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Correlation Between Hamstring Spasticity and Range of Motion and Selected Gait Parameters in Pediatric Clients with Spastic Diplegia

Erin McCain Glace
Old Dominion University

CORRELATION BETWEEN HAMSTRING SPASTICITY AND RANGE OF MOTION AND SELECTED GAIT PARAMETERS IN PEDIATRIC CLIENTS WITH SPASTIC DIPLEGIA

by

Erin McCain Glace
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Approved by:

Dr. John L. Echternach

Professor Evangeline Yoder

Jan Henderson, MSPT

ABSTRACT

CORRELATION BETWEEN HAMSTRING SPASTICITY AND RANGE OF MOTION AND SELECTED GAIT PARAMETERS IN PEDIATRIC CLIENTS WITH SPASTIC DIPLEGIA

Erin McCain Glace

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Spasticity is often considered the primary limitation to function in children with spastic cerebral palsy. The purpose of this study was to study the relationship between hamstring spasticity and a functional activity, specifically gait. The gait parameters chosen were step length, stride length and velocity. A secondary purpose was to study the relationship between hamstring contracture and the same gait parameters. Reliability data were calculated for tone and ROM measurements. Eleven subjects (8 male and 3 female) between the ages of three years and fifteen years with a primary diagnosis of spastic diplegia were recruited for this study.

Hamstring spasticity was graded using the modified Ashworth scale. Hamstring ROM measurements were taken by measuring popliteal angle using standard goniometric procedure. Velocity was measured with a stopwatch and a

measured paper walkway. The subjects ambulated on the paper walkway with inked pads on their shoes for the temporal measurements (stride and step length). Spearman rank correlation coefficients and Pearson product moment correlation coefficients were used for spasticity and ROM calculations, respectively. Critical values were used to determine significance at the .05 alpha level. Intraclass correlation coefficients were used for reliability measures.

The correlation coefficients for hamstring spasticity and the gait parameters ranged from poor (r_s=-.305) to good (r_s=-.877). Ashworth scale intertester reliabilty for this population was poor (ICC=.242) to fair (ICC=.613) and intratester reliability was fair (ICC=.487) to good (ICC=.941). Pearson product moment correlation coefficients for hamstring ROM and gait parameters was moderate (r=.685) to good (r=.840). Reliablity was good to high for intertester and intratester measurements.

Based on the results of this study, the relationship between spasticity and functional gait parameters is unclear, and the reliability of the modified Ashworth scale as applied to this type of pediatric client is questionable. The relationship between hamstring ROM and selected functional gait parameters was significant and measurement of the popliteal angle was reliable. Study findings suggest that treatment of hamstring range of motion deficits may be one key to improving functional gait.

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CHAPTER ONE

INTRODUCTION

Patients with upper motor neuron lesions present with a constellation of complex clinical signs and symptoms. Spasticity is one component or impairment resulting from an upper motor neuron lesion which is very difficult to assess. The impact of spasticity on function is controversial.

The International Classification of Impairments, Disabilities and Handicaps (ICIDH)[1] defines impairment as any loss or abnormality of psychological, physiological or anatomical structure or function. A disability is defined as any restriction or lack of ability (resulting from an impairment) to perform an activity in the manner or within the range considered normal for a human being.[1] Spasticity is an impairment which may cause disability by limiting function. Dekker et al.[2] state that relationships found between the impairment and disabilities are chosen as a basis for forming treatment goals and the interventions that are applied. One of the primary goals of physical therapists is to improve function. In order to improve function, therapists must be knowledgeable about which

manifestations of disease are the impairments limiting function.

Many studies have addressed the issue of defining and quantifying spasticity, but few have addressed the issue of determining the relationship between spasticity as an impairment and its direct relationship to functional activities. Limited range of motion is also a motor control impairment and its direct relationship to functional disability is assumed, but studies defining clear relationships to function are few.

This study was initiated to study the effects of two common upper motor neuron clinical impairments, spasticity and range of motion limitation, on functional gait.

