Left Ventricular Structure and Function in Children with Developmental Coordination Disorder

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Abstract

Developmental coordination disorder (p-DCD) is a neuro-developmental disorder featuring impairment in developing motor coordination. This study examined left ventricular mass (LVM) in children with p-DCD (n=63) and controls (n=63). LVM was measured using echocardiography. Body composition was determined using BOD POD and peak oxygen uptake (peak VO₂) was measured by a progressive exercise test. Height, weight and blood pressure were also measured. LVM was not significantly elevated in p-DCD compared to controls. Peak VO₂ was lower and SBP, BMI, HR, and BF(%) were significantly higher in p-DCD. They also demonstrated elevated stroke volume (SV), cardiac output (CO), end-diastolic volume, and ventricular diameter in diastole. In regression analyses, p-DCD was a significant predictor of SV and CO after accounting for height, FFM, VO₂FFM, and sex. These differences in children with p-DCD indicate obesity related changes in the left ventricle and may represent early stages of developing hypertrophy of the left ventricle.
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### Abbreviations

<table>
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<th>Abbreviation</th>
<th>Definition</th>
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<tbody>
<tr>
<td>ADL</td>
<td>Activities of daily living</td>
</tr>
<tr>
<td>ASE</td>
<td>American Society of Echocardiography</td>
</tr>
<tr>
<td>BF(%)</td>
<td>Percentage body fat</td>
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<tr>
<td>BMI</td>
<td>Body mass index</td>
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<tr>
<td>BOTMP</td>
<td>Bruininks-Oseretski test of motor proficiency</td>
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<tr>
<td>BP</td>
<td>Blood pressure</td>
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<tr>
<td>BSA</td>
<td>Body surface area</td>
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<tr>
<td>CARDIA</td>
<td>Coronary artery risk development in young adults</td>
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<tr>
<td>CHS</td>
<td>Cardiovascular health study</td>
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<tr>
<td>CO</td>
<td>Cardiac output</td>
</tr>
<tr>
<td>CSA</td>
<td>Cross-sectional area</td>
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<tr>
<td>CVD</td>
<td>Cardiovascular disease</td>
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<tr>
<td>DBP</td>
<td>Diastolic blood pressure</td>
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<tr>
<td>DCD</td>
<td>Developmental coordination disorder</td>
</tr>
<tr>
<td>DEXA</td>
<td>Dual x-ray absorptiometry</td>
</tr>
<tr>
<td>DSBN</td>
<td>District school board of Niagara</td>
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<tr>
<td>DSM-IV</td>
<td>Diagnostic and statistical manual</td>
</tr>
<tr>
<td>EDV</td>
<td>End-diastolic volume</td>
</tr>
<tr>
<td>EF</td>
<td>Ejection fraction</td>
</tr>
<tr>
<td>ESV</td>
<td>End-systolic volume</td>
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<tr>
<td>FFM</td>
<td>Fat free mass</td>
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<tr>
<td>FS</td>
<td>Fractional shortening</td>
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<tr>
<td>g</td>
<td>grams</td>
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<tr>
<td>HR</td>
<td>Heart rate</td>
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<tr>
<td>IVS</td>
<td>Interventricular septum</td>
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<tr>
<td>Kg</td>
<td>Kilograms</td>
</tr>
<tr>
<td>K-BIT</td>
<td>Kaufman Brief Intelligence Test</td>
</tr>
<tr>
<td>LVDd</td>
<td>Left ventricular diameter in diastole</td>
</tr>
<tr>
<td>LVM</td>
<td>Left ventricular mass</td>
</tr>
<tr>
<td>LVM/Ht&lt;sup&gt;2.7&lt;/sup&gt;</td>
<td>Left ventricular mass normalized to height&lt;sup&gt;2.7&lt;/sup&gt;</td>
</tr>
<tr>
<td>LVH</td>
<td>Left ventricular hypertrophy</td>
</tr>
<tr>
<td>M-ABC</td>
<td>Movement assessment batter for children</td>
</tr>
<tr>
<td>p-DCD</td>
<td>Probable developmental coordination disorder</td>
</tr>
<tr>
<td>peak VO&lt;sub&gt;2&lt;/sub&gt;</td>
<td>Maximal aerobic fitness</td>
</tr>
<tr>
<td>PHAST</td>
<td>Physical health activity study team</td>
</tr>
<tr>
<td>PW</td>
<td>Posterior wall thickness</td>
</tr>
<tr>
<td>SBP</td>
<td>Systolic blood pressure</td>
</tr>
<tr>
<td>SV</td>
<td>Stroke volume</td>
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<tr>
<td>RPE</td>
<td>Rating of perceived exertion</td>
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Chapter 1: Introduction

1.1 Preamble

Developmental coordination disorder (DCD) is a neuro-developmental disorder featuring marked impairment in motor coordination development. Motor abilities in children with DCD are well below that expected for age and intelligence, interfering with social and academic functioning in the absence of mental retardation. The prevalence of DCD using stringent cut-off criteria and severe cases has been shown to be 1.8% at a mean age of 7.5 years, with a 1.9:1 male to female ratio. When considering probable cases of DCD (M-ABC 5th to 15th percentile) prevalence has been reported to range between 4.9% and 6%. 

Children with DCD lack the fine and gross motor skills to perform activities of daily living such as tying shoelaces, buttoning shirts, drawing, and playing. As a result of their poor motor skills, children with DCD are excluded from participating in recreational activities or often exclude themselves due to fear of ridicule. This exclusion leads to reduced movement experiences and further impaired motor development. It has been shown that children with DCD participate less in free play and organized activities than their typically developing peers. The pathway linking reduced physical activity participation and DCD is hypothesized to be reduced generalized self-efficacy towards physical activity. This reduction in physical activity patterns can have a negative impact on health, as regular participation in physical activity is important for the development of many aspects of physical fitness.
Studies have reported that children with DCD demonstrate reduced muscular strength and endurance, flexibility, and cardio-respiratory fitness, as well as an increased risk of obesity.\textsuperscript{10-13} Faught et al. (2005)\textsuperscript{14} found that children with DCD were at a higher risk of developing cardiovascular disease (CVD) risk factors such as increased percentage body fat and reduced cardio-respiratory fitness. In particular, obesity is a major risk factor for hypertension, type-2 diabetes and overall CVD.\textsuperscript{15} However, data assessing the health of the cardiovascular system in individuals with DCD is limited,\textsuperscript{11} particularly in children.

Left ventricular hypertrophy (LVH) is a physiological adaptation which develops when there is a chronic increase in work load imposed on the heart\textsuperscript{16} and occurs in an attempt to normalize wall stress and maintain systolic function.\textsuperscript{17} Left ventricular hypertrophy has been shown to be a strong, independent predictor of cardiovascular morbidity in adults.\textsuperscript{18, 19} As well, elevated left ventricular mass (LVM), in the absence of hypertrophy, has also been shown to be an independent predictor of cardiovascular morbidity and mortality.\textsuperscript{20, 21} Left ventricle mass has been assessed in children and adolescents, demonstrating increased risk of LVH in overweight/obese and hypertensive children.\textsuperscript{22-25} The purpose of this study was to examine the risk of CVD in children with DCD by assessing left ventricular structure and function using non-invasive ultrasound techniques to estimate LVM.
1.2 Rationale

Although it has been well documented that children with DCD are at an increased risk of developing CVD risk factors such as obesity, reduced cardio-respiratory fitness, there are limited data using metabolic indices and laboratory measures assessing cardiovascular health of individuals with DCD.

1.3 Objective

The purpose of this investigation was to determine whether children diagnosed with probable DCD (p-DCD) demonstrate elevated LVM compared to controls, and whether they demonstrate differences in cardiac dimensions and systolic function.

1.4 Hypothesis

We hypothesized that children with DCD would demonstrate elevated LVM and cardiac dimensions compared to age, gender, and school matched controls. Also, that there would not be a difference in left ventricular function between groups.
Chapter 2: Review of Literature

2.1 Developmental Coordination Disorder

Awareness of different levels of motor performance has been identified as early as the beginning of the 20th century. The term developmental dyspraxia has been used by neurologists, pediatricians and neuropsychologists in an attempt to describe children with coordination disorders. Several other terms such as clumsy, clumsy child syndrome and “physically awkward” have been used to describe and incorrectly label children with coordination difficulties. This is due, in part, to the fact that there was a lack of an agreed upon definition. At a consensus meeting in 1994, a group of researchers agreed on the term developmental coordination disorder (DCD) as described by the American Psychiatric Association in the Diagnostic and Statistical Manual (DSM-IV 1994) to distinguish this term from apraxia or developmental dyspraxia. This section will discuss the characteristics of this condition.

2.1.1 Background of DCD

According to the DSM-IV (1994), DCD is a motor skill disorder which features marked impairment in the development of motor coordination. Motor abilities in children with DCD are well below that expected for age and intelligence, interfering with social and academic functioning in the absence of mental retardation. Developmental coordination disorder is diagnosed based on the following four criteria:

A. Performance in daily activities that require motor coordination is substantially below that expected given the person’s chronological age and measured intelligence. This may be manifested by marked delays in achieving motor milestones (e.g., walking, crawling, and sitting), dropping things, "clumsiness," poor performance in sports, or poor handwriting.
B. The disturbance in Criterion A significantly interferes with academic achievement or activities of daily living.

C. The disturbance is not due to a general medical condition (e.g., cerebral palsy or muscular dystrophy) and does not meet criteria for a pervasive developmental disorder.

D. If mental retardation is present, the motor difficulties are in excess of those usually associated with it.¹

Children with DCD are continually incorrectly labelled as clumsy, awkward, and lazy because they tend to have poor sport skills, drop objects, and have increased general clumsiness. Children with DCD differ in regards to the severity and type of motor impairment and performance in other domains such as intellect, education, and behaviour.²⁸ They are often noted by parents and teachers to have problems with daily tasks such as dressing themselves, tripping when they run, spilling things frequently, and to have messy handwriting and drawing.²⁹ Manifestations of DCD vary with age; however, children often lack fine and gross motor skills to perform activities of daily living. Younger children tend to have delayed developmental milestones such as walking, crawling, tying shoelaces, and buttoning shirts.³ Older children have difficulties with writing and drawing, playing, dressing, and speech.³ Recently, Wang et al. (2009)³⁰ assessed the functional performance of daily activities at home and at school in children with DCD compared to controls. This study reported that children with motor coordination problems exhibited significantly poorer functional performance of daily activities such as personal living skills (eating and drinking, grooming, dressing etc.),
gross motor skills (walking, running, and playing activities), and fine motor skills
(manipulation, painting, and using scissors) compared to typically developing children.
These findings support the fact that children with motor coordination problems
encounter difficulties in academic and daily life, suggesting a need for parents, teachers,
and clinicians to identify deficits in motor performance.

According to the DSM-IV (1994)\(^1\), the prevalence of DCD ranges between 5% -6%
of all school aged children, and may approach up to 10% for those with milder forms of
the condition.\(^2\)\(^8\) Several studies have reported the prevalence of DCD to range anywhere
from 5%-10% in children,\(^7\)\(^,\)\(^14\) with one study reporting up to 19%.\(^3\)\(^1\) The higher
prevalence rates observed in previous studies may reflect moderate cases of DCD (<15\(^{th}\)
percentile) rather than more severe cases (<5\(^{th}\) percentile) who have functional
impairment in their activities of daily living (ADL).\(^2\) A study conducted by Lingam et al.
(2009)\(^2\) aimed to determine the prevalence of DCD in children 7-8 years old from the
United Kingdom and was the first to use strict inclusion and exclusion criterion from the
DSM-IV to define DCD. The Movement Assessment Battery for Children (M-ABC) was
used as the coordination test and below the 5\(^{th}\) percentile was used to describe severe
motor difficulties, while probable motor difficulty was identified as scores between the
5\(^{th}\) and 15\(^{th}\) percentile. Academic achievement was measured by handwriting using a
literacy test and a score below level 2 in their writing task was considered as having
significant difficulty in handwriting. A parent-completed questionnaire was used to
measure ADL, which included questions on key areas in which children with DCD
struggle such as, self-care skills, playing skills, drawing etc. A stringent cut-off of at or
below the 10th percentile was used to define significant difficulties in ADL. An IQ test was used to identify mental retardation and those with a score of <70 were excluded from the study. As well, children with known medical conditions were excluded from the study. A total of 123 children met the full criteria for DCD. The prevalence was determined to be 1.8% at a mean age of 7.5 years, with a gender ratio of 1.9:1 males to females. When considering cases of probable DCD (pDCD; i.e. impairment score between 5th and 15th percentile) and an ADL score between the 10th and 15th percentile or a failed writing test, the prevalence rate was 4.9% for 7 to 8 year olds. The results reported from this study more accurately depict the prevalence rate of DCD in school aged children as it uses stringent criteria for identifying DCD.2

2.1.2 Diagnosing DCD

Currently, there is no widely accepted gold standard of assessment for identifying DCD due to its heterogeneous nature32, resulting in the use of several assessment tools to identify children with coordination problems. The two most commonly administered tests for children above 4 years of age are the Bruininks-Oseretsky Test of Motor Proficiency (BOTMP) and the M-ABC. The BOTMP-SF is a short form of the BOTMP and has been previously used in several school based studies.14,33 The short form has been validated against the full scale with inter-correlations of 0.90 and 0.91 reported for children aged 8 to 14.34 This is a more suitable test for large school based studies when the M-ABC test is not feasible, as it does not require clinical judgement by research assistants.35 The BOTMP-SF has been reported as the most frequently used field-based test, however, the reliability and validity have been
questioned. Dewey et al. (2001) report inconsistencies between the BOTMP and the M-ABC in identifying children with motor impairments. One of the greatest concerns regarding the use of the BOTMP-SF is that it does not measure quality of movement, but rather the ability to perform an activity. However, the BOTMP-SF has been reported as a reasonable alternative when the M-ABC is not feasible.

The M-ABC was designed to test motor performance of children between 4 and 12 years. It is one of the most widely used assessment tools by occupational therapists, physiotherapists, and education professionals for identifying motor impairment. The M-ABC was developed using children from the United Kingdom, Canada, and the United States and has shown to be a reliable and valid measure clearly distinguishing children with motor problems. The M-ABC2 is a revision of the M-ABC and is composed of two parts, a performance task and checklist. The performance task requires the child to complete a series of fine and gross motor tasks grouped into three categories: Manual Dexterity, Aiming and Catching, and Balance. The performance-based portion is developed from the Test of Motor Impairment created by Stott (1972) and revised by Henderson in 1984. The M-ABC2 also includes a checklist to provide information about a child’s motor skill difficulties as assessed by their parents.

2.1.3 Consequences of DCD

Children with DCD experience difficulties performing basic movement skills necessary for participating in sports or recreational activities. Because the child has poor motor skills, they tend to either exclude themselves from participating or are excluded by peers. This results in reduced movement experiences and impaired motor
development. Furthermore, these children demonstrate reduced physical activity levels by avoiding participation in free play activities and organized sports.\textsuperscript{4, 7} Several studies demonstrate that children with low motor competence in childhood do not grow out of the disorder in adolescence,\textsuperscript{5, 39} indicating that this disorder extends far beyond being a clumsy child. This can have serious health implications as childhood physical activity levels are important for developing physical fitness.

2.1.3.1 Physical Activity and DCD

Recent research in children with low motor proficiency suggest reduced levels of physical activity,\textsuperscript{5, 6, 40} as well as participating less in free play and organized activities.\textsuperscript{7, 8} It is hypothesized that the pathway linking DCD to reduced physical activity can be explained by reduced generalized self-efficacy.\textsuperscript{4, 8} Cairney et al. (2005)\textsuperscript{7} examined the influence of generalized self-efficacy on the association between DCD and physical activity (adequacy in, and preference for physical activity, and enjoyment of physical education). Children with DCD reported less participation in free play and organized activities and lower adequacy in, and preference toward physical activity, as well as less enjoyment of physical education classes. The results also indicated that the reduced physical activity observed in children with DCD is mediated through a decrease in generalized self-efficacy.\textsuperscript{7} A similar study by Cairney and colleagues (2005)\textsuperscript{41} examined the effects of generalized self-efficacy on physical activity patterns and whether gender played a role. Results from this study support previous work suggesting that children with DCD report lower levels of generalized self-efficacy and participate less in organized activities and free play. Girls with DCD reported the lowest levels of
generalized self-efficacy, participation in organized activities and free play compared to boys with DCD, and boys and girls without DCD.\textsuperscript{41}

Previous work (Klentrou, 2003)\textsuperscript{42} has demonstrated that children who enjoy physical education are more likely to be physically fit, and gender differences in participation of physical activity can be explained by lower enjoyment in girls than in boys. Children with DCD report lower levels of enjoyment in physical education class and perceived adequacy towards physical activity has been shown to account for the greatest proportion\textsuperscript{6} because of fear of embarrassment and ridicule.\textsuperscript{43} In order to test the affect of DCD on physical activity over time, Cairney and colleagues (2009)\textsuperscript{44} tested the affect of DCD on physical fitness and whether it widens or diminishes over time. The study population included children enrolled in Grade 4 in 2004 and collected over 3 years, ending in Grade 6. Children with DCD reported overall lower levels of participation compared to their peers. This remained constant over time in organized play, but increased over time with free play. Furthermore, among males, free play increased over time but decreased for females. Overall, there is consistency in the literature demonstrating that children with DCD are less likely to be physically active compared to their non-DCD counterparts as a result of lower generalized self-efficacy toward physical activity, which persists over time.

