SEATED POSTURAL CONTROL IN TYPICALLY DEVELOPING CHILDREN AND IN CHILDREN WITH TRAUMATIC BRAIN INJURY

by

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B.Sc., The University of British Columbia, 1988

A THESIS SUBMITTED IN PARTIAL FULFILLMENT OF THE

REQUIREMENTS FOR THE DEGREE OF

MASTER OF SCIENCE

in

THE FACULTY OF GRADUATE STUDIES

(School of Rehabilitation Sciences)

We accept this thesis as conforming to the required standard

THE UNIVERSITY OF BRITISH COLUMBIA

July 2002

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ABSTRACT

Purpose: Seated postural control is important for children with and without disabilities and is a requirement for many occupational, functional, and recreational activities. Traumatic brain injury (TBI) is the most frequent diagnosis of all traumatic injuries reported in children and often results in multiple limitations in function, however, there have been no studies on the seated postural control in these children. The objectives of this study were to evaluate the (1) seated postural control of typically developing children, including the test-retest reliability of these measures, and (2) seated postural control of children with severe TBI during re-acquisition of independent sitting.

Methods: Ten typically developing children were assessed on two separate occasions and two children with TBI (6 and 15 year old males) were assessed longitudinally. For all tests, children sat on a force plate on top of a raised bench which could be translated forward or backward. Surface EMG electrodes recorded bilateral trunk and leg muscle activity. The static, volitional, and reactive postural control of the children was assessed during quiet sitting, self-paced maximal leans, and platform translations, respectively. Intraclass correlation coefficients were used to determine the test-retest reliability of the postural control in typically developing children. Correlations were calculated to determine the effects of age on the postural control of typically developing children. Analysis of the postural control data in the children with TBI was descriptive.

Results: There was moderate to high test-retest reliability for all measures of postural control in the typically developing children. A statistically significant correlation was found between age and the static postural measure in the typically developing children. Initially, the postural control of the children with TBI differed considerably from that of the typically

developing children. Over time, the postural control of the children with TBI improved but still differed from that of the typically developing children.

Conclusions: Measures of seated postural control of typically developing children were reliable. The effects of age on these measures are dependent on the type of postural control. The recovery of seated postural control in children with TBI occurs in all three types of postural control concurrently.

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Acknowledgements

There are numerous people to whom I would like to express my sincere gratitude for their contributions to this thesis. To:

Dr. Janice Eng, my thesis advisor, for sharing her extensive, expert knowledge and for her unwavering support, encouragement, and enthusiasm throughout this thesis;

Dr. Susan Harris, Dr. George Hahn, and Dr. Tim Inglis, my committee members, for their invaluable advice, support, and time;

The children (and their parents), for their willingness to participate in this research;

Diane Wickenheiser, Kirsten Hall, and Bentley Leong, for their support in the recruitment of children with traumatic brain injury;

Kelly Chu and Maria Kim, for their camaraderie and assistance in data collection;

Laurent Mingo, for his technical support;

My supervisor and colleagues at work, for their enthusiasm and understanding;

The Rick Hansen Neurotrauma Initiative, for financial assistance; and last, but not least,

My wonderful family and loving husband, Fred Ott, for their unwavering support and encouragement.

CHAPTER 1: INTRODUCTION

Traumatic brain injury typically refers to brain damage caused by forces to the head and skull. Traumatic brain injury (TBI) is the most frequent diagnosis of all traumatic injuries reported in children and often results in multiple limitations in function and long-term disabilities (National Pediatric Trauma Registry, 1992). The particular area of function examined in this thesis was sitting balance. In this chapter, the incidence and motor outcomes of children with TBI are reviewed prior to a discussion about the relevance of sitting balance in these children.

TBI is differentiated from other forms of acquired brain injury that are caused by such mechanisms as vascular bleeds (e.g. cerebral vascular accident), infection (e.g. encephalitis), or anoxia (e.g. near-drowning). TBI is typically classified as mild, moderate, or severe based on the severity of the initial brain damage (Appendix A). Although several indices of severity of brain injury are used in the literature, the most commonly used classification of traumatic brain injury severity is the initial level of consciousness, measured by the Glasgow Coma Scale score (Teasdale & Jennett, 1974) (Appendix B). In this study, children with severe TBI were studied because the functional mobility of these children has been shown to be impaired.

Based on findings from the 1991 U.S. Census Bureau, Iverson (1998) estimated that 185 000 Canadians, including more than 24 000 British Columbia residents would sustain a traumatic brain injury resulting in a loss of consciousness during 1998. Epidemiology studies suggest that 10% to 15% of all traumatic brain injuries are moderate or severe. Thus, using the estimates from Iverson, between 18 500 and 27 750 Canadians, including 2400 to 3600 British Columbian residents, will sustain a moderate or severe brain injury each year.

This is consistent with estimates contained in the recent "Restoring Hope, British Columbia's Strategic Plan For Brain Injury" document (Higenbottam, 1994) of 4100 annual admissions to hospital for TBI. The Office of Injury Prevention of B.C. (1993) estimated an average of 2660 children and youth (ages 0 to 24 years) were hospitalized each year in British Columbia between 1986 and 1991 as a result of head injuries.

Several studies have evaluated the long-term outcomes of traumatic brain injury in children (Bruce, Schut, Bruno, Wood, & Sutton, 1978; Costeff, Groswasser, & Goldstein, 1990; Coster, Haley, & Baryza, 1994; Eiben et al.,1984; Emanuelson, von Wendt, Lundälv & Larsson, 1996; Jaffe, Polissar, Gay, & Liao, 1995; Klonoff, Low & Clark, 1977; O'Flaherty et al, 2000; Strauss, Shavelle, & Anderson, 1998). Only three of these papers specified the long-term mobility status of the children studied.

Costeff et al. (1990) prospectively studied 31 children for at least five years who were 3 to 15 years at the time of acquiring severe brain injury. Twenty-four of the children had major permanent disability in areas of motor, cognitive, speech, and/or behavior & social function. Four of the children were wheelchair-dependent until 3 years after injury and two children became wheelchair dependent due to late motor deterioration. In a case-control prospective study, O'Flaherty et al. (2000) assessed 51 children with mild to severe TBI three times within 2 years after injury. Subjects were 0 to fourteen years old at the time of injury. Fifty percent of the 26 children with severe TBI had gross motor impairments at both 6 months and 2 years after injury. Three of these 26 subjects (11.5%) were dependent for their mobility at 2 years. From 1987 to 1995, Strauss et al. (1998) evaluated the status of 946 children and adolescents approximately 6 months after suffering severe TBI. Subjects were 5 to 21 years at the time of injury. The authors evaluated five different aspects of mobility,

including: hand use, arm use, ability to creep and crawl, ability to roll and sit, and ability to ambulate. Thirty percent of the subjects had poor to no mobility, including inability to ambulate (with or without aide).

Therefore, as many as 30% of children and adolescents with severe TBI were dependent on wheelchairs for their mobility six months following injury and approximately 12% of children with severe TBI were wheelchair-dependent 2 to 3 years following injury. For children who are wheelchair-dependent, the ability to maintain an upright sitting posture may have a profound effect on their ability to perform many activities of daily living. Thus, detailed knowledge about the seated postural control of children with severe TBI may have considerable implications for their subsequent rehabilitation intervention and long-term functional outcomes.

Postural control is the ability to maintain the centre of mass over a base of support (Shumway-Cook & Woollacott, 1995), often referred to as "balance". Different types of postural control or balance are required in order to perform functional activities. There are several terms used in the literature to describe types of postural control. For the purposes of this study, types of postural control are: (1) static, which is used to maintain stability during "quiet" upright positions; (2) anticipatory, which is used to maintain stability in anticipation of changes in the orientation of the centre of mass caused by self-initiated movement of an extremity, (3) reactive, which is used to regain stability after it is lost due to changes in the orientation of the centre of mass caused by external forces, and (4) voluntary, which is the purposeful movement of the centre of mass required for functional tasks such as reaching.

In addition to its functional importance, seated postural control following brain injury may be predictive of future outcomes (Black et al., 2000; Feigin, Sharon, Czaczkes, & Rosin,

1996). In 134 subjects who had suffered stroke, Feigin et al. (1996) found a strong correlation ($r= 0.675$, $p< 0.0001$) between sitting balance at three weeks and walking at six months post-stroke. Black et al. (2000) studied 235 subjects with mild to severe traumatic brain injury who were 16 to 85 years old at the time of injury. Of several possible predictor factors, initial sitting balance was the second strongest predictor of the Functional Independence Measure at discharge. Further, independent sitting may have a profound effect on the long-term survival of children with severe TBI (Eyman, Grossman, Chaney & Call, 1993; Strauss et al. 1998). Over a 9-year period, Strauss et al. evaluated the risk factors for mortality in 946 children and adolescents with severe TBI. Compared to those with fair or good mobility, the mortality rate increased by 3.73 times in the subjects with no mobility (unable to independently roll, sit, creep, crawl, or walk), and 1.96 times in subjects with poor mobility. As well, in an 11-year follow-up study, Eyman et al. found that long-term survival of people with severe physical and mental impairment was strongly correlated to their ability to roll and sit independently.

Although a number of studies have evaluated seated postural control in typically developing children (Butterworth & Hicks, 1977; Hadders-Algra, Brogren, & Forssberg, 1996; Hirschfeld & Forssberg, 1994; Reid, Sochaniwskyj, & Milner, 1991; Woollacott et al., 1987), and in children with cerebral palsy, (Brogren, Forssberg, & Hadders-Algra, 2001; Brogren, Hadders-Algra, & Forssberg, 1998; Fife et al, 1991; McClenaghan, 1989; Yang et al., 1996), only one study evaluated the seated postural control of children with mild to moderate TBI (Reid, Sochaniwskyj, & Milner, 1991). No comparable studies have been found on children with severe traumatic brain injury. Therefore, the objectives of this study were: (1) to evaluate the seated postural control of children with severe TBI during re-

acquisition of independent sitting and, (2) to determine how the seated postural control of children with severe TBI differs from that of typically developing children.

The following questions were addressed in this study:

1. What is the static, reactive, and voluntary seated postural control in typically developing children?
2. Is the seated postural control studied in typically developing children consistent when evaluated at two separate times?
3. On the same tasks, what is the static, reactive, and voluntary seated postural control in children with severe TBI?
4. How does the static, reactive, and voluntary seated postural control change during re-acquisition of independent sitting?
5. How does the seated postural control in typically developing children compare with that of children with TBI?

As there are no studies of the seated postural control in children with severe TBI, this study may have clinical implications for both the assessment and treatment of postural control of children with TBI.

CHAPTER 2: SEATED POSTURAL CONTROL IN TYPICALLY DEVELOPING CHILDREN

2.1 Introduction

Postural control refers to the ability to maintain the centre of mass over a base of support (Shumway-Cook & Woollacott, 1995). The majority of postural control studies have evaluated the postural responses in standing non-disabled children and adults. However, seated postural control is important for many activities of daily living for children with and without disabilities. Children need to have good postural control in sitting in order to participate in many activities in school, to travel in vehicles, and to engage in recreational activities that require precise hand use.

Although seated postural control is important for occupational, functional, and recreational activities, there are relatively few studies on seated postural control in typically developing children. While numerous studies have evaluated postural control during quiet standing, only one study evaluated postural control during quiet sitting in children (Reid, Sochaniwskyj, & Milner, 1991). A number of studies have evaluated the limits of stability in sitting during leaning or reaching activities in non-disabled adults. However, there are no studies on the limits of stability in sitting in typically developing children. The majority of the studies on seated postural control in typically developing children have evaluated postural control responses following perturbations (Butterworth & Hicks, 1977; Hadders-Algra, Brogren, & Forssberg, 1996; Hirschfeld & Forssberg, 1994; Woollacott, Debû, & Mowatt, 1987).

In the quiet sitting study, children sat on a bench with full foot support (Reid et al., 1991). Although not specified by the authors, from the photographic illustration of

the protocol, it appears that the children also had full thigh support and rested their hands on their thighs. In the four studies of seated postural control responses to perturbation, typically developing children sat either in long-legged or 'ring-sitting' positions (Butterworth & Hicks, 1977; Hadders-Algra et al., 1996; Hirschfeld & Forssberg, 1994; Woollacott et al., 1987). The current study evaluated the seated postural control of children with feet dangling and the distal one-third of the thigh unsupported. Thus, the sitting position used in the current study was more challenging for postural control than the position used in the five other seated postural control studies of typically developing children.

Very few studies have evaluated the reliability of postural control measures. As yet, there are no reliability studies of seated postural control in typically developing children. Two studies examined the test-retest reliability of COP values during quiet standing in adults (Brouwer, Culham, Liston, & Grant, 1998; Geurts, Nienhuis, & Mulder, 1993). Three studies evaluated the reliability of postural control measures during maximal leans. One of these studies evaluated the test-retest reliability of maximal leans in standing young adults (Brouwer et al., 1998). Fisher and Bundy (1982) examined interrater and intrarater reliability during a study of maximal leans in standing children. In a recent study, Kerr & Eng (in press) evaluated the test-retest reliability of seated postural control in elderly adults. To date, no studies have evaluated the reliability of muscle responses following perturbations in either standing or sitting individuals.

There is evidence of an effect of age on some types of postural control in typically developing children. A number of researchers found an effect of age on postural control during quiet standing (Foudriat, DiFabio, & Anderson, 1993; Kirshenbaum, Riach, &

Starkes, 2001; Odenrick & Sandstedt, 1984; Riach & Hayes, 1987; Shimizu, Asai, Takata, & Watanabe, 1994). Researchers have also reported an effect of age on muscles responses following perturbations in seated children (Hadders-Algra, Brogren, & Forssberg, 1996). Fisher and Bundy (1982) found no effect of age on lateral trunk angle while children stood on a flat surface and leaned sideways. However, the effects of age on quiet sitting or on the limits of sitting stability have not been assessed previously.

2.1.1 Research questions

Based on a review of the literature, there is a need for further study of the seated postural control in typically developing children. Additionally, test-retest reliability of seated postural control in typically developing children needs further examination. Finally, although the effect of age on standing postural control has been studied by a number of researchers, there is less evidence of the effect of age on the sitting postural control of typically developing children.

Therefore, the following questions were addressed in this study:

1. What is the static, reactive, and volitional seated postural control in typically developing children?
2. Is the seated postural control in typically developing children consistent when evaluated on two separate occasions?
3. What is the effect of age on the seated postural control of typically developing children?

2.2 Methods

This study employed a prospective, test-retest design to assess the seated postural control of ten children with the same measures on two different days. Time between assessments ranged from 2 to 7 days.

2.2.1 Participants

Ten typically developing children, aged 6 to 15 years, were recruited from a sample of convenience. This age range was chosen in order to provide age-matched controls for the children with TBI who were recruited concurrently. Prior to the first evaluation, parental report insured that the children were: 1) attending age-appropriate academic and physical education programs and, 2) free of neurological or orthopedic conditions which would affect seated postural control. Written consent was obtained from parents and children at the first evaluation. Each child received an honorarium of $50 for participating in the study.

2.2.2 Instrumentation and Procedure

Testing occurred at the Rehab Research Lab at the G F Strong Rehabilitation Centre in Vancouver, B.C. between August and November 2000. Prior to testing, University of British Columbia and hospital ethics approval was received. For all postural control tests, children sat unsupported on a force plate on top of a raised bench (Appendix C). The bench was on top of a platform which could be moved forward and backward. Children sat on top of the force plate/bench so that their feet dangled freely. Their thighs were positioned with the distal one-third off the force plate surface. Unless

otherwise instructed, children were requested to sit in their normal, comfortable sitting posture with their arms crossed across the waist. No other attempts were made to standardize the sitting position. The children typically sat in a slouched, kyphotic posture with the pelvis posteriorly tilted and thighs slightly abducted.

In order to compare the seated postural control of typically developing children with that of children with TBI (see Chapter 3), the same testing procedure and sequence was used. Because it was anticipated that the children with TBI might fatigue during the testing procedure, the number of trials for each postural control task was chosen in order for adequate sampling without undue fatigue. The following testing procedure and sequence were used for children at both testing times: 1) four 30-second quiet sitting trials, alternating eyes open and eyes closed; 2) five repeated, self-paced maximal lean trials in each direction: forward, backward, right, then left; and 3) five repeated platform translations: forward then backward. For lean trials, children were instructed to sit in an upright posture and lean as far out as possible. During the leans, children were not allowed to prop with forearms against thighs or hook legs on the bench for stability. No other restrictions were made on the way the maximal leans were achieved. One practice lean in each direction was allowed and additional trials were conducted if the child did not perform a lean according to instructions.

The children sat on a 45 cm x 50 cm six-component Bertec force plate which has a maximum measurement error of 3% of body weight. Force plate data were collected at 600 Hz and filtered (second order, 50 Hz low pass filter) to measure the centre of pressure (COP) in anterior-posterior and medial-lateral directions.

An Optotrak (Northern Digital) imaging system was used to locate the vertical position of an infrared emitting diode (IRED) placed on a head band just above ear-level. The vertical position of the IRED at the beginning of each data collection session represented the sitting height of each subject. Optotrak data were collected at a sampling rate of 60 Hz. The imaging system has a measurement accuracy of ± 0.1 mm.

A 16 channel Bortec electromyographic (EMG) system was used to measure the activity in six thigh and trunk muscle groups. Surface EMG electrodes recorded bilateral muscle activity of: rectus femoris (RF), abdominals (ABD), sternocleidomastoid (SCM), hamstrings (HAMS), erector spinae at the 1st lumbar spine level (L1 ES) and erector spinae at the 4th cervical spine level (C4 ES). Electrode placement was standardized as described in Appendix D. Skin was prepped with alcohol swabs to reduce skin impedance and improve the quality of the EMG recordings. EMG data were collected at a sampling rate of 600 Hz and synchronized with the force plate and Optotrak data collection.