Purpose of Study

The purposes of this study were to examine the relationship between hamstring spasticity and these gait parameters- step length, stride length, and velocity, and to examine the relationship between hamstrings contracture and step length, stride length, and velocity. The chosen study population was a group of pediatric patients with a diagnosis of spastic diplegia.

Literature Review

The aims of this literature review are to:

1. Define the concept of upper motor neuron lesion and two impairments which frequently occur with an upper motor neuron lesion (spasticity and muscle contracture).
2. Report the literature available on clinical measures used to assess the impairments.
3. Review the mechanics of normal gait and the impact of clinical impairments on functional gait in the spastic diplegic population.

Upper Motor Neuron Lesions

Adams and Victor[3] define upper motor neuron paralysis as involvement of the several descending fiber systems that influence and modify the lower motor neuron. Afifi and Bergman[4] list these characteristics of upper motor neuron paralysis: (1) weakness or loss of movement, (2) exaggerated deep tendon reflexes, (3) clonus, (4) abnormal superficial reflexes (eg, Babinski) and (5) spasticity. Adams and Victor[3] add: (1) residual or associated movements are present and (2) groups of muscles are always involved, never individual muscles.

Spasticity, as a component of an upper motor neuron lesion, is poorly understood. Spasticity is often associated with injury to the corticospinal tract or pyramidal system as opposed to the extrapyramidal system. The extrapyramidal system, when strictly interpreted, refers

to all motor pathways outside the pyramidal system. Adams and Victor[3] state that the term extrapyramidal becomes more meaningful when used to describe the basal ganglia and the cerebellum. Disease of these areas of the brain results in clinical syndromes that differ significantly from disease manifestations associated with the pyramidal system. For example, a patient with an extrapyramidal lesion would experience disturbed muscle tone in the form of rigidity rather than spasticity.[3] The corticospinal tract originates in the cortex (primarily from areas 4 and 6, and supplemental areas 3,1,2 and 5) and descends through the posterior limb of the internal capsule, midbrain, pons and into the pyramids of the medulla. Within the pyramids, the majority of fibers decussate at the medulla-spinal cord junction.[3,5] This pathway is the only motor control pathway which directly controls voluntary movement through its direct connection to the alpha motor neuron (lower motor neuron).

In addition to the corticospinal pathway, several other descending pathways exist which influence motor control. These are the rubrospinal, reticulospinal, vestibulospinal and tectospinal.[3,5,6,7] These tracts are thought to have significant influence on voluntary movement and when lesioned may be involved in spasticity.[3,5,6] Studies have shown that isolated lesions of the medullary pyramids

actually produce hypotonia[3,5,6,7,8] and significant recovery often occurs.[3] Noback et al.[5] state that the only neurologic sign associated exclusively with a corticospinal tract lesion is the Babinski sign. Thus, a lesion producing classic upper motor neuron signs which occurs in the internal capsule or cortical white matter may be attributed to the corticospinal tract, but in fact, there are several descending pathways that may play a role in producing spasticity which is secondary to an upper motor neuron lesion.

Neurophysiological mechanism of spasticity

Numerous studies have explored the entity of spasticity to determine its physiological basis. The classic definition of spasticity is:

> a motor disorder characterized by a velocity dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks from the hyperexcitability of the stretch reflex as one component of the Upper Motor Neuron Syndrome.[9]

A prevailing theory of the mechanism of spasticity is that there is impaired reciprocal inhibition of antagonistic muscles during voluntary active movement.[10,11,12] This impaired inhibition results in "synchronous activation" or cocontraction between agonist and antagonistic muscle groups.[10] Clinically, this is believed to be a major

limitation to function. Iles et al.[12] state that there appears to be a relationship between loss of inhibition and the severity of weakness. Gottlieb et al.[13] and Myklebust et al.[14], in studies using electromyographic measurements evoked in soleus and anterior tibialis muscles by tendon tap found that reciprocal excitation may be the cause of cocontraction and decreased function in contrast to impaired reciprocal inhibition.