2.1.3.2 Physical Fitness, Body Composition, and DCD

Participation in regular physical activity is important for the development of many aspects of physical fitness\textsuperscript{9} and a reduction in activity levels may have a negative impact on health. Health related physical fitness is associated with disease prevention
and includes cardio-respiratory endurance, muscle strength and endurance, flexibility, and body composition. Children with DCD are at risk for low levels of physical fitness because most children develop fitness through their daily activities while performing movements such as running, walking, and climbing. Consequently, children with DCD have been associated with factors that increase risk of developing CVD. Hypoactivity among children with DCD can contribute to higher levels of body fat. The association between body composition and DCD has been reported in children with DCD as having a higher body mass index (BMI) and a greater likelihood to be overweight and obese than children without DCD.

Hands and Larkin (2006) found BMI to be significantly higher in children with motor learning difficulties (17.5 kg/m²) compared to controls (15.5 kg/m²), which was attributed to weight. There were eight children in the motor learning difficulty group with a BMI greater than 20, two of which were above 25. The control group had three children with a BMI above 20, none exceeding 22. Cantell et al. (2008) found a significant association between motor competence and being overweight and obese. In total, 52% of the low motor competence group were overweight or obese compared to 30% in the high motor competence group. When stratified for gender, this was only true for females. Furthermore, a study by Faught et al. (2005) linked motor impairment to factors associated with increased risk of CVD. Specifically, it was determined that p-DCD was directly linked to an increase in body fat percentage and a decrease in cardio-respiratory fitness with physical activity being a significant mediator. In one of the first longitudinal studies examining body composition in children with probable DCD, Cairney
and colleagues (2010) found that BMI and waist circumference were increased in children with p-DCD at baseline and after a two year period. The children were examined at three time points over a two year period from grades 4 to 6. It was determined that over time the differences in BMI and waist circumference between the two groups increased. Moreover, it was established that children with p-DCD had an odds ratio of 3.44 of becoming overweight over the study period, and an odds ratio of 4.00 of becoming obese over the study period. These results are different from Hands (2008) who found no between group differences in body composition in 5 to 7 year olds during a five year follow-up. Age differences and sample size may account for the differences in findings between studies.

More than a few studies have also demonstrated that low motor proficiency is associated with a significantly poorer performance on cardio-respiratory fitness, along with several other components of physical fitness such as muscular strength and endurance. A negative relationship between motor proficiency and fitness has been observed in various age groups. Hands and Larkin (2006) found that children aged 5 to 8 years old with motor learning difficulties performed worse on fitness components such as sit and reach, sit-ups, standing broad jump, and the multi-stage shuttle run compared to controls. Similarly, Cairney and colleagues (2007) found that children with p-DCD aged 9-14 years old had a lower predicted peak VO\textsubscript{2} than those without the disorder, which is consistent with several other studies also using the 20-meter shuttle run. Moreover, results from this study indicated that 61% of children with p-DCD were in the bottom 20\textsuperscript{th} percentile for VO\textsubscript{2}, increasing their risk of developing CVD risk
factors.\textsuperscript{47} However, it should be noted that there are limitations to using field based
tests to determine fitness in children with DCD. A study by Cairney and colleagues
(2006)\textsuperscript{49} found that a significant portion of the reduced fitness observed in children with
p-DCD could be attributed to differences in perceived adequacy, indicating a significant
psychological aspect to the differences seen in fitness.\textsuperscript{49}

Nevertheless, differences in fitness levels are supported by Wu et al. (2009)\textsuperscript{50}
after conducting a lab-based peak VO\textsubscript{2} test using the Bruce treadmill protocol to
determine cardio-respiratory fitness in children with and without DCD. The results from
this study demonstrated that children with DCD had significantly lower levels of cardio-
respiratory fitness (47.6 ml/kg/min vs. 39.7 ml/kg/min) than typically developing
children. Also of interest to note, no difference in max heart rate (HR) was evident,
indicating that fitness levels differed even though they appeared to be working just as
hard as typically developing children (196.8 beats/min vs. 196.6 beats/min).\textsuperscript{50}

A growing concern for children with DCD is that the differences observed in
fitness will persist and possibly worsen into adolescence and adulthood. Barnett et al.
(2008)\textsuperscript{13} suggested that childhood motor skill proficiency may be a determinant for
adolescent fitness. A multistage fitness test to determine cardio-respiratory fitness was
performed in adolescents who were previously tested for motor skill proficiency using
object control (kicking, throwing, and catching) and locomotor skills (hopping) 6 years
earlier. Results from this study revealed that 26% of the variation in adolescent cardio-
respiratory fitness was accounted for by childhood object control proficiency. Several
limitations to this study exist, including the lack of control for weight and testing both motor skill proficiency and cardio-respiratory fitness at baseline and follow-up. A recent study by Haga and colleagues (2009) examined how physical fitness develops over time in children with low and high motor competence. Motor competence was assessed using the M-ABC test and several aspects of physical fitness were tested such as jumping, throwing, climbing, and running. Results from this study revealed a significant difference in physical fitness measures between the low and high motor competence group. It is important to note that both groups scored significantly higher on physical fitness measure after 32 months; however, the low motor competent group remained significantly lower. In order to test the affect of DCD on cardio-respiratory fitness over time, Cairney and colleagues (2010) examined a cohort of children enrolled in Grade 4 at baseline, and followed them over a 2 year period, ending in Grade 6. Children with DCD reported lower levels of predicted peak $\text{VO}_2$ from the Léger 20-m shuttle run at baseline, which persisted over time. Furthermore, it was found that predicted peak $\text{VO}_2$ declined at a much greater rate in the DCD group compared to typically developing children. These results confirm that differences in cardio-respiratory fitness between children with and without DCD persist over time, highlighting a concern for poor cardiovascular health. A study by Hands (2008) also demonstrates a significant difference in fitness levels in children with DCD and typically developing children and this difference persisted over time.
2.1.3.3 Cardiovascular Risk and DCD

Developmental coordination disorder has been linked to several CVD risk factors such as increased prevalence of overweight and obesity,\textsuperscript{3,4,11} as well as reduced physical fitness and/or cardio-respiratory fitness.\textsuperscript{10,12,47,50} Data is limited in regards to laboratory measures of cardiovascular health in relation to motor competence. One study by Cantell and colleagues (2008)\textsuperscript{11} assessed physical activity, fitness, and health indices of children, adolescents, and adults with low motor competence compared to those with high motor competence. The metabolic indices associated with negative health showed that individuals with high and low motor competence are different. Those with low motor competence tend to have lower HDL and higher triglyceride levels.\textsuperscript{11} As well, consistent with previous studies, BMI was significantly higher in the low motor competence group. This result remained consistent after using a more accurate measure of body composition (DEXA scan), indicating that adults with low motor competence had a higher percentage of trunk fat compared to the high motor competence group.\textsuperscript{11} Results from this study demonstrate that in children with low motor competence, metabolic indices related to negative health such as obesity and CVD are evident.

2.2.0 Left Ventricular Function

With respect to the left ventricle, blood flows through the heart such that oxygen-rich blood returns to the left atrium via the pulmonary veins and then passes through the mitral valve into the left ventricle. Contraction of the left ventricle pumps oxygenated blood across the aortic valve where it is then distributed throughout the
body. During the contraction phase (systole) the pressure in the left ventricle begins to build up and communication between the atrium and ventricle is open. As ventricular pressure increases and exceeds that of the atrium, the mitral valve abruptly closes. During this phase the volume in the ventricle is fixed (isovolumetric contraction) because the aortic and mitral valves are shut. At this time pressure builds in the ventricle until exceeding that of the aorta, whereupon the aortic valve opens resulting in rapid ejection. During physiological diastole, the relaxation phase of the cardiac cycle, left ventricular and aortic pressures start to decline. The aortic and mitral valves are shut at this point, and volume in the ventricle remains unchanged (isovolumetric relaxation). This relaxation continues until ventricular pressure falls below that of the atrium, whereby the mitral valve opens and ventricular filling occurs. The early phase of filling is passive as left ventricular pressure falls below atrium pressure. The ventricle fills quite rapidly and soon pressure equalizes between the ventricle and atrium. Further filling is a result of left atrium contraction. The cycle restarts as the next phase of isovolumetric contraction commences.

Preload is the force acting to stretch the left ventricular fibres at the end of diastole (ventricular filling) and determines maximal resting length of sarcomeres. At the end of systole, an increased afterload will communicate itself to the ventricle as increasing wall stress. Afterload can be approximated using arterial blood pressure (BP). An increase in afterload increases the intraventricular pressure needed to open the aortic valve, and is the pressure in which the myocardium contracts against during the ejection phase.
2.2.1 Left Ventricular Hypertrophy

When the heart faces a burden of hemodynamic overload, two different compensatory mechanisms may result: (1) use of the Frank Starling mechanism to increase cross-bridge formation; and/or an (2) increase in muscle mass to bear the extra load. The Frank-Starling mechanism intrinsically regulates myocardial performance such that an increase in end-diastolic ventricular filling volume results in increased diastolic fibre length, corresponding to a more forceful contraction. However, this mechanism is limited in its scope as it greatly increases LV pressure, causing chamber dilation and a resultant increase in wall stress. Several studies have shown that enhanced performance of a chronically enlarged left ventricle is mediated by a normal performance of each unit of the enlarged chamber (increased cross-sectional area), as opposed to increasing contractility. A study by Lee et al. (1985) examined adaptations of the left ventricle in response to chronic volume overload in dogs. After 8 days of volume overload there was a significant increase in wall stress, end-diastolic diameter, and circumferential shortening, while wall thickness decreased and cross-sectional area (CSA) remained unchanged. After several weeks end-diastolic diameter progressively increased, wall thickness returned to normal, CSA increased and wall stress was reduced while circumferential shortening remained unchanged. These findings suggest that the Frank-Starling mechanism is utilized early on, with no apparent influence after chamber dilation. Thus, an increase in heart size assumes an essential role in hemodynamic overload.
Left ventricular hypertrophy (LVH) occurs in an attempt to normalize left ventricular wall stress and maintain systolic function. Normalization of systolic stress (afterload) helps to maintain ejection fraction even during high systolic pressure. According to the law of LaPlace:

\[
Wall\ stress = \frac{pressure \times radius}{2 \times wall\ thickness}
\]

Chamber dilation increases wall stress, which increases afterload. As well, at any given radius the greater the pressure developed by the left ventricle will in turn result in greater wall stress. In accordance to the law of LaPlace, the increase in wall thickness balances the increase in pressure allowing wall stress to remain unchanged.

The pattern of cardiac hypertrophy is dependent on the type of stress encountered. Grossman et al. (1975) demonstrated that LVH develops in a pattern which is unique to its stress. In response to pressure overload such as hypertension, parallel addition of sarcomeres increases myocyte width, in turn, increasing wall thickness. This type of remodelling is referred to as concentric hypertrophy and is characterized by an increase in the ratio of wall thickness to chamber size. In contrast, volume overload causes myocyte lengthening by sarcomeres replicating in series with an increase in chamber volume. This type of remodelling is known as eccentric hypertrophy and is characterized by no change in the ratio of wall thickness to chamber dimension. Chronic hypertrophy may be deleterious as it increases the risk of premature death. The increase in LVM has been shown to be an independent risk factor
for CVD morbidity and mortality, even at levels that are not considered to be hypertrophic.\textsuperscript{18}

The functional performance, or efficiency, and oxygen delivering capacity of the hypertrophied left ventricle, along with its reversibility once the overload is removed, have led to the classification of either pathological or physiological hypertrophy. Pathological hypertrophy as a result of increased pressure (hypertension) or volume (obesity) overload results when the hypertrophic responses do not successfully normalize wall stress or meet oxygen demands, in turn resulting in pathological chamber features.\textsuperscript{58} Pathological concentric ventricular morphology is characterized by parallel addition of new myofibrils with an associated increase in fibrosis and myocyte necrosis.\textsuperscript{58} Furthermore, an increase in diastolic stiffness and decreased contractility characterizes ventricular mechanics in pathological concentric hypertrophy. In pathological eccentric hypertrophy sarcomeres are added in series, resulting in wall thinning and elongation, frequently with myocyte necrosis.\textsuperscript{58} This is often associated with slippage of myocytes, decreasing contractility.\textsuperscript{54} Additionally, the occurrence of pathological hypertrophy is associated with abnormal ventricular function to the extent of which changes cannot be reversed.\textsuperscript{58}

Exercise induces a physiological hypertrophic adaptation of the left ventricle, allowing the heart to meet increased myocardial demands while still maintaining normal function.\textsuperscript{58, 59, 60} Physiological remodelling of the left ventricle can occur concentrically to pressure overload (strength training, anaerobic exercise), or eccentrically to volume overload (aerobic exercise).\textsuperscript{58} There are several important differences that distinguish
pathological and physiological remodelling of the left ventricle. Physiological concentric remodelling is accompanied by increased capillary density in absence of fibrosis and necrosis, while eccentric hypertrophy is accompanied by chamber volume enlargement in absence of dilation and wall thinning.\textsuperscript{58,61} This physiological increase in LVM is often seen in highly trained adult athletes as a result of repeated bouts of exercise stimuli\textsuperscript{62} and is identified as ‘athletes heart’.\textsuperscript{59,62-65}

2.2.1.1 Prognostic implications of LVH and LVM

After initial compensatory cardiac hypertrophy due to sustained overload, hypertrophy can further degenerate into inappropriate LVM; increasing risk of myocardial heart failure.\textsuperscript{52} Investigators of the Framingham Heart Study have shown that an increase in left ventricular cavity size is associated with adverse outcomes, including heart failure.\textsuperscript{66} This is primarily the result of continued ventricular dilation, increased wall stress and pressure, without a compensatory increase in wall thickness and a decrease in contractility. In most patients with hypertension, systolic performance appears normal and even supranormal,\textsuperscript{67} however, this may be a result of geometric changes in the left ventricle.\textsuperscript{68,69} Deterioration of the hypertrophied ventricle is evidenced by an inverse relationship between myocardial contractility (midwall fractional shortening) and LVM, in the presence of normal ejection fraction.\textsuperscript{68,70} Geometric changes in the left ventricle are associated with abnormal midwall shortening that is not apparent when ejection fraction is used as a measure of systolic function.\textsuperscript{69}

The ability to measure LVM by echocardiography has led to the important understanding of the contribution of increased LVM to the pathogenesis of
cardiovascular morbidity and mortality. Left ventricular hypertrophy is categorized as the upper end of "normalized" LVM.\textsuperscript{71} Previous studies have reported an increased risk for cardiovascular morbidity and mortality, all-cause mortality, heart failure and sudden cardiac death in subjects with echocardiographically determined LVH independent of other known risk factors.\textsuperscript{18, 19, 66, 72-75}

The definition of hypertrophy in various studies has been plagued by the method in which to index LVM in order to define hypertrophy. In the Framingham Heart Study, hypertrophy was defined by mean values $\pm$ 2 SD and corresponded to 131 and 100 g/m$^2$ in men and women respectively for BSA.\textsuperscript{71} Corresponding values for LVM/height were 143 and 102 g/m in men and women respectively. The partition values reported by de Simone et al.\textsuperscript{(1995)}\textsuperscript{76} for the 97.5 percentile of LVM/BSA, LVM/height, and LVM/height$^{2.7}$ were 117 g/m$^2$, 126 g/m, and 50 g/m$^{2.7}$ for men and 104 g/m$^2$, 105 g/m, and 47 g/m$^{2.7}$ for women respectively. The non-gender specific upper limit of 51 g/m$^{2.7}$ has also been suggested.\textsuperscript{77} In addition, the partition value of 125 g/m$^2$ for both men and women has been used for mass indexed by BSA.\textsuperscript{78} Liao et al. (1997)\textsuperscript{79} aimed to determine the most appropriate method of defining LVH to predict mortality risk in adults with and without CVD. Indexes of LVM that were compared in this study include height, height$^{2.7}$, and BSA using the partition values previously mentioned. Findings from this study indicate that LVH defined by the various methods of indexation similarly predict mortality risk in both patients with and without CVD. The prevalence of LVH was lower for LVM/BSA than LVM/height$^{2.7}$ in both patients with and without CVD, indicating that indexing to BSA may erroneously categorize obesity related LVH as normal.\textsuperscript{79}
Despite different partition values of LVH, mortality risk was significantly increased in all patients, which may indicate that elevated indexed LVM is an important predictor of morbidity and mortality.

There is evidence that echocardiographically determined elevated LVM, in the absence of hypertrophy, is also associated with cardiovascular morbidity and mortality and sudden cardiac death. In a study of hypertensive adults split into four quintiles of LVM below the limits of LVH, those in the upper quintile for LVM were at a 4-fold increase in risk for CVD compared to the lowest quintile. Previously in the Framingham Heart Study, Levy et al. (1990) also found that LVM was significantly associated with CVD, deaths from CVD, and all-cause mortality in both males and females after adjusting for age, diastolic BP, pulse pressure, antihypertensive treatment, cigarettes smoked per day, diabetes, BMI, high/low density lipoprotein ratio, and electrocardiographically determined LVH. The relative risk of CVD for men and women were 1.49 (95% CI, 1.20 to 1.85) and 1.57 (95% CI, 1.20 to 2.04) for each increment of 50g/m in LVM respectively. The corresponding values for death from CVD were 1.73 (95% CI, 1.19 to 2.52) in men and 2.12 (95% CI, 1.28 to 3.49) in women. The relative risk of death from all causes was 1.49 (95% CI, 1.14 to 1.94) in men and 2.01 (95% CI 1.44 to 2.81) in women.

