home programme treated group. It was noted that more intensively treated children attended special schools or

special classes (n=17) than did infrequently treated children (n=11). This might be because children who live outside the Greater Montreal area do not have opportunity to attend special schools but they would have the opportunity to attend special classes. It is interesting to note that at follow-up every intensively treated child was receiving extra help even if he/she were in a regular class (and one was functioning in an average range on all functional tests at follow-up) and five infrequently treated children were not receiving any extra help (four of these children were functioning in an average range on all of the functional tests given at follow-up). Thus it may be that the parents of intensively treated children reported poor school performance because their children were receiving extra help. The parents of intensively treated children also rated their children higher on the hyperactivity scale than did parents of non-delayed children while the infrequently treated group did not differ significantly from the other two groups. Achenbach used a sample of children who were referred and accepted for psychiatric treatment as his sample and children who had never been referred for treatment as his comparison group. He also found that one question, 'performs poorly in school', which is in the list of questions for the hyperactivity scale was the strongest predictor of psychiatric or psychological problems (Achenbach & Edelbrock, 1981). Thus, it would appear that these children (intensively treated) were having more psychological problems than the non-delayed group of children. It should be noted that some observations which connote hyperactivity overlap with measures of school

performance (e.g. "performs poorly at school", "has difficulty concentrating"). Thus if a child is thought to be doing poorly at school he/ she is likely to be scored higher on this hyperactivity scale. Another interpretation of these findings, therefore, is that parents of the intensively treated children may be sensitized towards looking at their children as being difficult and having problems. Alternately therapists may tend to refer these children to special schools or suggest special classes because they believe that these children need these services. Since most therapists are aware of the lack of motor interventions within the regular school systems it is likely that the therapists make referrals to special schools for motor delayed children. Irrespective of which interpretation is correct, the fact that a child has had special services nearly all of his/her life appears to have caused him/her to have a low self concept. The intensively treated children felt less accepted by their peers than did the children in Harter's standardization sample (Harter & Pike, 1984). As well, the parents of these children who have received so many special services appear to perceive their children as having more problems.

It would appear that there may be a need for a better balance between special services and no services. For example, two of the intensively treated children who at follow-up demonstrated normal intelligence were in special schools, while two of the retarded children at follow-up who were infrequently treated were in a regular class at school.

To summarize, it appears that although there may be shorter-term

gains for a child in the intensively treated programme there are longer-term losses both in terms of the child's self concept (feelings of peer acceptance) and in terms of the parents' attitude toward the child (parents reported poorer performance in school than both infrequently treated children and non-delayed children and more symptoms of hyperactivity than non-delayed children) . That is, intensive therapy appears to have the potential of damaging not only a child's feeling of self-esteem and self-worth, but also parental perception of the child, without having significant lasting effects on the child's motor development. It is, therefore, evident that if intensive therapy is to be offered to a child some programme changes are necessary. First, changes are necessary to ensure that the decrease in motor delay is maintained. These changes may include, as suggested previously, an increase in the intensity and duration of therapy. Second, therapy might include treatment within the home to provide greater normalization and greater generalization of gains. Preventative psychotherapy as well as treatment of existing behaviour problems would be a desirable objective. Fourth, and possibly most important the programme could be changed to encourage more family involvement in the actual therapy of the child. The intensively treated children could be encouraged to integrate into normal nursery schools and to become more involved in physical and social, peer related activities in order to encourage normalization. Parents need to be advised of the potential damaging effects of an intensive programme on their children's self-concept and on their own concept of their children and be advised how to combat

these effects. They need to learn to perceive their children as something other than deviant. Finally, the negative side effects of infrequent treatment must not be neglected. In particular there is a need for better follow-up in order to identify infrequently treated children who should not be in regular classes at school and intensively treated children who do not need special schools.

Significance of the Hypothesized Differential Effects of Intervention Therapy

Age distribution. As discussed in the introduction, Piaget's theory (1959) suggests that one should consider the necessity of early as opposed to late intervention for the motor delayed child. In the present study it was found at follow-up that children treated at a younger age functioned at a higher level than older treated children in both motor and intellectual measures, even though the former children were not treated for a longer period of time. This finding lends weight to Piaget's proposition that motor capabilities underlie later intellectual development and are necessary for them (Piaget, 1959). The finding that older treated children were more delayed than younger treated children at the time therapy began supports the thesis presented in the introduction of this paper and based on Piaget's work, that unremediated motor delay in a child could have a circular effect which would cause the child to become more and more delayed.

It becomes more and more important, therefore, for referring physicians to be aware that an early referral for therapy is essential even when the child's delay has a non-specifiable etiology (Fox, 1979).

The old idea of 'wait and the child will grow out of it' (Denhoff & Hyman, 1976) should be rejected. Only 5 of the 48 children evaluated in the follow-up study had no problems by age 7 or 8 (i.e. 'grew out of it'). This represents only 10% of all the delayed children. All 5 of the normally developing children at follow-up were treated before they were 2 years of age. When considering cost-effectiveness it would have been better to have treated all 48 children early, including the 10% who may not have needed treatment, rather than to have left some of these children untreated until later thus leaving them with the potential of becoming functionally retarded or of having a lower functional level than they might have had, by the time they entered school. These findings, of course, cannot be considered definitive because it is not known if other events occurred which might have caused the retardation. No specific reason could be found for the difference between the retarded-borderline children and the children of at least low average intelligence other than the age at which treatment began. In this retrospective study it was shown that younger treated children were more responsive to therapy in the shorter-term and maintained the decrease in motor delay at follow-up several years later. Duration of therapy was not different for the younger treated and older treated children so it cannot be said that the younger treated children are functioning at a higher level because they had more therapy. It is thus suggested by the data of this study that the younger treated children were functioning at a higher level because the compounding effects of motor delay on later intellectual functioning was negated or reduced by therapy at an earlier

age. Clearly, a prospective study examining the differential effects of age at initiation of therapy is necessary.

Significance of Non-hypothesized Findings

Intelligence distribution. It was shown in the follow-up phase of this study that a significant proportion of the children with a motor delay of non-specifiable etiology (n = 23 of 48 motor delayed children in the follow-up study) were either mildly mentally retarded, had borderline intelligence or could not be tested; non-testability was presumably due to mental retardation or severe emotional problems. This sub-population of delayed children differed from the delayed children who were of at least low average intelligence in that they tended to be evenly delayed in all levels of function and were in special schools or special classes. For example, all six children who were untestable were in special schools. Seven of the 17 testable children who were retarded were in special schools, three were in special classes within the regular school system and one was in a regular class but had failed. The six children with borderline intelligence quotients were divided evenly between special schools, special classes, and regular classes, each child having failed one year in the regular class.

Twenty-five of the 48 children evaluated at follow-up had achieved at least a low average intelligence quotient at follow-up. Only 2 of these 25 children were attending special schools but five were in learning disability classes. The rest (n = 18) were attending regular classes in a regular school, but 12 of these children had either repeated a grade or had received free flow help, help from a resource

teacher or extra tutoring. Thus only 6 of these 25 children had been apparently functioning at an average level in school.