Separate studies by Powers et al.[15] and Corcos et al.[16] have focused on the stretch reflex. Both authors concluded that a decreased threshold or hyperexcitable stretch reflex is related to spasticity and corresponding movement deficits. Corcos et al.[16] expanded on the relationship of spasticity relative to the individual patient. They stated that hyperactive reflexes cause movement deficits only in select patients. Based on EMG studies, Sahrmann and Norton[17] refuted the hyperexcitable stretch reflex as being a significant factor in causing functional limitation in clients with upper motor neuron syndrome because blocking fusimotor activity with local anaesthesia did not significantly improve function. Moreover, they found that the movement deficit may be caused instead by limited and prolonged recruitment in the agonist muscle at its termination of movement and by inappropriate overflow and maintenance of contraction when the agonistic movement is

reversed.

Thilmann et al.[18] have suggested that there are clinical differences between spasticity in the initial stages of a disorder and spasticity as a chronic impairment. Through EMG studies of the biceps in hemiparetic and normal subjects, these authors have found late EMG activity in the hemiparetic subjects. They concluded that this reflex EMG activity arose, not from a reduction in the threshold of stretch reflexes, but from a pathological increase in stretch reflex gain. In clinically impaired subjects where spasticity had been established for a year or more, the major cause of spasticity appeared to be a pathological increase in stretch reflex activity.

Functional Characteristics of Spasticity

Although many theories exist about the mechanism of spasticity, there are specific criteria used to identify its clinical characteristics. Adams and Victor[3] list the following characteristics of spasticity: (1) a predilection for involvement of certain muscle groups (predominantly antigravity muscle groups-- flexors of the upper extremities and extensors of the lower extremities), (2) a specific pattern of response to stretch (resistance increases linearly in relation to the velocity of stretch)

and (3) a manifest exaggeration of tendon reflexes. Patterns of paralysis due to disease of the upper motor neuron also exist and are described as follows: (1) monoplegia- weakness or paralysis of all muscles of one arm or leg, (2) hemiplegia- weakness or paralysis involving the arm, leg and sometimes face on one side of the body, (3) paraplegia- weakness or paralysis of both legs, (4) quadriplegia or tetraplegia- weakness or paralysis of all four extremities and (5) diplegia- a form of quadriplegia in which the legs are more affected than the arms.[3]

Management of Spasticity

Parizale et al.[19] have described several different methods for the management of spasticity including physical modalities, pharmacologic treatment, nerve blocks and surgical management. Orthopedic surgery as a mode of treatment remediates by lengthening or realigning the abnormal muscle tendon unit and using bony reconstruction.[20] Other surgical management includes the relatively recent acceptance of the selective dorsal rhizotomy as a means to diminish spasticity. Selective ablation of the posterior roots during the rhizotomy interrupts fibers in the spinal reflex arc. The operation appears to diminish spasticity, which can increase range of motion and improve functional ambulation.[19,21] Patients must be carefully selected for the

procedure. In some cases, functional lower extremity weakness continues following the surgery ,especially if the child had poor selective control prior to surgery.[22]

Quantification of Spasticity

Although universal agreement regarding the clinical definition of spasticity does not exist, clinicians evaluate the phenomenon as a part of upper motor neuron syndrome and consider it to be an impairment to a patients functional ability. Quantification of spasticity has been addressed in many ways. Bohannon et al.[23] and Brown et al.[24], in separate studies, used the concept of pendulum testing. Brown et al.[24] used an electrogoniometer with subsequent analysis of a microcomputer to assess resistance to movement as the subjects knees were allowed to swing freely, hence, the name pendulum. The subjects were healthy, elderly volunteers. Brown[24] states that voluntary contraction may be a factor limiting the validity of the test. Bohannon et al.[23] have described how the Cybex II isokinetic dynamometer can be used to document spasticity using the pendulum method. The Cybex II dynamometer is used to measure passive resistance in the patient's quadriceps. Katz[6] points out that the pendulum model suffers from the questionable assumption that mechanical properties of knee extensor and flexor muscles are equal.

A number of studies[25,26,27] have addressed the issue of measuring muscle spasticity by measuring muscle torque. Katz[6] defines torque as "the amount of force elicited by moving a limb over a specified angle". He states that torque measurements at a specified angle are the best index of hypertonia available at the time.