The importance of ventricular remodelling and cardiovascular risk has also been investigated. In the Multi-Ethnic Study of Atherosclerosis study, concentric remodelling was associated with increased risk of coronary heart disease and stroke, while body-size adjusted LVM alone predicted heart failure. Conversely, knowledge of geometric remodelling provided little additional prognostic information beyond LVM
and traditional CVD risk factors in the Framingham Heart Study. These findings provide insight into the importance of elevated LVM and its effect on cardiovascular morbidity and mortality. The mechanism linking an increase in LVM to morbidity and mortality remains elusive, however, it is hypothesized that an increase in LVM increases oxygen consumption while reducing coronary blood flow, enhancing arrhythmias, and has been found to be associated with increased atherosclerotic lesions.

2.2.1.2 Prognosis of LVM in children

Prevalence of abnormal LVM and severe LVH has been established in children and estimation of cardiovascular risk is based on adult data due to the fact that outcome-based standards for indexed LVM in children are not available. In a study by Daniels and colleagues, a partition value for LVH in children was set at ≥95th percentile, while severe hypertrophy was considered ≥51 g/m².7, a value shown to increase adverse cardiovascular outcomes 4-fold in adults. In total, 44% demonstrated LVH defined as ≥95th percentile of LVM (g/m².7) and 14% demonstrated severe LVH, corresponding to ≥99th percentile of LVM (g/m².7). It is evident that LVH is prevalent in children when using adult partition values, which is concerning given the increased risk identified in adults.

In light of these findings, it is suggested that a single cut-point of 51 g/m².7 to define hypertrophy is recommended in children, corresponding to the 99th percentile. However, as suggested by Khoury et al. (2009), a single cut point may lead to inaccurate diagnosis of LVH across the pediatric age range especially in younger children. In this study of children aged 0-18 years, age-specific reference values for
indexed LVM were determined. For children greater than 9 years LVM/height\textsuperscript{2.7} varied little and values of \( > 40 \text{ g/m}^2 \) for girls and 45 g/m\textsuperscript{2.7} for boys were considered to be abnormal, corresponding to \( > 95^{th} \) percentile. In children younger than 9 years, there was considerable variation and therefore, a single cut point could not be determined. In contrast, findings from de Simone et al. (1995)\textsuperscript{76} showed that the variability in LVM/height\textsuperscript{2.7} increases up to the age of 18, and remains stable thereafter. Therefore, the existing discrepancy in the literature supports the notion that a single cut point in children may be difficult due to the complex relationship between heart growth and body growth in children.

To determine the risk associated with elevated LVM in children, the Medical College of Virginia Twin Study evaluated tracking of LVM in children. Left ventricle mass was found to track significantly in adolescence from age 11-17 years, however, the importance of this tracking to predict premature CVD in adulthood remains unknown.\textsuperscript{87}

Findings indicate that when partition values for LVH for adult criteria are assessed in children, LVH is prevalent (\( > 95^{th} \) percentile). These findings are important as these criteria in adults have been shown to predict mortality. Therefore, presence of LVH in children should be an indication to initiate therapeutic intervention in order to reduce the risk of mortality in adulthood.
2.3.0 Echocardiographic Assessment of LVM

2.3.1 Basic Principles of Doppler Ultrasound Echocardiography

Ultrasound is sound whose frequency is above the range of human hearing and is widely used in medical imaging. Ultrasound energy is transmitted into a patient; then, because various internal structures reflect and scatter sound differently, returning echoes can be used to form an image of a structure. Sound waves propagate through matter by causing molecules to vibrate successively along the sound path. Transducers are used to generate ultrasonic energy, the major component being the piezoelectric element. Pulsed transducers can use the same piezoelectric element for sending and receiving because separate time intervals are provided for emission and reception. This material is capable of converting electrical energy into mechanical energy during transmission and mechanical energy back into electric energy during reception, which forms a visual image of the studied structure. Waves travel through body tissue and are reflected at interfaces where there are differences in acoustic impedance of adjacent tissues.

Ultrasound transducers periodically send short bursts of sound energy into the area being studied. The machine measures time elapsed between the initiation and reception of the sound waves, and calculates the distance from the transducer and each anatomic reflecting surface. Shock excitation is used to generate sound energy and the frequency of excitation, which is the pulse repetition frequency, is normally great than 1000 pulses/sec (1KHz). Echoes are produced when changes in the characteristic of materials are encountered and when these echoes return to the transducer they are
converted into electrical signals, producing an image. Reflections are most frequently received at a normal, perpendicular incidence. This is true of larger interfaces such as heart walls, which reflect sound waves in a specular fashion. Nonspecular interfaces cause scattering. Attenuation is described as the decrease in energy of a wave as it travels through a medium. This can be caused by absorption, reflection and scattering. Scattering increases with an increase in transmission frequency. Therefore, lower frequencies (2 to 4 MHz) are used for deeper penetrations while higher frequencies (7 to 10 MHz) are used for best resolution of superficial structures.

2.3.2 Display Modes and Transducers

Several display modes exist for returning echo information: A mode, B mode, and M-mode. A mode provides an amplitude-modulated display representing the relative strength of returning echoes. B mode provides a brightness-modulated display in which there is a change in spot brightness for echoes received by the transducer. B mode displays the structure of interest as a cross-sectional picture in shades of gray. M-mode is a graphic B-mode display that is a single-dimension time display representing the motion of a structure along a single line penetrated by a single beam. It provides a system through which quantitative measurements of cardiac structures can be obtained. M-mode is a tool for the evaluation of subtle changes of the heart that may not be visible from real-time examination.

Two real-time scanning formats used for cross-sectional imaging are linear and sector. The linear format provides a rectangular view displaying a large field of view for structures close to the transducer. This format transmits a series of acoustic lines in a
direction that is parallel to the previous acoustic line. This format is difficult to obtain an image of a structure that is beneath other anatomic areas. Ultrasound scanners producing a sector image overcome this difficulty by transmitting acoustic lines at a different angle from the previous acoustic line (phasing out like a fan). A phased array transducer contains a number of piezoelectric elements along a small scanning surface to produce a sector image. Each acoustic line in a phased array transducer is steered by pulsing all of the elements as one group with a small time difference between them.

2.3.3 Sonographic Appearance

In two-dimension (2-D) echocardiography, there are several standard views in which to transect the heart. The parasternal long axis view (Figure 2.1) is the most commonly used when examining LVM in research studies. This view transects the heart from the base to the apex, as seen in Figure 2.1. Most anteriorly, the right ventricle can be visualized and is separated from the left ventricle by the interventricular septum (IVS). The IVS is continuous with the anterior portion of the aortic root, of which, only two of the leaflets are visible from this view. Posterior to the aortic root is the left atrium. The posterior portion of the aortic root is continuous with the mitral valve leaflets, which open and close during diastole and systole respectively. Patients are connected to an ECG monitor that runs simultaneously along the image, assisting in timing the cardiac cycle.
Figure 2.1 Parasternal long axis view of the left ventricle in systole. AV = aortic valve, LV = left ventricle, LA = left atrium, MV = mitral valve.

2.3.4 Calculating LVM

Echocardiography is the most popular method of evaluating cardiac structure and function. It has been clinically employed for over 30 years and is widely used as a non-invasive method of measuring LVM. Generally, LVM is calculated by subtracting the left ventricular cavity volume by the epicardium bordered volume, which provides a "shell volume". The shell volume is converted to mass by multiplying by an estimate of myocardial density. Most LVM calculations are made using measurements from 2-D targeted M-mode echocardiography. There are discrepancies in the overall measurement and whether or not inclusion of the edges of different layers is necessary. Standard recommendations indicate inclusion of septum edges, but exclusion of the posterior wall epicardial layer. The Penn convention excludes thickness of endocardial interfaces from septal and posterior wall thickness measurements, but includes them in the left ventricular internal dimensions. The criteria proposed by the American Society
of Echocardiography (ASE) is the most accepted, which uses all leading edges. Figure 2.2 provides a visual representation of the various conventions in layer measurements using M-mode echocardiography.

Figure 2.2 A comparison between M-mode border measurements.

There are several formulas available for determining LVM. A regression equation using the Penn convention proposed by Deveraux et al. (1977) strongly correlated to necropsy LVM ($r=0.92\ p<0.001$) with 100% sensitivity and 86% specificity. The Penn equation is represented in Formula 1 where LVIDD is left ventricular internal diameter in diastole; PWTD is posterior wall thickness in diastole, and IVSTD is interventricular septum thickness in diastole.

Formula 1: $LVM\ (Penn) = 1.04\ ([LVIDD + PWTD + IVSTD]^3 - [LVIDD]^3) - 13.6g$
However, calculating LVM using the more widely accepted ASE convention resulted in a reduced correlation agreement with necropsy ($r=0.86$), as well as specificity and sensitivity. Therefore, Devereux et al. (1986)\textsuperscript{97} corrected LVM measurements using this convention and proposed a new adjusted equation that has been validated against necropsy;

\textit{Formula 2:} $LVM (ASE) = 0.8 \times \frac{1.04([LVDD + PWTD + IVSTD]^{3} - [LVIDD]^{3})}{IVSTD} + 0.6 \text{ g.}$

This equation proposed by Devereux is the most widely used method of determining LVM in both adult and child studies.

\textbf{2.3.4.1 Indexing LVM}

There is an increase in the variability of LVM during normal growth, which is associated with body size. Several studies have demonstrated a positive relationship between LVM and body weight, height and BSA.\textsuperscript{76, 98, 99} As a result, several indexes have been created in order to adjust for physiological variations in LVM\textsuperscript{93}, the most widely used methods of normalization are body surface area (BSA) and height\textsuperscript{2.7}. These methods of indexation allow comparisons across individuals of varying body sizes. The best method for normalization is still debatable. Indexing for BSA is useful for identifying LVH but has been found to underestimate the prevalence of LVH in overweight and obese individuals.\textsuperscript{75, 94, 99} A physiological approach to account for this problem is to index by lean body mass (LBM)\textsuperscript{100} but this is difficult for routine purposes. Alternatively, indexing by body height to its allometric power (height\textsuperscript{2.7}) reduces the variability of LVM in normal subjects and increases the ability to detect LVH related to obesity and CVD.\textsuperscript{77, 99}
In a study of normotensive children and adults, it was found that the normalizing LVM to height\(^{2.7}\) (LVM/\(Ht^{2.7}\)) reduced the variability between LVM and height\(^9\), suggesting that normalization of height\(^{2.7}\) reduces the physiological impact of height on LVM. It has also been shown that LVM/\(Ht^{2.7}\) better predicts cardiovascular events in children and adults with arterial hypertension\(^{76}\), and correlates best with LBM\(^{76,77}\). Therefore, it seems that dividing LVM by height\(^{2.7}\) is a better method of normalization for children and adolescents.

2.4.0 Determinants of LVM

The left ventricle normally grows continuously from infancy to adulthood, with hypertrophy accounting for most of the increase in size\(^9\). Several determinants of LVM, both in children and adults, are unalterable in their effects and are therefore considered to be physiological determinants of LVM. The left ventricle increases normally during growth, maturation and physical conditioning\(^{53}\), as well as pathologically as a result of pressure and volume overload\(^{101}\). Increases in adult LVM have been linked to factors associated with body size (weight, height, and BSA), as well as hemodynamic factors such as obesity and hypertension. In childhood, similar determinants of LVM have been shown to influence LVM including age, weight, height, sex, BP, and obesity\(^{102,103}\).

2.4.1 Age and Sex

The effects of age on LVM have been controversial to date. Some studies suggest an increase during adolescence\(^{104}\) and a steady decline thereafter\(^{105}\), while others have found LVM to progressively increase in both normotensive and hypertensive individuals\(^7\). In the Framingham Heart Study a strong association between age and
prevalence of LVH, independent of the confounding influences of BP, obesity, and coronary and valvular heart disease exists in adults (age, 17 to 90 years).\textsuperscript{75} This independent association between age and LVH is significant in both sexes, with a 15% increase in men and 67% increase in women for every 10-year increment of age.\textsuperscript{75} Further investigation into the effects of age on LVM in the Cardiovascular Health Study (CHS)\textsuperscript{106,107} revealed a weak association between age and LVM. This study population consisted of adults aged 65 to 100 years split into three groups: 1) with clinical coronary heart disease, 2) hypertension in the absence of coronary heart disease, and 3) no clinical coronary heart disease or hypertension. They were further categorized across five year age intervals (65-69, 70-74, 75-79, and 80+ years). The effect of age on LVM was small, with a weight-adjusted increase in LVM of less than one gram per year increase in age for each disease status group.\textsuperscript{106} In an extension of this study by Gardin et al. (1997),\textsuperscript{107} age was not a significant explanatory variable of LVM when other independent variables were entered into the model. In contrast, age demonstrated a positive relationship with IVS and posterior wall (PW) thickness, and inversely associated with end-diastolic diameter.\textsuperscript{107} The absence of association between age and LVM may be a result of the older truncated age range; i.e. 65-100 years.\textsuperscript{107} However, in comparison to a study of normal adults using a wider age range (17-72 years), Devereaux and colleagues (1984)\textsuperscript{108} also found no significant correlation between age and LVM/BSA in men ($r=-0.16$) or women ($r=0.11$). It appears that in adults aged 20-100 years old, LVM remains relatively stable with increasing age in the absence of other risk factors.\textsuperscript{109}
To further elucidate this association, de Simone et al. (1995) extended a study to children to determine if age was associated with LVM independent of body size and sex in children/adolescents (birth to 17 years) and adults (>17 years). Age was related to LVM both in children/adolescents (p<0.002) and adults (p<0.0001) independent of height or weight and sex. Consistent with previous findings however, the relationship between age and LVM was weak (r=.31) in adults. There was a close relationship (r=.77) between LVM and age in preadolescent children (birth to 12 years), with the growth rate being similar in both boys and girls (5.7 and 4.9 g/yr, p=.19). In contrast, left ventricle growth rate became more rapid in boys than in girls (7.3 and 5.4 g/yr, p<0.0001) between the ages of 12-17 years. An interesting finding from this study was that left ventricular growth was slower than that of body weight and height throughout infancy and adolescence. Accordingly, LVM/Ht decreased from birth to adolescence, followed by a gradual increase during adulthood. This is consistent with another study in which age was found to be linearly associated with LVM and was identified as an independent predictor of LVM in children aged 6-16 years old. This study further revealed a significant interaction effect between age and allometric height on LVM, suggesting the effect of height on LVM is not the same for all ages. The effect of height on LVM is more important than the effect of age after the age of 8 years old.

Sex related differences in LVM have also been investigated, indicating that males demonstrate greater LVM than females. In adults, it appears that LVM/Ht is greater in men than women, despite its ability to account for LBM. In fact, LVM/Ht is 20%
greater in men than women. Likewise, studies including children have demonstrated a sex-related difference in LVM even after adjusting for body size. 

Sex differences in myocardial mass are thought to develop early in life (number of myocytes), or, occur due to a difference in cardiac growth. In a study of children and adults aged 4 months to 70 years, de Simone and colleagues (1995) found no significant difference in LVM (g) between girls and boys during infancy and childhood. However, a sex-related difference became evident at age 9 to 12 years, where LVM grew faster in boys. Although these results suggest that the initial number of cardiac monocytes is similar in males and females, there is evidence indicating that a sex difference in LVM can be seen in preadolescents as a result of growth. Several studies have shown boys to have greater absolute LVM compared to girls which persists throughout puberty and into adolescence. In the Multi Center Virginia Twin study of children 11 years old, boys demonstrated significantly larger LVM compared to age-matched girls and this difference remained significant even after adjusting for BSA. These findings are consistent with those of the Bogalusa Heart Study, in which sex differences were identified in children aged 7 to 11 years old. The difference between boys and girls is beyond the effect of body size, and may be attributed to differences in LBM. As well, sex-related differences in LVM have been attributed to aerobic fitness such that LVM has been shown to be greater in males due to a higher VO2 max and lower resting HR.
Nevertheless, the sex related differences in both children and adults are attenuated when LVM is normalized to height^{2,7}, suggesting a role in LBM accounting for sex differences.^{93,98,113,115} This is consistent with a study by Daniels et al. (1995)^{77} who found that when LVM was indexed by LBM, the significant gender differences in children were eliminated. Despite these controversial findings, it appears beneficial to account for age and sex when investigating LVM in epidemiological studies.^{86,93}

2.4.2 Maturation

Maturation is the process towards attaining full development, or progress toward the mature state.^{110} Growth and maturation are distinct in that growth refers to size at a given time point (age), whereas maturation focuses on progress towards adult state.^{110} There is limited research regarding the effect of childhood maturation on LVM. Daniels and colleagues (1995)^{116} found a univariate association between sexual maturation and LVM. However, after adjusting for age, height, weight, BSA, LBM, fat mass, systolic and diastolic BP in multivariate analysis, the relationship was no longer significant. This is consistent with a study by Janz et al. (1995)^{117} which showed no significant association between testosterone levels, sexual maturation and LVM. However, when comparing LVM values normalized to BSA, Peralta-Huertas et al. (2008)^{22} suggested that the difference between their results and Goble and colleagues (1992)^{113} may in part be attributed to the difference in sexual maturation levels between the study samples. The exact role of sexual maturation on LVM is not well understood, however, it is likely mediated through hormonal changes that also influence body size,
in particular, FFM. Further investigation in the role of pubertal maturation is necessary to gain a better understanding of its role on LVM.

2.4.3 Ethnicity

Ethnic differences have been demonstrated in the prevalence and progression of hypertension and CVD, primarily among African Americans. There appears to be ethnic differences in relation to LVM in normotensive and hypertensive patients. In the Coronary Artery Risk Development in Young Adults (CARDIA) Study, black men had higher LVM adjusted for covariates than white men and no difference was found in women. In a large cohort of hypertensive adults, blacks had significantly greater LVM/Ht than whites even after multivariate analysis adjusting for age, gender, BMI, mean arterial pressure, cardiac index, and beta-blockers.