A custom computer program provided anterior and posterior platform translations of an acceleration of 300 cm/sec^2 to reach a velocity of 15 cm/sec for a distance of 8 cm. Each perturbation was separated by approximately 5 seconds to allow the child to return to a steady sitting position prior to subsequent perturbations.

2.2.3 Data Analysis

Postural control in quiet sitting was measured by the root mean square (RMS) of the COP displacement and velocity in the anterior-posterior and medial-lateral directions during four 30-second trials, alternating eyes opened with eyes closed conditions. In

studies of quiet standing, researchers have found that the RMS of the COP displacement and velocity had better test-retest reliability than several other commonly used measures of COP (Geurts, Nienhuis, & Mulder, 1993). Other researchers found that trial durations of 30 seconds or more yielded more reliable COP outcomes than trial durations of 15 seconds (Carpenter, Frank, Winter, & Peysar, 2001). Thirty-second trials were chosen in order to avoid problems of inattention or restlessness which may have occurred with longer duration trials. Paired t-tests were used to determine whether there was a difference in COP parameters in eyes open versus eyes closed conditions.

Postural control during self-paced leans was measured by the maximal displacement of the COP in the forward, backward, left, and right directions. The maximal displacements of the COP reflect the limits that the centre of mass (COM) can be moved over the base of support before losing balance, or 'the limits of stability'. Using custom Matlab programs, the baseline and maximal COP values were determined and used to derive the COP displacement for each trial. Means and standard deviations were calculated from 5 trials for each lean direction.

To remove the effect of sitting height on COP parameters for quiet sitting and lean tasks, data were normalized. To normalize for sitting height, individual trial values for each child were divided by the corresponding child's sitting height prior to calculating the mean and standard deviation of 5 trials. The resultant normalized COP displacement values are unitless because displacement (cm) was divided by sitting height (cm). Similarly, the resultant normalized COP velocity values are expressed in seconds^{-1}.

Postural control during platform translations was measured by the onset of bilateral muscle responses of the six representative trunk and thigh muscle groups. Raw

EMG data were filtered at 100 Hz and full-wave rectified. Using custom Matlab programs, the onset of muscle responses was determined by the difference between the start of the perturbation and the start of a muscle burst. The start of the perturbation was determined by visual inspection of the force plate recordings and defined as the first change from baseline of the anterior-posterior force. A muscle burst was determined by visual inspection of the EMG recordings and defined as a sharp increase of at least twice the resting muscle activity. Muscle bursts that occurred before 60 msec were removed from further analysis to exclude short-latency stretch reflex responses. In addition, muscle bursts that occurred after 300 msec were removed to exclude later voluntary responses. When bursts were present in at least 3 out of 5 trials, the means and standard deviations of the onset times were calculated and used for data analysis.

To determine the test-retest reliability of the measures between the two testing times, intraclass correlation coefficients (ICCs) were calculated. Correlations were classified using the descriptors of Munro (1997), where 0.26-0.49=low correlation; 0.05-0.69=moderate correlation; 0.07-0.89=high correlation; and 0.09-1.00=very high correlation.

To determine the effects of age on postural control, Pearson product moment correlations or Spearman's rho correlations were calculated. The non-parametric correlation (i.e. Spearman's rho) was used when the Shapiro-Wilk test of normality was significant, indicating that the outcome variables in question were not normally distributed.

Descriptive data analysis was used to further examine the muscle responses which occurred following platform translations.

2.3 Results

The characteristics of the ten typically developing children evaluated in this study are listed in Table 1. The children ranged from 6.3 years, to 15.6 years old (mean 11.4 years; standard deviation 3.6 years). There were 4 boys and 6 girls, fairly evenly dispersed across the age range, although the three youngest children were boys. All but one child was right-hand dominant. Sitting height ranged from 51.7 cm to 78.5 cm (mean 65.2 cm; standard deviation 10.2 cm).

2.3.1 Postural control during quiet sitting

Postural control during quiet sitting was measured by the COP displacement and velocity in the anterior-posterior and medial-lateral directions. The ICCs (1,2) for the four COP parameters ranged from 0.83 (anterior-posterior velocity) to 0.97 (medial-lateral velocity), for the COP parameters (Table 2). Because the ICCs were high to very high, the data from Time 1 for all subjects were subsequently analyzed. Time 1 data were chosen to minimize possible confounding effects such as the time between trials (which varied between subjects from 2 to 7 days).

The means and standard deviations of non-normalized and normalized COP displacement and velocity values were calculated (Tables 3 and 4). The COP displacement in the anterior-posterior direction (6.19×10^{-2} cm) was approximately one-third greater than in the medial-lateral direction (4.07×10^{-2} cm). The larger anterior-posterior excursion was accompanied by a faster anterior-posterior velocity (5.47×10^{-1} cm versus 3.42×10^{-1} cm).

Table 1. Characteristics of 10 typically developing children

Subject Code	Subject Age (year, month)	Gender	Dominant Side	Sitting Height (cm)
tn03	6, 4	Male	Right	54.1
tn04	11, 8	Female	Right	60.0
tn05	9, 11	Female	Left	56.8
tn06	13, 11	Female	Right	78.5
tn07	10, 8	Female	Right	68.1
tn08	7, 11	Male	Right	57.7
tn09	15, 4	Female	Right	74.9
tn10	7, 1	Male	Right	51.7
tn11	15, 7	Male	Right	72.4
tn12	15,7	Female	Right	77.6

tn = typically developing subject

Table 2. Intraclass correlation coefficients for COP displacement & velocity in typical subjects –values not normalized to sitting height (N=10)

	Eyes Open	Eyes Closed
AP Displacement (cm)	0.87	0.93
ML Displacement (cm)	0.84	0.94
AP Velocity (cm/sec)	0.84	0.83
ML Velocity (cm/sec)	0.90	0.97

ICC (1,2) one-way random effect, average measure

Table 3. Means (STD) for COP displacement & velocity in typical subjects – values not normalized to sitting height (N=10)

	Eyes Open	Eyes Closed
AP Displacement (cm)	6.19×10^{-2} (2.74×10^{-2})	6.84×10^{-2} (3.36×10^{-2})
ML Displacement (cm)	4.07×10^{-2} (2.51×10^{-2})	4.47×10^{-2} (3.09×10^{-2})
AP Velocity (cm/sec)	5.47×10^{-1} (1.49×10^{-1})	5.40×10^{-1} (2.00×10^{-1})
ML Velocity (cm/sec)	3.42×10^{-1} (1.64×10^{-1})	3.57×10^{-1} (2.05×10^{-1})

Table 4. Mean (STD) for COP displacement & velocity in typical subjects – values normalized to sitting height (N=10)

	Eyes Open	Eyes Closed
AP Displacement (unitless)	1.00×10^{-3} (0.54×10^{-3})	1.10×10^{-3} (0.65×10^{-3})
ML Displacement (unitless)	0.67×10^{-3} (0.49×10^{-3})	0.73×10^{-3} (0.59×10^{-3})
AP Velocity (sec^{-1})	7.96×10^{-3} (4.06×10^{-3})	8.65×10^{-3} (4.00×10^{-3})
ML Velocity (sec^{-1})	5.56×10^{-3} (3.20×10^{-3})	5.80×10^{-3} (3.89×10^{-3})

For eyes open and eyes closed conditions, there was no significant difference between any of the COP displacement and velocity values for normalized or non-normalized data (Tables 5 and 6).

The COP displacement and velocity values were not normally distributed. Therefore, to determine the effect of age on displacement and velocity, Spearman's rho correlations were calculated for normalized and non-normalized values (Tables 7 and 8). There was a significant non-linear relationship between age and all normalized COP parameters (Table 7). However, when sitting height was included in the analysis (i.e. non-normalized values), only three of the COP parameters were significantly related to age (Table 8). Four representative scatter plots of age versus normalized COP parameters demonstrated that the COP displacement and velocity decreased with increasing age (Figure 1). Age appeared to have a stronger effect for younger children, depicted by a steeper slope below age ten and a flatter slope above age ten. In addition, the plots indicate that there was greater variability between younger children, depicted by more variable COP values in the seven year olds than in the 15 year old children.

2.3.2 Postural control during maximal leans

The limits of stability in sitting were measured by the maximal displacement of the COP during self-paced leans in the forward, backward, and sideways (non-dominant and dominant) directions. The ICCs (1, 5) ranged from 0.78 (non-dominant direction) to 0.96 (dominant direction) for maximal COP displacements (Table 9). Because the ICCs were high to very high for the four lean directions, the means and standard deviations for Time 1 were used for further data analysis. The forward COP displacement was

Table 5. Paired samples t-test for eyes open and eyes closed conditions for normalized COP values (N=10)

COP Parameter	t value[†]	p value
Anterior-posterior Displacement	-1.57	0.15
Medial-lateral Displacement	-1.07	0.32
Anterior-posterior Velocity	-0.76	0.47
Medial-lateral Velocity	1.80	0.11

[†]degrees of freedom=9

Table 6. Paired samples t-test for eyes open and eyes closed conditions for non-normalized COP values (N=10)

COP Parameter	t value[†]	p value
Anterior-posterior Displacement	-1.74	0.12
Medial-lateral Displacement	-1.18	0.27
Anterior-posterior Velocity	-0.77	0.46
Medial-lateral Velocity	-0.59	0.57

[†]degrees of freedom=9

Table 7. Spearman's rho correlation between age and COP displacement and velocity with in typical subjects (normalized to sitting height) (N=10)

	Eyes Open	Eyes Closed
AP Displacement (unitless)	-0.71*	-0.70*
ML Displacement (unitless)	-0.79**	-0.81**
AP Velocity (sec^{-1})	-0.79**	-0.77**
ML Velocity (sec^{-1})	-0.81**	-0.79**

*significant at $p<0.05$; **significant at $p<0.01$, (2-tailed)

Table 8. Spearman's rho correlation between age and COP displacement and velocity with in typical subjects (not normalized to sitting height) (N=10)

	Eyes Open	Eyes Closed
AP Displacement (cm)	-0.42	-0.25
ML Displacement (cm)	-0.58	-0.51
AP Velocity (cm/sec)	-0.44	-0.36
ML Velocity (cm/sec)	-0.57	-0.48

AP = anterior-posterior direction, ML = medial-lateral direction

Figure 1. Sample scatter plots for age versus COP displacement and velocity in typical subjects – sway values normalized to sitting height (Time 1 data)

Table 9. Mean (STD) and intraclass correlation coefficients for maximal forward, backward, dominant side, and non-dominant leans in typical children (N=10) – values non-normalized

Direction of Lean	Time 1	Time 2	ICC
Forward (cm)	8.3 (2.5)	8.5 (1.8)	.83
Backward (cm)	16.8 (4.7)	17.6 (4.4)	.94
Dominant Side (cm)	13.4 (2.2)	13.7 (2.8)	.96
Non-Dominant Side (cm)	13.1 (2.3)	14.8 (2.7)	.78

ICC = intraclass correlation coefficient: (1,5) one-way random effect, average measure

approximately one-half of the distance of the backward displacement, and two-thirds the distance of sideways leans (Table 9). Non-dominant and dominant side COP displacements were very similar to each other and approximately three-fourths of the distance of the backward displacement.

The effect of age on maximal COP displacement was calculated for the normalized Time 1 values (Table 10). There were no significant correlations between COP displacement and age for any of the lean directions.

2.3.3 Postural control during platform perturbations

Anterior and posterior perturbations were applied to the sitting platform in order to rapidly move the centre of mass (COM) of the upper body over the base of support (BOS). Anterior perturbations cause backward displacements of the COM, requiring anterior muscles to contract to regain an upright posture. Correspondingly, posterior perturbations cause the COM to move forward, requiring posterior muscles to contract to restore an upright posture. Postural control during platform perturbations was measured by the onset of bilateral muscle responses of six representative trunk and thigh muscle groups (rectus femoris, abdominals, sternocleidomastoid, hamstrings, L1 erector spinae, and C4 erector spinae). Although statistical analyses where not performed, on visual inspection there were no obvious individual or group trends in the muscle onset times over the 5 consecutive anterior or posterior perturbations. Intraclass correlation coefficients were calculated for the onset of muscle responses following anterior platform perturbation (Table 11) and posterior platform perturbation (Table 12). ICCs (1,5) were only calculated when activation of a muscle group occurred both at Time 1 and Time 2

Table 10. Pearson product moment correlations between age and maximal forward, backward, dominant side, and non-dominant side leans (normalized to sitting height) in typical children (N=10)

	Forward	Backward	Dominant	Non-Dominant
Age	0.094	0.077	-0.044	0.092

Table 11. Mean (STD) and intraclass correlation coefficients for onset of muscle activation (msec) following anterior perturbations in typical children (N=10)

Muscle Group	Time 1 (msec)		Time 2 (msec)		ICC	
	Dominant	Non-Dominant	Dominant	Non-Dominant	Dominant	Non-Dominant
Rectus Femoris	103.4 (10.3) n=10	106.9 (14.6) n=10	110.3 (11.6) n=10	112.5 (14.5) n=10	0.71 np=10	0.86 np=10
Abdominals	110.0 (23.6) n=10	105.1 (19.2) n=10	106.9 (12.0) n=10	112.3 (17.4) n=10	0.84 np=10	0.85 np=10
Sternocleido-mastoid	113.1 (29.5) n=9	106.1 (18.5) n=10	109.6 (30.6) n=9	108.4 (22.5) n=10	0.93 np=8	0.73 np=10
Hamstrings	157.2 (53.1) n=8	172.0 (34.1) n=8	162.7 (22.8) n=6	175.2 (27.0) n=7	N/A np=6	N/A np=6
L1 Erector Spinae	159.7 (61.3) n=6	140.8 (49.8) n=7	146.6 (40.9) n=5	163.6 (44.9) n=6	N/A np=5	N/A np=5
C4 Erector Spinae	133.0 (29.8) n=7	148.3 (21.6) n=4	164.6 (24.4) n=5	142.5 (27.4) n=4	N/A np=5	N/A np=3

Italic muscle groups = all anterior muscles groups; n = number of subjects with muscle group response; np = number of subjects with muscle group responses at time 1 and time 2 (number of paired muscle responses); N/A = ICC not calculated when fewer than 7 paired muscle responses.
ICC = intraclass correlation coefficient: (1,5) one-way random effect, average measure

Table 12. Mean (STD) and intraclass correlation coefficients for onset of muscle activation following posterior perturbations in typical children (N=10)

Muscle Group	Time 1 (msec)		Time 2 (msec)		ICC	
	Dominant	Non-Dominant	Dominant	Non-Dominant	Dominant	Non-Dominant
Hamstrings	120.0 (25.3) n=9	114.2 (23.1) n=9	120.9 (22.9) n=9	125.9 (25.4) n=10	0.51 np=8	0.83 np=9
L1 Erector Spinae	132.5 (19.5) n=10	138.0 (28.9) n=9	131.4 (25.2) n=9	133.9 (22.4) n=9	0.84 np=9	0.73 np=9
C4 Erector Spinae	154.5 (37.1) n=9	151.7 (33.4) n=8	168. 8 (44.4) n=7	155.4 (41.1) n=8	0.79 np=7	0.65 np=8
Rectus Femoris	153.6 (30.5) n=5	157.1 (40.2) n=7	166.3 (59.1) n=4	173.0 (76.0) n=4	N/A np=4	N/A np=3
Abdominals	148.2 (58.0) n=5	146.1 (62.4) n=5	145.5 (64.4) n=4	115.0 (8.3) n=2	N/A np=3	N/A np=2
Sternocleido-mastoid	129.2 (13.1) n=3	124.9 (11.0) n=3	158.0 (24.5) n=4	170.9 (2.9) n=2	N/A np=2	N/A np=1

Italic muscle groups = all posterior muscle groups; n = number of subjects with muscle group response; np = number of subjects with muscle group responses at time 1 and time 2 (number of paired muscle responses); N/A = ICC not calculated when fewer than 7 paired muscle responses. ICC = intraclass correlation coefficient: (1,5) one-way random effect, average measure

in at least 7 of the 10 children. In response to anterior platform perturbation, only the rectus femoris (RF), abdominals (ABD), and sternocleidomastoid (SCM) muscles were activated consistently at both Time 1 and Time 2. The ICCs were high to very high for RF, ABD, and SCM for both the dominant and non-dominant sides. In response to posterior platform perturbations, only the hamstrings (HAMS), L1 erector spinae (L1 ES), and C4 erector spinae (C4) were activated consistently at both testing times. ICCs were moderate for the dominant side HAMS and non-dominant side C4 ES (0.51 and 0.65 respectively). ICCs were high and ranged from 0.73 to 0.84 for the non-dominant side HAMS, bilateral L1 ES, and dominant side C4 ES.

Onset of muscle responses to anterior platform perturbation ranged from a mean and standard deviation of 103 (±10) msec for rectus femoris muscles to 175 (±27) msec for hamstrings muscles (Table 11). The onset of muscle responses to posterior perturbation ranged from a mean and standard deviation of 114 (±23) msec for hamstrings muscles to 173 (±76) for rectus femoris muscles (Table 12). According to the group data, there was no clear sequence of anterior muscle responses to the anterior perturbation. The mean onsets of the RF, ABD, and SCM muscles were within 10 msec and had considerable overlap, considering the standard deviations ranged from 10.26 to 30.57 msec. In contrast, the onset of posterior muscle responses to posterior perturbation followed a caudal to cephalic pattern (i.e. HAMS→L1 ES→C4 ES). Although there was a larger spread between mean onsets of the posterior muscles (i.e. 30 to 48 msec), there was still considerable overlap, with standard deviations ranging from 19.47 to 44.36 msec. Because the group variability of onset times for many muscles was quite large, a second analysis was performed to examine the sequence of muscle responses for

individual children (Tables 13 and 14). In most children, the order of muscle activation was not the same on the non-dominant and dominant sides. Therefore, the activation order was tabulated for both sides for all 10 children. There were seven different sequences of anterior muscle activation in response to anterior perturbation (Table 13). The two most common sequences of anterior muscle activation each occurred four times, (i.e. ABD→SCM→RF and RF→ABD→SCM). Thus, only four out of a possible 20 responses were in a caudal-cephalic sequence. There were nine difference sequences of posterior muscle activation in response to posterior perturbation (Table 14). The most common sequence of posterior muscle activation occurred seven times and was the same caudal-cephalic pattern as that of the group data (i.e. HAMS→L1 ES→C4 ES).