Three major variables were found which discriminated between children with at least a low average intelligence and children who were retarded or had borderline to average intelligence. These were: 1) the average percentage motor delay at initial assessment; 2) age at initial assessment, and; 3) gender. Children who had average intelligence at follow-up were treated earlier and were less delayed at the onset of treatment than the others. The children of average intelligence were mostly males. In contrast the retarded or borderline children consisted of about one-half males and females. The children with borderline or retarded intelligence quotients also tended to come from families of higher socioeconomic levels who had more social problems and more often had other delayed members in the family.

Clearly there must be some reason why male children were referred at an earlier date for assessment and therapy. It could be speculated that it is because families tend to refer their delayed boys for early assessment more readily than their delayed girls. This idea certainly is supported by the finding that many more non-delayed males than non-delayed females were referred for assessment of a potential delay (34 males to 12 females). Perhaps family problems could account for difference in referral for therapy. There is evidence to indicate that in some families a child's poor response to therapy is symptomatic of a generalized family dysfunction (Kearsley, 1978; Posnanski, 1973). In many of these families there appears to be a need for the child not to

get well too quickly (Kord, 1982). A common example of this is a family in which the child's problems function as a distancing mechanism between the spouses (Kord, 1982). One could speculate that for some of these children this malfunctioning family system operated and caused both a late referral and poor response to therapy. Yet, it is also known that the delayed child can be a factor in causing social problems in the family (Kord, 1982; Posnanski, 1973).

One suggestion as to why two different sub-populations of children with a motor delay of non-specifiable etiology exist at follow-up is that the sub-populations were separate from the beginning. If this is so there must be some natural selection factor that determined early versus late treatment. For example, a child who is developing unevenly (i.e. a child who has higher level language skills than gross motor skills) would probably be referred earlier than a child who is developing more evenly. In this study no indication was found, however, that the early treated children were developing more unevenly than the late treated children. There was no difference between the number of early and late treated motor delayed children who had no delay in language.

One conclusion is certain. There is a need to look at the variables which influence early child referral for therapy. The importance of this, as mentioned previously, is due to the fact that earlier treated children function on many variables at a significantly higher level when they are 7 and 8 years of age than later treated children.

Gender distribution. In the delayed population of the present study there were slightly more males than females (ratio of male to females was 1.2/1). Males are said to be affected more often than females with many different neuro-psychiatric and developmental disorders (Rutter, 1977). In this study males were referred for evaluation more often than females even when they had no delay. It may be that society is sensitized to the idea that males are more often affected (Rutter, 1977) and therefore tend to refer males more readily but in reality males may not be more often affected. At least males were not more often affected in the present study which consisted of children with a motor delay of non-specifiable etiology. It was also noted in this study that males who were treated intensively, outnumber females by nearly 4 to 1. Although gender did appear to influence whether treatment was received (more females did not receive early therapy and did not receive intensive therapy), gender did not appear to influence the effectiveness of therapy. That is, both males and females did benefit from therapy as demonstrated by a decrease in their motor delay from initial to discharge assessment. Most females who did receive therapy were older (over 2 years of age) when the assessment was provided. It would appear that somewhere along the continuum (parent, doctor, therapist) that brings a motor delayed child into therapy, there is a bias against treating females and towards treating males. How can this be explained? There was no difference between the ratio of males to females treated by infrequent home programme therapy (1.08/1) than in the general delayed population (1.2/1). It would appear therefore, that

the bias was not with the therapists who offered home programme service. They offered therapy to all delayed children who were assessed in their department. Whether parents showed a gender bias in home treatment and whether only some parents carried out the home programme could not be determined in this study. However, many more males than females were accepted into treatment for intensive therapy. It is not known if more females were referred for this type of therapy than were accepted for therapy. Family motivation is an essential criterion for acceptance into the intensive therapy programme; the child of a family that does not appear highly motivated will not be accepted. As well, it is known that doctors are reluctant to refer for therapy any child with a delay of non-specifiable etiology (Fox, 1979). These two facts suggest that the bias may come from the families because it is likely that only a motivated family will manage to get a child with a delay of non-specifiable etiology referred for intensive therapy or the parents themselves will refer the child for intensive therapy. It would appear, therefore, that parents are more motivated to have their sons rather than their daughters treated in a programme which is intensive and that more often parents choose not to have their daughters treated until later. This bias against therapy for females crosses all social classes and may reflect a cultural bias that a female who is delayed can adapt more readily to many societal expectations than can a delayed male. Thus it appears that it is more important for parents that a delayed male receive therapy than a delayed female. Moreover it seems that sexual inequality is very basic at least as manifested by the treatment of

delayed children. The consequences, according to this study appear to be great. The percentage of females at follow-up who were either retarded or of borderline intelligence was much greater than the percentage of females in the average intelligence range.

Conclusions and Research Suggestions

Since this study was retrospective, it should be treated as a pilot project for a prospective study. It should be considered more as a study from which questions for future research are derived than one from which definitive answers were found. Suggestions for possible answers, however, could be promulgated. One clear finding was that a population of children exists with a delay of non-specifiable etiology for which no demonstrable variables other than motor delay could separate it from a normal non-delayed population at initial assessment. At follow-up assessment these children with a delay of non-specifiable etiology differed in motor capabilities, intelligence and school performance from non-delayed children. It could be concluded, as well, that this population is indeed significant since it included nearly one-third of all delayed children referred for assessment. Therefore this population should be given further attention and further study by those interested in child development.

Another important indication from this study is that at least in the shorter-term these children appear not only to be responsive to therapy, but actually more responsive than do children with a specifiable etiology for their delay. There is a need, therefore, to investigate in the future, the variables which tend to influence the

most effective therapy. This study's results indicated that in the shorter-term intensive(in-centre) therapist mediated therapy is more effective in reducing children's motor delay than infrequent (home programme) parent mediated therapy but this greater decrease in motor delay is not maintained at follow-up several years after therapy had stopped. It would appear that research needs to examine other variables in order to determine a therapy which is effective in maintaining a decrease in motor delay. The findings that for some variables children who were treated before 2 years of age were functioning at a significantly higher level at follow-up than children who were treated later suggest that; the parameter to determine whether intervention has an effect on subsequent development of the motor delayed child is age at initiation of therapy. If we consider the age at onset of therapy it has been shown in this study that therapy does have a longer-term effect. We cannot conclude, however, on the basis of therapy modality (intensive, in-centre, therapist mediated therapy versus infrequent, home programme, parent mediated therapy) that therapy has any beneficial effect in terms of remediating motor delay. There is the possibility, never-the-less, that therapy modality affects other parameters of behaviour such as hyperactivity. This was seen in the present study. If the findings that therapy is effective if initiated before the age of 2 can be replicated then further demographic research is necessary in order to identify the parameters which influence early referral for evaluation and therapy. Moreover, one would want to determine how physicians and parents can be influenced to refer children (females

as well as males) for therapy at as young an age as possible.