Research regarding ethnic differences in LVM has been extended to children and similar results have been reported. A collaborative study of hypertensive children and adolescents from three sites belonging to the International Pediatric Hypertension Association examined the prevalence of LVH. In pediatric patients, LVH was classified as greater than the 95th percentile of LVM/height based on normative values for males (39.36 g/m) and females (36.88 g/m). This study found that both Hispanic and African Americans had an increased prevalence of LVH than did white children. In the Medical College of Virginia Twin study, race comparisons in children indicated that at younger ages black boys had higher LVM than white boys, but this was not evident at older ages. At a younger age, black boys were heavier, which could possibly explain the differences seen in LVM at a younger age.
2.4.4 Obesity

Obesity is associated with multiple risk factors for CVD, including LVH. Obese individuals are subject to LVH as a result of chronic pressure overload due to obesity related hypertension or volume overload due to an increase in cardiac output (CO). Chronic volume overload causes left ventricular dilation and increases risk of eccentric LVH, whereas chronic pressure overload induces concentric LVH. Several studies in adults have demonstrated that obesity increases the risk of LVH, independent of hypertension. A study conducted by Rider et al. (2009) aimed to identify the determinants of LVM in otherwise healthy obese adults. Consistent with previous research, it was found that LVM was significantly greater in the obese group (126 ± 27g) compared to the normal weight group (90 ± 20g). They found total fat mass, visceral fat mass, LBM, waist hip ratio, and BMI to be positively related to LVM. Systolic and diastolic BP showed no relationship in this normotensive population. Furthermore, this study revealed that only LBM, stroke volume (SV), and visceral fat mass independently predicted LVM. Similarly in the Strong Heart Study, results from Bella and colleagues (1998) demonstrated that LBM is the strongest correlate of LVM in a population based sample of elderly adults. Conversely, they found that adipose mass was not an independent predictor of LVM in either gender. These findings suggest that although obesity is a strong clinical predictor of LVM in adults, LBM and not fat mass is the main determinant of LVM.

Research has been extended to children and various studies have demonstrated an impact of obesity on LVM in both adolescent and preadolescent individuals.
which can persist into adulthood. Friberg et al. (2004) aimed to determine whether LVM was increased in obese adolescents aged 11-17 years old using cardiac magnetic resonance imaging. Results from this study demonstrated a 16% increase in LVM normalized to height in the obese group compared to the lean group (76g/m vs. 66g/m, p<0.0043). Likewise, in a study of overweight and normal-weight preadolescent children, Peralta-Huertas et al. (2008) found that LVM normalized to BSA and height was significantly greater in the overweight compared to the normal-weight group. This finding is consistent with results of Maggio et al. (2008) who found that obese prepubertal children had significantly greater LVM normalized to height compared to lean controls. It is evident that changes in LVM can occur in children prior to reaching puberty as a result of obesity.

The increase in LVM observed in children has been associated with similar variables as seen in adults such as body fatness, percent body fat, lean tissue mass, and 24-h SBP. Much attention has been directed towards percent body fat and adiposity as major determinants leading to increased LVM beyond normal growth in children and adults. In the study previously mentioned by Maggio et al. (2008), percent body fat and fat mass (kg) were significantly associated with LVM/height. Similarly, in a study of obese children aged 7-11 years, Humphries and colleagues (2002) found that percent body fat was associated with LVM normalized to height, suggesting that increased adiposity is an independent predictor of LVM. Contrary to these findings, two important studies by Daniels et al. (1995) and Dai and colleagues (2009) found that fat mass was weakly, although significantly correlated to LVM and only played a minor
role in determining $LVM/height^{2.7}$. For example, in the study by Dai and colleagues$^{126}$ every 10 kg increase in fat mass, resulted in an 11 g increase in $LVM$, however, $LBM$ was the strongest determinant of $LVM$ resulting in a 21 g increase in $LVM$ for every 10 kg of $LBM$. Therefore, the strongest determinant of $LVM$ in children and adolescents appears to be $LBM$.\textsuperscript{116,126}

As previously mentioned, $LBM$ is strongly associated with $LVM$\textsuperscript{23,116,125} and has been suggested to be a better predictor of increased $LVM$ than fat mass.\textsuperscript{23,113,116,126} Using bioelectrical impedance, Janz and colleagues (1995)\textsuperscript{117} found that fat-free mass accounted for 72\% of $LVM$ variability in boys and 62\% in girls. Similar results have been found using DEXA, a more accurate measure of body composition. For example, Humphries et al. (2002)\textsuperscript{125} found a significant ($r=0.83$, $p<0.01$) correlation between $FFM$ and $LVM$ normalized to height$^{2.7}$ in children. As well, a similar association between lean tissue mass and $LVM/height^{2.7}$ was found by Maggio (2008).\textsuperscript{23} Furthermore, Daniels et al. (1995)\textsuperscript{116} assessed body composition using DEXA and reported that $LBM$ was the most significant predictor of $LVM$, accounting for 75\% of the variance. In fact, $LBM$ was still the most important correlate of $LVM$ after accounting for race and sex.\textsuperscript{116} A more recent study of healthy children aged 8, 11, and 14 years confirmed that body size, especially when measured in $LBM$ was the strongest predictor of $LVM$.\textsuperscript{126} Possible explanations for the strong relationship between $LBM$ and $LVM$ have been postulated, which include an increase in $CO$, and hormonal factors that affect skeletal muscle mass and organ mass.\textsuperscript{23,100,120} In order to compensate for the additional physical demand imposed by increased body weight, an associated increase in skeletal muscle mass is
necessary and has been confirmed in several studies.\textsuperscript{22,23,120} Lean body mass generates nearly all metabolic activity and oxygen demand, which determines the required CO and SV.\textsuperscript{100,120} It has been shown that SV is closely associated to LVM. Therefore, it is not surprising that the associated increase in LBM observed in overweight individuals is a powerful determinant of LVM.

Current research has uncovered the fact that childhood obesity has lasting effects into adulthood. Research has found that childhood obesity is a predictor of LVH in adulthood, and this risk increases even more when childhood obesity persists into adulthood.\textsuperscript{124} In males and females, initial weight, and measures of obesity (ponderal index, triceps skinfold) were important determinants of LVM/height\textsuperscript{2.7} in a five year follow-up study in children.\textsuperscript{101} Body mass index has also been found to be related to LVM in children.\textsuperscript{127} As well, the degree of increase in LVM from childhood to adulthood is dependent on the increase in BMI, regardless of childhood BMI.\textsuperscript{128} Nevertheless, there are shortcomings to using BMI as a measure of obesity as there is no distinction between lean mass and adiposity, general adiposity and/or visceral adiposity.

Body fat distribution, specifically central adiposity, increases the risk of CVD.\textsuperscript{129} Body fat distribution associated with an increase in central/truncal obesity shows a stronger correlation to increased LVM than percent body fat.\textsuperscript{130} Epicardial adipose tissue, a true measure of visceral adipose tissue deposited around the heart, is strongly correlated to an increase in LVM/height\textsuperscript{2.7} in adults.\textsuperscript{131} These finding are also supported in children. Humphries et al. (2002)\textsuperscript{125} demonstrated that in obese children, visceral
adipose tissue was associated with an increase in unadjusted LVM, however, no
information was provided for adjusted LVM.

Based on past research, there is sufficient evidence suggesting obesity in
children plays an important role in the pathological increase in LVM, and this risk tracks
into adulthood. Early interventions in the prevention and treatment of childhood
obesity are important for the prevention of cardiovascular morbidity and mortality seen
with increased LVM.23,124

2.4.5 Hypertension

It has been established that the risk of morbidity and mortality increases as BP
rises.132 Left ventricular hypertrophy develops as an adaptation to normalize afterload
and preserve systolic function in those individuals with hypertension.133 Hypertension
causes a pressure overload, which stimulates growth in cardiac muscle cells and induces
concentric LVH.119 A correlation between persistently high SBP and increased LVM has
been found in adults.104,132,134 In adults aged 28 to 62 years, Levy et al. (1988)75
demonstrated a continuous and independent association of SBP with LVH in a dose-
response relationship at levels <140 mmHg in both sexes. Furthermore, de Simone et al.
(1994)122 found that in a pooled analysis of hypertensive and normotensive adults,
systolic BP (SBP) was an independent predictor of LVM/height2.7.

Results of studies in hypertensive children indicate that LVH is prevalent and can
be severe.104,135 Hanevold and colleagues (2004)25 found the prevalence of LVH in
hypertensive children and adolescents to be 41%. More recently, a study of
hypertensive children identified with BP ≥ 95th percentile for age, height, and gender found LVH to be more prevalent with increasing severity of hypertension. Although there was no significant difference in LVM/HT².7 between stages of hypertension, individuals identified with stage 1 hypertension (≥95th percentile to 99th percentile+5 mm Hg) and stage 2 hypertension (≥99th percentile+5 mm Hg) demonstrated a prevalence of LVH of 14.5% and 30% respectively, compared to 9% in normotensive. In a more recent study by Stabouli et al. (2009), the effect of hypertension and prehypertension on LVM were assessed on adolescents (14 to 15 years old). Important findings from this study revealed that both hypertensive (36.8 g/m².7) and prehypertensive (34.1 g/m².7) individuals demonstrated greater LVM/HT².7 compared to normotensive individuals (29.5 g/m².7). The differences in LVM/HT².7 remained significant even after adjusting for age, sex and BMI z-score. Moreover, prevalence of LVH was equal in both the hypertensive and prehypertensive groups (20%), and significantly lower in the normotensive group (6.7%, p<0.01). Also of importance is that all individuals with LVH in the normotensive group were categorized as overweight and obese. Another important finding from this study demonstrated that 38.5% of patients with LVH had LMV >51 g/m².7, a value that has been associated with a fourfold greater risk of adverse health outcomes in adults. The results of this study provide insight into the impact of hypertension on LVH in children, suggesting that chronically elevated BP increases the prevalence of LVH in children, subsequently increasing their risk of CVD.

Not only has research focused on the relationship between hypertension and LVH, but it has also focused on the overall role of BP on LVM. In a study by Daniels and
colleagues (1995) SBP was found to have a weak, yet significant independent association with LVM, explaining 0.5% (p=0.04) of the variability after accounting for LBM and fat mass. Malcolm et al. (1993) also found that SBP had a low to moderate association with LVM in both boys (r=0.54) and girls (r=0.43). These findings are consistent with other studies indicating a weak association between BP and LVM. Because of its close association with body weight, the effect of BP on LVM is greatly reduced when controlling for body size parameters. Additionally, a possible explanation for the small impact of BP on LVM is that casual BP, used in the majority of studies, may not represent an individual’s true BP. The use of 24-hour ambulatory BP in adults has produced a better correlation with LVM (g/m²), predicting LVH better than casual BP.

Therefore, it appears that persistently elevated BP, independent of obesity, plays a role in determining LVM. Individuals with hypertension have been shown to be at an increased risk for LVH. Hence, it is important to identify children with elevated BP when assessing LVM.

2.4.5 Cardiovascular Fitness

It has been found that increased fitness attenuates the development of LVH in both hypertensive and prehypertensive adults. In a recent study by Kokkinos et al., (2007) it was found that moderate and high fit prehypertensive individuals have significantly lower left ventricular wall thickness and LVM than low fit individuals. Furthermore, the prevalence of LVH was significantly higher in the low fit (48.3%) compared to the moderate (18.7%) and high fit (21.6%) individuals, and the likelihood of
developing LVH decreases 42% for every 1 MET increase in exercise capacity.\textsuperscript{139} There are several studies suggesting that long-term, high-intensity training causes an increase in LVM in children\textsuperscript{65,140-143}. In one study, Triposkiadis et al. (2002)\textsuperscript{140} compared a group of 25 prepubertal male and female elite swimmers (mean age 11.9 years) to controls (mean age 11.3 years) and found that LVM (g/m\textsuperscript{2}) in swimmers was significantly greater (91.3 \pm 21.7 g/m\textsuperscript{2}) than controls (60 \pm 11.5 g/m\textsuperscript{2}). Furthermore, in a more recent study of male prepubertal swimmers, Ayabakan et al. (2006)\textsuperscript{143} found a significant difference in LVM (g/m\textsuperscript{2.7}) between swimmers (35.50 g/m\textsuperscript{2.7}) and controls (27.02 g/m\textsuperscript{2.7}).

In spite of these findings, the association between short-term exercise training and increases in LVM in previously sedentary individuals is less clear in both children and adults. In a group of previously sedentary adults, Wolfe and colleagues (1979)\textsuperscript{144} found no change in LVM after completion of a 6-month training program that consisted of jogging 4 days per week. In this study, an overall significant increase in pre-post VO\textsubscript{2} max (ml/kg/min) was found, however, no effect of exercise was found on LVM. In contrast, Cox et al. (1986)\textsuperscript{145} found that their exercise program of training 6 days/week for 7 weeks consisting of alternating days of running and cycling significantly increased both VO\textsubscript{2} max (ml/kg/min) and LVM (g/m\textsuperscript{2}) post training. However, this study failed to compare results to an adult control group. In a study of prepubertal children aged 10-11 years old, a 13-week endurance training program significantly increased LVM normalized to BSA.\textsuperscript{141} However, a similar increase in LVM/BSA was also seen in the control group.\textsuperscript{141} To determine the effects of training on LVM, Humphries and colleagues (2002)\textsuperscript{125} explored the effect of aerobic training in obese adolescents on left ventricular
parameters. Results indicated that individuals with higher body fatness had higher LVM/\text{Ht}^{2.7}. Although aerobic training was able to significantly improve \text{VO}_2 \text{max} and body composition, there were no significant changes in LVM/\text{Ht}^{2.7}. It is likely that the magnitude of change in body fatness and \text{VO}_2 \text{max}, although significant, were not enough to elicit a change in LVM.

It appears that in order for an individual to obtain a physiological increase in LVM, the training program must be long-term (years) and at a high intensity, equivalent to that of elite athletes. In fact, several studies indicate that a correlation exists between LVM and \text{VO}_2 \text{max}, and a greater increase in \text{VO}_2 \text{max} results in a greater increase in LVM.\textsuperscript{146, 147}

2.5 Objective

Although it has been well documented that children with DCD are at an increased risk of developing CVD risk factors such as overweight and obesity, reduced cardio-respiratory fitness, there are limited data using metabolic indices and laboratory measures assessing cardiovascular health in children with DCD.\textsuperscript{11} The need to assess left ventricular structure and function in this population is important as children with DCD exhibit risk factors for developing LVH. The purpose of this investigation was to determine whether children diagnosed with p-DCD demonstrate elevated LVM compared to controls, and whether they demonstrate differences in cardiac dimensions and systolic function.
2.6 Hypothesis

We hypothesized that children with DCD would demonstrate elevated LVM and cardiac dimensions compared to age, gender, and school matched controls. Also, that there would not be a significant difference in left ventricular function between groups.
Chapter 3: Methodology

3.1 Research Design

This investigation is part of a larger study and will make use of data from the Physical Activity Health Study Team (PHAST). This study was approved by the Brock University Research Ethics Board (Appendix A) and the District School Board of Niagara (DSBN). PHAST is a longitudinal investigation comprised of two phases. Phase I began in September 2004 when 2519 of a possible 3030 grade four students from the Niagara District School Board agreed to participate in bi-annual school based health assessments. Phase I ended September 2007, at which time the second phase of the study began and continued until June 2010. Of the 2519 students from phase I, 1785 agreed to participate in Phase II. This phase involved annual school health assessments as well as a lab-based component which utilized a case-control design.

3.2 Study Population

Of the 1785 students that agreed to participate in phase II, 963 (54% response rate) expressed interest in being contacted by telephone to participate in the laboratory-based component. There were 198 students diagnosed with probable DCD using the Bruininks-Oseretsky Test of Motor Proficiency-Short Form (BOTMP-SF) who score below the 10th percentile. Of these 198, 131 either declined to be contacted further or were unable to be contacted. A total of 67 were left to be contacted to participate (31 males and 36 females). Of the 67 students with probable DCD, 63 cases (37M and 26F) fully agreed to participate in the study. Healthy control subjects (63) were selected randomly from consenting students and were matched for gender, school and age within 6 months.
3.3 Experimental Protocol

Subjects were scheduled for an appointment at the Human Hemodynamic Laboratory of Brock University. Upon arrival, subject and parent(s) were reminded of the study purpose and consent forms were signed (Appendix B). The laboratory-based component was multifaceted and included a large battery of physiological and survey based assessments. Figure 3 below summarizes the measurements performed during data collection in the Applied Health Sciences labs at Brock University. The entire lab component took approximately 2 hours. Measurements pertinent to this study are described in more detail in the following sections.

![Figure 3.1 Experimental protocol for PHAST lab testing.](figure)

Testing took place at Brock University and all information was recorded on the Advanced Health Information sheet (Appendix C). Upon arrival, consent forms were completed and subject demographics (age, gender, ethnicity etc.) were recorded.

Subjects were then taken into a private room with their parent or guardian. Each subject was measured for height, weight, and waist and hip circumference. Body composition
was measured by air-displacement plethysmography using the BOD POD. Once
measures of body composition were complete, subjects entered the hemodynamic
laboratory for cardiovascular measures.

Subjects lied in the supine position while BP was taken manually three times
(sphygmomanometer) with each measure separated by one minute. Subjects continued
to lie in the supine position for 15 minutes before data collection began. Five minutes of
baseline beat-by-beat HR with a one-lead electrocardiogram was obtained. Following
baseline measures, images of the left ventricle were taken using echocardiography.
Three final manual measurements of BP were taken at the very end to ensure that the
subject was still at rest.

Subjects were then taken to perform the movement assessment battery for
children, 2\textsuperscript{nd} edition (M-ABC2), with a trained occupational therapist (Appendix D). The
final station completed was the peak oxygen uptake test. Participants completed a
gradual test (approximately 10 minutes) ending at 100\% maximal effort using a
programmed cycle ergometer. Lastly, Tanner staging was completed by a trained
research assistant 7-8 days following laboratory testing.