The relative onset of activation of the flexor and extensor muscle groups located at the thigh, trunk, and neck was examined (Figures 2 and 3). For comparison, the muscles have been grouped in pairs based on their location or 'segment': RF-HAMS, ABS-L1 ES, and SCM-C4 ES. The muscles were not activated in segment pairs in all children. For example, following anterior platform perturbation, ABS were activated in 10 children, but L1 ES were only activated in 6 children. When there was activation of both the flexor and extensor groups, RF were consistently and distinctly activated prior to HAMS following anterior platform perturbation. However, this flexor-extensor activation pattern was not consistently present in the ABS-L1 ES or SCM-C4 ES segment pairs following anterior perturbation. In fact, extensor-flexor or synchronous contraction patterns occurred frequently in ABS-L1 ES and SCM-C4 ES segment pairs. Following posterior platform perturbations, HAMS were consistently activated prior to RF in an extensor-flexor pattern. There was not a clear extensor-flexor pattern in the ABS-L1 ES

Table 13. Sequence of anterior muscle group activation following anterior platform perturbations (bilateral muscle responses)

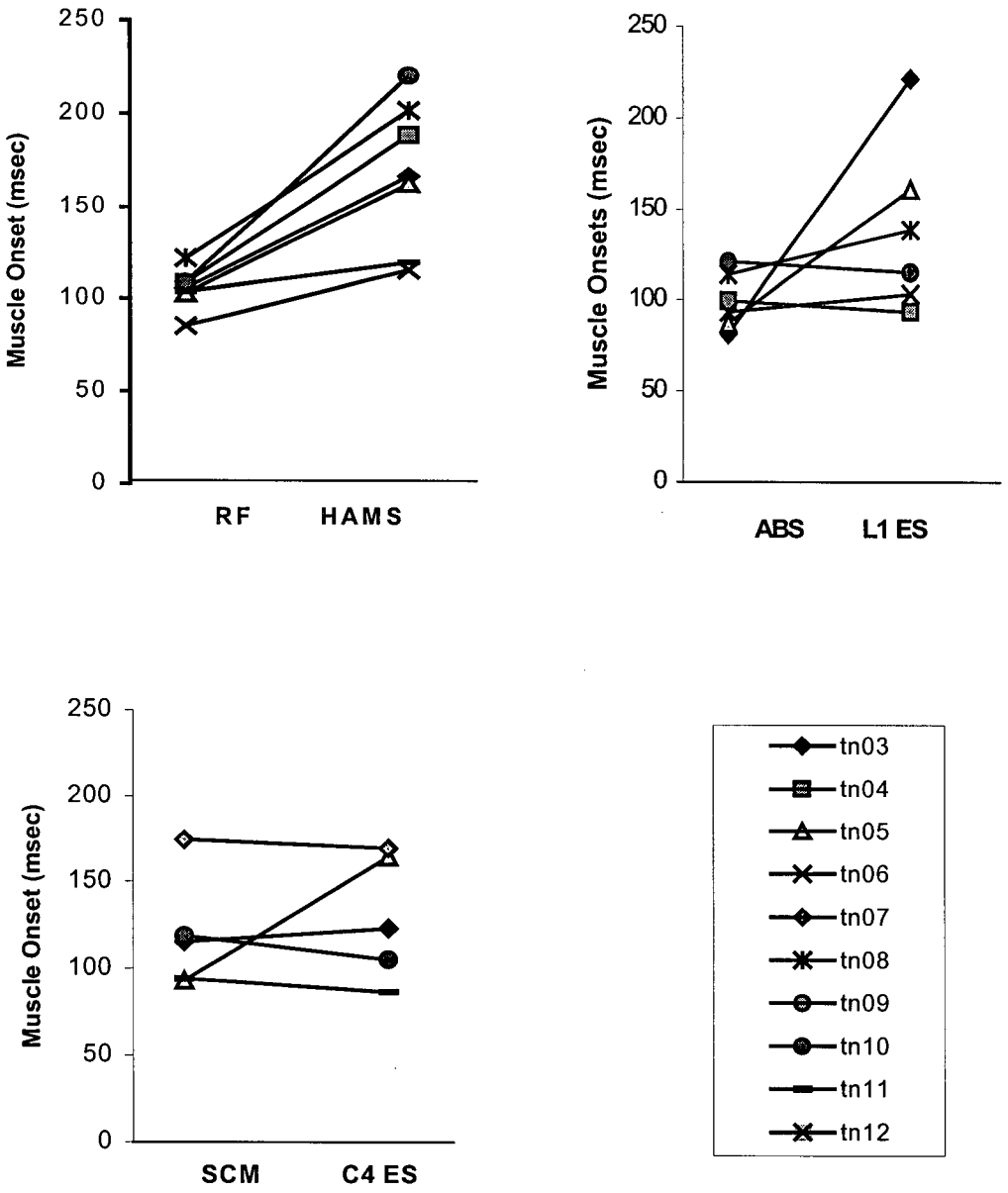
Sequence	Frequency (n=20)*
Abdominals, Sternocleidomastoid, Rectus Femoris	4
Rectus Femoris, Abdominals, Sternocleidomastoid	4
Rectus Femoris, Sternocleidomastoid, Abdominals	3
Sternocleidomastoid, Abdominals, Rectus Femoris	3
Sternocleidomastoid, Rectus Femoris, Abdominals	3
Abdominals, Rectus Femoris, Sternocleidomastoid	2
Rectus Femoris, Abdominals	1

*bilateral muscle responses for 10 typical children

Table 14. Sequence of posterior muscle group activation following posterior platform perturbations (bilateral muscle responses)

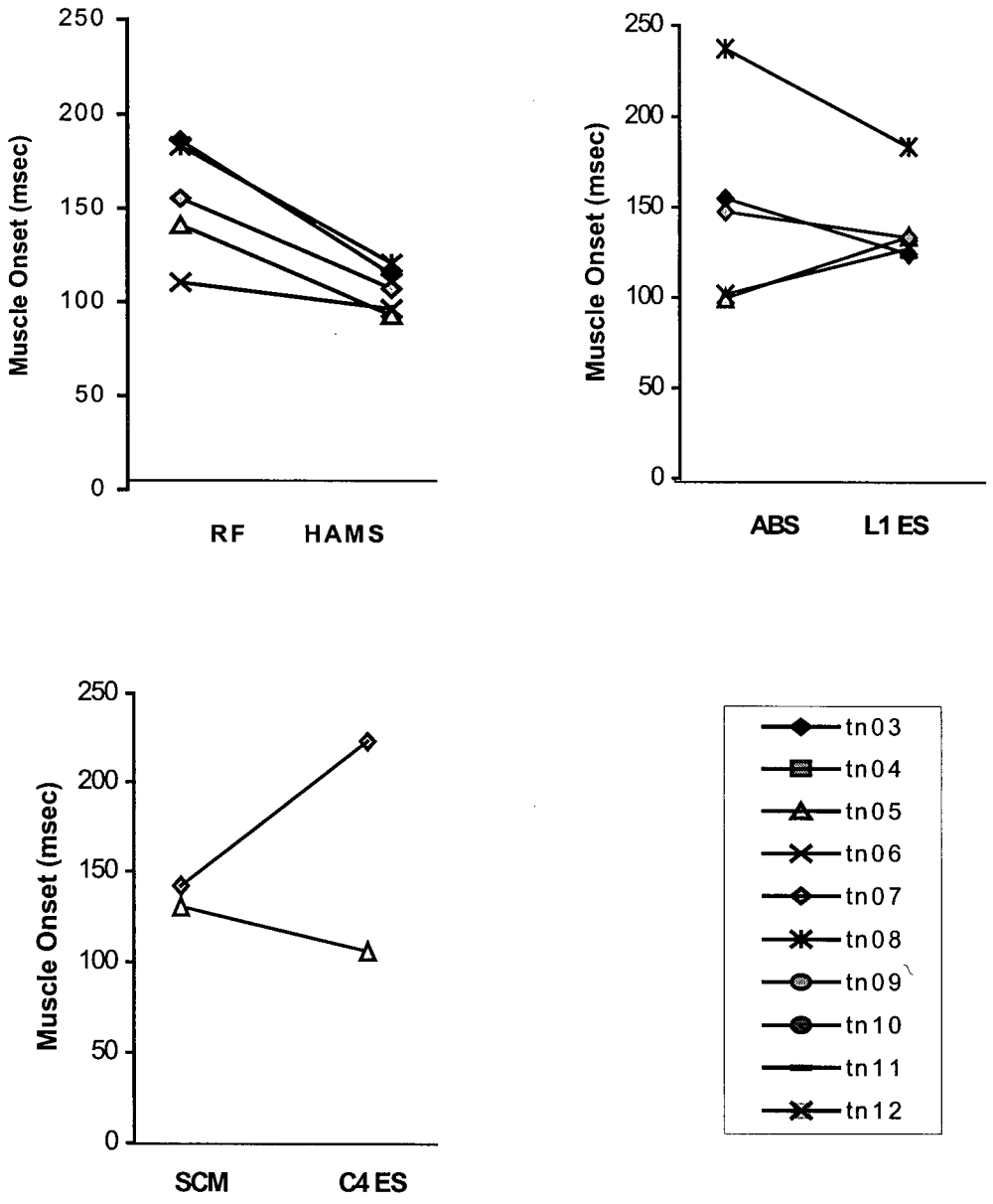
Sequence	Frequency (n=20)*
Hamstrings, L1 Erector Spinae, C4 Erector Spinae	7
Hamstrings, C4 Erector Spinae, L1 Erector Spinae	4
L1 Erector Spinae, Hamstrings, C4 Erector Spinae	2
C4 Erector Spinae, Hamstrings, L1 Erector Spinae	2
C4 Erector Spinae, L1 Erector Spinae, Hamstrings	1
Hamstrings, L1 Erector Spinae	1
L1 Erector Spinae, Hamstrings	1
Hamstrings	1
L1 Erector Spinae	1

*bilateral muscle responses for 10 typical children



RF = rectus femoris, HAMS = hamstrings, ABS = rectus abdominus, L1 ES = erector spinae at L1 level, SCM = sternocleidomastoid, C4 ES = erector spinae at C4 level

Figure 2. Time of muscle onset of flexor-extensor muscle pairs for the dominant side following anterior perturbation in typical children.



RF = rectus femoris, HAMS = hamstrings, ABS = rectus abdominus, L1 ES = erector spinae at L1 level, SCM = sternocleidomastoid, C4 ES = erector spinae at C4 level

Figure 3. Time of muscle onset of flexor-extensor muscle pairs for the dominant side following posterior perturbation in typical children.

or SCM-C4 ES segment pairs. Instead, a flexor-extensor pattern occurred approximately 50% of the time in the ABS-L1 ES and SCM-C4 ES segment pairs.

The onsets of muscle activation for the RF, ABS, and SCM following anterior perturbation were normally distributed. Similarly, activation onsets for HAMS, L1 ES, and C4 ES following posterior perturbation were normally distributed. In order to determine whether there was an effect of age on the onset times of these muscles, Pearson product moment correlations were calculated for Time 1 non-dominant side responses (Tables 15 and 16). The non-dominant side was chosen because there was more consistent activation of these muscles at Time 1 and Time 2. There was no significant effect of age on onset times for the six muscles of interest.

Due to the large variability of individual responses, the effect of age on activation sequence was visually analyzed. There was no apparent relationship between age and the sequence of muscle activation following anterior or posterior perturbations. For example, the most common posterior muscle activation sequence was found across the age span of the children studied.

Table 15. Pearson product moment correlations between age and onset of anterior muscle activation following anterior perturbation – non-dominant side muscles

Muscle group	r value	p value	N
Rectus Femoris	-0.40	0.25	10
Abdominals	0.063	0.86	10
Sternocleidomastoid	-0.41	0.24	10

N=number of muscles activated

Table 16. Pearson product moment correlations between age and onset of posterior muscle activation following posterior perturbation – non-dominant side muscles

Muscle group	r value	p value	N
Hamstrings	-0.13	0.74	9
L 1 Erector spinae	-0.33	0.39	9
C 4 Erector spinae	-0.54	0.17	8

N=number of muscles activated

2.4 Discussion

One of the purposes of this study was to describe the static, voluntary, and reactive seated postural control of typically developing children. These aspects of postural control were examined during quiet sitting, maximal lean, and platform perturbation tasks. Another purpose of this study was to determine the test-retest reliability of seated postural control in typically developing children. Test-retest reliability was particularly important to evaluate for later comparisons with the seated postural control of children with traumatic brain injury (Chapter 3). The final purpose of this study was to determine the effect of age on the seated postural control of typically developing children.

2.4.1 Seated postural control in typically developing children

The seated postural control of typically developing children is described and discussed for each type of postural control task evaluated in this study. The test-retest reliability for all postural control tasks will be discussed and compared in a separate section.

2.4.1.1 Postural control during quiet sitting

Postural control during quiet sitting was determined by the COP displacement and velocity in the anterior-posterior and medial-lateral directions, alternating eyes opened with eyes closed conditions. Both COP displacement and velocity were approximately one-third greater in the anterior-posterior direction than in the medial-lateral direction. Odenrick and Sandstedt (1984) also found anterior-posterior COP excursions were

greater than medial-lateral COP excursions during quiet standing in non-disabled children. The differences in anterior-posterior and medial-lateral displacement and velocity in sitting may be due to the musculoskeletal structure of the spine, pelvis, and hips. Recently, researchers have argued that the position of COM of the body during quiet standing is primarily controlled by 'stiffness' of the ankles and hips (Winter, Patla, Prince, Ishac, & Gielo-Perczak, 1998; Winter, Patla, Rietdyk, & Ishac, 2001). Stiffness is defined as the initial resistance to movement due to the inherent properties of the muscles and passive tissues (Rietdyk, Patla, Winter, Ishac, & Little, 1999). Thus, a greater stiffness to medial-lateral movement of the COM may be present in the musculoskeletal structures of spine, pelvis, and hips in sitting individuals. This in turn would lead to reduced displacement and velocity of the COP in the medial-lateral direction.

As there is only one study of quiet sitting in children (Reid et al., 1991), the results of this study will also be compared to the literature on quiet standing in non-disabled children. Researchers have evaluated the effects of height, gender, age, and/or vision on quiet standing in non-disabled children (Foudriat, et al., 1993; Kirshenbaum, et al., 2001; Odenrick & Sandstedt, 1984; Riach & Hayes, 1987; Shimizu, et al., 1994). Many of these studies also evaluated the effects of support surface conditions and visual conditions on postural stability during standing. For the purposes of comparison with this study, only the data of quiet standing while on stable surfaces with eyes open and closed will be considered.

In a study of quiet standing in 3-17 year old non-disabled children, Odenrick and Sandstedt (1984) found height to be positively correlated with lateral sway in boys but

not girls. The effect of sitting height on COP displacement and velocity was not specifically evaluated in this study. However, when the correlations between age and COP values are compared between normalized and non-normalized data, it is clear that height has an effect, since there is only an age effect when sitting height is normalized (Tables 7 and 8). This suggests that increases in sitting height lead to larger non-normalized COP displacement and velocity. Because older children had larger sitting heights and smaller normalized COP values, the effects of height and age would counter each other, leading to non-significant correlations of age for non-normalized COP values.

The effect of eyes open versus eyes closed on quiet standing was evaluated in three studies (Foudriat et al., 1993; Odenrick & Sandstedt, 1984; Riach & Hayes, 1987). There was no common finding across these studies. Odenrick & Sandstedt (1984) found that 68% of children aged 3-17 years had small increases in anterior-posterior and medial-lateral COP excursions with eyes closed, irrespective of age. Foudriat et al. (1993) found no differences between postural stability in eyes open or closed conditions in 3-6 year olds except in 5years olds who had decreased stability with eyes closed. In contrast, Riach & Hayes (1987) found that COP excursions were greater in eyes open conditions for children age 2-14 years. The differences in the findings of these studies could be due to varied foot positions and protocols. In the present study, there were no significant differences in any of the COP displacement or velocity values for eyes open and eyes closed conditions. This suggests that vision does not have an effect on the maintenance of quiet sitting balance. It is possible that the large base of support afforded by sitting and the stiffness of the spine, pelvis and hips made the regulation of the COM in quiet sitting vision-independent. Even in quiet standing, Winter et al. (1998) found no

significant differences between eyes open and closed conditions in the anterior-posterior and medial-lateral COP or COM excursions of non-disabled adults.

In the current study, age had a significant non-linear effect on COP displacement and velocity, with a greater effect on children younger than 10 years. Reid et al. (1991) determined the 3-dimensional movement of the C7 spine during quiet sitting in forty-six 5-15 year old non-disabled children. The authors found the movement of the C7 spine decreased significantly with age. Using the error of the sum of squares, the authors concluded that the effect of age on C7 spine movement was linear rather that non-linear. In the current study, age had a non-linear correlation with normalized COP displacement and velocity values but not with non-normalized COP values. The effects of normalizing for sitting height, the smaller sample size, and a more challenging sitting position in the current study may explain the differences in the correlations found between the current study and the Reid et al. study. The different postural control outcome measures used in the current study and in the study by Reid et al. may also have lead to the discrepancies in the type of correlations found between the two studies.