There are several easily used clinical scales that offer ordinal measurement methods to measure the degree of resistance in a limb to passive movement, including the Pedersons scale and the Ashworth scales. Ashworth developed a five point (0-4) standardized scale that Bohannon modified with the addition of the 1+ grade (See Table 1). Bohannon proved high statistically significant interrater reliability of the modified Ashworth scale on assessment of spasticity in elbow flexor muscle groups in adult hemiplegic subjects.[28] Limitations to this scale include the clustering effect -most patients are grouped in the middle grades, the test has not been proven reliable on all muscle groups or on clients with cerebral palsy and, at the higher end, the Ashworth scale measures only resistance to passive movement and does not distinguish spasticity from other causes of increased tone such as rigidity.[6,29] Although limitations are present, the Ashworth scale is one of the most clinically used means of quantifying spasticity. Katz[6] states that:

> although the Ashworth (and Pederson) scales offer only qualitative information, they have been widely used in the study of spasticity and are the present yardstick against which newer more exact methods must be compared.

Muscle Contracture

A directly proportional relationship between long standing spasticity and joint contracture has been assumed by many clinicians.[30] Spasticity and contracture appear to be directly correlated in upper motor neuron syndrome.

Gage[30] states that:

> children with cerebral palsy have spasticity and abnormal gait patterns which prevent normal growth and favor static muscle contracture of certain muscle groups, therefore, anything which will normalize muscle tone will help to prevent contracture.

Nash et al.[31] conducted a study with three spastic diplegic children in which they trained the children (using biofeedback of the gain on the tonic stretch reflex) to increase ankle rotation. They stated that if some control of spasticity could be achieved, it could retard the development of muscle contracture. Their results showed that two of the children gained significant increases in ankle rotation movements (plantarflexion and dorsiflexion) due to the control they achieved over their spasticity. Although contracture and spasticity are related clinically, they must

assessed separately. Glenn and Whyte[29] state that assessing the relative contributions of contracture and spasticity to loss of joint mobility can be difficult, but agree that this determination will often affect the clinicians approach to treatment. Perry[22] has performed several studies which examine the relationship between spasticity, contracture and function. She suggests that recent findings clarify the need to distinguish between spasticity and contracture.

Normal Gait

Normal human gait is traditionally defined in terms of a cycle of gait. One gait cycle begins when one foot hits the ground and ends when that same foot strikes the ground again.[30] The two phases of gait are referred to as swing phase and stance phase. Stance phase consists of heel strike (initial contact), foot flat (loading response), midstance, heel off (terminal stance) and toe off (preswing). Swing phase consists of acceleration (initial swing), midswing and deceleration of gait (terminal swing). Stance phase comprises 60% of the gait cycle and swing phase comprises 40%.[30]

Swing phase gait analysis is most pertinent to this study. Gage[30] describes the knee-swing phase. The

hamstrings will affect the step length and stride length during this phase of gait. The rectus femoris muscle accelerates the pendulum (lower leg) and the hamstring muscle decelerates it. At a normal pace, knee flexion is passive. In order to accelerate, knee flexion and ankle plantarflexion become more active. During midswing, there is the switching of control from rectus femoris to hamstrings, with no muscle control across the knee joint. At terminal swing, eccentric hamstrings activity begins to decelerate both the knee and the hip. If gait is slower than normal, passive knee flexion is inadequate during initial swing and must be augmented by the gracilis, sartorius and short head of the biceps. Gage[30] states that to achieve normal gait, "the timing of the biarticular muscles must be precise".

Spastic Diplegic Gait

Spastic diplegia, defined as motor impairment of the lower extremities with a lesser degree of involvement of the upper extremities, is one of the most common types of cerebral palsy. The usual etiology is hemorrhage into the third ventricle with associated periventricular leukomalacia and this is usually associated with premature birth.[30] More than 50 percent of children diagnosed with spastic diplegia will ambulate unassisted and the rest will use assistive

devices or will not ambulate at all.[32] The typical gait pattern of a child with diplegia is one of flexion, adduction and internal rotation at the hips with flexion at the knees.[30] This is often referred to as a crouch posture. Gage[30] adds that the characteristic foot position is one of valgus hindfoot with supination and an abducted forefoot. The children are often toe walkers with a toe-toe or toe-heel gait pattern. This may be due to hindfoot equinus or to knee flexion that is persistent throughout the stance phase.[30] These defects may cause secondary complications, such as excessive hip internal rotation resulting from overactive spastic muscles (gluteus medius and medial hamstrings) leading to excessive femoral anteversion.[33] Muscle contractures are another common complication.