In conclusion, several questions for future research are raised. There has been little information about the effect of therapy for these children with a motor delay of non-specifiable etiology. There has not been much information, also, as to whether an etiology can be established for these children's delays. The present study has made an important beginning, however, in providing some information about the motor delayed child with a non-specifiable etiology for the delay. Prospective controlled trials of therapy are needed to determine the necessity for treatment, the choice of various treatment methods, the duration of treatment, and the optimal age at which therapy should be introduced. This retrospective study has indicated that(in-centre) therapist mediated therapy which is at least 6 hours per week for at least 12 months (averaging 18 months) is more effective in decreasing a child's delay by the time of discharge from therapy, than infrequent (home programme) parent mediated therapy of no more than 10 hours of therapist mediated therapy in a year. The decrease in delay promoted by the intensive therapy, however, was not maintained when the child was assessed several years after discharge from therapy while the results have indicated the possibility that there are some negative side effects of intensive therapy on both child and parent attitudes. It is not known, what can be done to reduce these negative side effects although suggestions have been given and research is necessary to verify the effectiveness of these suggestions. It was not possible to determine from this study whether untreated

delayed children would be any different from treated children at school age. This study did indicate that therapy is effective if it is initiated before the age of 2 because earlier treated children achieved higher motor and intelligence scores, functioned better at school and participated in more social activities than later treated children when they were all 7 or 8 years of age. A more definitive answer to the therapy effectiveness question would entail a longitudinal study of a population in which therapy is initiated before 2 years of age with monitoring of motor, behavioural and cognitive development into the first years of school. Such a study could compare three groups of motor delayed children with a non-specifiable etiology of delay: 1) non-treated children who did not follow-up on suggestions to receive therapy; 2) in-centre, therapist mediated treated children; and, 3) home programme parent mediated treated children. All children in all groups would be assessed before 2 years of age and the treated children would begin therapy before 2 years of age.

References

Achenbach, T. M. (1978). The child behaviour profile, I: Boys age 6-11. Journal of Consulting and Clinical Pychology, 46, 759-776.

Achenbach, T. M. (1979). The child behavior profile: an empirically based system for assessing children's behavioral problems and competencies. International Journal of Health, 1979, 7, 24-42.

Achenbach, T.M. (1982). The child behavior check list and child behavior profile. Child, adolescent, family and community psychiatry. University of Vermont, 1 South Prospect St., Burlington, Vermont.

Achenbach, T. M. & Edelbrock, C. S. (1979). The child behavior profile, II: Boys aged 12-16 and girls aged 6-11 and 12-16. Journal of Consulting and Clinical Psychology, 47, 223-233.

Achenbach, T. M., & Edelbrock, C. S. (1981). Behavioral problems and competencies reported by parents of normal and disturbed children aged four through sixteen. Monographs of the Society for Research in Child Development, 46, (Serial No. 188).

Anwar, F. & Hermelin, B. (1979). Kinaesthetic movement after-effects in children with Down's Syndrome. Journal of Mental Deficiency Research, 23, 287-297.

Asher, E. J. (1939). The inadequacy of current intelligence tests for testing Kentucky Mountain children. Journal of Genetic Psychology, 46, 480-486.

Barna, S., Bidder, R. T., Gray, O. P., Clements, J. & Gardner, S. (1980). The progress of developmentally delayed preschool children in a training scheme. Child Care Health and Development, 6(3), 157-164.

Berkson, G. (1960a). An analysis of reaction time in normal and mentally deficient young men. I. Duration threshold experiment. Journal of Mental Deficiency Research, 4, 51-58.

Berkson, G. (1960b). An analysis of reaction time in normal and mentally deficient young men. II. Variations of complexity in reaction time. Journal of Mental Deficiency Research, 4,59-67.

Blatt, B., & Garfunkel, F. (1967). Educating intelligence: Determinants of school behavior of disadvantaged children. Exceptional Children, 23, 601-608.

Bobath, K. & Bobath, B. (1972). Cerebral palsy. In P. H. Pearson and C. A. Williams (Eds.) Physical Therapy Services in the Developmental Disabilities. Springfield, Ill., Charles C. Thomas, 31-185.

Braun, J. J. (1978). Time and recovery from brain damage. In S. Finger (Ed.) Recovery from brain damage. New York: Plenum Press.

Brenner, M. W. & Gillman, S. (1966). Visuomotor ability in school age children - a survey. Developmental Medicine & Child Neurology, 8, 686-703.

Brewer, N. & Nettlebeck, T. (1977). Influence of contextual cues on the choice reaction time of mildly retarded adults. American Journal of Mental Deficiency, 82, 37-43.

Broman, S. H., Nichols, P. L. & Kennedy, W. A. (1975). Preschool IQ: Prenatal and early developmental correlates. New Jersey: Laurence Erlbaum, Assoc.

Bromwich, R. M. (1977). Stimulation in the first year of life? A perspect on infant development. Young Children, 1, 71-82.

Bromwich, R. M. & Parmelee, A. H. (1979). An intervention program for preterm infants. In T. R. Field, A. Sostek, S. Goldberg & H. H. Shuman (Ed.) Infants born at risk. Jamaica, New York: Spectrum.

Bronfenbrener, U. (1968). Early deprivation: A cross species analysis. In S. Levine & G. Newton (Eds.) Early experience in behavior. Illinois: Charles C. Thomas.

Bronfenbrenner, U. (1975). Is early intervention effective? In B. Z. Friedlander, G. M. Sterrit & G. E. Kirk (Eds.) Exceptional infant: Assessment and intervention. Vol. 3. New York: Brunner/Mazel.

Browder, J. A. (1981). The pediatrician's orientation to infant stimulation programs. Pediatrics, 67, 42-46.

Bruinicks, R. H. (1964). Physical and motor development in retarded persons. In N. R. Ellis (Ed.) International review of research in mental retardation, Vol. 4. London: Academic Press.

Bruinicks, R. H. (1978). Bruinicks- Oseretsky Test of Motor Proficiency. American Guidance Service Inc., Circle Pines, Minnesota.

Caldwell, B. M., Bradley, R. H. & Elardo, R. (1975). Early stimulation. In E. Wortis (Ed.) Mental retardation and developmental disabilities. Vol. 7. New York: Brunner/Mazel.

Campbell, S. (1974). Facilitation of cognitive and motor development in infants with central nervous system dysfunctions. Physical Therapy, 45(1), 346-353.

Cicirelli, V. (1969). The impact of Head Start: An evaluation of the efforts of Head Start on children's cognitive and affective development. Vol. 1. A report to the Office of Economic Opportunity. Athens, Ohio: Westinghouse Learning Corporation and Ohio University.

Clarke, A.M. & Clarke, A. D. B. (1976). Early experience: Myth and evidence. New York: The Free Press.

Coopersmith, S. (1967). The antecedents of self-esteem. San Francisco: W. H. Freeman.

Costeff, H., Cohen, B. E. & Weller, L. E. (1983). Biological factors in mild mental retardation. Developmental Medicine and Child Neurology, 25, 580-587.

Czeizel, A., Lanyi-Engelmayer, A., Klugber, L., Metneki, J., & Tusnady, G. (1980). Etiological study of mental retardation in Budapest, Hungary. American Journal of Mental Deficiency, 85(2), 120-128.

Davis, K. (1940). Extreme social isolation of a child. American Journal of Sociology, 45, 554-565.

Davis, K. (1947). Final note on a case of extreme isolation. American Journal of Sociology, 52, 432-437.

Day, M. C. & Parker, R. D. (Eds). (1977). The preschool in action: Exploring early childhood education programs. (2nd ed.). Boston: Allyn & Bacon.