A non-linear effect of age was also found on postural control during quiet standing in non-disabled children (Foudriat et al., 1993; Kirshenbaum et al., 2001; Odenrick & Sandstedt, 1984; Riach & Hayes, 1987; Shimizu et al., 1994). Foudriat et al. found that 3-4 year olds were significantly less stable than 5-6 year olds for both eyes open and eyes closed conditions. Kirshenbaum et al. found that anterior-posterior COP velocity decreased non-linearly in children studied longitudinally from age 5 to 8 years. When studying 100 children between 5-13 years of age, Shimizu et al. found that anterior-posterior COP excursions with eyes open were significantly greater in children

less than 9 years of age compared to adults. With eyes closed, the anterior-posterior COP excursions were significantly greater in children less than 7 years of age compared to adults. Odenrick & Sandstedt (1984) were the only researchers to find a significant difference in the effect of age between boys and girls aged 3-17 years. These researchers found that anterior-posterior and medial-lateral COP excursions decreased with eyes open in boys but not in girls. With eyes closed, COP excursions decreased with age in both boys and girls.

In the current study, the variability of COP displacement and velocity appeared to be larger in younger children. Riach and Hayes (1987) also found considerable variability in the standing stability of younger children which diminished with age.

2.4.1.2 Postural control during maximal leans

The limits of sitting stability in typically developing children were measured by the maximal displacement of COP during self-paced leans in the forward, backward, and sideways (non-dominant and dominant) directions. No other studies have examined the limits of sitting stability in non-disabled children. However, Fisher and Bundy (1982) examined the maximal self-paced lateral reach in standing of 4-12 year old non-disabled children. When children reached laterally while standing on a firm surface, there was no correlation between trunk angle and age in either girls or boys. In the present study, there was no correlation between age and normalized maximal COP displacements in any direction. Although further research is necessary to verify these findings, the results of the two studies suggest that there is not an effect of age on maximal lateral leans in non-disabled children.

Two studies have examined the limits of stability during maximal forward, backward, left, and right leans in non-disabled adults (Brouwer et al.,1998; Kerr & Eng, in press). Brouwer et al. evaluated the limits of standing stability of young non-disabled adults during maximal self-paced leans. The maximal backward COP excursion was approximately one-sixth of the maximal excursions in the forward, left, and right directions [1.12 degrees, 7.46 degrees, 7.08 degrees, 7.46 degrees, respectively]. Kerr and Eng evaluated the COP displacement and velocity of non-disabled elderly adults during fast-paced maximal leans. Subjects sat unsupported on a force plate with approximately 80% thigh support and feet dangling. COP displacements in the forward, backward, dominant side, and non-dominant side directions were within 1 cm [13.20 cm, 13.28 cm, 12.59 cm, 12.31 cm, respectively].

Because Brouwer et al. (1998) expressed COP excursion in degrees, the absolute values cannot be compared with those obtained by Kerr and Eng (in press) or for this current study. However, the relative COP excursions in each study can be compared. Whereas the COP excursion was the smallest for backward leans during standing, the COP excursion was largest in the backward direction during sitting. Although the base of support is similarly limiting in the posterior direction in both sitting and standing, in this current study the children relied on their abdominal and hip flexor strength to maintain stability during backwards leans. Compared to the backward COP displacement in the Kerr and Eng study, the backward COP displacement in this study was 3.5 cm larger (13.28 cm, 16.8 cm, respectively). This discrepancy may be due to differences in the task (i.e. fast-paced versus self-paced leans) and/or differences in the abdominal and hip flexor strength in the elderly adults and children.

In each study, sideways leans had very similar COP excursions. Interestingly, the COP displacements for sideways leans were within 1 cm in the elderly adults and children [e.g. 12.59 cm, 13.4 cm, non-dominant side leans, respectively]. Thus, the combined findings from Brouwer et al. (1998), Fisher and Bundy (1982), Kerr and Eng (in press), and the current study suggest that COP displacement during sideways leans in non-disabled individuals is minimally affected by dominance and age.

It is likely that the sitting position used during this study (i.e. approximately one third of the distal thigh length was unsupported) had a substantial limiting effect on the maximal forward COP displacement. In the study by Kerr and Eng (in press) where subjects sat with 80% of the thigh supported, forward and backward COP displacements were similar. Chari and Kirby (1986) found that forward reach with both feet off the ground was significantly greater when the front edge of the seat was 7.5 cm behind the knees compared to 25 cm behind the knees [69.7 cm, 88.1 cm, respectively]. Thus, in this study if more of the thigh was supported, the maximal forward COP displacements would probably be the same or greater than the maximal backward COP displacements.

2.4.1.3 Postural control during platform perturbations

Postural control during platform perturbations was measured by the onset of bilateral muscle responses of six representative trunk and thigh muscle groups (rectus femoris, abdominals, sternocleidomastoid, hamstrings, L1 erector spinae, and C4 erector spinae). Three studies have evaluated the muscle responses to anterior and posterior perturbations in non-disabled children (Hadders-Algra et al., 1996; Hirschfeld & Forssberg, 1994; Woollacott et al., 1987). Researchers in each of the three studies were

interested in the development of postural responses in the young child. For comparison with this current study, only the data on the postural responses of independently sitting young children will be considered. Because the infants and children in the three studies were much younger than the children in the present study, two additional studies of seated postural control in children will be discussed (Brogren et al., 1998; Brogren et al., 2001). Both studies evaluated the postural responses to perturbations in older children (ranged between 3-11 years) with cerebral palsy and age-matched non-disabled controls. For comparison with the current study, only data on the seated postural responses of the non-disabled children will be considered.

In the current study, there was considerable variability between subjects in the onset times for many muscles in response to both anterior and posterior perturbations. This variability was also reported in the literature, with large differences in the muscle onsets evident between studies (Forssberg & Hirschfeld, 1994; Hirschfeld & Forssberg, 1994; Woollacott, et al., 1987). In the current study, muscle onsets following anterior perturbations ranged from 103 msec for rectus femoris to 175 msec for hamstrings. In 8 month to 3 year olds, Woollacott et al. (1987) reported muscle onsets ranging from 123 msec for cervical extensors to 158 msec for neck flexors. In 7-8 month old infants, Hirschfeld and Forssberg (1994) reported onsets ranging from 151 msec for rectus femoris to 305 msec for lumbar extensors. Although there is considerable variability in the ranges of muscle onsets reported in the literature, Hadders-Algra et al. (1996) reported no statistically significant effect of age on muscle onsets in 5-10 month old children. Similarly, in the current study, there was no correlation between age and onset of muscle activation following perturbations (Tables 15 and 16). Therefore, the

variability of muscle onsets reported in the literature may, instead, be due to the different testing protocols and data analysis techniques used by the researchers.

In the current study, responses of posterior muscles following posterior perturbation were slightly more variable than responses of anterior muscles following anterior perturbation. This is somewhat inconsistent with the literature, which regularly reported extremely variable activation of muscles following posterior perturbations (Brogren et al., 1998; Brogren et al., 2001; Forssberg & Hirschfeld, 1994; Hadders-Algra, et al., 1996; Hirschfeld & Forssberg, 1994; Woollacott, et al., 1987). This discrepancy may be explained by the differences in the sitting positions used in the literature compared with the position used in the current study.

In the current study, children sat with feet dangling and the distal one-third of the thigh unsupported. Thus, the base of support in the forward direction was considerably shorter than that of the other studies where children sat either cross-legged or long-legged. Following posterior perturbations of the same magnitudes, the movement of the centre of mass would be closer to the forward limits of the shorter base of support than longer base of support. Therefore, more muscles would be required to activate in order to stabilize the centre of mass over the shorter base of support. Only Woollacott et al. (1987) reported muscle onsets following posterior perturbation. In infants and young children, muscle onsets ranged from 105 msec for trunk extensors to 132 msec for neck extensors. In the current study, onset of muscle responses to posterior perturbation ranged from 114 msec for hamstrings to 173 for rectus femoris muscles. According to the results of Woollacott et al. (1987) and to those of the current study, the onset times of

muscle responses do not appear to have greater variability than muscle onsets following anterior perturbations.

In the current study, the sequence of muscle activation in response to both anterior and posterior perturbations was quite variable. According to the group data, there was no clear sequence of anterior muscle response to anterior perturbation. Due to very similar mean onsets and overlapping ranges, anterior muscle activation could be considered simultaneous. However, analysis of individual responses yielded 7 different sequences of anterior muscle activation. The two most common sequences of anterior muscle activation each occurred 4 times, (i.e. ABD→SCM→RF and RF→ABD→SCM). Three studies reported the activation sequence of anterior muscle responses following anterior perturbations (Forssberg & Hirschfeld, 1994; Hirschfeld & Forssberg, 1994; Woollacott, et al., 1987). Although Forssberg and Hirschfeld reported a tendency toward a caudal-cephalic order of anterior muscle responses, there were no significant differences between the muscle onsets in young adults. In their study of 7-8 month old infants, Hirschfeld and Forssberg reported simultaneous onsets of anterior muscle responses. In addition, Woollacott et al. reported simultaneous onsets of the abdominals and neck flexors in infants and young children following anterior perturbation. Thus, the findings in the literature of similar or simultaneous mean onsets in anterior muscles following anterior perturbation are consistent with the current study.

In this study, the sequence of posterior muscle activation following posterior perturbations was generally less variable than that seen in anterior muscles following anterior perturbations. A caudal-cephalic sequence was more clearly apparent in both the group and individual data (i.e. HAMS→L1 ES→C4 ES). Although no researchers

reported the sequence of posterior muscle activation following posterior perturbations, both Brogren et al. (1998) and Brogren et al. (2001) reported that the sequence of activation usually started in the hamstrings. This is consistent with the results of the current study, in which 13 out of 20 muscle responses started with the hamstrings.

The relative onsets of flexor and extensor muscles of the thigh, trunk, and neck 'segments' were evaluated in the current study. In response to anterior perturbations, rectus femoris was consistently activated before hamstrings, but a consistent flexor-extensor activation pattern was not present for the trunk and neck muscles. Other researchers found more consistent flexor-extensor muscle activation patterns following anterior perturbations (Brogren et al., 2001; Brogren et al., 1998; Forssberg & Hirschfeld, 1994; Hirschfeld & Forssberg, 1994). Forssberg and Hirschfeld found that all the extensor muscles were activated after the flexor muscles of the same segment. Hirschfeld and Forssberg reported that hamstrings onsets were not significantly later than the flexor muscles, but trunk and neck extensor muscles activated significantly later than the flexors. Similarly, Brogren et al. found that the onsets of neck and trunk extensor muscles usually followed the flexor muscles of the same segment but the onset of hamstrings was occasionally simultaneous with rectus femoris. A possible explanation for the discrepancy between the literature and this study could be differences in the magnitudes of the perturbations used. It is not possible to compare the magnitudes of perturbations used in other studies with the current study because the researchers did not provide information about acceleration. However, a perturbation of a greater magnitude could possibly cause more simultaneous activation of muscles to maintain stability of the body.

In the current study, following posterior perturbations, hamstrings were consistently activated prior to rectus femoris, but a clear extensor-flexor pattern was not present in the trunk and neck muscles. Brogren et al. (2001) reported that the activation of flexor muscles following posterior perturbations was infrequent. However, when activated, rectus femoris always followed hamstrings activation. The results are consistent with the current study which also found inconsistent activation of the trunk and neck flexors following posterior perturbation.

Researchers have suggested that differences in muscle responses following anterior perturbation versus posterior perturbation reflect differences in the limits of stability during backward and forward trunk movement (Brogren et al., 2001; Brogren et al., 1998; Forssberg & Hirschfeld, 1994; Hirschfeld & Forssberg, 1994). In quiet sitting, the COM is generally located much closer to the back edge than the front edge of the base of support. Thus, perturbations of the same magnitude but in opposite directions would move the COM nearer to the limits of stability in the backward direction than in the forward direction. In order for the COM to be moved near to the forward limits of stability, posterior perturbations would need to be of much greater magnitude. In the current study, anterior and posterior perturbations were the same magnitude. Thus, the posterior perturbations would not have moved the COM as close to the limits of stability as the anterior perturbations. This may explain why fewer posterior muscles were activated following posterior perturbations.

An additional explanation for the differences in muscle responses to forward and backward perturbations may be related to the concept of muscle and joint 'stiffness'. Recently Rietdyk, Patla, Winter, Ishac and Little (1999) studied the joint moments

(rotational forces) that occurred following medial-lateral perturbations of non-disabled standing adults. They found that the start of joint moments in the hip and spine was synchronous with the change in joint angle. Because the joint moments occurred between 56-116 msec, these researchers suggested that muscle stiffness and not muscle contraction contributed to the initial stabilization following perturbation. In sitting, it is possible that the posterior musculo-tendinous structures of the spine and thighs may provide more stiffness than the anterior structures. This would be particularly true in long-legged sitting positions (used by some researchers), when the hamstrings would likely provide substantial resistance to forward movement of the pelvis. Thus, it is possible that greater posterior stiffness acts to stabilize the forward movement of the body so that fewer muscle responses are required.

2.4.2 Test-retest reliability of seated postural control

The quiet sitting study of non-disabled children by Reid et al. (1991) did not evaluate the reliability of their postural control outcome measure. In addition, the studies reviewed of quiet standing in non-disabled children did not report the test-retest reliability of their measures. However, Brouwer et al. (1998) examined the test-retest reliability of COP values during quiet standing in young adults on three separate occasions. Although mean COP values were not significantly different on each occasion, the ICCs (2,1) were low for eyes open and eyes closed conditions (0.45 and 0.38, respectively). In this study, ICCs (1,2) of the root mean square (RMS) of the COP displacement and velocity were high to very high (range of 0.83 to 0.97). Discrepancies in the ICCs between the two studies may be due to the different testing positions used.

Better test-retest reliability of COP values in the sitting position may be due to greater stability afforded by a larger base of support, fewer joints requiring stabilization, and a closer proximity of the COM to the base of support. The results of these studies suggest that quiet sitting may be a more reliable measure of postural control than quiet standing. This is an important consideration, particularly when making comparisons of the postural control of children with and without disabilities.

Three of the studies reviewed evaluated the reliability of their measures of maximal leans (Brouwer et al., 1998; Fisher & Bundy, 1982; Kerr & Eng, in press). Brouwer et al. (1998) evaluated the ICCs of forward, backward, and sideways leans in standing young adults who were tested on three occasions one week apart. The authors reported ICCs (2,1) that ranged from 0.88 to 0.93 for the maximal leans. Fisher and Bundy (1982) used Pearson correlation coefficients to assess the interrater and intrarater reliability of the investigators. These researchers reported reliabilities that exceeded r = .98 for trunk angle scores during maximal lateral reaches in 4-12 year old standing children. Kerr and Eng evaluated the ICCs and the standard error of measurement (SEM) of forward, backward, and sideways COP displacements in sitting elderly adults who were tested on two occasions 2-4 days apart. The ICCs ranged from 0.74 to 0.94 and SEMs ranged from 0.83 cm to 1.12 cm for fast-paced maximal leans without foot support. In this current study, the ICCs (1,5) for COP displacements during maximal self-paced leans ranged from 0.78 to 0.94. The combined results of these studies suggest that maximal leans are very reliable measures of the limits of stability in non-disabled children and adults. Therefore, maximal leans may be appropriate measures for making comparisons of the seated postural control of children with and without disabilities.

To date, no studies have reported reliability values of muscle responses to perturbations. In this study, ICCs (1,5) were calculated when muscle responses were present at both Time 1 and Time 2 in at least seven out of ten children. Anterior muscles were activated consistently at Time 1 and Time 2, with high to very high ICCs. Posterior muscles were activated slightly less consistently at Time 1 and Time 2, with moderate to high ICCs. Thus, it appears that, using the perturbation magnitude and sitting position of the current study, the onset of muscle responses following anterior and posterior perturbations is a reliable measure of the seated postural control in children.

2.4.3 Limitations

A larger sample size would improve the power to detect differences in the postural control of children of varying ages. There is some evidence that gender may have an effect on static postural control (Odenrick and Sandstedt, 1984) and volitional postural control (Fisher & Bundy, 1982) in typically developing children. Therefore, an even distribution of males and females at each age would help to clarify the gender effect on seated postural control in typically developing children.

Reactive postural control was only measured by muscle onsets following perturbations. The effectiveness of muscle responses used to stabilize the upper body after perturbation may have been elucidated by the evaluation of the magnitude of muscle responses and centre of pressure excursions. Further, comparisons of the muscle response effectiveness in typically developing children with that of children with TBI (Chapter 3) may have yielded important clinical information.

This study did not evaluate the anticipatory postural control of typically developing children. Anticipatory postural control may be an important aspect of seated postural control, particularly while lifting and moving objects.

Although the current study evaluated test-retest reliability, other forms of reliability were not established. As well, the use of more clinically accessible measures of seated postural control in combination with the measures used in the current study may have led to greater clinical utility of this research.

2.4.4 Future directions

There is a need for further research on the seated postural control of typically developing children, particularly of the static, anticipatory, and volitional types of postural control. Additional information is needed about the test-retest, interrater, and intrarater reliability of postural control evaluation in typically developing children. More studies are also needed to examine the effects of different sitting positions on postural control in seated children. In particular, in order to determine the seated postural control of children in every day activities, studies should examine common sitting positions children obtain when sitting in a standard chair.

CHAPTER 3: SEATED POSTURAL CONTROL IN CHILDREN WITH TRAUMATIC BRAIN INJURY

3.1 Introduction

Traumatic brain injury typically refers to brain damage caused by forces to the head and skull. Traumatic brain injury (TBI) is the most frequent diagnosis of all traumatic injuries reported in children and often results in multiple limitations in function and long-term disabilities (National Pediatric Trauma Registry, 1992). TBI is typically classified as mild, moderate or severe based on the severity of the initial brain damage (Appendix A). Although several indices of severity of brain injury are used in the literature, the most commonly used classification of traumatic brain injury severity is the initial level of consciousness, measured by the Glasgow Coma Scale score (Teasdale & Jennett, 1974). The Glasgow Coma Scale (GCS) is used to determine the level of consciousness following injury by rating the motor, verbal, and eye-opening responses (Appendix B). An initial GCS of 3 to 8 indicates a severe traumatic brain injury (Iverson, 1998). In the current study, children with severe TBI were assessed because they often have long-term impairments of their sitting balance and functional mobility.