3.4 Experimental Measurements
3.4.1 Anthropometry

All measurements of body composition were performed in a private room with
the parent(s) present. Body mass index was calculated using height and weight (kg/m\(^2\)).
Standing height (cm) was measured using a stadiometer (Stat 7X, Ellard Instrumentation
so Ltd Monroe, WA, USA) and recorded to the nearest 0.1 cm. Weight (kg) was measured using a digital scale (BWB-800S, Tanita Digital Scale, Tokyo, Japan) and recorded to the nearest 0.1 kg. The scale was calibrated using a 10 kg weight and subjects were weighed wearing only tight fitting swimsuits or undergarments.

Relative body fat was assessed using whole body air-displacement plethysmography with the BOD POD (Life Measurement, Inc, Concord, CA). The BOD POD is a reliable and valid technique that can evaluate body composition in children, adults, and obese individuals. The surface area of hair and clothing has an impact on the measure of air volume; therefore, subjects were instructed to wear tight fitting swim suits and were provided with a Lycra swim cap. The BOD POD was calibrated using a cylinder of known volume. Subjects were then seated in the chamber and body volume measurements were taken twice, each lasting 40 seconds. Subjects were advised to breathe normally and remain relaxed. If both measures were within 150 ml of each other, the mean of the two measures were taken. If the difference between the two measures was greater than 150 ml, a third measure was taken and the mean between the two closest were used.

3.4.2 Blood Pressure and Heart Rate

Auscultation is the recommended method of BP measurement in children. As recommended, subjects lied supine with their arm resting at heart level for 5 minutes. Blood pressure was measured using a non-invasive, standard inflatable cuff and a sphygmomanometer placed on the right arm. A stethoscope was placed over the brachial artery pulse, below the bottom edge of the cuff (2 cm above the cubital fossa).
Correct measurement of BP requires an appropriate sized cuff; each child was fitted with either a pediatric or adult cuff based on arm size. Systolic blood pressure was determined by the onset of the Korotkoff sounds (K1), and the fifth Korotkoff sound (K5), or the disappearance of Korotkoff sounds was used to determine DBP. A standard one-lead electrocardiogram was used to collect HR.

### 3.4.3 Echocardiography

LVM was measured using non-invasive Doppler echocardiography (Vivid I, GE Medical Systems, Horten, Norway). Images were taken using the parasternal long-axis view while subjects lied in the supine position. Three images consisting of 5 cardiac cycles each were taken for each subject using high resolution (3.25 MHz probe) B-mode. After obtaining a clear image in B-mode, images were then converted to anatomical M-mode for analysis.

Analyses of M-mode images were performed according to the recommendations of the American Society of Echocardiography. Dimensions at end diastole were obtained at the onset of the QRS complex on the electrocardiogram in a plane through a standard position. Measurements included the diameter of the left ventricle at end diastole, measured from the septum edge to the endocardium mean border; the posterior wall, measured as the distance from the anterior wall to the epicardial surface; and the interventricular septum, measured as the distance from the surface of the left ventricle border to the right ventricle septum surface. Each measure was completed for three beats per image for a total of two images and were averaged together. These
averaged values were used to calculate LVM using a validated equation by Devereux et al (1986). \(^9^7\)

\[
\text{LVM (g)} = 0.8 \times (1.04(\text{LVIDD} + \text{PWTD} + \text{IVSTD})^3 - \text{LVIDD}^3)) + 0.6 \text{ g,}
\]

where IVSd represents interventricular septum at end diastole (cm); LVDd represents the left ventricle diameter at end diastole (cm); and PWd represents the left ventricle posterior wall at end diastole (cm). Left ventricle mass was normalized to height\(^{2.7}\) which has been shown to predict cardiovascular risk in adults. \(^7^6,^9^4\) Left ventricle hypertrophy was defined using age-specific reference values of LVM/height\(^{2.7}\) >95\(^{th}\) percentile for males and females. \(^8^6\)

Stroke volume was estimated by subtracting end systolic volume (ESV) from end diastolic volume (EDV), and ejection fraction was calculated as \((\text{EDV} - \text{ESV})/\text{EDV} \times 100\%\). Fractional shortening was calculated as \((\text{LVDd}-\text{LVDs})/\text{LVDd} \times 100\%\), where LVDd and LVDs represent left ventricular diameter at end diastole and end systole respectively. Cardiac output was estimated as the product of HR and SV.

3.4.4 Aerobic Fitness

A maximal oxygen uptake test using a programmed cycle ergometer (Lode Excalibur, Groningen, Netherlands) was used to measure peak aerobic power in children. This procedure is a continuous and incremental exercise protocol. A mask and nose clip was used to collect expired gases. Gases were analyzed using an Oxygen analyzer (Model S-3A, AIE Technologies, Pittsburgh, Pennsylvania). Each subject completed a practice period to familiarize themselves with the equipment and protocol.
HR was monitored continuously using a Polar HR monitor. Prior to assessment, subjects were instructed on the importance of exercising until they reached maximum volitional fatigue.

Subjects began cycling at a rate of 65-75 rpm with an initial power output of 20 watts for the first three minutes. Work rate was increased every minute by 20 watts until subjects reached the estimated final stage whereby work rate increased by 15 watts. The subjects were encouraged throughout the assessment until volitional fatigue. Subjects were instructed to rate their perceived exertion throughout using the standardized Borg rating of perceived exertion scale (Appendix E). The criteria used to determine if peak aerobic power was achieved were two of the following: HR was greater than 85% of theoretical maximal level for age (220-age), subject showed signs of intense effort (facial flushing, or difficulty maintaining speed cycle) or the respiratory exchange ratio reached 1.0. For between group comparisons, peak VO₂ was adjusted for body mass in kilograms of FFM, which has been shown to better correlate with peak VO₂.

3.4.5 Developmental Coordination Disorder

The Movement Assessment Battery for Children, 2nd edition (M-ABC2) was used to assess motor coordination. The test required children to complete a series of fine and gross motor tasks (8-items) grouped into three categories: Manual dexterity, Aiming and Catching, and Balance. A standard score was provided for each item. From each of these standard scores an age-adjusted cumulative score and percentile was provided. All tests were performed by a pediatric occupational therapist that was familiar with the
M-ABC2 test. However, to confirm diagnosis of DCD all criteria from the DSM-IV (1994) is required. For the current study, information on criteria B from the DSM-IV (1994) was missing, therefore the term probable DCD (p-DCD) was used. Children with a score below the 16th percentile were identified as p-DCD. The Kaufman Brief Intelligence Test is a quick and reliable measure of intelligence that can be administered by health professionals. It is a cognitive screening tool that measure two areas of cognitive functioning (verbal and non-verbal). Children with an estimated intelligence quotient below 70 are considered to be well below average intelligence.

3.4.6 Pubertal Maturation

To classify subjects into their maturation grouping, pubertal maturation was self-reported using pictures of the Sexual Maturation Scale by Tanner taken from Taylor et al (2001). To reduce embarrassment, each subject completed the self-assessment at home in the presence of their parent(s). Once complete, the self-assessment was placed in a folder and handed to the research assistant to maintain anonymity.

3.5 Statistical Analysis

Analyses were completed using SPSS software version 16.0 (SPSS Inc, Chicago, IL), and level of significance for all measures was set to $p \leq 0.05$. All analyses compared p-DCD ($\text{M-ABC2}<16^{th}$ percentile) and controls ($\text{M-ABC2}>16^{th}$ percentile). Descriptive statistics were calculated using an independent T-test for all variables and are presented as mean and standard deviation for subject demographics/characteristics (age, gender,
body composition, K-Bit, M-ABC2, BP, HR, SV, CO, peak VO$_2$) and all LVM measurements.

A chi square test was used to determine differences between groups for maturation.

Pearson correlation was performed to determine all factors associated with LVM, LVM/Ht$^{2.7}$, CO and SV. A multivariate linear regression was performed to examine the effect of p-DCD on SV and CO, controlling for the effects of body size (height), sex, aerobic fitness (peak VO$_2$), and body fatness (FFM). For SV, an interaction was entered into the model for p-DCD and VO$_2$FFM, and for p-DCD and FFM. For CO, an interaction was entered into the model for p-DCD and FFM. In order to address the problem of multicollinearity, all continuous predictor variables included in the interaction term were centered, prior to calculation of the interaction term. This was done by subtracting the mean from each individual value in the variable. Then the centered variable was entered in the model with the interaction term. Tests for the presence of multicollinearity were conducted using variance inflation factors (VIFs). Values over 10 indicate problems with collinearity.
Chapter 4: Results

4.1 Sample Characteristics

This study was part of a laboratory-based prospective case-control design examining the cardiovascular health of children with p-DCD. In total there were 126 subjects eligible to participate in the study. This included 63 with p-DCD and 63 age, gender, and school matched controls. Participants were categorized as p-DCD if they scored below the 16th percentile on the M-ABC2 test. Table 4.1 shows the demographic and cardiovascular characteristics for p-DCD and control groups. There were no significant differences in demographic variables such as age, gender, and height. Table 4.2 presents the distribution of participants in tanner stages; there was no difference in sexual maturation between groups. As expected there were significant differences between groups in the M-ABC2 test of motor coordination, where children with p-DCD demonstrate significantly lower percentile scores than controls (p<0.001). Significant differences were also evident in body composition variables such as weight, BMI, and percent body fat (p≤0.001); all of which were higher in the p-DCD group compared to controls. As for the cardiovascular variables, SBP, DBP, and HR were higher in the p-DCD group (p<0.05), while peak VO\textsubscript{2} normalized to FFM (VO\textsubscript{2FFM}) was significantly lower (p=0.001).
Table 4.1 Demographic and cardiovascular measures in children with p-DCD and controls.

<table>
<thead>
<tr>
<th>Demographic Variable</th>
<th>p-DCD</th>
<th>Control</th>
<th>t-value</th>
<th>Df</th>
<th>p-value</th>
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<td>Age</td>
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<td>12.4 ± 0.5</td>
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</tr>
<tr>
<td>Sex (n=Male, n=Female)</td>
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<td>37, 26</td>
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<tr>
<td>Height (cm)</td>
<td>158.4 ± 7.9</td>
<td>156.9 ± 7.7</td>
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<tr>
<td>Weight (kg)</td>
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<td>50.2 ± 11.3</td>
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<td>124</td>
<td>0.001</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>23.4 ± 5.9</td>
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</tr>
<tr>
<td>BF (%)</td>
<td>28.3 ± 11.2</td>
<td>20.0 ± 9.9</td>
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<td>124</td>
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<tr>
<td>FFM (kg)</td>
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<td>124</td>
<td>ns</td>
</tr>
<tr>
<td>M-ABC2 (percentile)</td>
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<td>42.4 ± 21.7</td>
<td>13.9</td>
<td>124</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>K-BIT</td>
<td>89.5 ± 13</td>
<td>100.3 ± 10</td>
<td>5.3</td>
<td>124</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cardiovascular Variable</th>
<th>p-DCD</th>
<th>Control</th>
<th>t-value</th>
<th>Df</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP (mmHg)</td>
<td>110 ± 11.1</td>
<td>105 ± 9.2</td>
<td>-2.5</td>
<td>124</td>
<td>0.01</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>69 ± 9.5</td>
<td>65 ± 9.6</td>
<td>-2.5</td>
<td>124</td>
<td>0.01</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>80 ± 11</td>
<td>75 ± 10</td>
<td>-2.9</td>
<td>124</td>
<td>0.005</td>
</tr>
<tr>
<td>Peak VO₂ (L/min)</td>
<td>2.0 ± 0.5</td>
<td>2.1 ± 0.5</td>
<td>1.4</td>
<td>123</td>
<td>Ns</td>
</tr>
<tr>
<td>Peak VO₂ (mL/kgFFM/min)</td>
<td>48.5 ± 7.5</td>
<td>53.2 ± 8.1</td>
<td>3.4</td>
<td>123</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Mean ± SD, independent-samples T test. BMI = Body Mass Index, BF = Body Fat, FFM = Fat Free Mass, M-ABC2 = Movement Assessment Battery for Children 2nd edition, K-BIT = Kaufman Brief Intelligence Test, SBP = Systolic Blood Pressure, DBP = Diastolic Blood Pressure, HR = Heart Rate, bpm = beats per minute, Peak VO₂ = peak aerobic fitness, ns = not significant.

4.2 Cardiovascular Measurements

All participants received an echocardiogram in order to determine cardiac dimensions and LVM. Of the 126 participants who received an echocardiogram only 121 were adequate for analysis. The remaining five were technically difficult to determine accurate cardiac dimensions and therefore excluded from the study. Cardiac dimensions and LVM are shown in Table 4.3. Left ventricular diameter in diastole (p=0.02), EDV (p=0.03), SV (p=0.02) and CO (p<0.001) were significantly higher in the p-DCD compared to controls. As for LVM estimates, there were no significant difference between groups.
Table 4.2 Distribution of Participants across Tanner Stages

<table>
<thead>
<tr>
<th>Tanner Stage</th>
<th>Breast/ Penis</th>
<th>Pubic Hair</th>
<th>Breast/ Penis</th>
<th>Pubic Hair</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2</td>
<td>3</td>
<td>3</td>
<td>4</td>
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<tr>
<td>2</td>
<td>16</td>
<td>14</td>
<td>20</td>
<td>21</td>
</tr>
<tr>
<td>3</td>
<td>28</td>
<td>20</td>
<td>25</td>
<td>18</td>
</tr>
<tr>
<td>4</td>
<td>12</td>
<td>18</td>
<td>12</td>
<td>14</td>
</tr>
<tr>
<td>5</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>6</td>
<td>-</td>
<td>2</td>
<td>-</td>
<td>0</td>
</tr>
</tbody>
</table>

Table 4.3 Cardiac dimensions in children with p-DCD and controls.

<table>
<thead>
<tr>
<th></th>
<th>p-DCD</th>
<th>Controls</th>
<th>t-value</th>
<th>Df</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>IVSd (cm)</td>
<td>0.68 ± 0.07</td>
<td>0.68 ± 0.09</td>
<td>0.1</td>
<td>119</td>
<td>Ns</td>
</tr>
<tr>
<td>LVDd (cm)</td>
<td>4.48 ± 0.32</td>
<td>4.35 ± 0.33</td>
<td>-2.3</td>
<td>119</td>
<td>0.02</td>
</tr>
<tr>
<td>PWd (cm)</td>
<td>0.65 ± 0.08</td>
<td>0.67 ± 0.09</td>
<td>1.5</td>
<td>119</td>
<td>Ns</td>
</tr>
<tr>
<td>LVDs (cm)</td>
<td>3.06 ± 0.32</td>
<td>2.98 ± 0.30</td>
<td>-1.4</td>
<td>119</td>
<td>Ns</td>
</tr>
<tr>
<td>EDV (mL)</td>
<td>91 ± 19</td>
<td>84 ± 19</td>
<td>-2.3</td>
<td>119</td>
<td>0.03</td>
</tr>
<tr>
<td>ESV (mL)</td>
<td>30 ± 9</td>
<td>27 ± 8</td>
<td>-1.5</td>
<td>119</td>
<td>Ns</td>
</tr>
<tr>
<td>SV (mL)</td>
<td>62 ± 14</td>
<td>56 ± 13</td>
<td>-2.3</td>
<td>119</td>
<td>0.02</td>
</tr>
<tr>
<td>CO (L/min)</td>
<td>4.9 ± 1.2</td>
<td>4.2 ± 0.9</td>
<td>-3.9</td>
<td>118</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>EF (%)</td>
<td>68 ± 6</td>
<td>67 ± 6</td>
<td>-0.4</td>
<td>119</td>
<td>Ns</td>
</tr>
<tr>
<td>FS (%)</td>
<td>32 ± 4</td>
<td>32 ± 4</td>
<td>-0.4</td>
<td>119</td>
<td>Ns</td>
</tr>
<tr>
<td>LVM (g)</td>
<td>89 ± 17</td>
<td>87 ± 21</td>
<td>-0.6</td>
<td>119</td>
<td>Ns</td>
</tr>
<tr>
<td>LVM/Ht^{2.7} (g/m^{2.7})</td>
<td>26 ± 5</td>
<td>26 ± 6</td>
<td>-0.1</td>
<td>119</td>
<td>Ns</td>
</tr>
</tbody>
</table>

Mean ± SD, independent-samples T test. IVSd = interventricular septum in diastole, LVDd = left ventricular diameter in diastole, PWd = left ventricular posterior wall in diastole, LVDs = left ventricular diameter in systole, EDV = end diastolic volume, ESV = end systolic volume, SV = stroke volume, CO = cardiac output, EF = ejection fraction, FS = fractional shortening, LVM = left ventricular mass, LVM/Ht^{2.7} = left ventricular mass normalized to height^{2.7}. Ns = not significant.