Denhoff, E. & Hyman, I. (1976). Parent programs for developmental management. In T. D. Tjossem, (Ed.). Intervention strategies for high risk infants and young children. Baltimore: University Park Press.

Dennis, W. & Nazarian, P. (1957). Infant development under environmental handicap. Psychology Monographs, 71, 1-13.

Drillien, C. M., Thomson, A. J. M., & Burgoyne, K. (1980). Low-birthweight children at early school-age: A longitudinal study. Developmental Medicine and Child Neurology, 22, 26-46.

Edelbrock, C. S. & Achenbach, T. M. (1980). A typology of child behavior profile patterns: Distribution and correlates for disturbed children aged 6-16. Journal of Abnormal Child Psychology, 8, 441-470.

Ellison, P. H., Horn L. & Browning, C. (1981). Multiple variable predictors of motor dysfunction of infancy (MDI) in neonatal intensive care unit (NICU) population. Medical College of Wisconsin, Milwaukee, University of Denver, Colorado.

Field, T. (1980). Supplemental stimulation of preterm neonates. Early Human Development, 3, 301-314.

Field, T., Hallock, N., Ring, G., Demprey, J., Dabini, D., & Shuman, H. (1978). A first year follow-up of high-risk infants: Formulating a cumulative risk. Child Development, 49, 1-4.

Field, T., Sostek, A., Goldberg, S. & Shuman, H. H. (1979). Infants born at risk. Jamaica, New York: Spectrum.

Findlay, E. (1979). Remediation of developmental delay in infants: A comparative study of two methods. Unpublished Master's thesis, McGill University, Montreal.

Fox, N. (1979). Attitudes of referring physicians to some aspects of developmental therapy (London District Crippled Children's Treatment Centre), 385, Hill Street, London, Ontario N6B 1E4. Developmental Medicine and Child Neurology, 21, 113.

Francis, S. H. (1971). The effects of own-home and institutional rearing on the behavioral development of normal and mongol children. Journal of Child Psychology and Psychiatry, 12, 173-190.

Gallagher, J. J. (1973). Preventative intervention. Pediatric Clinics of North America, 20(3), 681-693.

Garber, H. & Heber, F. R. (1977). The Milwaukee project. Indications of effectiveness of early intervention in preventing mental retardation In P. Mittler (Ed.) Research to practice in mental retardation. Vol. 1. Baltimore: University Park Press, 1977.

Gillberg, C. (1980). Maternal age and infantile autism. Journal of Autistic Developmental Disorders, 10, 293-297.

Gillberg, I. C. & Gillberg, C. (1983). Three year follow-up at age 10 of children with minor neurodevelopmental disorders. I. Behavioural problems. Developmental Medicine and Child Neurology. 25, 437-449.

Gillberg, I. C., Gillberg, C. & Rasmussen, P. (1983). Three year follow-up at age 10 of children with minor neurodevelopmental disorders. II. School achievement problems. Developmental Medicine and Child Neurology, 25, 566-579.

Gillberg, C. & Rasmussen, P. (1982). Perceptual, motor and attentional

deficits in 7 year old children: Background factors. Developmental Medicine and Child Neurology, 24, 753-770.

Gillberg, C., Rasmussen, P., & Wahlstrom, J. (1982). Minor neurodevelopmental disorders in children born to older mothers. Developmental Medicine and Child Neurology. 24, 437-447.

Gilmer, B., Miller, J. O. & Gray, S. W. (1975). Intervention with mothers and young children: Study of intra-family effects. Nashville, Tenn.: DARCEE Demonstration and Research Center for Early Education, 1970. Cited in Bronfenbrenner, U. Is early intervention effective? In B. Z. Friedlander, G. M. Sterrit & B. E. Kirk (Eds.) Exceptional infant. Vol. 3. New York: Brunner/Mazel.

Goldfarb, W. (1945). Effects of psychological deprivation in infancy and subsequent stimulation. American Journal of Psychiatry, 102, 18-33.

Goodson, B. & Hess, R. (1983). The effects of parent training programs on child performance and parent behavior. Unpublished manuscript, Standford University 1976. Cited in Slauter, D. Early intervention and its effects on maternal and child development. Monographs of the Society for Research in Child Development, 4,(Serial No. 202).

Gordon, H. (1923). Mental and scholastic tests among retarded children. Education pamphlet 44. London: Board of Education.

Gordon, I. J. (1973). Early child stimulation through parent education. In L. Stone, J. Smith & L. Murphy (Eds.) The competent infant. New York: Brunner/Mazel.

Gordon, I. J., Guinagh, B. & Jester, R. D. (1970). The Florida parent education infant and todler programs. In M. C. Day and R. K. Parker (Eds.). The preschool in action: Exploring early childhood education programs. (2nd ed.). Boston: Allyn & Bacon.

Gordon, N. & Mckinlay, I. (Eds). (1980). Helping clumsy children. Edinburgh: Churchhill, Livingstone, Great Britain.

Gray, S. W. & Wandersman, L. P. (1980). The methodology of home-based intervention studies: Problems and Promising strategies. Child Development, 51, 993-1009.

Gubbay, S. S. (1975). The clumsy child. Philadelphia: Saunder.

Gustavson, K. H., Hagberg, B., Hagberg, G., & Sars, K. (1977). Severe mental retardation in a Swedish country. II. Etiology and pathogenic aspects of children born in 1959-1970. Neuropaediatrie , 8, 293-304.

Harris, S. R. (1982). Effects of neurodevelopmental therapy on motor

performance of infants with Down syndrome. Developmental Medicine and Child Neurology, 23, 477-483.

Harter, S. (1978). Effectance motivation reconsidered: Toward a developmental model. Human Development, 1, 34-64.

Harter, S. (1981). A model of master motivation in children: Individual differences and developmental change. In Collins (Ed.), Minnesota symposium on child psychology, Vol. 14. Hillsdale, New Jersey: Lawrence Erlbaum.

Harter, S. (1982). The perceived competence scale for children. Child Development, 53, 87-97.

Harter, S & Pike, R. (1984). The pictorial scale of perceived competence and social acceptance for young children. Child Development, 55, 1969- 1982.

Hebb, D. O. (1947). The effects of early experience on problem-solving at maturity. American Psychologists, 2, 306-307.

Heber, F. R. (1978). Sociocultural mental retardation: A longitudinal study. In D. Forays (Ed.) Primary prevention of psychopathology. Vol. 2. Hanover, N. H.: University Press of New England.

Hecean, H. & Albert, M. C. (1978). Human neuropsychology. New York: Wiley.

Henderson, A. (1981). Research in occupational therapy and physical therapy with children. In B.W. Camp (Ed.) Advances in behavioral pediatrics, Greenwich, Conn: Jai Press Inc.

Henderson, S. E. & Hall, D. (1982). Concomits of clumsiness in young school children. Developmental Medicine and Child Neurology, 24, 448-460.

Hertzig, M. (1981). Neurological 'soft' signs in low-birth weight children. Developmental Medicine and Child Neurology, 1981, 23, 778-791.

Hewison, J. (1982). The current status of remedial intervention for children with reading problems. Developmental Medicine and Child Neurology, 24, 183-186.