During swing phase the child with spastic diplegia may exhibit decreased extension range of the knee.[30] EMG studies have shown that when comparing spastic diplegic gait to normal gait, the medial hamstrings in the diplegic tend to be active more than twice the length of time they are normally active during a complete gait cycle.[34] Therefore, step length and stride length are likely to be decreased secondary to restriction of full knee extension.

Summary

Upper motor neuron lesions produce a multitude of

impairments to function of which spasticity is one. Researchers continue to examine the neurophysiological aspects of spasticity. Treatment of patients with upper motor neuron lesions focuses on techniques to improve function in these patients. Research aimed at determining the relationship between spasticity and functional activity is needed. This study focused on the relationship between spasticity and function and also examined the relationship between muscle contracture and specific components of walking.

Research Questions

1. Is there a relationship between the degree of hamstring spasticity and step length, stride length and velocity in the child with spastic diplegia?
2. Is there a relationship between the degree of hamstring contracture (as measured by the popliteal angle) and step length, stride length and velocity in the child with spastic diplegia?

Hypotheses

1. There is a significant relationship between the degree of hamstring spasticity and selected gait characteristics in the spastic diplegic child.
2. There is a significant relationship between the degree

of hamstring range of motion and selected gait characteristics.

Subhypotheses

1. There is a significant negative correlation between hamstring spasticity (Ashworth scale measurement) and stride length, step length and walking velocity at the .05 alpha level (eg, as the Ashworth score increases, the gait parameters will be adversely affected).

2. There is a significant positive correlation between hamstring length (popliteal angle measurement) and stride length, step length and walking velocity at the .05 alpha level (eg, as the hamstrings range of motion increases, the gait parameters will approach more normal values).

CHAPTER TWO

METHODS AND PROCEDURES

This chapter will describe the sample selection, instrumentation and the procedures. This study was conducted in accordance with the ethical standards of the Human Subjects Institutional Review Board of Old Dominion University.

Selection of the Sample

The study sample of cerebral palsied children with spastic diplegia was obtained by recruitment from physical therapists and physicians in the Tidewater area of Virginia. A letter was sent to therapists and physicians (see Appendix A) requesting that they recruit children with a primary diagnosis of spastic diplegia who were able to ambulate at least five feet and able to follow simple verbal commands. There were no disqualifications for participation secondary to previous surgical intervention and the children were allowed to use any assistive devices that they would ordinarily use for daily walking. The parents were contacted by phone to explain the procedures thoroughly and to confirm the intention for their child to participate in

the study.

Instrumentation

A clinical evaluation form was devised that included categories for walking assessment, range of motion measurements, spasticity assessment and strength assessment of specified muscle groups (Appendix B). The evaluation included goniometric measurements for range of motion assessment, strength measurements using manual muscle testing and tone assessments using the Ashworth scale (Table 1).

Operational Definitions

1. Spasticity: a motor impairment characterized by a velocity dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks from the hyperexcitability of the stretch reflex as one component of an upper motor neuron lesion[9], which can be measured by degree of resistance to passive range of motion.
2. Contracture: a permanent shortening of muscle, tendon, other soft tissue or scar tissue producing deformity.
3. Cerebral Palsy: a lesion to an immature brain acquired prenatally, perinatally, or postnatally which always affects the motor system and is nonprogressive.[35]
4. Spastic diplegia: a form of quadriplegia in which the

legs are more affected than the arms.[3]

5. Stride length: linear distance between two successive events that are accomplished by the same lower extremity during gait.[36]