4.3 Determinants of Left Ventricular Mass, Stroke Volume, and Cardiac Output

Results of the correlations for the entire cohort are presented in Table 4.4. Left ventricular mass was correlated with BMI (p<0.001), FFM (p<0.001), SBP (p=0.03), and peak VO_{2FFM} (p=0.02). Stroke volume was positively correlated with BMI (p=0.001), FFM (p<0.001), and peak VO_{2FFM} (p=0.03). Cardiac output was correlated with M-ABC2 percentile (p=0.001), BMI (p<0.001), FFM (p<0.001), percent body fat (p=0.002) and SBP (p<0.001). Normalized LVM correlated with BMI (p=0.006) and VO_{2FFM} (p=0.02).
Multivariate linear regression analyses were used to determine the independent effect of p-DCD on SV and CO. Table 4.5 shows the results of the regression analyses for SV and CO. In the first model, SV was regressed on p-DCD only. In this model, the main effect of p-DCD on SV was positive and significant \( (p=0.02) \), accounting for 3.5% of the variability in SV \( (\text{adj. } R^2 = 0.035) \). When height, sex, \( VO_{2\text{FFM}} \), and FFM were entered into model 2, the main effect of p-DCD remained significant \( (p=0.001) \) and the unstandardized b-coefficient increased from 5.6 to 7.3. In this model height \( (p=0.006) \), \( VO_{2\text{FFM}} \) \( (p=0.002) \), and FFM \( (p=0.03) \) were all significant predictors of SV. These results did not show a significant difference for sex. Together these variables accounted for 33.5% of the variability in SV. An interaction was entered into the model for p-DCD and \( VO_{2\text{FFM}} \), and for p-DCD and FFM. Neither of these interactions reached the threshold for statistical significance.

The regression analysis for CO on p-DCD in model 1 demonstrated a main effect of p-DCD \( (p<0.001) \), accounting for 10.6% of the variability. In model 2, height, sex, \( VO_{2\text{FFM}} \), and FFM were entered and these variables accounted for 32.5% of the variability in CO. The effect of p-DCD remained significant \( (p<0.001) \) and the unstandardized b-coefficient increased from 0.76 to 0.82 from model 1 to 2. In this model height \( (p=0.01) \) and FFM \( (p=0.02) \) were significant predictors of CO. An interaction was entered into the model for p-DCD and FFM and there was no significant interaction effect.
Table 4.4 Univariate Correlations

<table>
<thead>
<tr>
<th></th>
<th>M-ABC2</th>
<th>BMI</th>
<th>FFM</th>
<th>BF(%)</th>
<th>SBP</th>
<th>DBP</th>
<th>VO\textsubscript{2FFM}</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI</td>
<td>-0.324*</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FFM</td>
<td>0.01</td>
<td>0.533*</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PBF</td>
<td>-0.391*</td>
<td>0.813*</td>
<td>0.161</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP</td>
<td>-0.162</td>
<td>0.541*</td>
<td>0.416*</td>
<td>0.376*</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DBP</td>
<td>-0.081</td>
<td>0.090</td>
<td>0.060</td>
<td>0.124</td>
<td>0.377*</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>VO\textsubscript{2FFM}</td>
<td>0.332*</td>
<td>-0.142</td>
<td>-0.184*</td>
<td>-0.140</td>
<td>-0.042</td>
<td>0.134</td>
<td>-</td>
</tr>
<tr>
<td>SV</td>
<td>-0.148</td>
<td>0.368*</td>
<td>0.368*</td>
<td>0.134</td>
<td>0.140</td>
<td>0.016</td>
<td>0.196*</td>
</tr>
<tr>
<td>CO</td>
<td>-0.293*</td>
<td>0.391*</td>
<td>0.401*</td>
<td>0.280*</td>
<td>0.316*</td>
<td>0.158</td>
<td>-0.009</td>
</tr>
<tr>
<td>LVM</td>
<td>-0.083</td>
<td>0.383*</td>
<td>0.474*</td>
<td>0.149</td>
<td>0.264*</td>
<td>0.074</td>
<td>0.213*</td>
</tr>
<tr>
<td>LVM/\textsubscript{Ht}\textsuperscript{2.7}</td>
<td>-0.042</td>
<td>0.251*</td>
<td>0.100</td>
<td>0.067</td>
<td>0.155</td>
<td>0.066</td>
<td>0.209*</td>
</tr>
</tbody>
</table>

*Significant at the level of \(p \leq 0.05\) (2-tailed). M-ABC2 = movement assessment battery for children \(2^{nd}\) edition, BMI = body mass index, FFM = fat free mass, BF(%) = percentage body fat, SBP = systolic blood pressure, DBP = diastolic blood pressure, VO\textsubscript{2} = peak oxygen uptake, SV = stroke volume, CO = cardiac output, LVM = left ventricular mass, LVM/\textsubscript{Ht}\textsuperscript{2.7} = left ventricle mass normalized to height\textsuperscript{2.7}.

Table 4.5 Regression analyses of stroke volume and cardiac output on p-DCD

<table>
<thead>
<tr>
<th>Stroke Volume</th>
<th>Model 1</th>
<th>p-value</th>
<th>Model 2</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>p-DCD</td>
<td>5.6 (0.21)</td>
<td>0.02</td>
<td>7.3 (0.27)</td>
<td>0.001</td>
</tr>
<tr>
<td>Height</td>
<td>0.47 (0.27)</td>
<td>0.006</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td>-3.7 (-0.14)</td>
<td>0.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>VO\textsubscript{2FFM}</td>
<td>0.45 (0.28)</td>
<td>0.002</td>
<td></td>
<td></td>
</tr>
<tr>
<td>FFM</td>
<td>0.37 (0.23)</td>
<td>0.03</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>56.3</td>
<td>-51.002</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adjusted R-squared</td>
<td>0.035</td>
<td>0.335</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cardiac Output</th>
<th>Model 1</th>
<th>p-value</th>
<th>Model 2</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>p-DCD</td>
<td>0.76 (0.34)</td>
<td>&lt;0.001</td>
<td>0.82 (0.37)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Height</td>
<td>0.036 (0.25)</td>
<td>0.01</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td>-0.21 (-0.094)</td>
<td>0.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>VO\textsubscript{2FFM}</td>
<td>0.016 (0.12)</td>
<td>0.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>FFM</td>
<td>0.031 (0.23)</td>
<td>0.02</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>4.17</td>
<td>-3.23</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adjusted R-squared</td>
<td>0.106</td>
<td>0.325</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Values are unstandardized b-coefficients with Betas in parentheses. p-DCD = probably developmental coordination disorder, FFM = fat free mass, VO\textsubscript{2FFM} = peak oxygen uptake normalized to FFM.
Chapter 5: Discussion

5.1 Introduction

The objective of this study was to compare LVM and cardiac function in children with and without significant motor impairments. In order to fulfill these objectives, echocardiography, as well as a test of motor coordination (M-ABC2) were performed on 126 participants. Of these participants, 63 cases (p-DCD) and 63 controls (without p-DCD) were assessed. We hypothesized that children with p-DCD would demonstrate elevated LVM/Ht^{2.7} compared to controls. The principle findings of this study indicated that children with p-DCD do not demonstrate an increased risk of CVD compared to controls, with normalized LVM not being significantly different between groups. However, a significant difference was evident in cardiac performance where SV and CO were elevated in children with p-DCD. Also, novel findings from this study demonstrate significant differences in basic cardiovascular measures such as SBP, DBP, and HR.

5.2 Body Composition and Cardiovascular Measures

This study used an accurate and reliable method of determining relative body fat in children with DCD, using whole body air-displacement plethysmography. Previous studies have shown, using bioelectrical impedance, BMI, and sum of skin folds that children with DCD have greater percentage body fat and are at increased risk of being overweight and obese. One study by Faught found that in 11.5 year old children, those with DCD had a higher percentage body fat than the non-DCD group using bioelectrical impedance. In a study by Cairney and colleagues (2005) bioelectrical impedance showed that boys with DCD demonstrated a greater body fat percentage
compared to controls, however, there was no significant difference in percent body fat between DCD and non-DCD in girls. Results from our study confirm the fact that those children with p-DCD have a greater percentage body fat and demonstrate increased prevalence of overweight and obesity. In the present study, percentage body fat for children with p-DCD compared to controls was 28% and 20% respectively, ranging from 6.7 to 48.6% and 5.6 to 51.6% in the p-DCD and control groups, respectively.

Furthermore, it was found that children with p-DCD had significantly higher BMI than controls. When subjects were grouped into BMI categories of normal weight, overweight, and obese according to recommended guidelines, 50.9% of the p-DCD were overweight/obese compared to only 22.2% of the controls. In total, 15 children were overweight and 17 were obese in the p-DCD group compared to 11 overweight and 3 obese in the controls. Interestingly, there was no difference observed in FFM between groups. In previous studies examining LVM in obese children, it has been shown that obese individuals have a greater amount of FFM compared to lean individuals which is likely an adaptation to the increased physical demands associated with increased weight. The fact that there was only a small difference in BMI between groups in our study may explain why we did not see a difference in FFM between groups.

Results from our study also provide further support that individuals with p-DCD demonstrate significantly lower peak VO₂ compared to their typically developing peers. It has previously been shown that children with DCD demonstrate reduced fitness in both field based studies as well as lab-based studies of peak VO₂. Wu et al.
(2009) reported peak VO₂ (ml/kg/min) in 9-11 year-old children from Taiwan to be 47.6 ± 6.1 ml/kg/min in typically developing children and 39.7 ± 6.6 ml/kg/min in children with DCD. In our study of 12 and 13-year olds we found that although the pDCD group reached a similar maximum HR (187 ± 15) as the controls (192 ± 13), peak VO₂ was 48.5 ± 7.5 ml/kgFFM/min in those with p-DCD compared to 53.2 ± 8.1 ml/kgFFM/min in controls.

Novel findings from this study were the differences found in basic cardiovascular measures in individuals with p-DCD compared to controls, such as resting HR, SBP, and DBP. In a study of physical fitness and health indices in children, adolescents, and adults of high and low motor competence, Cantell et al. (2008) found no significant differences in HR, SBP, and DBP between those with low and high motor competence. In our study, SBP was found to be 110 mmHg and 105 mmHg in the p-DCD and control groups, respectively. Diastolic BP was 69 mmHg and 65 mmHg, while resting HR in p-DCD was 80 beats/min compared to 75 beats/min in the controls. Seeing as how it has previously been shown that children with DCD are more likely to be overweight and obese, less physically active, and demonstrate reduced cardio-respiratory fitness, our findings are not surprising. Further investigation into BP and BP regulation is necessary to determine risk of future hypertension and CVD in children with p-DCD.

5.3 Left Ventricular Structure

Left ventricular hypertrophy and elevated LVM have been shown to be strong independent predictors of cardiovascular morbidity and mortality in adults. Major
determinants for pathological changes in LVM include obesity and hypertension. Given the growing rates of obesity and hypertension in children\textsuperscript{157}, research has been extended to children to determine whether obesity and hypertension increases risk of LVH. A recent study by Peralta-Huertas et al. (2008)\textsuperscript{22} found a significant difference in LVM/Ht\textsuperscript{2.7} between obese and normal weight boys and girls. This is consistent with findings from Maggio and colleagues (2007)\textsuperscript{23} who also found that in prepubertal children, LVM/Ht\textsuperscript{2.7} was significantly greater in obese compared to lean subjects. In this study, LVM/Ht\textsuperscript{2.7} was 36.1 ± 5.8 g/m\textsuperscript{2.7} in obese subjects, compared to 30.9 ± 5.7 g/m\textsuperscript{2.7} in lean controls. Our results show no difference in LVM/Ht\textsuperscript{2.7} between p-DCD and controls, indicating no increased risk for children with p-DCD compared to control subjects. The values for LVM normalized to height\textsuperscript{2.7} for p-DCD and controls in our study correspond to 26 ± 5 g/m\textsuperscript{2.7} and 26 ± 6 g/m\textsuperscript{2.7} respectively.

Several differences exist between our study population and that of Maggio et al. (2007)\textsuperscript{23} and Peralta-Huertas et al. (2009).\textsuperscript{22} Primarily, in our study BMI in the p-DCD was 23.4 ± 9 kg/m\textsuperscript{2} and 20.2 ± 4 kg/m\textsuperscript{2} in the control group, with a mean difference of 3.2 kg/m\textsuperscript{2}. Whereas BMI in Maggio’s study population was 25.3 ± 4.6 kg/m\textsuperscript{2} and 15.5 ± 1.5 kg/m\textsuperscript{2} in the obese and lean groups respectively. In Peralta-Huertas et al. (2009), BMI for normal weight boys and girls was 16.9 ± 0.3 kg/m\textsuperscript{2} and 17.1 ± 0.4 kg/m\textsuperscript{2} respectively, and 24.0 ± 0.9 kg/m\textsuperscript{2} and 25.7 ± 1.7 kg/m\textsuperscript{2} for overweight boys and girls respectively. Although examining obesity was not the purpose of this investigation, the fact that there was only a small difference in BMI between groups in our study may explain why we did not see a difference in LVM/Ht\textsuperscript{2.7} between groups. Another reasonable
explanation as to why there was no observed difference in LVM/Ht\textsuperscript{2.7} between groups could be due to the fact that the groups did not demonstrate a difference in FFM. Fat free mass has been strongly associated with LVM\textsuperscript{23,116,125} and has been suggested to be a better predictor of increased LVM than fat mass.\textsuperscript{23,113,116,126} In our study, FFM was 40.9 \pm 8.6 kg and 40.1 \pm 8.8 kg in the p-DCD and control groups, respectively.

5.4 Left Ventricular Function

Our results also demonstrate significant differences between groups in left ventricular diameter in diastole, EDV, SV, and CO where these values were consistently higher in the p-DCD group. Stroke volume and EDV in children with p-DCD were 62 \pm 14 ml and 91 \pm 19 ml respectively, and 56 \pm 13 ml and 84 \pm 19 ml in the controls. Our values for SV were slightly lower than those of Peralta-Huertas,\textsuperscript{22} however, EDV in the p-DCD was comparable to the values of the overweight boys and girls, 87.3 \pm 4.3 ml and 94.8 \pm 4.8 ml respectively.\textsuperscript{22} It has been well established from previous studies that SV and CO are elevated in obese subjects compared to normal-weight individuals.\textsuperscript{158-160} The increased values in SV and CO were not accompanied by changes in fractional shortening or ejection fraction, which suggests there is no evidence of altered contractility in the p-DCD group.

In multiple linear regression analyses, p-DCD alone explained 3.5\% of the variability in SV and 10.6\% of the variability in CO. Probable DCD remained a significant predictor of cardiac output and SV even after height, sex, VO\textsubscript{2FFM}, and FFM were entered into the model. In fact, in the model for SV, the unstandardized b-coefficient increased
from 5.6 to 7.3 when height, sex, VO_{2\text{FFM}}, and FFM, were included. This is similar to past studies where height, VO_{2\text{FFM}}, and FFM were also found to be independent predictors of SV.\textsuperscript{159,160,161} In contrast, for CO only height and FFM were significant predictors along with p-DCD. There is an observable suppression effect for p-DCD on SV and CO in which adding the variables of height, sex, cardio-respiratory fitness, and FFM caused the b-coefficient for p-DCD to increase. However, no interaction effects were significant in the model. Explanations into the effect of p-DCD on SV and CO output are beyond the scope of this investigation and are likely a result of one or several unmeasured variables.

As evidenced by the increased EDV in the p-DCD group, the difference in SV between groups is likely a result of obesity related changes in blood volume. Obesity in children has been associated with increased blood volume, SV, and CO.\textsuperscript{158} The difference observed in CO is not surprising given the elevated HR and SV. The elevated SV and CO do not indicate any pathological changes as LVM/Ht\textsuperscript{2,7\textsuperscript{2}}, ejection fraction, and fractional shortening are not different between groups. However, the results of this study are still important as it has been established that LVH occurs as a response to normalize left ventricular wall tension. An increase in SV, as a result of an increased preload, increases left ventricular cavity size.\textsuperscript{16,52} This leads to an increase in wall tension and therefore, increased left ventricular pressure. In order to normalize wall stress the left ventricle hypertrophies.\textsuperscript{52} Although the difference is small, LVd is significantly different between groups (p<0.05), indicating a slightly larger chamber size. Therefore, subjects in the p-DCD may be at risk of developing LVH in the future.
5.5 Limitations

It is important to note that several limitations to this study do exist. Primarily, we refer to children as having probable DCD (p-DCD) because we are unable to test whether these children meet the full criteria of the DSM-IV. We could not confirm that the disturbance in criterion A (e.g. Performance in daily activities that require motor coordination is substantially below that expected given the person's chronological age and measured intelligence) significantly interfered with academic achievement or ADL (criterion B). Also, four participants were included in the p-DCD group despite scoring a standard score below 70 on the intelligence test. Secondly, this is a cross-sectional study within a larger longitudinal study. Therefore, we cannot make any causal links between p-DCD and cardiovascular risk. Tracking these subjects over a period of time will provide a better understanding of the development of CVD risk factors in children with p-DCD, and to determine for certain whether these individuals are at an increased risk.

A final and important limitation to address in this study is the use of ultrasound echocardiography as a measurement tool for obtaining left ventricular measurements. Although it is widely used as a standard procedure for determining cardiac structure and function\textsuperscript{93, 94}, it comes with several limitations. The most significant echocardiographic limitation is inadequate image quality.\textsuperscript{93} Accurate measurements require optimal visualization of the endocardial border. Proper probe placement and imaging requires great technique and experience. For this study an experienced sonographer imaged the left ventricle. As well, to maintain consistency, the same sonographer completed the analysis of all images according to the proper guidelines set forth by the American
Society for Echocardiography\textsuperscript{94} and standard reference values were followed for proper landmarking.