Hogg, J. (1982). Motor development and performance of severely mentally handicapped children. Developmental Medicine and Child Neurology, 24, 188-193.

Hollingshead, A. B. (1957). Two factor index of social position. New Haven, Conn,: Yale University, Department of Sociology.

Hollingshead, A. B. & Redlick, F. C. (1958). Social class and mental illness, John Wiley & Sons, New York.

Horowitz, F. D. & Paden, L. Y. (1973). The effectiveness of environmental intervention programs. In B. M. Caldwell & H. N. Ricciuti (Eds.) Review of child development research. Chicago: University of Chicago Press.

Horton, D. B. (1976). Early intervention for hearing-impaired infants and young children. In T. D. Tjossem (Ed.) Intervention strategies for high risk infants and young children. Baltimore: University Park Press.

Huck, S. W. & McLean, R. A. (1975). Using a repeated measures ANOVA to analyze data from a pretest-post-test design: A potentially confusing task. Psychological Bulliton, 82(4), 511-518.

Hunt, J. McV. (1961). Intelligence and experience. New York: Ronald Press.

Itard, J. M. G. (1932). The wild boy of Aveyron. Translated by G. and M. Humphrey. New York: Appleton-Century-Crofts.

Jayaxkara, R. & Street, J. (1978). Parental age and parity in dyslexic boys. Journal of Biosocial Science, 10, 225-261.

Jensen, A. R. (1981). Raising the IQ: The Ramey and Haskins study. Intelligence, 5, 29-40.

Karlsen, B., Madden, R. & Gardner, D. (1976). Stanford diagnostic reading test., New York:Harcourt Brace Jonanovich.

Kames, M. (1969). Evaluation and implication of research with young handicapped children and low income children. In J. Stanley (Ed.) Research and development program on preschool disadvantaged children: Final report. Washington, D. C.: U. S. Office of Education.

Karnes, M. B., Teska, J., Hodgens, A. & Badger, E. (1970). Educational intervention at home by mothers of disadvantaged infants. Child Development, 41, 925-935.

Kearsley, R. B. (1978). Introgenic retardation: A Syndrome of learned incompetence. In R. B. Kearsley and I. Sigel (Eds.) Infants at risk: Assessment of cognitive functioning. Hillsdale, N. J.:Lawrence Erlbaum.

Kertesz, A. (1979). Recovery and treatment. In K. M. Heilman & E. Valenstein (Eds.) Clinical neuropsychology. New York: Oxford University Press.

Kirk, S. A. (1958), Early education of the mentally retarded: An experimental study. Urbana: University of Illinois Press.

Kirk, S. A. (1969). The effects of early education with disadvantaged infant In M. B. Karnes (Ed.) Research and development program on preschool disadvantaged children: Final report. Washington, D. C.: U. S. Office of Education.

Klaus, R. A. & Gray, S. W. (1967). The early training project for disadvantaged children: A report after five years. Nashville, Tenn: George Peabody College for Teachers.

Kord, D. (1982). A family-oriented approach to the treatent of developmentally delayed preschoolers. Paper presented at the 59th Annual Meeting of the American Orthopsychiatry Association, San Francisco.

Langer, J. (1969). Theories of development. New York: Holt, Rinehart & Winston, Inc.

Lazar, I. & Darlington R. (1982). Lasting effects of early education: A repo from the consortium for longitudinal studies. Monographs of the Society for Research in Child Development, 47(2-3),(Serial No. 195).

Levenstein, P. (1970).Cognitive growth in preschoolers through verbal interaction with mothers. American Journal of Orthopsychiatry, 40, 426-432.

Levenstein, P. (1977). The mother-child home program. In M. C. Day & R. K. Parker (eds.) The preschool in action: Exploring early childhood education programs. (2nd ed.). Boston: Allyn & Bacon.

Liff,, S. (1976). Early intervention and language development in hearing impaired children. Unpublished master's thesis, Vanderbilt University, Nashville. Cited in T. D. Tjossem (Ed.) Intervention strategies for high risk infants and young children. Baltimore: University Park Press.

Llorens, L.A. (1970). 1969 Eleanor Clark Slagle lecture: Facilitating growth and development: The promise of occupational therapy. American Journal of Occupational Therapy, 26, 1-9.

Mackay, R. I. (1982). The causes of severe mental handicap. Developmental Medicine and Child Neurology, 24, 386-393.

Masi, W. (1979). Supplemental stimulation of the premature infant. In T. Field, A.Sostek, S. Goldberg, & H. H. Shuman (Eds.) Infants born at risk. Jamaica, New York: Spectrum.

Meier, J.H. (1976). Developmental and learning disabilities. Vol. 1. Baltimore: University Park Press.

Miller, L. B & Bizzell, R. P. (1984). Long-term effects of four preschool programs: Ninth and tenth grade results. Child Development, 55, 1570-1587.

Milman, D. H. (1979). Minimal brain dysfunction in childhood: Outcome in late adolescence and early adult years. Journal of Clinical Psychiatry, 40(9), 371-380.

Mittler, P. (Ed). (1977). Research to practice in mental retardation: Care and intervention. Vol. 1. Baltimore: University Park Press.

Molnar, G. H. (1975). Analyses of motor disorder in retarded infants and young children. American Journal of Mental Deficiency, 83, 213-222.

Morrison, D. & Pothier, P. (1972). Two different remedial motor training programs and the development of mentally retarded preschoolers. American Journal of Mental Deficiency, 77, 251-258.

Moxley-Haegert, L. (1977). The influence of verbal reinforcement and tactile plus verbal reinforcement on learning. Unpublished Honour's thesis, Concordia University, Montreal.

Moxley-Haegert, L. (1981). Parent developmental training and its effects on parental compliance. Master's thesis, Concordia University, Montreal.

Moxley-Haegert, L. & Serbin, L. (1983). Developmental education for parents of delayed infants: Effects on parental motivation and children's development. Child Development, 54, 1324-1333.

Multhern T. & Baumeister, A. A. (1971). Effects of stimulus-response compatibility and complexity upon reaction time of normals and retardates. Journal of Comparitive and Physiological Psychology, 75, 459-463.

Nettlebeck, T. & Brewer, N. (1976). Effects of stimulus-response variables on the choice reaction time of mildly retarded adults. American Journal of Mental Deficiency, 81, 85-92.

Northcott, W. (1973). Implementing programs for young hearing impaired children. Exceptional Children, 39, 455-463.

Oberlaide, R., Dworkin, R. H. & Levine, N. (1979). Developmental behavioral dysfunction in preschool children. American Journal of the Disabled Child, 133, 1126-1131.

Ohwaki, S. & Stayton, S. E. (1978). The reaction of length of institutionalization on intellectual functioning of the profoundly retarded. Child Development, 49, 105-109.

O'Reilly, D. E. & Walentynowicz, J. C. (1981). Etiological factors in cerebral palsy: An historical review. Developmental Medicine and Child Neurology, 23,633-642.

Orton,, S. T. (1937). Reading, writing and speech problems in children. New York: Norton.

Penrose, L. S. (1963). The biology of mental deficit, Vol 3. London: Sidgwick & Jackson.

Penrose, L. S. (1972). The biology of mental deficit, Vol. 4. London: Sidgwick & Jackson.