6. Step length: linear distance between two successive points of contact of opposite extremities.[36]

7. Initial contact: the instant the foot of the leading extremity strikes the ground.[36]

8. Gait velocity: measured in feet/second. (Operational definition)

9. Popliteal Angle: measure of passive knee extension (hamstring length). In this study, popliteal angle is measured with the subject in supine with hip supported at 90^0 flexion and in standing with the hip maintained at 30^0 flexion. Knee extension is measured with the goniometer fulcrum of motion aligned with the lateral femoral epicondyle; the proximal stationary lever arm is aligned with the greater trochanter and the distal goniometer lever arm is aligned with the lateral malleolus.[37]

Procedures

Each subject was requested to wear shorts in order to facilitate ease of measurement. Parents were present during testing and testing was conducted in the same facility in all cases except one. Both lower extremities were tested in

each subject. Reliability data was collected for all measures throughout the study. The same two testers were used for each subject.

Gait analysis

Robinson and Smidt[38] describe a quantitative gait evaluation technique using a grid pattern as the walkway. Gage[30] describes a similar measurement technique using a measured walkway with talcum powder. The gait analysis data was collected first. Each subject had inked moleskin attached to the bottom of his/her shoes. The child was asked to sit in a chair placed at the end of a fifteen foot strip of paper. A one foot section was marked off that allowed the subject to stand prior to initiating the walk. Each child was allowed to use any assistive devices s/he ordinarily used. The subject was then asked to walk at his/her normal pace towards the end of the paper where another researcher or their parent stood. The child was then allowed to sit down and rest for one to three minutes while his/her shoes were removed. Assistive devices used by subjects are listed in Table 2. Step and stride length were calculated by averaging the middle two gait cycles to account for acceleration and deceleration. The measurements were taken by measuring toe off to toe off for each subject. Norkin and Levangie[36] state that stride length may be

measured by using events such as two successive toe off's of the same extremity, although in normal gait stride is usually measured by two successive heel strikes.

Tone and Range of Motion assessments

The clinical assessment of hamstring tone and range of motion included testing in two positions- standing and supine. The standing position included having the subject stand on a block that allowed the extremity being tested to swing freely. The subject was allowed to support himself/herself or, if necessary, a researcher helped support the subject. The subject maintained weight bearing on the extremity not being tested. Standing was used in order to simulate the position of ambulation when assessing hamstring tone and hamstring length. Research has shown that change of body position changes the degree of tone.[22] The subject's hip was supported in approximately thirty degrees of flexion to simulate the maximum flexion normally achieved by the hip during swing phase of gait.[30] Tone was then assessed in standing using the modified Ashworth scale. The subject's lower leg was moved through available range of motion three times with the hip in thirty degrees of flexion. The highest Ashworth score was recorded. Popliteal angle was also measured in this position using the greater trochanter, lateral femoral epicondyle and lateral

malleolus as landmarks.

Supine measurements were taken for comparison purposes. The supine measurements were taken with the subject lying on a plinth with his knees bent over the edge of the plinth. During measurements, the opposite extremity was supported with the hip and knee flexed and the foot resting on the supporting surface to assure pelvic stability. The measuring protocol used for tone and range of motion measurements in supine were the same as those used in standing. Standing measurements will be used for data analysis in this study.

CHAPTER THREE

RESULTS

Demographics

Subjects for this study included three females and eight males with a mean age of eight years and one month. The range of ages was three years nine months to fifteen years. Three of the subjects had not had any orthopedic surgeries and two subjects did not use any assistive devices for walking. Demographic information including subject age, sex, assistive devices used, orthopedic and related surgeries and etiology of disorder is presented in Table 2.