Recently, Swaine and Sullivan (1996) documented early motor recovery following severe traumatic brain injury in adults. These authors used a three-point ordinal scale (can perform, performs with assistance, cannot perform) to describe the changes in a wide range of motor functions. Sixty percent of the subjects progressed from dependent to independent sitting by six weeks after TBI. Although Swaine and Sullivan documented important information about the natural course of motor recovery following severe TBI in adults, the study did not examine the characteristics of postural

control that could have accounted for the changes in motor function. The current study documented the changes that occurred in three types of seated postural control as children with TBI progressed from dependent to independent sitting.

Standing balance has been examined in adults with TBI and the amount of sway has been found to be increased compared to normal subjects, especially when vision is not used and in subjects with severe TBI (Geurts, Ribbers, Knoop, & van Limbeek, 1996; Ingersoll & Armstrong, 1992; Lehmann et al., 1990; Wöber et al., 1993). This information suggests that vision can partially compensate for postural imbalance in subjects with brain injury. The current study evaluated the effects of vision on the 'static' postural control of children with TBI during quiet sitting.

One study documented changes in postural control over time for adults with TBI but no similar studies have been done with children (Wade, Canning, Fowler, Felmingham, & Baguley, 1997). Wade et al. studied changes in postural sway during standing in subjects who had TBI and were able to stand for 10 seconds for the initial assessment. When the subjects were reassessed 2 to 6 weeks later, Wade et al. found significant decreases in postural sway in standing.

Only three studies have examined postural control in children with TBI (Chaplin, Deitz, & Jaffe, 1993; Lahat et al., 1996; Reid et al., 1991). Reid et al. determined the 3-dimensional movement of the C7 spine during quiet sitting in seven 13-15 year old children with mild to moderate TBI. At the time of testing, the children with TBI could sit independently with or without hand support. There was no significant difference in the C7 spinal movement of the children with TBI compared to that of the age-matched typically developing children. Lahat et al. examined the postural sway of standing

children aged 8 to 15 years who had sustained mild TBI 24 to 36 hours prior to testing. Postural sway of the children with mild TBI was significantly greater than normal age-matched controls for standing tasks. Chaplin et al. evaluated the standing and walking balance of children aged 5 to 15 years who had sustained severe TBI 16 or more months prior to testing. On the balance subtest of a norm-referenced standardized gross motor test (Bruininks-Oseretsky Test of Motor Proficiency, 1978), children with severe TBI had significantly lower scores than their age-matched peers.

Several scales have been developed to evaluate sitting balance of people with neurological impairment (Berg, Wood-Dauphinee, Williams, & Gayton, 1989; Carr & Sheppard, 1987; Feigin et al., 1996; Fife et al., 1991; Mulcahy, Pountney, Nelham, Green & Billington, 1988). Only two of these scales have been used to evaluate sitting balance in children (Fife et al., 1991; Mulcahy et al., 1988). Fife et al. modified the sitting scale of Mulcahy et al. and evaluated the interrater and test-retest reliability of this seven-point scale on 40 children with developmental disabilities. The Kappa values indicated that the reliability was fair to good, (i.e. Kappa values ranged from .54 to .62). The Level of Sitting Scale (Fife et al., 1991) was used in this study since it is the only sitting scale validated on children with disabilities. An additional advantage of this scale is that, similar to the sitting position used in the current study, it evaluates the sitting ability of children with their feet unsupported.

Although a number of studies have evaluated seated postural control in typically developing children (Butterworth & Hicks, 1977; Hadders-Algra et al., 1996; Hirschfeld & Forssberg, 1994; Reid et al., 1991; Woollacott et al., 1987), and in children with cerebral palsy, (Brogren et al., 2001; Brogren et al., 1998; Fife et al, 1991; McClenaghan,

1989; Yang et al., 1996), only one study evaluated the seated postural control of children with mild to moderate TBI (Reid et al., 1991). No comparable studies have been found on children with severe traumatic brain injury. Therefore, the objectives of this study were to evaluate the seated postural control of children with severe TBI during re-acquisition of independent sitting.

3.1.1 Research questions

The following questions were addressed in this study:

1. What is the static, reactive, and volitional seated postural control in children with severe TBI?
2. How does the static, reactive, and volitional seated postural control change in children with severe TBI during re-acquisition of independent sitting?
3. How does the seated postural control in typically developing children compare with that of children with severe TBI?

As there are no studies of the seated postural control in children with severe TBI, this study may have clinical implications for both the assessment and treatment of postural control of children with TBI.

3.2 Methods

This study employed a prospective, longitudinal design to assess the seated postural control of children with severe TBI, using the same measures on two different occasions. In order to determine the changes that occurred in postural control from dependent to independent sitting, the children were required to meet specific levels of sitting ability at each occasion, based on the Level of Sitting Scale by Fife et al. (1991). The scale was modified slightly for use in the current study. In the original scale, level 5 is distinguished from level 6 only by the criterion that the child is able to lean forward and re-erect whereas at level 6 the child is able to lean to the side and re-erect. Based on the clinical experience of this researcher, the distinction between the two levels did not seem appropriate for children with TBI since anterior-posterior postural stability does not predictably precede medial-lateral postural stability. Thus, level 5 and level 6 were collapsed into one level and designated as level 5 in this modified version (Appendix E). At Time 1, children were required to be able to maintain 'static' independent sitting for 30 seconds with feet unsupported (i.e. level 4). At Time 2, children were required to be able to lean 20 degrees in the forward or sideways direction and re-erect without using hands for support (i.e. level 5).

3.2.1 Participants

From August 1999 to July 2000, children with severe TBI were recruited from two local acute care hospitals and one local rehabilitation hospital. Hospital and university ethics approvals were received prior to recruitment. Hospital physiotherapists were informed of the study through telephone contact and an initial letter of intent. Initial

contact with the parents was through an introductory letter given to the parents of appropriate candidates by the hospital physiotherapist. If the parents indicated interest in their child participating in the study, an informational video of the procedure was reviewed with the parents and children prior to the first evaluation. Written consent was obtained from parents and children (when possible) at the first evaluation.

During the 12-month recruitment period, three children with severe TBI met the inclusion criteria and volunteered for the study. However, one subject was subsequently excluded as a pre-injury attention deficit disorder impaired his ability to adequately perform the postural control tasks as directed. For example, he was unable to sit quietly for more than a few seconds at a time. Only one child who was known to meet the inclusion criteria declined to participate in the study.

To ensure that the children were tested at the appropriate times according to the modified Levels of Sitting Scale (Fife et al., 1991), hospital physiotherapists regularly assessed the sitting ability of the children. In addition, the children were required to be: 1) between 6 to 15 years of age, 2) medically stable, 3) able to follow simple commands, 4) able to tolerate sitting (supported) for one hour, and 5) able to maintain head upright for 30 seconds. In order to improve homogeneity of the sample, a narrower age range of 8 to 12 years was initially considered as the age criterion. However, in order to increase the potential number of participants in the study, the age range was expanded. Children younger than 6 years of age were not included because of concerns of tolerance and cooperation with the testing procedure.

Children with TBI were excluded from the study if the following conditions existed: 1) a concurrent agitated state of consciousness, 2) the presence of orthopedic or

peripheral neurological injuries which would interfere with the ability to perform tasks, or 3) history of previous brain injury or other neurological impairment.

3.2.2 Instrumentation and Procedure

Testing occurred at the Rehab Research Lab at the G F Strong Rehabilitation Centre in Vancouver, B.C. between October 1999 and July 2000. The children were assessed at times when they would be most alert and rested. With the following exceptions, all testing procedures and data analysis were the same as that used for control group (refer to Chapter 2 for details). During the trials, the examiners monitored signs of fatigue and provided rest breaks as needed. At Time 1, both children required regular rests between trials. This was provided by a padded removable backboard that was placed behind the children for support. At Time 1, the six year old was not able to maintain an upright sitting posture with the distal one-third of the thighs unsupported. Therefore, for the 6 year old, the thighs were fully supported on the seating surface at both Time 1 and Time 2 in order to ensure consistency between his test positions. In addition, at Time 1 both children were unable to sit with arms crossed across the waist. Therefore, at Time 1 they were allowed to support themselves with hands on thighs.

The number of trials for quiet sitting and maximal leans varied from the original testing protocol. At Time 1, the 15 year old had marked difficultly maintaining his sitting balance for 30 seconds and thus, data were collected from only one trial each of quiet sitting with eyes open and with eyes closed. The 6 year old was not able to sit with eyes closed without distress at Time 1, so this data was not collected. For both children, maximal leans were very destabilizing and difficult at Time 1, therefore data were

collected for only 2 or 3 trials for each lean direction. As well, the 6 year old was not able to perform backward leans at either testing time without falling, so this data was not collected. During Time 2, particularly for the 6 year old, additional trials were required to ensure that enough data were collected where the performance adhered closely to the task criteria.

Surface EMG electrodes were placed on the 15 year old at the same locations as described in Appendix D. However, because the 6 year old did not tolerate the electrodes on the front of his neck, no data were collected for the sternocleidomastoid muscles at Time 1 or Time 2.

3.2.3 Data Analysis

Data processing and analysis were the same as that for control group (refer to Chapter 2) with the following exceptions. Because only two children with TBI were assessed, descriptive data analysis was used to summarize the postural control for each child at Time 1 to Time 2. The postural control of children with TBI during quiet sitting was compared with age-matched controls because a significant effect of age was found for all COP values in the typically developing children (see Chapter 2 for details). For the control group, there were no statistically significant effects of age found for the postural control measures of the maximal lean and perturbation trials. In addition, ICCs for the postural control measures of the maximal lean and perturbation trials in the control group were moderate to high. Therefore, the limits of stability during maximal leans and the onset of muscle responses during perturbation trials in the children with TBI were compared with the Time 1 group data of control group.

3.3 Results

Two children with severe TBI were evaluated in this study. The characteristics of the 15 year old and 6 year old male children are listed in Table 17. The mechanism of injury, initial GCS score, level of consciousness at Time 1 and Time 2, and the extent of brain injury were similar in the two children. Additional information about the concurrent motor status of the children with TBI was obtained by chart review (Appendix F). This information was drawn from physician, physiotherapist, and occupational therapist reports within one week of the Time 1 and Time 2 assessments of each child with TBI.

At Time 1, both children required the use of their hands to support their upper body in upright sitting. At both testing times, each child tended to sit with marked spinal kyphosis and posterior pelvic tilt. At Time 2, there was little change in the sitting posture in the 6 year old, but the 15 year old had less of a slouched posture. The duration between Time 1 and Time 2 was twenty-eight days for the 15 year old and forty-two days for the 6 year old. Thus, the 15 year old showed a greater acceleration in the re-acquisition of independent sitting after the Time 1 assessment.

The seated postural control at Time 1 and Time 2 will be described for each child with TBI. In the discussion, the postural control of the children with TBI will be compared with the seated postural control of typically developing children (as described in Chapter 2).

3.3.1 Postural control during quiet sitting

Table 17. Characteristics of two male children with traumatic brain injury

	ti01	ti03
Age	15 years	6 years
Initial GCS	5	7
Mechanism of injury	MVA (passenger)	MVA (pedestrian)
Extent of brain injury	multiple shear hemorrhages of hemispheres, left thalamus, and midbrain	diffuse shear hemorrhages of hemispheres, cerebellar peduncle, and midbrain
LOC at Time 1 / Time 2	level 2 / some level 1 items	level 2 / some level 1 items
Hand dominance	right	left
Duration between injury and Time 1	98 days	78 days
Duration between Time 1 and Time 2	28 days	42 days
Level of sitting* at Time 1 / Time 2	level 4 / level 5	level 4 / level 5

ti01 = traumatic injury subject 1, ti03 = traumatic injury subject 3
LOC – Level of Consciousness, Pediatric LOC Scale (Appendix G)
GCS – Glasgow Coma Score (Appendix B)
MVA – motor vehicle accident
* modified Level of Sitting Scale (Appendix E)

Postural control during quiet sitting was measured by the COP displacement and velocity in the anterior-posterior and medial-lateral directions. The means and standard deviations of normalized and non-normalized COP values were calculated for the two children with TBI (Tables 18 and 19). The anterior-posterior COP displacement was greater than the medial-lateral COP displacement for both children at Time 1 and Time 2 in eyes open and eyes closed conditions. For both children, the COP velocity showed a similar pattern, with faster speeds in the anterior-posterior direction than in the medial-lateral direction for all times and conditions. At Time 1 for the 15 year old male, data for only one trial for each eyes open and eyes closed condition were collected, hence the absence of standard deviation values. Variability of values between trials for each child was low except for the velocity values for the 6 year old at Time 1 (eyes open) and Time 2 (eyes closed).

There is no appreciable difference (i.e. generally less than 1 standard deviation difference) in the COP displacement and velocity values between eyes open and closed conditions in each child with TBI.

Because a significant effect of age was found for all COP values during quiet sitting in the typically developing children, the COP values of children with TBI were compared with age-matched controls (Figures 4-7). At Time 1, the COP displacements and the anterior-posterior velocity of the 15 year old with TBI were more than two times larger than the corresponding COP values for the typically developing 15 year old. The medial-lateral COP velocity was the same at Time 1 in both 15 year old children. At Time 1, the COP displacements for the 6 year old with TBI were approximately 4 times larger than the corresponding COP displacements of the typically developing 6 year old.

Table 18. Means (STD) of COP displacement and velocity for eyes open and closed in children – non-normalized values

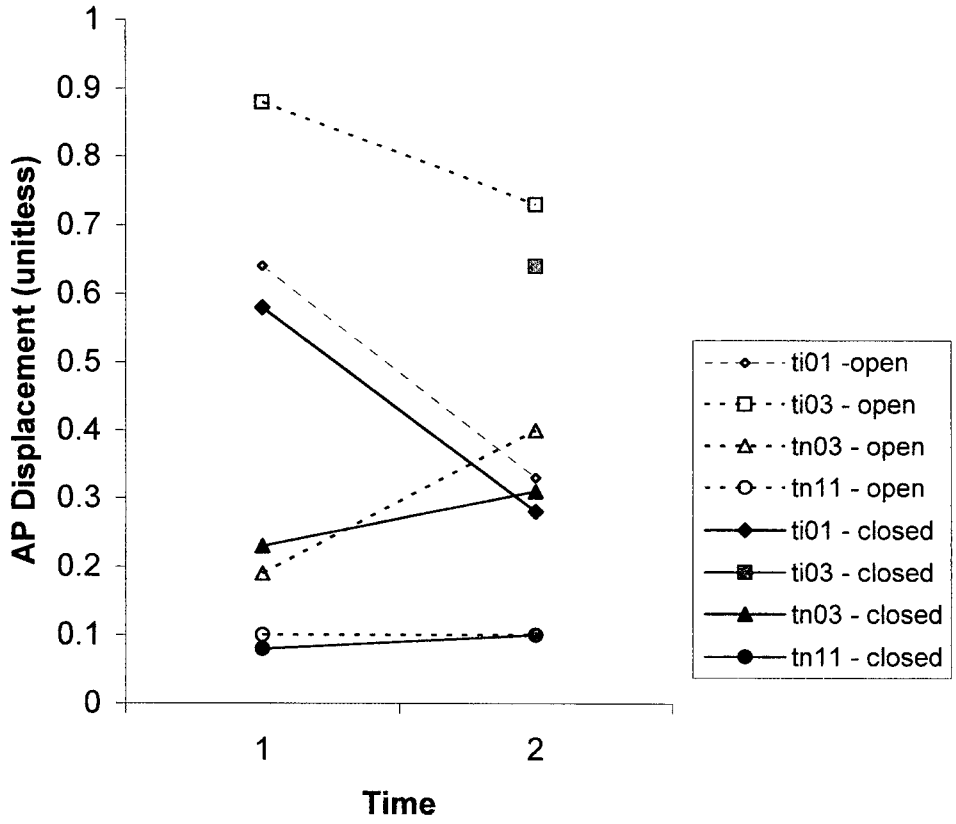
	ti01 (15 year old male)		ti03 (6 year old male)	
	Time 1	Time 2	Time 1	Time 2
EYES OPEN				
AP Displacement (cm)	0.54	0.28 (0.01)	0.42 (0.19)	0.35 (0.08)
ML Displacement (cm)	0.16	0.13 (0.02)	0.29 (0.11)	0.26 (0.04)
AP Velocity (cm/sec)	1.82	1.26 (0.16)	2.14 (1.15)	2.32 (0.31)
ML Velocity (cm/sec)	0.80	0.62 (0.04)	1.58 (0.92)	1.28 (0.17)
EYES CLOSED				
AP Displacement (cm)	0.49	0.23(0.07)	N/A	0.31 (0.26)
ML Displacement (cm)	0.21	0.11 (0.03)	N/A	0.25 (0.26)
AP Velocity (cm/sec)	1.81	1.19 (0.21)	N/A	1.60 (1.37)
ML Velocity (cm/sec)	0.79	0.50 (0.14)	N/A	1.04 (0.98)

ti01 = traumatic injury subject 1, ti03 = traumatic injury subject 3
N/A: data not available, ti03 unable to sit with eyes closed

Table 19. Means (STD) of COP displacement and velocity for eyes open and closed in children – normalized values (x 10^{-2})