Piaget, J. (1951). Plays, dreams and imitations in childhood. (C. Gattegno & F.M. Hodgson, Trans.). New York: Norten.(Original work published 1945)

Piaget, J. (1952). The origins of intelligence in children. (Margaret Cook,Trans.). New York: International University Press.(Original work published 1936)

Piaget, J. (1954). The construction of reality in the child.(Margaret Cook,Trans.). New York: Basic Books.(Original work published 1937)

Piaget, J. (1959). The psychology of intelligence. (M. Piercy & D.E. Berlyne,Trans.). London: Routledge & Kegan Paul. (Original work published 1947)

Piaget, J. (1970). Piaget's theory. In P. H. Mussen (Ed.) Carmichael's Manual of Child Psychology. Vol. 1. New York: John Wiley.

Piers, E. & Harris, D. (1969) The Piers-Harris children's self-concept scale. Nashville, Tenn: Counselor Recordings & Tests.

Piper, M. C. & Pless, I. B. (1980). Early intervention for infants with Down Syndrome: A controlled trial. Pediatrics, 65(3), 463-468.

Poznanski, E. (1973). Emotional issues in raising handicapped children. Rehabilitative Literature, 34,(11), 322-326.

Provence, S. & Liton, R. C. (1962). Infants in institutions: A comparision of their development with family reared infants during the first years of life. New York: International University Press.

Ramey, C. T. & Campbell, R. A. (1979). Compensatory education for disadvantaged children. School Review, 87, 171-189.

Ramey, C. T. & Haskins, R. (1981a). The modification of intelligence through early experience. Intelligence, 5, 5-19.

Ramey, C. T. & Haskins, R. (1981b). Response to Jensen and Hunt. Intelligence, 5, 41-48.

Ramey, C. T. & Smith, B. J. (1977). Assessing the intellectual consequences of early intervention with high-risk infants. American Journal of Mental Deficiency, 81, 318-324.

Rarick, G. L. (1973). Motor performance of mentally retarded children. In G. L. Rarick (Ed.) Physical activity: Human growth and development. London: Academic Press.

Rosenberg, S. A. (1978). Family and parent variables affecting outcomes of parent-mediated intervention. Doctoral dissertation, George Peabody College for Teachers, 1977. Dissertations Abstracts International, 38(8-B), 3904.

Ruben, R. A. & Barlow B. (1980). Infant neurological abnormalities as indicators of cognitive impairment. Developmental Medicine and Child Neurology, 22, 336-343.

Rutter, M. (1977) Speech delay. In M. Rutter & L. Hersov (Eds.) Child psychiatry- Modern approaches. Oxford: Blackwell.

Rutter, M. (1980). The longterm effects of early experience. Developmental Medicine and Child Neurology, 22, 800-815.

Scarr-Salapatek, S. & Williams, N. (1973). The effects of early stimulation on low birth weight infants. Child Development, 29, 210-217.

Sandown, S. A., Clarke, A. D. B., Cox, M. V. & Stewart, F. L. (1981). Home intervention with parents of severely subnormal pre-school children: A final report. Child: Care, Health and Development, 7, 135- 144.

Schafer, L. & Moersch, M. (1977). Developmental programming for infants and young children. Vol. 2. Ann Arbor: University of Michigan Press.

Schweinhart, L. J. & Weikart, K. P. (1980). Young children grow up: The effects of the Perry preschool program on youth through age 15, Ypsilanti, Mich: High/Scope Press.

Sherman, M. & Key, C. B. (1932). The intelligence scores of isolated

mountain children,. Child Development, 3, 279-290.

Simeonsson, R. J., Cooper, D. H. & Scheiner, A. P. (1982). A review and analysis of the effectiness of early interventions. Pediatrics, 69, 635-641.

Skeels, H. M. & Dye, H. B. (1939). A study of the effects of differential stimulation on mentally retarded children. Proceedings and Addresses of American Association on Mental Deficiency, 44, 114-136.

Skodak, M. & Skeels, H. M. (1945). A follow-up study of children in adoptive homes. Journal of Genetic Psychology, 66, 21-58.

Skodak, M. & Skeels, H. M. (1949). A final follow-up study of one hundred adopted children. Journal of Genetic Psychology, 75, 85-125.

Smith, R. (1981). Abnormal head circumference and learning disabled children. Developmental Medicine and Child Neurology, 23, 626-632.

Spicker, H. H., Hodges, W. L. & McCandless, B. R. (1966). A diagnostically based curriculum for psychosocially deprived, preschool, mentally retarded children: Interim report. Exceptional children, 33, 215-220.

Spitz, R. A. (1946). Hospitalism: a follow-up report. Psychoanalytic Study of the Child, 2, 113-117.

Stipek, D. J. (1981). Children's perceptions of their own and their classmates ability. Journal of Educational Psychology, 73, 404-410.

Talbot, G. (1974,1977). Batterie d'evaluation Talbot. Service d'ergotherapie, Hopital Ste-Justine. 3175 Cote Ste. Catherine, Montreal.

Tizard, J.& Grad, J. (1971). The mentally handicapped and their families, London: University Press.

Turner, R. (1978). Multivariate assessment of therapy outcome research. Therapy and Experimental Psychology, 9, 309-314.

Tjossem, T. D. (Ed.). (1976). Intervention strategies for high risk infants and young children. Baltimore: University Park Press.

Wade, M., Newell,,K. M. & Wallace, S. A. (1978). Decision time and movement time as a function of response complexity in retarded persons. American Journal of Mental Defieiciency, 83, 125-144.

Wechsler, D. (1974). Wechsler intelligence scale for children-revised. The psychological corporation, New York, New York.

Webb, R. C. & Koller, J. R. (1979). Effects of sensorimotor training on intellectual and adaptive skills of profoundly retarded adults. American Journal of Mental Deficiency, 83, 490-496.

Weikart, D. P. (1967). Results of preschool intervention programs. In D. Weikart (Ed.) Preschool intervention: A preliminary report of the Perry Preschool Project. Ann Arbor, Mich.: Campus Publishers.

Werner, E. E., Beinman, J. M. & French, F. E. (1971). The children of Kauai. Vol. 3. Honolulu: University of Hawaii Press.

Wheeler, L. T. (1932). The intelligence of east Tennessee mountain children. Journal of education and psychology, 23, 353-371.

White, B. (1977). Early stimulation and behavioral development. In Oliverio (Ed.) Genetics, environment and intelligence, Elsevier/North Holland: Biomedical Press.

White, R. (1959). Motivation reconsidered: The concept of competence. Psychological Review, 66, 297-323.

White, S. H. (1973). Federal programs for young children: Review and recommendations. 3 Vols. Washington, D. C.: Education and Welfare.

Williams M. & Scarr, S. (1971). Effects of short term intervention on program performance in low birth weight disadvantaged children. Pediatrics, 37(1), 289-297.

Wright, T. & Nicholson, J. (1973). Physiotherapy for the spastic child. An evaluation. Developmental Medicine and Child Neurology, 15, 146-163.

Younes, R. P., Rosner, B. & Webb, G. (1983). Neuroimmaturity of learning-disabled children: A controlled study. Developmental Medicine and Child Neurology, 25, 574-579.