5.6 Future Considerations

Although we may not see evidence of elevated LVM in children with p-DCD, there was evidence of early ventricular remodelling. Future studies should assess not only LVM in this population of children, but also if there is any evidence of concentric or eccentric remodelling, which may precede hypertrophy. Additionally, the findings from this study suggest that although there is no LVH in children with p-DCD, they do demonstrate differences in factors associated with LVH and elevated LVM such as obesity, elevated BP, and reduced cardio-respiratory fitness. As well, children with p-DCD demonstrate elevated SV and CO, which are likely to increase wall stress, resulting in future hypertrophy. In order to better understand the cardiovascular risk involved with p-DCD, longitudinal studies are necessary to determine if these differences are associated with future LVH. Longitudinal tracking will allow observations over time and therefore will give a better understanding of the importance of elevated SV and CO in those with p-DCD and whether it leads to elevated LVM or LVH.
Chapter 6: Conclusion

This is the first study to use laboratory measures to assess individuals identified with p-DCD. This investigation showed that children with p-DCD demonstrate negative health outcomes in body composition measures such as greater BMI and percentage body fat, cardiovascular measures such as elevated BP and HR, and reduced aerobic fitness compared to controls. Despite these findings however, there is no observable difference in LVM between groups. However, children with p-DCD demonstrate significantly elevated EDV, diastolic chamber size, SV, and CO. These differences may represent the early stages towards the development of left ventricle hypertrophy.
References


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95. Devereux RB. Detection of left ventricular hypertrophy by M-mode echocardiography. anatomic validation, standardization, and comparison to other methods. Hypertension 1987;9:119-126.


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121. Bella IN, Devereux RB, Roman MJ, O'Grady MJ, Welty TJ, Lee ET, Fabsitz RR, Howard BV. Relations of left ventricular mass to fat-free and adipose body mass: The strong heart study. Circulation 1998 December 8;98(23):2538-44.


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Appendix A
Research Ethics Approval

DATE: January 10, 2008

FROM: Michelle McGinn, Chair
       Research Ethics Board (REB)

TO: Brent FAUGHT, CHSC
    John Hay

FILE: 07-106 FAUGHT

TITLE: Establishing the Health Profile of Children with Motor Coordination Challenges

The Brock University Research Ethics Board has reviewed the above research proposal.

DECISION: Accepted as clarified

This project has received ethics clearance for the period of January 10, 2008 to December 30, 2011 subject to full REB ratification at the Research Ethics Board's next scheduled meeting. The clearance period may be extended upon request. The study may now proceed.
Appendix B
Informed Consent

CHILD LETTER OF INFORMED CONSENT

Principal Investigators: Dr. John A. Hay, Brock University
Dr. John Cairney, University of Toronto & Brock University
Dr. Brent E. Faught, Brock University

Dear Parent and Child:
Thank you for your interest in our study. Please read the following information together. If you both feel comfortable and willing to participate in the tests described below, please check the boxes at the end of this consent form indicating child and parent consent.

Purpose: The purpose of this study is to look at healthy growth and development of children for the next three years.

Procedures: This assessment will take approximately 2.5 to 3 hours long and is divided into three parts. We thank you for participating. As promised, we have agreed to provide transportation for you to and from Brock University as well as $50 ($20 for lab measures and $30 for home measures) for your family's participation in this study. Your participation is voluntary and you are free to withdraw from this study at any time without penalty from Brock University. Further, you are under no obligation to answer any or all questions or to participate in any aspect of this project. If you wish to stop participating in this study at any time, you and your parent will still receive free transportation from us as well as $20 for your participation in the lab measures. Each part is described below.

PART I
This part of the study will be conducted in our laboratory at Brock University and requires 2.5 to 3 hours of your time. First, we would like you to complete the following forms, which will take about 10 minutes.
1. Medical Screening Questionnaire
2. Edinburgh Survey – Handedness Questionnaire

Next, we would like to complete a number of physical assessments on your child with the parent/guardian present. These assessments include:
1. Body composition:
   a. Height and weight will be measured using a dual purpose stadiometer.
   b. 9 skinfold sites using painless pinch calipers. (It does not hurt).
   c. Measure around the waist and hip using a flexible tape measure.
   d. Bioelectric impedance analysis requires your child to stand on a weight scale and grasp handles. An electrical impulse travels from your child’s hands to their feet. The impulse cannot be felt and causes no harm.
   e. Lengths of your child’s ring and index fingers.
   f. Body muscle and fat weight will be measured while your child sits in the BOD POD chamber. If your child expressing previous or current anxiety for confined
spaces, they will not be allowed to participate in this portion of the study. The BOD POD incorporates a built-in window on the front of the chamber in the event of a claustrophobic event or for communication purposes as well as a safety latch on the inside of the chamber for the subject to voluntarily exit on their own. During this 5-minute assessment, your child will be asked to relax and breathe normally.

2. **Cardiovascular health measures:** The carotid ultrasound method will be performed using a probe and pen-like devices. Heart rate will be measured using sensors placed on the skin of your child’s chest. These sensors are used to detect the electrical activity generated by the heart and are not used to transmit electrical signals into their body from the heart rate monitor. Blood pressure is monitored using an automated arm cuff system that is similar to the method used in a doctor’s office. A cuff is wrapped around the upper arm and is inflated then deflated. No risk is involved.

3. **Movement ABC² assessment:** This motor coordination assessment involving 8 short activities, including tasks such as tracing, cutting on a line and throwing a ball.

4. **Physical fitness assessment:** This assessment uses a bicycle to measure the maximum amount of heavy exercise. The bicycle tension will gradually get more difficult to pedal. A mask over the mouth and nose will be used to collect oxygen and carbon dioxide. The assessment will be finished when your child decides. One of the common risks of this assessment is the brief sensation of exhaustion. At the end of the assessment, your child will be asked to continue to pedal the bicycle at a very easy level until this sensation goes away. The risk of serious illness or death is extremely rare and is reduced by completing the medical screening questionnaire before the assessment and the continuous monitoring we will perform during the assessment.

5. **Accelerometer assessment:** This assessment will require your child to wear a small box the size of a smaller pager clipped onto their pant waist. The accelerometer is designed to measure activity movement that your child performs. We wish for your child to wear the accelerometer from the time they wake up, until the go to bed at night for 7 days. We also ask that the parent complete the Habitual Activity Estimation Scale and our Activity Log. There is no risk associated with this assessment. We will make arrangements to pick the accelerometer unit at your home.

**PART II**

The second part of the study would take place approximately 7 days from now at your home. We would come in the morning (before your child has breakfast) and it will only take about 10 minutes. We wish to collect a sample of your child’s blood using a finger pinprick technique. The middle finger of your child’s non-dominant hand (e.g. if they are right-handed, we will use the middle finger of their left hand) will be pricked so two drops of blood can be sampled. Your child will feel a small prick, but will not feel any pain or discomfort for the remainder of the assessment. The tip of that finger may feel sensitive and a little bit sore for about a day. It is important to keep the site clean and covered with an adhesive bandage until it is healed to reduce the risk of infection. We will also use this moment to pick up the accelerometer that you will have had for the past week.

**PART III**

For this part of the study we would like you to allow your child’s homeroom teacher complete a survey on your child’s combined listening, speaking, reading, writing, mathematics and reasoning skills. The name of this survey is the Learning Disabilities Diagnostic Inventory. Despite the name
of this survey, we are not looking to diagnose any disabilities in your child’s learning ability, nor is the teacher expected to provide a learning disabilities’ diagnosis. We simply wish to see how able your child is while learning at school. The results of this assessment will not be shared with your child’s school.

*Participation and Withdrawal:* Your child’s participation is voluntary and they are free to withdraw from this study at any time without penalty from Brock University. Further, your child is not required to answer any or all questions or to participate in any aspect of this project.

*Confidentiality:* All personal data will be kept strictly confidential and all information will be coded so that your child is not associated with their answers. Only the researchers named above will have access to the complete data. Any information we receive will be entered immediately into computer records using a code number with no name attached. It is our intent to continue to publish the results of this research in scientific journals. Again, no personal information will be identified or be possible within any publication.

*Information:* This study has been reviewed and approved by the Brock University Research Ethics Board, (File#: 07-106) Research Services, Brock University, Room C315 - 905-688-5550 (Ext. 4315). We greatly appreciate your co-operation. If you would like to receive more information about the study, please contact Dr. Brent E. Faught at 905-688-5550, (Ext. 3586). If you are willing to grant permission to participate in this study, please complete the consent form below.

Thanks for your help!

Brent E. Faught, Ph.D. John A. Hay, Ph.D. John Cairney, Ph.D.

---

**PARENT CONSENT FORM**

I have read and understand the above explanation of the purpose and procedures of the project. My questions have been answered to my satisfaction.

- I give permission for my child to participate in **Part I** of the Brock University study conducted by Dr. John Hay, Dr. John Cairney and Dr. Brent E. Faught.

- As the participating child, I wish to participate in **Part I** of the Brock University study conducted by Dr. John Hay, Dr. John Cairney and Dr. Brent E. Faught.

- I give permission for my child to participate in **Part II** of the Brock University study conducted by Dr. John Hay, Dr. John Cairney and Dr. Brent E. Faught.
As the participating child, I wish to participate in Part II of the Brock University study conducted by Dr. John Hay, Dr. John Cairney and Dr. Brent E. Faught.

I give permission for my child to participate in Part III of the Brock University study conducted by Dr. John Hay, Dr. John Cairney and Dr. Brent E. Faught.

As the participating child, I wish to participate in Part III of the Brock University study conducted by Dr. John Hay, Dr. John Cairney and Dr. Brent E. Faught.

OR

I do NOT give permission for my child to participate in the Brock University study conducted by Dr. John Hay, Dr. John Cairney and Dr. Brent E. Faught.

As the participating child, I do NOT wish to participate in the Brock University study conducted by Dr. John Hay, Dr. John Cairney and Dr. Brent E. Faught.

Signature of Parent/Guardian: ___________________________ Date: ___________

Signature of Student: ________________________________ Date: ___________
Appendix C
ADVANCED HEALTH ASSESSMENT INFORMATION SHEET

Date: __________________________________________ Time (am/pm): _______________________

SECTION 1: STUDENT INFORMATION

<table>
<thead>
<tr>
<th>Student ID #:</th>
<th>Name:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Gender:</th>
<th>DOB:</th>
<th>Age:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male/Female</td>
<td><em><strong>/</strong></em>/____</td>
<td><em>(month)/</em>(day)/<em>(year)</em></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Height (cm):</th>
<th>Weight (kg):</th>
<th>BMI:</th>
</tr>
</thead>
<tbody>
<tr>
<td>___</td>
<td>___</td>
<td>___</td>
</tr>
</tbody>
</table>

SECTION 2: CONSENTS and QUESTIONNAIRES

<table>
<thead>
<tr>
<th>STUDENT (check for completeness)</th>
<th>PARENT (circle for completeness)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2. Medical Screening Questionnaire:</td>
<td>2. Medical Academic History Questionnaire:</td>
</tr>
<tr>
<td>4. Tanner Questionnaire:</td>
<td>4. Edinburgh Modified Parent Survey:</td>
</tr>
<tr>
<td>OR complete at home:</td>
<td></td>
</tr>
<tr>
<td>5. Accelerometer and Pkg (given):</td>
<td>5. Habitual Activity Estimation Scale:</td>
</tr>
<tr>
<td>Y     N (no consent)</td>
<td></td>
</tr>
<tr>
<td>6. Teacher Package (given):</td>
<td>6. Hypermobility Questionnaire:</td>
</tr>
<tr>
<td>Y     N (no consent)</td>
<td></td>
</tr>
<tr>
<td>7. Tanner Questionnaire Completed:</td>
<td>7. Accelerometer Log Completed:</td>
</tr>
<tr>
<td>Y     N</td>
<td>Y     N</td>
</tr>
<tr>
<td>8. “Two Days in My Life” Completed:</td>
<td>8. “Two Days in My Child’s Life” Completed:</td>
</tr>
<tr>
<td>Y     N</td>
<td>Y     N</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### SECTION 3: BODY COMPOSITION MEASURES

**Waist Circumference**

<table>
<thead>
<tr>
<th>Examiner:</th>
<th>Hip Circumference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trail #1:</td>
<td>Trail #1:</td>
</tr>
<tr>
<td>Trail #2:</td>
<td>Trail #2:</td>
</tr>
<tr>
<td>Mean:</td>
<td>Mean:</td>
</tr>
</tbody>
</table>

**Waist / Hip Ratio and Percentage**

<table>
<thead>
<tr>
<th>Ratio:</th>
<th>Percentage:</th>
</tr>
</thead>
</table>

**Bioelectric Impedence Analysis**

<table>
<thead>
<tr>
<th>Examiner:</th>
<th>Lean Body Mass:</th>
<th>Percent Body Fat:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basal Metabolic Rate:</td>
<td>kg</td>
<td>%</td>
</tr>
<tr>
<td>Body Fat Mass:</td>
<td>kg</td>
<td>kcal</td>
</tr>
</tbody>
</table>

### SECTION 3 CONTINUED: BODY COMPOSITION MEASURES

**Skinfold Measurements**

<table>
<thead>
<tr>
<th>Examiner:</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>SITE</th>
<th>TRIAL 1 (mm)</th>
<th>TRIAL 2 (mm)</th>
<th>TRIAL 3 (&gt;1mm)</th>
<th>MEAN (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>BICEPS</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TRICEPS</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CHEST</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SUBSCAPULAR</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MID-AXILLARY</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SUPRA-ILIAC</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ABDOMEN</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### THIGH

<table>
<thead>
<tr>
<th>MEDIAL CALF</th>
</tr>
</thead>
<tbody>
<tr>
<td>SUM OF SKIN FOLDS: __________ (mm)</td>
</tr>
</tbody>
</table>

### PERCENT BODY FAT

- 3 site – Jackson and Pollock: __________ (%)
- 4 site – Durnin and Wormersley: __________ (%)
- 7 site – Jackson and Pollock: __________ (%)

### BOD POD

**Examiner:**

<table>
<thead>
<tr>
<th>Fat Mass: __________ kg</th>
<th>Percent Body Fat: __________ %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fat Free Mass: __________ kg</td>
<td>Body Volume: __________ L</td>
</tr>
<tr>
<td>Body Mass: __________ kg</td>
<td>Body Density: __________ kg/L</td>
</tr>
<tr>
<td>Thoracic Gas Volume: __________ L</td>
<td></td>
</tr>
</tbody>
</table>

### Digits

**Examiner:**

<table>
<thead>
<tr>
<th>RIGHT HAND</th>
<th>LEFT HAND</th>
</tr>
</thead>
<tbody>
<tr>
<td>Digit #2 (pointer finger): __________ (mm)</td>
<td>Digit #2 (pointer finger): __________ (mm)</td>
</tr>
<tr>
<td>Digit #4 (ring finger): __________ (mm)</td>
<td>Digit #4 (ring finger): __________ (mm)</td>
</tr>
<tr>
<td>Right Hand Ratio (D2/D4): __________</td>
<td>Left Hand Ratio (D2/D4): __________</td>
</tr>
</tbody>
</table>

### SECTION 4: ARTERIAL MEASUREMENTS

**Doppler Settings**

**Examiner:**

- Frequency: 10.0 mHz
- Power: 0 dB
- Depth: __________ cm
- FPS: change focus # (decrease to 2) to increase fps
- Persistence: turn to minimum

**Blood Pressure - Manual**
<table>
<thead>
<tr>
<th>Pre</th>
<th>1</th>
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</thead>
<tbody>
<tr>
<td></td>
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<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
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<tr>
<td>Post</td>
<td>1</td>
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<td></td>
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<td></td>
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</tr>
</tbody>
</table>

**SECTION 5: LEFT VENTRICULAR MASS MEASUREMENTS**

**Examiner:**

**Probe:**

**Depth:** cm

**B-Mode Images:**

**M-Mode Images:**

**Distance Measurements**

Sternal notch to toe: cm | Sternal notch to carotid: cm

**Notes for Cardiovascular Component**

<p>| SECTION 5 CONTINUED: LEFT VENTRICULAR MASS MEASUREMENTS |</p>
<table>
<thead>
<tr>
<th>Interventricular Septum (end-distole):</th>
<th>Ejection Fraction:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Left Ventricular Diameter (end-diastole):</td>
<td>Circumferential Shortening:</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Posterior Wall (end-diastole):</td>
<td>LVM:</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Left Ventricular Diameter (end-systole):</td>
<td></td>
</tr>
</tbody>
</table>

**SECTION 6: VO₂ MAX**

**Examiner:**

**Bike Instructions**

RPM: 60 – 80 rpm

Begin Test: 20 watts

Increment Changes: 20 watt increase every 2 minutes

Finish Test: volitional drop out; heart rate reaches max (220-age), expiratory ratio is ≥ 1.1, or of the VO₂ peak plateaus

**Heart Rate**

<table>
<thead>
<tr>
<th>Rest: _______ bpm</th>
<th>W: _______ bpm</th>
</tr>
</thead>
<tbody>
<tr>
<td>______ W: _______ bpm</td>
<td>______ W: _______ bpm</td>
</tr>
<tr>
<td>______ W: _______ bpm</td>
<td>______ W: _______ bpm</td>
</tr>
<tr>
<td>______ W: _______ bpm</td>
<td>______ W: _______ bpm</td>
</tr>
<tr>
<td>______ W: _______ bpm</td>
<td>______ W: _______ bpm</td>
</tr>
</tbody>
</table>

Final VO₂: _______ ml/kg

MAX Heart Rate: _______ b/min

Final Duration: _______ min

Watts: _______ W

Final Stage: _______

Final RER: _______

Last RPE Report: _______

**Notes:** (Please note any changes to protocol, problems during testing, medical conditions that would hinder test results)
SECTION 7: BLOOD ANALYZER

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>TC:</td>
<td>______ mg/dL</td>
</tr>
<tr>
<td>HDL:</td>
<td>______ mg/dL</td>
</tr>
<tr>
<td>TRG:</td>
<td>______ mg/dL</td>
</tr>
<tr>
<td>LDL:</td>
<td>______ mg/dL</td>
</tr>
<tr>
<td>Non-HDL:</td>
<td>______ mg/dL</td>
</tr>
<tr>
<td>TC/HDL:</td>
<td>______ mg/dL</td>
</tr>
<tr>
<td>GLU:</td>
<td>______ mg/dL</td>
</tr>
<tr>
<td>GLU:</td>
<td>______ mmol/L</td>
</tr>
</tbody>
</table>