Data Analysis

Step length and stride length measures for each subject were normalized by dividing each by the subjects total body height.[36] Bohannon and Smith modified the Ashworth scale with a 1+ grade in order to make the scale more discrete at the lower end.[28] The 1+ score can not be analyzed statistically without conversion and therefore, a numerical grade of 1.5 was assigned to the 1+ score for statistical analysis purposes. Although the Ashworth scale is ordinal, the conversion of 1+ to 1.5 manipulated the scale the least and was consistent with the original intent of Bohannon and

Smith. The correlation between spasticity and velocity, step length and stride length was determined using Spearman rank correlation coefficient (r_s). Critical values were used to determine significance.[39] The correlation between popliteal angle measurements and velocity, stride length and step length was determined using Pearson product moment correlation coefficient (r). Critical values were used to determine significance.[39] Intertester and intratester reliability measures were calculated using the Intraclass correlation coefficient (ICC). The ICC was chosen because it reflects both the degree of correspondence and agreement among the ratings.[39] Although it is primarily used with interval level data, Portney and Watkins[39] state that the ICC can be applied without distortion to data on the ordinal scale when the intervals between such measurements are assumed to be equivalent. In addition, percent agreement reliability measurements were taken for Ashworth scale scores to allow appropriate interpretation. The ICC's for intertester reliability were based on the formula (2,1) and for intratester, the ICC formula was (3,1). There are no widely accepted criteria for defining the strength of association when interpreting correlational data. Interpretation will be based on guidelines suggested by Portney and Watkins[39]: .00- .25 indicate little or no relationship, .25- .50 suggest a fair degree of

relationship, .50- .75 are moderate to good, and above .75 are good to excellent. A general guideline for reliability is values of .75 and above are indicative of good reliability and below .75 are poor to moderate.

Results

The primary research question was: (1) Is there a relationship between the degree of hamstring spasticity and velocity, stride length and step length in the child with spastic diplegia? Table 3 lists the correlation coefficients and critical values for this comparison. The data in Table 3 reveals that Ashworth scores correlated negatively with velocity, stride length and step length. Right sided comparisons were only fairly correlated and not significant, while left sided comparisons showed moderate to good negative correlations (-.569- -.877) and were all significant (when compared to the critical values). Moderate correlation existed between spasticity and velocity while spasticity and step length had the strongest correlation. Graphs 1 through 6 are the scatterplots for these data points.

The second research question was: (2) Is there a relationship between hamstring range of motion (popliteal angle) and velocity, stride length and step length in the

child with spastic diplegia? Table 4 lists the correlation coefficients for popliteal angle and velocity, step length and stride length. The relationships were all positively and significantly correlated with moderate to good correlation coefficients (.671- .840). Unlike the spasticity measurements, there were no significant differences between left sided and right sided measurements. Graphs 7 through 12 show the scatterplots for these data points.

Table 5 shows the ICC intertester and intratester reliability coefficients for Ashworth scores. Table 6 shows percent of agreement for intertester and intratester reliability of the Ashworth scores. The intertester reliability for Ashworth scores was poor to moderate (ICC= .242- .613). Percent agreement was very poor (0%- 14%) and not significantly better when differences of one grade were taken into account (28%- 57%). Intratester reliability coefficients ranged from poor to good (ICC= .487- .947). Percent agreement for exact agreement ranged from 0% to 75% and when one grade difference was allowed, percent agreement was better and ranged from 50% to 100%. Table 7 shows intratester and intertester reliability coefficients for popliteal angle measurements. Intratester measurements showed high reliability (ICC= .884- .957). Intertester measurements also showed high reliability (ICC= .909- .962).

CHAPTER FOUR

DISCUSSION

The discussion will focus on each of the research questions and the reliability coefficients addressing each of these questions. The scope and limitations of the study and suggestions for further research will also be addressed in this chapter.

The primary research question dealt with the relationship between spasticity and functional gait parameters. The correlational results were difficult to interpret because tone and gait parameters were poorly correlated on the right, but on the left, there were moderate to good relationships. Reasons for this discrepancy may be that the Ashworth scores on the left side of the subjects (range of 0-3) tended to be higher than the right side (range of 0-2). This finding may indicate that spasticity scores of three or greater impact functional gait parameters to a significant degree.

While spasticity has been assumed to be one of the major deterrents to functional motor control[19], other primary and secondary impairments such as weakness, contracture, incoordination, and central timing

dysfunction[22,29] may, in fact, impact adversely on functional gait equally or more than spasticity. In this study, the inability to establish a clear cut relationship between spasticity and function may have been due to an insufficient degree of spasticity in the subjects, and/ or the presence of contracture, or other more detrimental impairments not addressed in this study.