Zigler, E. & Trickett, P. K. (1978). IQ, social competence, and evaluation of early childhood intervention programs. American Psychologist, 33, 789-798.

Appendix A

Medical Records Data Collection Form

SES_____ TREATED___yes___no

Intake Percentage Delay_____%

Date Information Recorded_____

Child's Name _____ I.D.No._____
(first) (last)

Age (in months) at first admission for treatment(assessment)___

Date of Birth_____ Chronological age Years__months___

Gender_____

Reason for referral - original admission _____

_____

Percentage Delay	Initial Assessment Developmental Age	Initial Assessment Percentage Delay	Dischage Assessment Developmental Age	Dischage Assessment Percentage Delay
Gross Motor				
Fine Motor				
Perception				
Language				
Socialization				
Mental Age				
Average Percentage Motor Delay (Gross, Fine and Perceptual Motor)				

Father's name (last)_____ (first)_____

Mother's name (maiden)_____ (first)_____

Appendix A (cont)

Date information recorded _____

Child's Name (last)_____(first)_____

I.D.No._____

Current Address:_____(Street)

_____(City)

_____(Province)

Telephone No. _____

Additional child information (e.g. foster parent address):

_____

_____

SUMMARY - BIRTH AND NEONATAL HISTORY

Father's age at child's birth_____(years)

Mother's age at child's birth_____(years)

Sibling Rank _____

Prenatal

Problems during pregnancy? Explain.

Bleeding_____

Sickness (what kind)_____

_____

Drugs or Alcohol used (specify)_____

_____

Other (specify)_____

_____

Appendix A (cont)

Date information recorded _____

Child's Name (Last) _____ (First) _____

I.D.No. _____

Labour-Delivery

Problems during birth? (Explain)

Labour - Hours _____

Medications Used (specify) _____

_____

Caesarean (specify reason for it - repeat, fetal distress ect:) _____

Induced (specify reason) _____

_____

Forceps used _____

Other problems (specify) _____

_____

Gestational Age (in weeks) _____

Birth Weight (in grams) _____

Post-natal neonatal Problems?

Problems (specify) _____

_____

Transient neurological signs (i.e. Abnormal EEG one time, Hypotonicity which was maintained) (specify) _____

_____

Appendix A (cont)

SUMMARY - MEDICAL HISTORY

Date infomation recorded_____

Child's Name (Last)_____(First)_____

I.D.No._____

Child's physician - Name, Address, and Specialty_____

_____

MAJOR MEDICAL HISTORY

Major Illnesses (Specify)_____

_____

Frequent Illnesses (Specify - i.e. ear infections)_____

_____

Surgery_____

_____

Special Tests (Specify - i.e.EEG,Cat Scan, etc.)_____

_____

Developmental delay - (delay area(s) & age in months when established)_____

_____

Treatment of Assessment for Rehabilitation

Reason for referral_____

_____

Referred by (specify whether parent,doctor, nursery school teacher etc.)_____

Appendix A (cont)

Date Information recorded _____

Child's Name (Last)_____ (First)_____

Therapy

Physical Therapy*_____

_____

Occupational Therapy*_____

_____

Speech Therapy*_____

_____

Psychology*_____

_____

Other*_____

_____

*Record commencement date, discharge date, treatment periods per week, missed sessions.

SUMMARY - SOCIAL HISTORY

Mother's marital status at child's birth_____

Mother's current marital status_____

Education Level Father_____

Mother_____

Occupational Status Father_____

Mother_____

Preferred Language_____

Appendix A (cont)

Date information recorded_____

Child's name (Last)_____(First)_____

I.D.No._____

Family information (including extended family)

Other delays in family (specify)_____

_____

Social problems (specify)_____

_____

Socio economic level (based on Hollingshead scale)_____

Appendix B

Initial Letter Sent to Parents

Date

Name

Address

Dear Mr. and Mrs. _____:

This letter is further to our recent telephone call to your home regarding your child _____. _____ is the individual who had called your home.

Your child _____ is one of the children selected to participate in our study of children who have had and of children who have not had a delay in motor behaviour during the first four years of life. Participation in the study will involve two (2) visits to your home with yourself and your child. Each visit will be for approximately two hours; and the person visiting you will be Mr._____. Mr._____ will measure various aspects of your child's current behaviour. This will be done by having your child participate in several standard tests and by talking with yourself.

The results obtained in these two sessions may be of benefit to you and your child as well as other children, and you will receive a complete report of your childs functioning on the tests. Your family name and first name of your child will be treated in complete confidence.

The individuals who are responsible for the study are, again, Dr. Charles Larson of the Montreal Children's Hospital and Dr. Herbert Ladd of Concordia University.

This letter will be followed by another phone call concerning your child's participation in the study and to arrange an appointment for Mr._____'s first visit.

Enclosed is the copy of a sample report similar to the one you would receive if your child participates in this project. Thank you for considering our request.

Yours very truly,

Herbert W. Ladd
Professor

Appendix C

Consent Form

I, _____ the parent of guardian of the child _____, give consent to the investigative procedure to be carried out under this research project.

I am aware that this is a study on the effects of treatment provided for children with possible delayed motor development during the first four years of age. This study will involve two (2) hour home visits with myself and the child. The visits will consist of evaluation of the physical skills and psychological behavior of my child. The assessments can include obtaining school information (e.g., class placement, retention in grade) where applicable. I understand that if the results of this project are published no parent or child will be identified by name.

Parent _____

Witness _____

Date _____

Appendix C

Consent Form

I, _____ the parent or guardian of the child _____, give consent to the investigative procedure to be carried out under this research project.

I am aware that this is a study on the effects of treatment provided for children with possible delayed motor development during the first four years of age. This study will involve two (2) hour home visits with myself and the child. The visits will consist of evaluation of the physical skills and psychological behavior of my child. The assessments can include obtaining school information (e.g., class placement, retention in grade) where applicable. I understand that if the results of this project are published no parent or child will be identified by name.

Parent _____

Witness _____

Date _____

Appendix D

Thank You Letter to the Parents

Date

Name

Address

Dear Mr. and Mrs. _____:

I wish to take this opportunity to thank you and your child for having participated in our study which is concerned with developmental processes of young children. I can assure you that your time and effort will have contributed importantly to the study. I would point out again that the data of the study will be used to help ourselves in providing improved health care to children.

We would inform you, additionally, that we have been gathering data on over 100 children. It is taking considerable time for us to collect the data from all of these children, and then to evaluate the data and present it in written form. We would expect, thus, that we will have a written report concerned with your participation to you in the late summer. We would make ourselves available for discussion with yourselves as well after you have received your report.

Sincerely,

Herbert W. Ladd
Professor

Appendix E

Sample Child Report

Address

Name:

Date of Birth:

Age at Date of Testing:

Dates Tested:

Tests

Wechsler Intelligence Scale for Children - Revised (WISC-R)
Bruinicks-Oseretsky Test of Motor Proficiency
The Pictorial Scale of Perceived Competence and Social Acceptance for Young Children (Harter)
Stanford Diagnostic Reading Test
The Achenbach Child Behaviour Checklist (Achenbach)

Purpose of Evaluation

_____ was assessed as part of a study conducted by the Montreal Children's Hospital, Constance-Lethbridge Rehabilitation Center and Concordia University evaluating child development and the effects of treatment provided for children with possible delayed motor function in the first four years of life.