Notes: (Please note any changes to protocol, problems during testing, other circumstances that would hinder test results)

EXTRA MEASUREMENTS:

Section 4 Continued: Arterial Measurements

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diastolic Diameter:</td>
<td>______ mm</td>
</tr>
<tr>
<td>Heart Rate:</td>
<td>______ bpm</td>
</tr>
<tr>
<td>Systolic Diameter:</td>
<td>______ mm</td>
</tr>
<tr>
<td>Automated Systolic Arterial Pressure:</td>
<td>______ mmHg</td>
</tr>
<tr>
<td>Diameter Change:</td>
<td>______ mm</td>
</tr>
<tr>
<td>Automated Diastolic Arterial Pressure:</td>
<td>______ mmHg</td>
</tr>
<tr>
<td>Carotid Pulse Pressure:</td>
<td>______ mmHg</td>
</tr>
<tr>
<td>Mean Arterial Pressure:</td>
<td>______ mmHg</td>
</tr>
<tr>
<td>Compliance:</td>
<td>______ mm/mmHg</td>
</tr>
<tr>
<td>Automated Pulse Pressure:</td>
<td>______ mmHg</td>
</tr>
<tr>
<td>Distensibility:</td>
<td>______ %</td>
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</table>

Section 5 Continued: Left Ventricular Mass Measurements

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>End Diastolic Volume:</td>
<td>______ ml</td>
</tr>
<tr>
<td>Stroke Volume:</td>
<td>______ ml</td>
</tr>
<tr>
<td>End Systolic Volume:</td>
<td>______ ml</td>
</tr>
<tr>
<td>LMVbsa:</td>
<td>______ g/m²</td>
</tr>
</tbody>
</table>
Appendix D
Movement Assessment Battery for Children, Version 2
Manual Dexterity 1: TURNING PEGS

Record: Preferred hand: R/L ( Humphreys' or as for Drawing Tool); Time taken (sec); "F" for failure; "R" for refusal; "I" if inappropriate (note reasons below)

<table>
<thead>
<tr>
<th>Preferred hand</th>
<th>Non-preferred hand</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Time (sec)</td>
</tr>
<tr>
<td>1st Peg</td>
<td></td>
</tr>
<tr>
<td>2nd Peg</td>
<td></td>
</tr>
</tbody>
</table>

Qualitative observations

Posture/body control

Sitting posture is poor

Hand movements are jerky

Hand held too close to task

Moves constantly/charges

Hand held at an odd angle

Adjustment to task requirements

Does not look while inserting peg

Misaligns pegs with respect to holes

Does not use proper grip to pick up pegs

Uses excessive force when inserting pegs

Exaggerates finger movements in releasing pegs

Is exceptionally slow/hexes not change speed from trial to trial

Does not use the supporting hand to hold base-headers

Greats will fall for accuracy

Does extremely poorly with one hand (asymmetry noticeable)

Other

Changes hands or uses both hands during a trial

Comments

---

Manual Dexterity 2: TRIANGLE WITH NUTS AND BOLTS

Record: Time taken (sec); "F" for failure; "R" for refusal; "I" if inappropriate (note reasons below)

<table>
<thead>
<tr>
<th>No. of seconds</th>
<th>Time (sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st Peg</td>
<td></td>
</tr>
<tr>
<td>2nd Peg</td>
<td></td>
</tr>
</tbody>
</table>

Qualitative observations

Posture/body control

Sitting posture is poor

Hand movements are jerky

Hand held too close to task

Moves constantly/charges

Hand held at an odd angle

Adjustment to task requirements

Does not look at hole while inserting bolt

Sometimes misses hole with tip of bolt

Does not use proper grip to hold nuts and bolts

Gets muddled in the construction sequence

Finds it difficult to hold bolt with one hand and screw nut on with the other

Is exceptionally slow/hexes not change speed from trial to trial

Changes hands or uses both hands during a trial

Comments
Manual Dexterity 3: DRAWING TRAIL 3

Note: Bic/Atlantic pen to be used

Record: H for hand; B for both; No. of errors if for failure; R for refusal; I if inappropriate (note reasons below)
Number of errors should be counted after testing using scoring criteria provided in Appendix A of the Manual.

<table>
<thead>
<tr>
<th>No. of errors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trial 1</td>
</tr>
<tr>
<td>Trial 2</td>
</tr>
</tbody>
</table>

Qualitative observations

Posture/body control
- Sitting position is poor
- Holds head too near paper
- Holds head at an odd angle
- Does not look at trail
- Holds pen with an odd/awkward grip
- Holds pen too far from palm
- Holds pen too close to paper
- Does not hold paper still

Comments:

Aiming & Catching 1: CATCHING WITH ONE HAND

Record: Number of incorrectly executed catches; R for refusal; I if inappropriate (note reasons below)

Right Hand Practice: 10 Trials: Total:____
Left Hand Practice: 10 Trials: Total:____

Qualitative observations

Posture/body control
- Sitting position is poor
- Does not follow trajectory of ball with eyes
- Turns away or closes eyes as ball approaches
- Holds hand out flat, with fingers stiff as the ball rebounds
- Hands and arms held wide apart, fingers extended
- Arm and hand swing out to meet impact of ball
- Fingers close too early or too late
- Does extremely poorly with one hand (paying very little)
- Movements last minute

Adjustment to task requirements:
- Does not adjust body position for catching
- Does not adjust position of feet
- Judges toss of throw poorly (too much or too little)
- Does not adjust to height of rebound
- Does not adjust to force of rebound
- Other
**Aiming & Catching 2: THROWING AT WALL TARGET**

Record: Hand used: R / L / Both, number of successful trials: R for refusal, 1 if inappropriate (note reasons below)

Practice: 

10 Trials: 

Total: 

Qualitative observations:

**Posture/Body control**
- Balance while throwing is poor
- Does not keep eye on target
- Does not follow through with the throwing arm
- Releases too early or too late
- Changes hands from side to side
- Movements lack fluency

**Adjustment to task requirements**
- Errors are consistently to one side of the target
- Inconsistent striking
- Control of direction is variable
- Litters form of throw poorly (too much or too little)
- Control of force is variable
- Other

Comments:

---

**Balance 1: TWO-BOARD BALANCE**

Record: Time balanced (seconds), R for refusal, 1 if inappropriate (note reasons below)

<table>
<thead>
<tr>
<th></th>
<th>No. of seconds</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trial 1</td>
<td></td>
</tr>
<tr>
<td>Trial 2</td>
<td></td>
</tr>
</tbody>
</table>

Qualitative observations:

**Posture/Body control**
- Body appears stable
- Body appears Tilted
- Sways widely to try to maintain balance
- Does not hold head and eyes steady
- Makes too few compensatory arm movements to help maintain balance

Notes: Do not administer a second trial if the child maintains balance for 30 seconds

Comments:
Balance 2: WALKING TOE-TO-HEEL BACKWARDS

Record: Number of correct Emirian steps from the beginning of the line. Whether entire line was walked successfully: YES / NO. If inappropriate, note reasons below.

<table>
<thead>
<tr>
<th>Trial</th>
<th>No. of steps</th>
<th>Line?</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Qualitative observations
- Posture/body control
  - Body appears rigid/tense
  - Body appears hunched
  - Swings widely to try to maintain balance
  - Does not look behind to check position on track
  - Does not alternate arm swing to maintain balance
  - Exaggerated arm movements disrupt balance
  - Wobbly while placing feet on line

Adjustments to task requirements
- Does not move for accuracy
- Individual movements lack smoothness and fluency
- Sequencing of steps is not smooth/pauses frequently
- Other

Comments:

Balance 3: ZIG-ZAG HOPPING

Record: Number of correct Zig-zag hops (maximum of 5). YES / NO. If inappropriate, note reasons below.

<table>
<thead>
<tr>
<th>Right Leg</th>
<th>Left Leg</th>
<th>Right Leg</th>
<th>Left Leg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trial 1</td>
<td>Trial 1</td>
<td>Trial 2</td>
<td>Trial 2</td>
</tr>
</tbody>
</table>

Qualitative observations
- Posture/body control
  - Body appears rigid/tense
  - Body appears hunched
  - Non-supporting leg held up in front of body
  - Legs with stiff legs and flat feet
  - Legs coming off push-off from leg
  - Arm movements are exaggerated
  - Does not use arms to assist hop
  - Stumbles on landing

Adjustments to task requirements
- Does extremely poorly with one leg (symmetry striking)
- Does too fast for accuracy
- Does not alternate arm swing and forward movements effectively
- Too much effort
- Other
Trial 1

Trial 2

Practice
### NON-MOTOR FACTORS THAT MIGHT AFFECT MOVEMENT

Complete the sections below by noting any features of the child's behaviour during testing that you suspect might have affected his or her motor performance. Headings (with examples) are given as guidelines only. Although negative aspects are given more emphasis, remember to note positive aspects of the child's behaviour.

<table>
<thead>
<tr>
<th></th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Disorganised (e.g., scattered clothes slows up dressing after PE; puts on shoes before socks).</td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>Hesitant/forgetful (e.g., slow to start complex actions; forgets what to do in the middle of an action sequence).</td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Passive (e.g., hard to interest, requires much encouragement to participate).</td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>Timid (e.g., fearful of activities such as jumping, climbing, constantly asks for assistance).</td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>Anxious (e.g., trembles, becomes flustered in a stressful situation).</td>
<td></td>
</tr>
<tr>
<td>6.</td>
<td>Impulsive (e.g., starts before instructions are complete; impatient with detail).</td>
<td></td>
</tr>
<tr>
<td>7.</td>
<td>Distractible (e.g., looks around, responds to irrelevant noise).</td>
<td></td>
</tr>
<tr>
<td>8.</td>
<td>Overactive (e.g., squirms and fidgets, moves constantly while listening to instructions, fiddles with clothes).</td>
<td></td>
</tr>
<tr>
<td>9.</td>
<td>Overestimates own ability (e.g., tries to make tasks more difficult; tries to do things too fast).</td>
<td></td>
</tr>
<tr>
<td>10.</td>
<td>Underestimates own ability (e.g., complains of task difficulty, anticipates failure before starting).</td>
<td></td>
</tr>
<tr>
<td>11.</td>
<td>Lacks persistence (e.g., gives up quickly, is easily frustrated).</td>
<td></td>
</tr>
<tr>
<td>12.</td>
<td>Upset by failure (e.g., looks fearful, refuses to try task again).</td>
<td></td>
</tr>
<tr>
<td>13.</td>
<td>Unable to get pleasure from success (e.g., fails to respond to praise).</td>
<td></td>
</tr>
</tbody>
</table>

Other (please specify)...

Overall, do you think these problems prevent the child from demonstrating his or her true movement capability? (Please circle)
- Not at all
- A little
- A great deal

### PHYSICAL FACTORS THAT MIGHT AFFECT MOVEMENT

<p>| |</p>
<table>
<thead>
<tr>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Anatomical/postural defect: YES/NO. Specify if possible.</td>
</tr>
<tr>
<td>Vision defect: YES/NO.</td>
</tr>
<tr>
<td>Hearing defect: YES/NO.</td>
</tr>
<tr>
<td>Judgement of weight: average/overweight/underweight.</td>
</tr>
<tr>
<td>Judgement of height: average/tall/short.</td>
</tr>
<tr>
<td>Other.</td>
</tr>
</tbody>
</table>
### Table 2a: Brief summary of changes made to AB1 - now covers ages 3 to 6 years

<table>
<thead>
<tr>
<th>Task</th>
<th>Movement ABC AB1</th>
<th>Movement ABC-2 AB1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Manual Dexterity 1</td>
<td>Posting Coins</td>
<td>Posting Coins</td>
</tr>
<tr>
<td>Manual Dexterity 2</td>
<td>Threading Beads</td>
<td>Threading Beads</td>
</tr>
<tr>
<td>Manual Dexterity 3</td>
<td>Bicycle Trail</td>
<td>Drawing Trail 1*</td>
</tr>
<tr>
<td>Aiming &amp; Catching 1</td>
<td>Catching Beanbag</td>
<td>Catching Beanbag</td>
</tr>
<tr>
<td>Aiming &amp; Catching 2</td>
<td>Rolling Ball into Goal</td>
<td>Throwing Beanbag onto Mat**</td>
</tr>
<tr>
<td>Balance 1</td>
<td>One-Leg Balance</td>
<td>One-Leg Balance</td>
</tr>
<tr>
<td>Balance 2</td>
<td>Walking Heels Raised</td>
<td>Walking Heels Raised</td>
</tr>
<tr>
<td>Balance 3</td>
<td>Jumping over Cord</td>
<td>Jumping on Mats**</td>
</tr>
</tbody>
</table>

* Altered item: shape of trail has changed
** New item

### Table 2b: Brief summary of changes made to AB2 and AB3 - now labelled AB2 and covers ages 7 to 10 years

<table>
<thead>
<tr>
<th>Task</th>
<th>Movement ABC AB2</th>
<th>Movement ABC AB3</th>
<th>Movement ABC-2 AB2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Manual Dexterity 1</td>
<td>Placing Pegs</td>
<td>Shifting Pegs by Rows</td>
<td>Placing Pegs</td>
</tr>
<tr>
<td>Manual Dexterity 2</td>
<td>Threading Lace</td>
<td>Threading Nuts on Bolt</td>
<td>Threading Lace^</td>
</tr>
<tr>
<td>Manual Dexterity 3</td>
<td>Flower Trail</td>
<td>Flower Trail</td>
<td>Drawing Trail 2*</td>
</tr>
<tr>
<td>Aiming &amp; Catching 1</td>
<td>Two-Hand Catch</td>
<td>One-Hand Bounce and Catch</td>
<td>Catching with Two Hands</td>
</tr>
<tr>
<td>Aiming &amp; Catching 2</td>
<td>Throwing Beanbag into Box</td>
<td>Throwing Beanbag into Box</td>
<td>Throwing Beanbag onto Mat**</td>
</tr>
<tr>
<td>Balance 1</td>
<td>Stork Balance</td>
<td>One-Board Balance</td>
<td>One-Board Balance</td>
</tr>
<tr>
<td>Balance 2</td>
<td>Heel-to-Toe Walking</td>
<td>Ball Balance</td>
<td>Walking Heel-to-Toe Forwards</td>
</tr>
<tr>
<td>Balance 3</td>
<td>Jumping in Squares</td>
<td>Hopping in Squares</td>
<td>Hopping on Mats+</td>
</tr>
</tbody>
</table>

Altered items:
- New start position/layout
-^ Lacing board now longer
- * Shape of trail has changed
- ** Mat with target now used instead of box
+ Mats used for this task

### Table 2c: Brief summary of changes made to AB4 - now labelled AB3, covering ages 11 to 16

<table>
<thead>
<tr>
<th>Task</th>
<th>Movement ABC</th>
<th>Movement ABC-2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Manual Dexterity 1</td>
<td>Turning Pegs</td>
<td>Turning Pegs</td>
</tr>
<tr>
<td>Manual Dexterity 2</td>
<td>Cutting-Out Elephant</td>
<td>Triangle with Nuts and Bolts^</td>
</tr>
<tr>
<td>Manual Dexterity 3</td>
<td>Flower Trail</td>
<td>Drawing Trail 3*</td>
</tr>
<tr>
<td>Aiming &amp; Catching 1</td>
<td>One-Hand Catch</td>
<td>Catching with One Hand</td>
</tr>
<tr>
<td>Aiming &amp; Catching 2</td>
<td>Throwing at Wall Target</td>
<td>Throwing at Wall Target</td>
</tr>
<tr>
<td>Balance 1</td>
<td>Two-Board Balance</td>
<td>Two-Board Balance</td>
</tr>
<tr>
<td>Balance 2</td>
<td>Walking Backwards</td>
<td>Walking Toe-to-Toe Backwards</td>
</tr>
<tr>
<td>Balance 3</td>
<td>Jumping and Clapping</td>
<td>Zig-Zag Hopping^</td>
</tr>
</tbody>
</table>

^ New items
* Altered item: Shape of trail has changed
Appendix E
Rating of Perceived Exertion

Ratings of Perceived Exertion

6
7   VERY, VERY LIGHT
8
9   VERY LIGHT
10
11  FAIRLY LIGHT
12
13  SOMewhat HARD
14
15  HARD
16
17  VERY HARD
18
19  VERY, VERY HARD
20
APPENDIX F
Tanner Staging Pictures

Male Pubertal Stage

FACULTY OF APPLIED HEALTH SCIENCES
BROCK UNIVERSITY

This survey will be used to assess the maturational levels of the participant. For each photo choose the appropriate stage and place an X in the corresponding square.

- Please circle the box that looks most like you
- Please look at the penis size only
- Please look at the pubic hair only
- Please circle the box that looks most like you
Female Pubertal Stage

BROCK UNIVERSITY

FACULTY OF APPLIED HEALTH SCIENCES

This survey will be used to assess the maturational levels of the participant. For each photo choose the appropriate stage and place an X in the corresponding square.

Directions: You should choose only one of the stages shown below. One stage for Breast development and one stage for Pubic Hair development.

Study Subject No:

- Please put a tick in the box that looks most like you now....

1. The breasts are flat.
   - No hairs

2. The breasts form small mounds.
   - Very little hair

3. The breasts form larger mounds than in 2.
   - Quite a lot of hair

4. Nipple, Areola, Breast
   - The hair has not spread over the thighs

5. Only the nipple sticks out beyond the breast.
   - The hair has spread over the thighs

Please answer the following questions:

1. Have you had your period? YES NO

2. How old were you when you had your first period? ___________________

3. How often do you get periods? (in days) ___________________