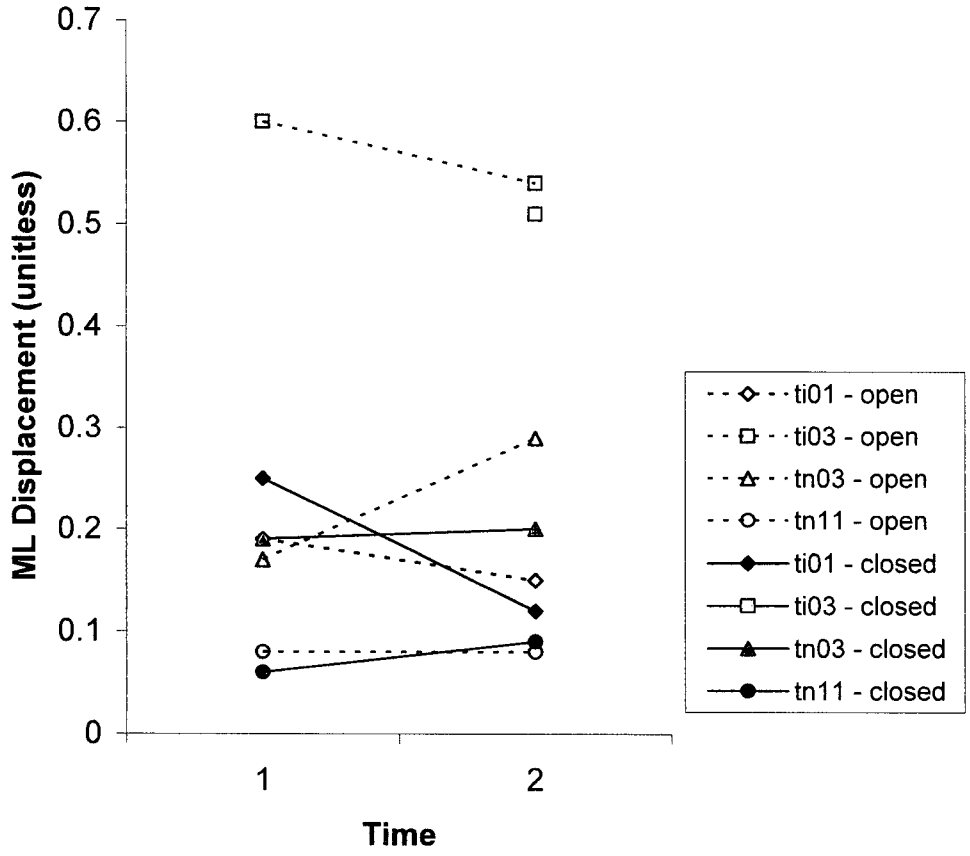
	ti01 (15 year old male)		ti03 (6 year old male)	
	Time 1	Time 2	Time 1	Time 2
EYES OPEN				
AP Displacement (unitless)	0.64	0.33 (0.01)	0.88 (0.40)	0.73 (0.16)
ML Displacement (unitless)	0.19	0.15 (0.02)	0.60 (0.23)	0.54 (0.09)
AP Velocity (sec^{-1})	2.16	1.49 (0.19)	4.42 (2.37)	4.78 (0.65)
ML Velocity (sec^{-1})	0.95	0.73 (0.05)	3.25 (1.90)	2.65 (0.35)
EYES CLOSED				
AP Displacement (unitless)	0.58	0.28 (0.09)	N/A	0.64 (0.54)
ML Displacement (unitless)	0.25	0.12 (0.03)	N/A	0.51 (0.53)
AP Velocity (sec^{-1})	2.15	1.41 (0.24)	N/A	3.30 (2.84)
ML Velocity (sec^{-1})	0.93	0.60 (0.17)	N/A	2.14 (2.02)

ti01 = traumatic injury subject 1, ti03 = traumatic injury subject 3
N/A: data not available, ti03 unable to sit with eyes closed



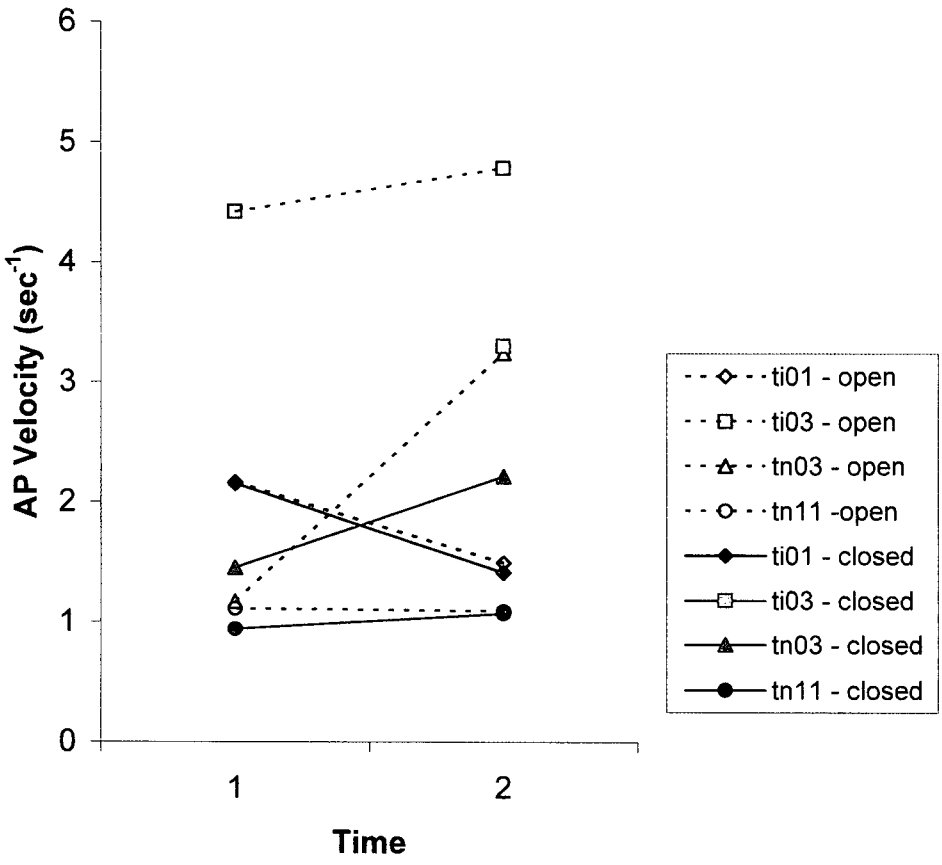
ti01 = 15 year old with TBI, ti03 = 6 year old with TBI, tn03 = typical 6 year old, tn11 = typical 15 year old

Figure 4. COP displacement in anterior-posterior direction for two children with TBI and age-matched typically developing children – normalized values



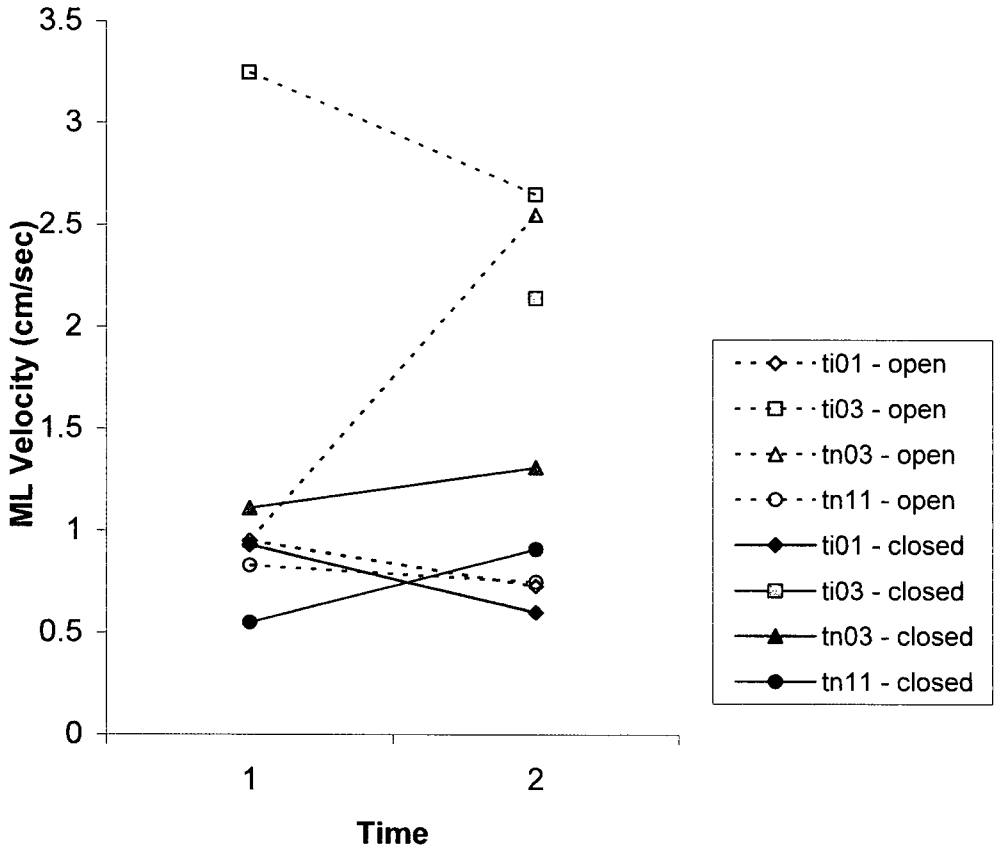
ti01 = 15 year old with TBI, ti03 = 6 year old with TBI, tn03 = typical 6 year old, tn11 = typical 15 year old

Figure 5. COP displacement in medial-lateral direction for two children with TBI and age-matched typically developing children – normalized values



ti01 = 15 year old with TBI, ti03 = 6 year old with TBI, tn03 = typical 6 year old, tn11 = typical 15 year old

Figure 6. COP velocity in anterior-posterior direction for two children with TBI and age-matched typically developing children – normalized values



ti01 = 15 year old with TBI, ti03 = 6 year old with TBI, tn03 = typical 6 year old, tn11 = typical 15 year old

Figure 7. COP velocity in medial-lateral direction for two children with TBI and age-matched typically developing children – normalized values

The COP velocities for the 6 year old with TBI were approximately 3 times larger than the corresponding COP values for the typically developing 6 year old at Time 1.

From Time 1 to Time 2, the medial-lateral COP displacement and COP velocity values for the 15 year old with TBI had decreased such that they were the same as the corresponding COP values of the typically developing 15 year old. At Time 2, the anterior-posterior COP displacements of the 15 year old with TBI were approximately 3 times larger than the corresponding COP displacements of the typically developing 15 year old.

The eyes open COP values for the 6 year old child with TBI decreased from Time 1 to Time 2, with the exception of anterior-posterior velocity which increased slightly. At Time 2, the anterior-posterior and medial lateral COP displacements of the 6 year old with TBI were still 3 times larger than the corresponding COP displacements of the typically developing 6 year old. At Time 2, the eyes closed COP velocity values for the 6 year old with TBI were 1.5-2 times larger than the corresponding eyes closed COP velocity values for the typically developing 6 year old. Interestingly, the COP velocities for the typically developing 6 year old increased 2-3 fold from Time 1 to Time 2. The change in COP velocities of the typically developing 6 year old supports the previous observation that younger typically developing children have more variable COP excursions. The combination of decreased COP velocities in the 6 year old with TBI and increased COP velocities in the typically developing 6 year old lead to converging COP velocity values at Time 2.

At Time 1 and Time 2, the COP displacements and velocities of the 6 year old with TBI were all much larger than the COP displacement and velocity values of the 15 year old with TBI.

3.3.2 Postural control during maximal leans

The limits of stability in sitting were measured by the maximal displacements of the COP during self-paced leans in the forward, backward, and sideways (non-dominant and dominant) directions. Normalized and non-normalized mean and standard deviation values for the maximal COP displacements were calculated for the children with TBI (Tables 20 and 21). Because there were no statistically significant effects of age found for maximal leans in typically developing children and the ICCs were high to very high for the four lean directions, the limits of stability during maximal leans in the children with TBI will be compared with the Time 1 normalized group data of the control group (Table 21). At Time 1, the maximal forward COP displacement for the 15 year old with TBI was almost the same as the group mean of the control group. However, the maximal backward and sideways COP displacements for the 15 year old with TBI were 1.8 to 2.6 times smaller than the corresponding control group mean COP displacements. At Time 1 and Time 2, the six year old was unable to maintain his balance during backward leans. In addition, in order to sit unsupported at Time 1, the six year old needed to have full thigh support on the sitting surface. Therefore, in order to keep his sitting position consistent between Time 1 and Time 2, the six year old sat with full thigh support at both times. At Time 1, the maximal forward COP displacement for the 6 year old was slightly greater than the mean forward COP displacement of the control group. However, the

Table 20. Mean (STD) of maximal forward, backward, dominant side and non-dominant side leans in children – values non-normalized (cm).

	Forward	Backward	Dominant	Non-Dominant
ti01				
(15 year old male)				
Time 1	8.3 (1.9)	11.7 (0.8)	6.7 (0.1)	8.4 (N/V)
Time 2	10.7 (2.9)	11.3 (2.6)	9.1 (0.5)	10.2 (0.5)
ti03				
(6 year old male)				
Time 1	6.6 (1.3)	N/A	4.2 (2.2)	4.8 (2.5)
Time 2	12.0 (1.4)	N/A	4.2 (0.6)	5.7 (1.1)
Typical Children				
(N = 10)				
Time 1	8.3 (2.5)	16.8 (4.7)	13.4 (2.2)	13.1 (2.3)

N/A – child unable to perform
N/V – no standard deviation value available because data for only one trial collected

Table 21. Mean (STD) of maximal forward, backward, dominant side and non-dominant side leans in children – values normalized (unitless).

	Forward	Backward	Dominant	Non-Dominant
ti01				
(15 year old male)				
Time 1	0.10 (0.02)	0.14 (0.01)	0.08 (0.00)	0.10 (N/V)
Time 2	0.13 (0.04)	0.13 (0.03)	0.11 (0.01)	0.12 (0.01)
ti03				
(6 year old male)				
Time 1	0.14 (0.03)	N/A	0.09 (0.05)	0.10 (0.05)
Time 2	0.18 (0.05)	N/A	0.09 (0.01)	0.12 (0.03)
Typical Children				
(N = 10)				
Time 1	0.13 (0.03)	0.26 (0.06)	0.21 (0.02)	0.20 (0.02)

N/A – child unable to perform
N/V – no standard deviation values available because data for only one trial collected

maximal sideways COP displacements for the six year old were 2 to 2.3 times smaller than the corresponding control group mean COP displacements.

At Time 2, the maximal COP displacement values for the 15 year old increased only slightly compared to Time 1 values. At Time 2, the forward COP displacement for the 15 year old was the same as the control group mean forward COP displacement. However, the maximal backward and sideways COP displacements for the 15 year old were still approximately two times smaller than the respective COP displacements of the control group. At Time 2, the non-dominant side COP displacement increased slightly for the 6 year old. There was no change in the dominant side COP displacements from Time 1 to Time 2. Thus, at Time 2 the non-dominant side and dominant side maximal COP displacements for the 6 year old were still approximately two times smaller than the respective mean COP displacements of the control group. At Time 2, the maximal forward COP displacement for the 6 year old had increased and was 1.4 times greater than the group mean forward COP displacement.

3.3.3 Postural control during platform perturbations

Anterior and posterior perturbations were applied to the sitting platform in order to rapidly move the centre of mass (COM) of the upper body over the base of support (BOS). Anterior perturbations cause backward displacements of the COM, requiring anterior muscles to contract to regain an upright posture. Correspondingly, posterior perturbations cause the COM to move forward, requiring posterior muscles to contract to restore an upright posture. Postural control during platform perturbations was measured by the onset of bilateral muscle responses of six representative trunk and thigh muscle

groups (rectus femoris, abdominals, sternocleidomastoid, hamstrings, L1 erector spinae, and C4 erector spinae). Mean and standard deviation values were calculated for the dominant side muscle responses following anterior and posterior perturbations (Tables 22 and 23). Because there were no statistically significant effects of age found for the onsets of muscle responses in typically developing children and the ICCs were moderate to high for the muscle responses which occurred regularly, the onsets of muscle responses in the children with TBI will be compared with the Time 1 group data of the control group (Tables 22 and 23).

At Time 1, there were no consistent muscle responses to anterior perturbations in the 15 year old. Anterior muscle responses were present in the 6 year old at Time 1, with longer mean onsets for the rectus femoris (RF) and abdominals (ABS) than the group mean onsets (ie. 72 msec longer, 36 msec longer, respectively). In the 6 year old, the onset of the RF was much more variable than that of the control group.

Posterior muscle responses following posterior perturbations were present at Time 1 in both the 15 year old and the 6 year old. Onsets of the hamstrings (HAMS) and level 4 cervical erector spinae (C4 ES) muscles in the 15 year old were similar to the group mean onsets for the respective muscles. The onset of the level 1 lumbar erector spinae (L1 ES) in the 15 year old was 38 msec longer than the group mean onset for the L1 ES. Onsets for the HAMS and L1 ES muscles in the 6 year old were 63 msec and 51 msec longer than the respective group mean muscle onsets. The onset for the C4 ES in the 6 year old was 27 msec shorter than the group mean onset. In the 15 year old, the onset of the HAMS was much more variable than that of the control group.