Summary

_____ is functioning overall in the borderline range of intelligence. His performance skills were much higher than his language skills (he achieved average scores on several performance subtests). _____ does not seem to be aware of what is appropriate behaviour in social and relationship situations. He needs help with what he should do in many situations which require common sense, judgement and reasoning. _____ appears to be seen by his parents as a very difficult child with many behavioural problems. It is suggested that the family might be helped or that _____ might be helped if they were to see a psychologist or a psychiatrist relating to these problems.

_____ demonstrated motor difficulties particularly in the gross motor area. He has very good perceptual and spatial capabilities.

Appendix E (cont)

Detailed information to the specific tests is contained in the following pages.

We would like to take this opportunity again to thank _____ and his parents for their participation in a time consuming evaluation for our study. If you have any questions do not hesitate to call:

Dr. Ladd, Ph.D. 482-0320, Ext. 254, Concordia University
Mrs. L. Haegert, M.A. 487-1770, Ext. 302, Constance-Lethbrige Rehabilitation Centre

History

_____ is the second child in his family. His mother had no complications during the pregnancy or birth. He weighed 3500 grams at birth.

The baby was slightly jaundiced at birth and was a sickly baby with stuffy breathing. He had many infections as a young child, severe pneumonia in February, 1978 and otitis media several times during 1978.

_____ was not walking at 22 months of age and was referred to the pediatric services of the Constance-Lethbridge Rehabilitation Centre at 26 months of age. At the time of assessment he was found to have a severe gross motor delay and a moderate fine motor, perceptual motor and language delay. He was assessed psychologically and found to be in the borderline range of intelligence. When he was discharged at four years, nine months of age he had no motor delay and a mild receptive language delay.

_____ is now attending a special learning disability class in his school. He has had difficulty at school. He took two years in kindergarten and is now taking a split grade 1/2 school programme.

This information was obtained from _____'s medical chart and from his parents' report at the time of evaluation.

Test Results and Impressions

_____ continues to function in the borderline range of intelligence according to his performance on the WISC-R. His performance subscale score is much higher (near the low average range) than his verbal subscale score (in the mentally retarded range). He achieved several average scores on the performance subscale demonstrating that he has good perceptual spatial awareness and pays good attention to detail. He had great difficulty with two subtests which evaluate (one verbally and one non-verbally) his understanding of the world around him. He seems not to make good use of common sense,

Appendix E (cont)

judgement, and reasoning. To help a child who has difficulties in these areas his parents or teacher should consider the assignment of minor responsibilities to the child in order to provide experiences for him to proceed on his own. Emphasis may be placed on "What to do if...?" situations and many "why?" type question will have to be asked. The teacher or parents should be prepared to guide the child in cause and effect relations. Inferential thinking on the part of the child should be encouraged, for example questions such as, "It's snowing outside and a fire is roaring in the fireplace..... what time of year is it?" should be given to him. _____ should be encouraged to participate in peer and group experiences with children of his own age or younger which will allow him to gain some level of independence.

_____ is just beginning to read and he knows his letters and some sounds according to his performance on the Stanford Reading Test. He is delayed in his motor proficiency according to the Bruinicks-Oseretsky test. He has greater difficulty with running speed and balance (gross motor skills) than with upper-limb co-ordination and bilateral co-ordination (fine motor skills). This is similar to his initial assessment at 26 months of age.

_____ appeared to tire before the end of the test on the Pictorial Scale of Perceived Competence and Social Acceptance. Early on in the test it appeared that he did not feel competent cognitively and did not feel accepted by his peers. Later on in the test he began pointing to the left hand picture on each page no matter what the question was.

_____ has many behavioural difficulties according to his parents' responses on the Achenbach behavioural checklist. He is high on seven out of the nine scales. These include the depression scale and several scales which include questions on aggressive, cruel and hyperactive behaviour. It is suggested that the family might consider help from a psychologist or psychiatrist in relation to _____'s behaviour as he is seen by his parents as a very difficult child.

Appendix F

Analysis of Variance Summary Table

Comparisons Among the Treatment Groups (Delayed Infrequent Home Programme Therapy, Delayed Intensive In-Centre Therapy and Non-delayed No Therapy) on Control Measures (Age at Follow-up and Socio-economic Level)

Variables	df	MS	F	p
Anova F's				
Age at Follow-up	2	36.76	.59	.55
Error	69	69.45		
Socio-Economic Level	2	1.26	.43	.65
Error	69	2.90		

Appendix F (cont)

Comparisons Among the Treatment Groups (Delayed Infrequent Home Programme Therapy, Delayed Intensive In-centre Therapy) On Control Measures (Average Percentage Motor Delay at Initial Assessment and Duration of Therapy)

Variables	df	MS	f	p
Anova F's				
Average Percentage Motor Delay	1	352.08	2.06	.16
Error	46	170.5		
Duration of Therapy	1	5.33	.062	.81
	46	86.24		

Appendix G

Analysis of Variance Summary Table

Comparisons Between Age Groups (Treated Between Birth and Two Years and Between Two and Four Years) on Control Measures (Average Percentage Motor Delay at Initial Assessment, Age at Follow-up, Treatment Received and Treatment Duration)

Variables	df	MS	F	p
Anova F's				
Average Percentage Motor Delay at Initial Assessment	1	38.78	.219	.64
Error	46	177.39		
Therapy Received	1	.003	.011	.92
Error	46	.304		
Age at Follow-up	1	8.64	.14	.71
Error	46	60.69		
Duration of Therapy	1	5.45	.063	.803
Error	46	86.22		
Socio-economic Level	1	.006	.002	.96
Error	46	3.36		

Appendix H

Discriminant Analysis Summary Table

Age at Initial Assessment, Gender, Average Percentage Motor Delay Handedness, Mother's Age at Child's Birth, Number of Prenatal, Perinatal, and Postnatal Problems, Gestational age, Birthweight, Seizures, Large and Small Head Circumference, Other Delays, Number of Social Problem and Hyperactivity

by Intellectual Level at Follow-up

Variables Entered	Wilks Lambda[a]	p[b]	B[c]
Average Percentage Motor Delay at Initial Assessment	.75	.001*	.76
Seizures	.66	.001*	-.73
Gender	.61	.001*	.77
Socioeconomic Level	.54	.001*	-.37
Number of Social Problems	.51	.001*	.65
Gestational Age(weeks)	.48	.001*	.54
Other Delays in Family	.45	.001*	-.41
Small Head Circumference	.41	.001*	-.70
Age at Initial Assessment	.36	.001*	.29
Percentile Hyperactivity	.35	.001*	.28
Birthweight (grams)	.34	.001*	-.23

*$p < .001$

Appendix H (cont)

Note: The Eigenvalue and its accompanying canonical correlation are 1.92 and .81 respectively.

[a]A measure of group homogeneity.

[b]Only those variables that significantly increase the distance between groups centroids were entered.

[c]Standardized linear weights that optiminze the distance between the group centroids (for the group with borderline-mentally retarded intelligence scores the centroid was 2.56 and for the group with low average-average intelligence scores the centroid was -.72. ($F = 6.65$, 12/38 df, $p < .001$)