Table 22. Mean (STD) for onset of muscle activation (msec) following anterior perturbations in children – dominant side values

	RF	*ABS*	*SCM*	HAMS	L1ES	C4ES
ti01						
(15 year old male)						
Time 1	N/R	N/R	N/R	N/R	N/R	N/R
Time 2	115.9	127.3	135.3	N/R	N/R	N/R
	(31.6)	(15.5)	(34.1)			
ti03						
(6 year old male)						
Time 1	175.0	147.6	N/C	N/R	N/R	196.3
	(78.1)	(31.0)				(86.8)
Time 2	N/R	145.6	N/C	68.9	250.0	174.2
		(20.8)		(9.8)	(39.3)	(46.3)
Typical Children						
(N = 10)						
Time 1	103.4	110.0	113.1	157.2	159.7	133.0
	(10.3)	(23.6)	(29.5)	(53.1)	(61.3)	(29.8)

RF – rectus femoris, ABS – abdominals, SCM – sternocleidomastoid, HAMS – hamstrings, L1ES – L1 erector spinae, C4ES – C4 erector spinae
Italicized muscle groups=anterior muscles
N/R – no muscle response; N/C – data were not collected for SCM for ti03

Table 23. Mean (STD) for onset of muscle activation (msec) following posterior perturbations in children – dominant side values

	HAMS	*L1ES*	*C4ES*	RF	ABS	SCM
ti01						
(15 year old male)						
Time 1	116.7	170.0	150.3	184.5	N/R	N/R
	(65.4)	(35.8)	(29.2)	(63.8)		
Time 2	93.9	135.0	140.0	N/R	N/R	N/R
	(44.4)	(26.6)	(8.7)			
ti03						
(6 year old male)						
Time 1	183.7	183.3	127.3	N/R	251.7	N/C
	(9.7)	(16.6)	(18.2)		(16.6)	
Time 2	147.3	172.0	122.5	215.6	N/R	N/C
	(25.9)	(25.6)	(20.7)	(25.8)		
Typical Children						
(N = 10)						
Time 1	120.0	132.5	154.5	153.6	148.2	129.2
	(25.3)	(19.5)	(37.1)	(30.5)	(58.0)	(13.1)

RF – rectus femoris, ABS – abdominals, SCM – sternocleidomastoid, HAMS – hamstrings, L1ES – L1 erector spinae, C4ES – C4 erector spinae
Italicized muscle groups=anterior muscles
N/R – no muscle response; N/C – data were not collected for SCM for ti03

At Time 2, the onsets of the anterior muscles following anterior perturbation in the 15 year old were essentially the same as the onsets of the respective muscles in the control group. The only anterior muscle response in the 6 year old was in the ABS which had the same onset as in Time 1. In the 15 year old at Time 2, the onset of the RF was more variable than that of the control group.

From Time 1 to Time 2, the onsets of all of the posterior muscles following posterior perturbation had decreased in the 15 year old. Thus, at Time 2 the onsets of the L1 ES and C4 ES muscles following posterior perturbation in the 15 year old were essentially the same as the onsets of the respective muscles in the control group. The onset of the HAMS was slightly shorter in the 15 year old than the group mean onset. From Time 1 to Time 2, the onsets of all the posterior muscles following posterior perturbation had also decreased in the 6 year old. However, at Time 2 the onsets for the HAMS and L1 ES muscles in the 6 year old were still longer than the respective group mean muscle onsets. In addition, at Time 2 the onset for the C4 ES in the 6 year old was even shorter than the group mean onset.

The sequence of muscle responses following perturbations was tabulated for the children with TBI (Tables 24 and 25). For the 6 year old at Time 1, the sequence of anterior muscle responses following anterior perturbations was the same in the dominant side and non-dominant side muscles. At Time 1, the sequence of posterior muscle responses following posterior perturbations was quite variable between the dominant side and non-dominant side for each child with TBI. At Time 2, there was much less variability in sequence of dominant side and non-dominant side muscle responses in both children. In

Table 24. Sequence of anterior muscle group activation following anterior platform perturbation for two children with traumatic brain injury at Time 1 and Time 2

	Time 1	Time 2
ti01 (15 year old male)		
Dominant side	N/R in any muscles (0)	RF, ABS, SCM (4)
Non-dominant side	N/R in any muscles (0)	RF, SCM, ABS (4)
ti03 (6 year old male)		
Dominant side	ABS, RF, [SCM]* (3)	ABS, [SCM]* (0)
Non-dominant side	ABS, RF, [SCM]* (3)	ABS, [SCM]* (0)

RF = rectus femoris, ABS = abdominals, SCM = sternocleidomastoid, N/R = no response
* SCM was not collected, may or may not have contracted in sequence
Numbers in parentheses = frequency of sequence (out of a possible 20 bilateral responses) in 10 typical children

Table 25. Sequence of posterior muscle group activation following posterior platform perturbation for two children with traumatic brain injury at Time 1 and Time 2

	Time 1	Time 2
ti01 (15 year old male)		
Dominant side	HAMS, C4 ES, L1 ES (4)	HAMS, L1 ES, C4 ES (7)
Non-dominant side	L1 ES, C4 ES (0)	HAMS, L1 ES (1)
ti03 (6 year old male)		
Dominant side	C4 ES, L1 ES, HAMS (1)	C4 ES, HAMS, L1 ES (2)
Non-dominant side	HAMS, C4 ES (0)	C4 ES, HAMS, L1 ES (2)

HAMS = hamstrings, C4 ES = cervical spine level 4 erector spinae, L1 ES = spine level 1 erector spinae
Numbers in parentheses = frequency of sequence (out of a possible 20 bilateral responses) in10 typical children

addition, the sequence of muscle responses in the 15 year old at Time 2 was the same as the most common sequences of responses observed in the control group.

Muscle activation of the flexors and extensors of the thigh, trunk, and neck 'segments' occurred infrequently in both children with TBI. Following anterior perturbations, only one segmental pair was activated in the 6 year old at Time 2. In this segmental pair, the trunk flexors (ABS) contracted 100 msec prior to the extensors (L1 ES). Following posterior perturbations at Time 1, the hip extensors (HAMS) activated 68 msec before the flexors (RF) in the 15 year old. In the 6 year old, following posterior perturbations, the trunk extensors (L1 ES) contracted 69 msec prior to the flexors (ABS) at Time 1. As well, the hip extensors (HAMS) activated 69 msec before the flexors (RF) in the 6 year old at Time 2.

3.4 Discussion

3.4.1 Seated postural control in children with traumatic brain injury

To date, the study by Reid et al. (1991) is the only one to evaluate the seated postural control of children with TBI. Reid et al. compared the quiet sitting of 13-15 year old typically developing children with that of children with mild to moderate TBI. For comparison with the current study, the findings of the Reid et al study will be discussed. As there are no other studies on the seated postural control of children with TBI, the results of the 2 children with TBI will be compared to the results of the 10 typically developing children discussed in Chapter 2.

3.4.1.1 Postural control during quiet sitting

Similar to the control group, both children with TBI consistently demonstrated greater COP displacement and velocity values in the anterior-posterior direction than in the medial-lateral direction (Tables 18 and 19). Because the children with TBI had been developing normally prior to the injury, it is likely that their spinal, hip, and pelvic structures would provide a greater 'stiffness' to medial-lateral movement of the COM, as is postulated to occur in the typically developing children. Alternately, the abnormal muscle tone that was present in both children with TBI may have created greater anterior-posterior excursions of the COM than medial-lateral excursions. This explanation is consistent with the clinical presentation of children with abnormal muscle tone affecting the trunk. Children with increased extensor tone tend to lose their sitting balance in the backward direction whereas children with low trunk tone tend to lose their balance in the forward or sideways directions.

There was not an appreciable difference in the COP displacement and velocity values between eyes open and closed conditions in either child with TBI (Tables 18 and 19). This is consistent with the findings for typically developing children where no significant differences were found in COP values for eyes open and closed conditions. These results suggest that, even in children with severe TBI, vision is not a required sensation for the maintenance of quiet sitting.

In the 15 year old with TBI, the COP displacements and velocities appear to be sensitive to the concurrent change in sitting ability from level 4 to level 5. From Time 1 to Time 2, the 6 year old with TBI also showed improvement in sitting ability from level 4 to level 5. However, the COP displacement and velocity values of the 6 year old with TBI do not appear to be sensitive indicators of this significant clinical change. A possible explanation for the lack of correspondence between these two postural control measures in the 6 year old with TBI is the extent to which the hands were used to stabilize the COM at Time 1. Although both children with TBI relied on the support of hands to stabilize the COM at Time 1, the 6 year old may have relied more heavily on the support of the hands for stability. Therefore, at Time 1 the COP displacements and velocities for the 6 year old may have been attenuated by the use of hand support. Thus, the 'true' change in COP values for the 6 year old with TBI may have been masked.

In the current study, the children with TBI tended to sit in more slouched postures than the control group. The slouched posture may have been an unconscious mechanism to provide greater stability during sitting. Reid et al. (1991) measured the 3-dimensional movement of the C7 spine in seven 13-15 year old children with mild to moderate TBI and age-matched non-disabled children. Children sat quietly on a flat bench with feet

supported for 5 minutes while viewing cartoons. Although Reid et al. (1991) did not find any statistical differences in the movement of the C7 spine in the two groups of children, they noted that the children with TBI sat in more rigid postures. These researchers suggested that the children with TBI may have been proximally "fixing" in order to compensate for disruptions of sensory regulation of postural control.

At both Time 1 and Time 2, the 15 year old with TBI had substantially smaller displacements and slower velocities than the 6 year old child with TBI. This difference may be a result of the effects of age, as was found in the control group. Alternately, the difference between the COP values of the children with TBI may reflect the disparate effects of brain injury on the postural control of each child.

3.4.1.2 Postural control during maximal leans

The limits of stability in sitting were measured by the maximal displacements of the COP during self-paced leans in the forward, backward, and sideways (non-dominant and dominant) directions.

At Time 1 and Time 2, the maximal forward COP displacement for the 6 year old with TBI was greater than the group mean forward COP displacement. Because the 6 year old had full thigh support, his base of support in the forward direction would have been relatively longer than that of the control group. This is probably the reason that the 6 year old with TBI had larger forward COP displacements than the control group. It is highly unlikely that the larger maximal forward COP displacements of the 6 year old reflect truly larger forward limits of stability, particularly since he had much smaller

limits of stability in the sideways and backward directions at both times compared with the typical children.

At Time 2, the maximal forward COP displacement of the 15 year old had decreased to the mean COP displacement value of the control group. However, at Time 2 the maximal backward and sideways COP displacements of the 15 year old were still approximately 2 times smaller than the respective group mean COP displacements. Thus, even at a level 5 on the sitting scale at Time 2, both children with TBI had much smaller limits of stability in the backward and sideways directions than the control group.

As in the quiet sitting tasks, both children with TBI relied on their hands for support during maximal leans at Time 1. Therefore, the maximal COP displacement values at Time 1 were probably much greater than they would have been if the children were required to sit without hand support. Thus, the 'true' changes in maximal COP displacements from Time 1 to Time 2 for the children with TBI may have been attenuated.

3.4.1.3 Postural control during platform perturbations

At Time 1, there were no consistent muscle responses to anterior perturbations in the 15 year old. This may have been partly due to the fact that he used his hands for stability at Time 1. At Time 1, several of the onsets of muscle responses in the children with TBI were longer than the onsets of the corresponding muscles in the control group. However, at Time 2 in the 15 year old, five out of six muscle groups had onsets that were the same as the onsets of the analogous muscles in the control group. Although all of the muscle onset times decreased from Time 1 to Time 2 in the 6 year old, with the exception

of the C4 ES, the muscle onsets were still longer than the onsets of the corresponding muscles in the control group.

At Time 2, the sequence of muscle group activation was less variable in both children with TBI than at Time1. Most of the activation sequences in the children with TBI at Time 2 were the same as those seen in the control group. These changes may also signal improvements in the cortical functioning of the children with TBI.

Muscle activation of the flexors and extensors of the thigh, trunk, and neck 'segments' occurred infrequently in both children with TBI. This is not dissimilar to the results found in the typically developing children where the highest frequency of segment pair responses occurred in the RF and HAMS in 7 children following anterior perturbations.

3.4.1.4 Mechanisms of improved postural control

Currently, three predominant theories of recovery after brain injury are postulated in the literature. Recovery after brain injury may be due to reversal of diaschisis, behavioral compensation, and/or vicariation of function (Finger & Almli, 1985; Nudo, Plautz, & Frost, 2001). Researchers have theorized that the initial improvements in function following brain injury may be due to the reversal of diaschisis. This theory maintains that damage to one part of the brain causes temporary but reversible depression of normal neuronal activity in other areas of the brain, or diaschisis. In time, the neuronal activity of these temporarily affected areas returns to normal (i.e. reversal of diaschisis), resulting in observed behavioral improvements.

A specific type of temporary neural depression is postulated for the two children with TBI in this study. Both children had diffuse cortical and subcortical shear hemorrhages (Table 17 and Appendix H). Because of the extent of this damage, it is possible that considerable interstitial edema was present up to 3 months following the injuries (G. Hahn, personal communication, July 25, 2002). Compression of peripheral nerves has been found to reduce their conduction velocity (Robertson, 1985, chap. 22). Similiarly, interstitial edema could lead to compression of cerebral axons and reduced axonal conduction velocities (G. Hahn, personal communication, July 25, 2002). It follows that, as the interstitial edema resolves, the conduction velocities of the cerebral axons may increase.

Increases in the axonal conduction velocities may account for the changes observed in the reactive postural control of the children with TBI. In these children, most of the muscle responses to perturbation had onsets between 100 – 185 msec. Onsets of this duration may represent automatic postural responses which have transcortical inputs (Haas, Diener, Bacher, and Dichgans, 1986; Nashner & Cordo, 1981). Thus, the decrease in the onsets of muscle responses which was observed in both children with TBI from Time 1 to Time 2 may reflect increased conduction velocities of the cerebral axons.

An alternate theory of the recovery of function following brain injury is known as behavioral compensation (Finger & Almli, 1985; Nudo et al., 2001). According to this theory, functional improvements are accomplished by individuals with brain injury through the use of new, compensatory strategies to accomplish tasks. This theory is not supported by the changes observed in the reactive postural control in children with TBI in the current study. In fact, from Time 1 to Time 2 muscle onset times and muscle

activation sequences in both children with TBI became more like those observed in the typically developing children. Therefore, the changes observed in reactive postural control in the children with TBI appear to be better explained by the reversal of diaschisis and/or vicariation of function.

A third theory of the recovery of function following brain injury is the process known as vicariation of function (Finger & Almli, 1985; Nudo et al., 2001). According to this theory, brain structures either remote or adjacent to the area of damage take over the function of the damaged area. Thus, neuronal reorganization accounts for the improvements in function following brain injury. There is increasing evidence of neuronal reorganization in humans. A recent study of adults with syndactyly has demonstrated reorganization of the somatosensory cortex within 1-5 weeks following the surgical separation of syndactylous digits (Mogilner et al., 1993). The durations between Time 1 and Time 2 for two children with TBI in this study were 28 and 42 days apart, respectively. Therefore, it is possible that neuronal reorganization could have occurred in both children, leading to improvements in their static, voluntary, and reactive seated postural control.

An additional explanation is postulated for the improvements observed in the static postural control of the children with TBI. Over several years of clinical experience, this researcher has observed that improvements in the motor function of children following TBI usually occur as the level of consciousness (LOC) increases. Rarely have children shown clinically significant motor improvements without concurrent increases in LOC. The LOC of both children with TBI in this study increased from Time 1 to Time 2 (Table 17 and Appendix G). The increased LOC may have allowed these children to

accurately perceive their body orientation. This in turn, may have improved their accuracy and efficiency of controlling the centre of mass (through movement of the centre of pressure) during quiet sitting.

The children with TBI in the current study received intensive in-patient rehabilitation for several months following injury. Between Time 1 and Time 2, the rehabilitation program consisted of physiotherapy, occupational therapy, recreational therapy, and rehabilitation nursing. Typically, 45 to 60 minute therapy sessions for each discipline occurred 3 to 5 times per week. Interdisciplinary goals included improved independent mobility and activities of daily living. Recent studies provide support for the putative effects of rehabilitation following brain injury (Liepert, Miltner, Bauder, Sommer, Dettmers, & Taub, 1998; Miltner, Bauder, Sommer, Dettmers, & Taub, 1999). In adults with chronic stroke, lasting improvements in the impaired upper extremity and changes in the motor cortex were demonstrated following 2 weeks of daily training of the impaired limb and constraint of the non-impaired limb. Therefore, it is possible that the intensive acute rehabilitation received by the two children with TBI contributed to the observed changes in the static, voluntary, and reactive seated postural control.

3.4.1.5 Limitations

Although the intention of this study was to examine changes in postural control measures which occurred as children with TBI progressed from dependent to independent sitting, more meaningful comparisons may have been possible if children were required to be at a level 5 sitting ability at Time 1. This would have allowed the children to perform the postural control tasks without using their hands for support. Thus,

comparisons with subsequent assessments would not be confounded with the effects of hand support as in this study. In addition, children at level 4 sitting ability were not able to perform some of the postural control tasks or modifications in the test protocol were required. Thus, requiring a level 5 sitting ability at Time 1 may have improved the quality of comparisons between assessments.

Clearly, a larger number of children with TBI between the ages of 6 and 15 would have made the findings of this study more generalizable to other children with severe TBI.

CHAPTER 4: CONCLUSIONS

This study evaluated the seated postural control of typically developing children and children with severe traumatic brain injury. Seated postural control is important for many activities of daily living for children with and without disabilities. Children need to have good postural control in sitting in order to participate in many occupational, functional, social and recreational activities. Seated postural control is of great significance to clinicians in rehabilitation settings as independent sitting reduces the risks of falls and decreases the need for costly seating equipment. In addition, seated postural control following brain injury may be predictive of future functional outcomes (Black et al., 2000; Feigin et al., 1996). Further, independent sitting may have a profound effect on the long-term survival of children with severe TBI (Eyman et al.,1993; Strauss et al., 1998).

For many years, normal motor development and neurological rehabilitation approaches have been based on the assumption that the control of movement is organized hierarchically (Horak, 1991; Woollacott & Shumway-Cook, 1990). Based on this hierarchical perspective, the development of normal movement is achieved through integration or inhibition of primitive reflexes. Thus, as a child develops, movement proceeds from primarily reflexive control to voluntary control. The development of motor skills is viewed as step-wise stereotyped achievement of motor milestones. In addition, according to the hierarchical perspective, all abnormal movement following brain injury has been thought to be due to disinhibition of lower-level reflexes caused by injury of higher-level cortical motor centres. Thus, recovery of postural control should progress from primitive reflexive movement to righting reactions and then to mature

equilibrium reactions. Based on the hierarchical model, rehabilitation has focused on inhibiting abnormal tone and reflexive movements while facilitating "normal" movement patterns. The rehabilitation of motor skills has focused on re-training earlier "motor milestones" prior to re-training later "motor milestones". For example, following brain injury, sitting balance would be re-trained and achieved prior to proceeding to re-training standing balance.

The results of this study tend to contradict a hierarchical, step-wise concept of motor development and recovery of postural control. For example, although 6-7 year old typically developing children had volitional and reactive postural control similar to that of older children, the 6-7 year olds' static postural control during quiet sitting was not similar to or as "mature" as the older children. In addition, although at the second evaluation the 6 year old with TBI demonstrated postural responses following perturbations that were similar to the postural responses seen in the control group, his COP displacement and velocity values were still 1.5-3 times larger than the age-matched typically developing child. Changes that were observed in the postural control of the 6 year old with TBI occurred in all three types of postural control, without return to "normal" of the static postural control prior to the more "mature" or challenging types of postural control.

In this study, seated postural control was direction-specific in both the control group and children with TBI. In particular, in both groups of children, anterior-posterior COP displacements and velocities were greater than medial-lateral displacements and velocities. In addition, anterior perturbations elicited more consistent motor responses than posterior perturbations in the control group. This is similar to the findings of other

researchers who postulated that the disparity of the motor responses between anterior and posterior perturbations is related to differences in stability during backward and forward trunk movement over the base of support (Brogren et al., 2001; Brogren et al., 1998; Forssberg & Hirschfeld, 1994; Hirschfeld & Forssberg, 1994). In contrast, in the current study the children with TBI had slightly more variable muscle responses to anterior perturbations. These findings may be due to the more slouched sitting postures of the children with TBI which would make them more stable than the controls to posterior perturbations. As well, the two children with TBI demonstrated greater increases in maximal leans in the anterior and posterior directions than in the lateral directions. These direction-specific differences cannot be adequately explained by a hierarchical reflex model of motor development. In order to explain direction-specific effects on postural control, biomechanical constraints such as the size and shape of the base of support, the position of the centre of mass relative to the base of support, and the effects of joint stiffness and muscle stiffness (particularly of two joint muscles) need to be considered.

The findings of this study have implications for neurorehabilitation. The effects of age need to be considered when evaluating and treating impairments of static postural control in children. That is, children younger than 10 years of age should not be expected to have the same static postural control of older children. In addition, because postural control does not appear to recover in a step-wise fashion, simultaneous re-training of different types of postural control is appropriate. Thus, static postural control may not have returned to "normal" even though other, more challenging or "mature" types of postural control may have recovered.

This study was the first to demonstrate changes in muscle onsets following brain injury. The muscle onsets in both children with TBI decreased as much as 35-36 msec from the first to the second evaluation. These changes suggest that there are central neural changes that occur during the recovery from traumatic brain injury which impact on automatic postural responses. Future descriptive research of postural control in individuals with brain injury may help to clarify whether the changes observed in this study are specific to children with TBI or generalizable to adults with TBI (or other forms of cerebral injury).

In addition, experimental research is needed to evaluate the effects of rehabilitation interventions on the recovery of postural control in individuals with acquired brain injury. The course and extent of recovery following severe TBI varies markedly from one child to another. Because of this variability and relatively small numbers of children with severe TBI, randomized control studies evaluating postural control interventions in two (homogeneous) groups are not practical. However, multiple baseline design single-subject research can control for the effects of "spontaneous" recovery in subjects while evaluating the effectiveness of postural control intervention (Backman, Harris, Chisholm, & Monette, 1997).

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Appendix A

Definition of Severity of TBI

Mild TBI

Traumatic injury to the head with any one or combination of the following:

a) confusion or disorientation for several minutes (with or without the loss of consciousness),

b) loss of consciousness for up to 30 minutes,

c) after return of consciousness, Glasgow Coma Scale score of 13 to 15,

d) symptoms such as headache, hypersensitivity to noise or light, dizziness, nausea, fatigue, irritability, or problems with concentration and thinking skills,

e) neuroradiologically detected abnormality or focal neurological deficits such as weakness, abnormal reflexes, or language disturbances.

Moderate TBI

Traumatic injury to the head with any one or combination of the following:

a) loss of consciousness for 30 minutes to 6 hours,

b) after return of consciousness, Glasgow Coma Scale score of 9 to 12.

Severe TBI

Traumatic injury to the head with any one or combination of the following:

a) loss of consciousness for more than 6 hours,

b) initial Glasgow Coma Scale score of 3 to 8.

From: Iverson, G. (1998, Fall). Epidemic in name. Recovery Magazine, 9, 4-7.

Appendix B

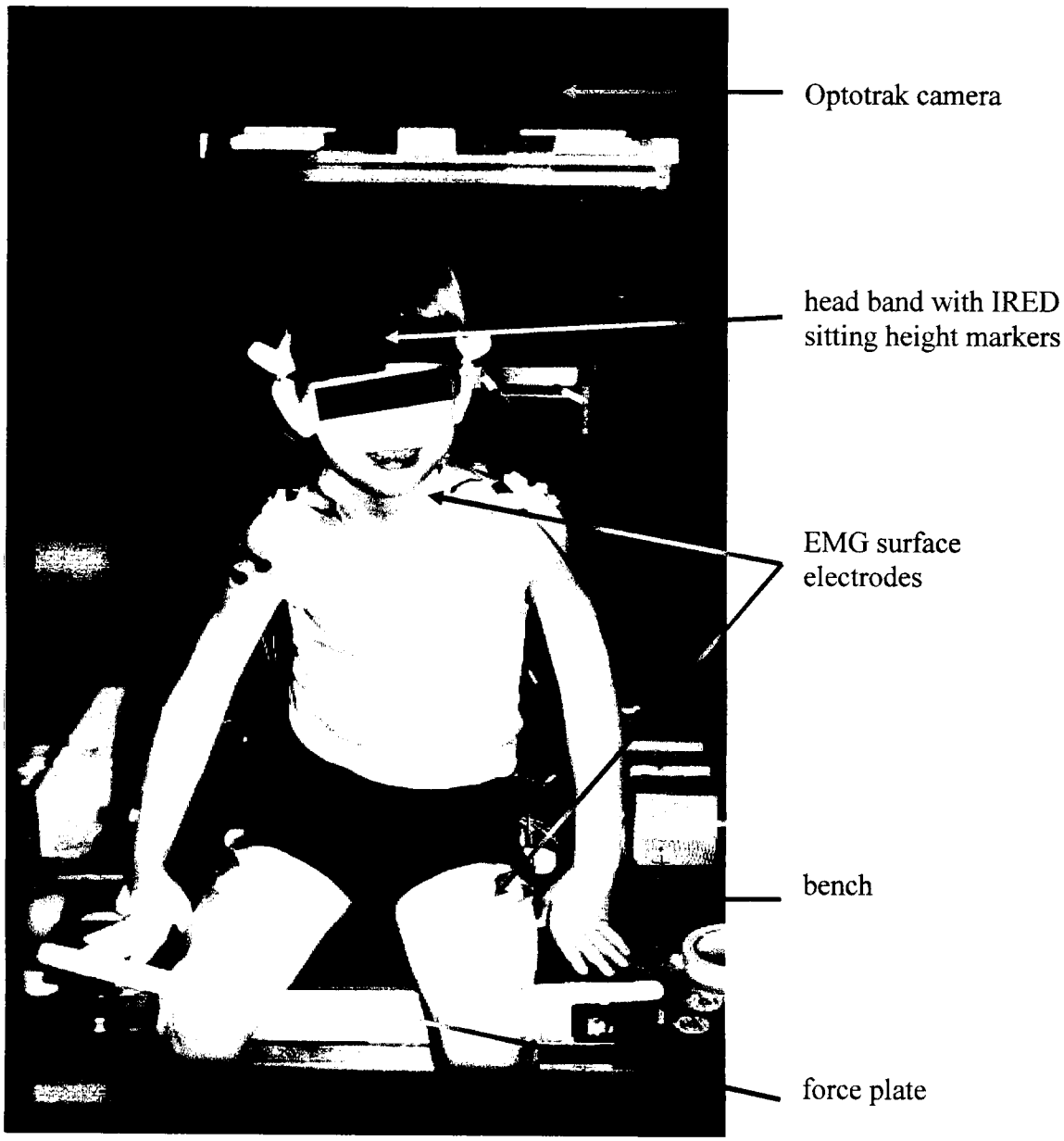
Glasgow Coma Scale

Aspects of Behaviour	Responses	Scores
Eye Opening	Spontaneous	4
	To speech	3
	To pain	2
	None	1
Best Verbal Response	Orientated	5
	Confused	4
	Inappropriate	3
	Incomprehensible	2
	None	1
Best Motor Response	Obey commands	5
	Localise to pain	4
	Flexion to pain	3
	Extension to pain	2
	None	1

From: Teasdale, G. & Jennett, B. (1974). Assessment of coma and impaired consciousness. Lancet, 2, 81-83.

Appendix C

Photograph of Data Collection Set-up



Appendix D

Electromyographic (EMG) Surface Electrode Placement

Muscle Group	Placement Description
Rectus Femoris	bisect girth of thigh, distal 3rd of thigh
Hamstrings	bisect girth of thigh, distal 3rd of thigh
Abdominals	3 cm lateral to the umbilicus
L1 Erector Spinae	3 cm lateral to the spinous processes, 3 spinous processes superior to the level of the iliac crests
C4 Erector Spinae	2 cm lateral to the spinous processes, midway between the base of the neck and T1 spine
Sternocleidomastoid	midpoint of muscle belly

Appendix E

Modified Level of Sitting Scale (Fife et al., 1991)

Test Conditions:

Child is in "sitting position" at the edge of a high mat or bench with feet unsupported.

Definition of "sitting position":

1. The child's hips and lower trunk can be flexed sufficiently so that the trunk (defined by a line joining T1 and sacrum) is inclined at least 60 degrees above the horizontal plane.
2. The child's head is either neutral with respect to the trunk or aligned vertically.
3. The position can be maintained for a minimum of 30 seconds with due regard for comfort and safety of the child.

Sitting Scale:

Level	Descriptor	Definition
0	Unplaceable	Child cannot be placed in sitting or cannot be held in sitting by one person
1	Supported from head downward	Child requires support of head, trunk, and pelvis to maintain sitting position

Appendix E continued

Modified Level of Sitting Scale (Fife et al., 1991)

Level	Descriptor	Definition
2	Supported from shoulders downward	Child requires maximal support from the shoulders downward to maintain sitting position
3	Supported from trunk downward	Child requires no more than moderate one-person support at mid-trunk level or lower
4	Maintains position, does not move	Child maintains position independently if he/she does not move limb or trunk
5	Leans, re-erects	Child, without using hands for support, can lean at least 20 degrees anterior to the vertical plane or 20 degrees to one side from midline; can recover balance/re-erect independently

Appendix F

Concurrent Motor Status of Two Children with TBI

	Time 1	Time 2
Mobility		
ti01	- uses supported seating and wheelchair for mobility - standing pivot transfer with moderate 2-person assist - stand with moderate 2-person assist - walk with moderate-maximal 2-person assist short distances	- uses wheelchair for distances - sit to stand with minimal 1-person assist - 'quiet' stand independently - walk 10 laps in parallel bars, holding onto bars 2-3 times each lap to maintain balance
ti03	- uses wheelchair and supported seating for mobility - sit to stand with maximal 1-person assist - stand with moderate-maximal 1-person assist - unable to walk	- uses wheelchair and seating for distances - stand up from floor independently - stand and throw 2-handed with moderate pelvic support - uses walker independently with pelvic support; walked 6-7 steps without walker with marked pelvic instability
Muscle Strength (manual muscle testing, 0 – 5)		
ti01	- hip and knee strength 3 to 4+ bilaterally	- hip and knee 4- to 5 bilaterally
ti03	- unable to test (child unable to perform resisted test)	- unable to test, general lower extremity weakness persists
Joint Range of Motion		
ti01	- 75°- 80° hip and knee flexion bilaterally	- full active hip flexion, knee flexion 100° bilaterally
ti03	- previous 20° left hip flexion contracture	- no limited lower extremity range noted
Extraneous Movement & Muscle tone		
ti01	- increased extensor tone of left extremities	- no abnormal muscle tone noted
ti03	- marked trunk and extremity ataxia and intentional tremor, right > left upper extremity	- trunk and extremity ataxia and tremor slightly decreased from Time 1

Appendix G

Pediatric Level of Consciousness Scale

School Age (5 Years and Older)

Level 5: No response to stimuli

Complete absence of observable change in behavior to visual, auditory or painful stimuli

Level 4: Gives generalized response to sensory stimuli

Gives generalized startle to loud sound

Responds to repeated auditory stimulation with increased or decreased activity

Gives generalized reflex response to painful stimuli

Level 3: Gives localized response to sensory stimuli

Blinks when strong light crosses field of vision

Follows moving object passed within visual field

Turns to or away from loud sound

Gives localized response to painful stimuli

Pediatric Level of Consciousness Scale

Level 2: Is responsive to environment

Follows simple verbal or gestured requests

Initiates purposeful activity

Actively participates in therapy program

Refuses to follow request by shaking head or saying "no"

Imitates examiner's gestures or facial expressions

Level 1: Oriented to time and place; is recording ongoing events

Can provide accurate, detailed information about self and present situation

Knows way to and from daily activities

Knows sequence of daily routine

Knows way around ward; recognizes own room

Cart find own bed; knows where personal belongings are kept

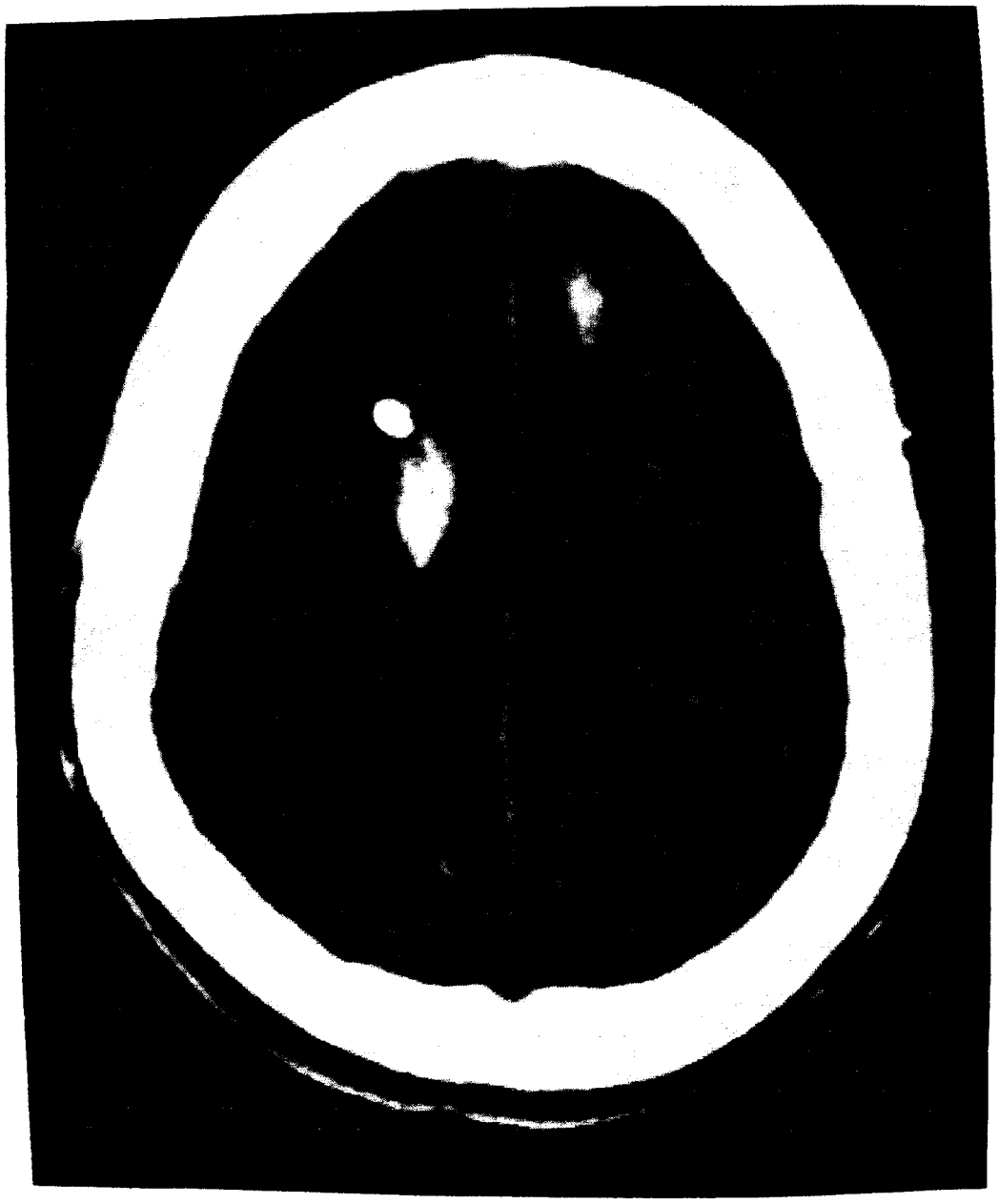
Is bowel and bladder trained

From: Sellars, C.W., & Vegter, C. R. (1993). Pediatric brain injury: a practical resource. Tuscon, AZ: Communication Skill Builders

Appendix H

Imaging Scans of Two Children with TBI

Coronal CT Scan of ti01 (15 year old male). White regions indicate injured areas.



Appendix H continued

Imaging Scans of Two Children with TBI

Sagittal and coronal MRI Scans of ti03 (6 year old male). White regions indicate injured areas.