Perry[22] has addressed the issues of the effects of spasticity versus contracture on functional activities. She states that recent experience with the selective posterior rhizotomy for spastic diplegia has allowed one to distinguish between spasticity and control dysfunction. Spasticity is diminished following the rhizotomy but there is often persistence of functional errors. Therefore, she supports the notion that it is important to elucidate the patients mode of control as well as to identify spasticity and contracture.

Another issue to consider in explaining the lack of correlation between tone and gait characteristics in this study is the tool used for the measurement of spasticity. Bohannon and Smith[28] found the modified Ashworth scale to have high intertester reliability when used to test elbow flexor spasticity in adult stroke patients. Bohannon and Smith[28] tested patients in a very stable supine position. In the present study, tone measures were taken in standing

because it was considered to be a position specifically related to functional gait. However, the upright position contributed to postural instability which would be compensated for by voluntary and involuntary muscle tone fluctuations. Testers noted increased associated reactions when the children were distracted, laughing and engaged in other activities versus being completely still. Brown[24] showed in his study using the electrogoniometer that voluntary contractions limit the validity of the test to assess spasticity. Adults are generally able to relax more easily on command and attend better for clinical tests than children. Therefore, although the modified Ashworth scale has been proven reliable for adult stroke patients, it has not been proven reliable for the pediatric population with spastic diplegia. Review of the literature revealed no studies reporting use of the modified Ashworth scale on a pediatric population.

The second research question focused on the relationship between hamstring range of motion (as measured by the popliteal angle) and the specified functional gait parameters. Significant and moderate correlations existed between hamstring range of motion and velocity measurements. This was consistent with the hypothesis.

There was a significant and good relationship, as defined previously, between hamstring range of motion

measurements and stride length and step length. Intertester and intratester reliability also proved to be good consistently for the popliteal angle measurements. This appears to indicate that restricted hamstring range of motion plays a role in limiting functional gait, through decreased step length, decreased stride length or decreased gait velocity.[30] A logical treatment implication and follow up study would be to assess if improving hamstring range of motion will have a significant effect on increasing velocity, step length and stride length in the spastic diplegic child.

Treatment aimed at improving hamstring length would be a reasonable option to improve step length, stride length and gait velocity when it is determined that hamstring contracture is a limiting factor in the individual patient. Consequences of shortened hamstrings include inability to fully extend the knee for maximum step length and stride length, in addition to other functional limitations in sitting and other positions.[30] Several points should be considered when determining the best mode of treatment. Range of motion in standing (as in this study) may give a more accurate assessment of limitations that may be present during gait as compared to measures taken in supine. DeLuca[33] has noted that joint or muscle range limitations identified in recumbent positions may not actually interfere

with functional gait in upright standing. A decision for treatment should be based on a thorough gait evaluation as well as a clinical range of motion exam performed in standing.

Documentation of objective evaluation techniques (such as goniometric measurement) is necessary pre and post treatment in order to support any benefit of treatment that is directly related to improving hamstring length. The methods described in this research for measuring hamstring length and for assessing step length, stride length and velocity could easily be duplicated in any clinical setting.

Several factors should be considered when interpreting the data and results of this research project. There were a number of variables which could not be held constant between subjects (eg, strength, emotional state, cognitive level). Another consideration is the questionable reliability of the modified Ashworth scale for the pediatric population; and finally, the users of correlational data must be careful not to assume cause and effect.

Further research regarding the relationship between spasticity and functional limitations is certainly warranted. The importance of spasticity continues to be debatable in terms of its specific relationship to functional deficits. Complications of spasticity or secondary impairments, such as contracture, may be more

limiting than spasticity alone. A reliable and valid measurement tool that would be clinically accessible and which could distinguish spasticity from other components of UMNS from a functional standpoint is needed. Further research postoperatively following the selective posterior rhizotomy which eliminates spasticity would be helpful in distinguishing the relative importance of limiting factors on functional gait in patients with UMN lesions.

This research suggests that there is a stronger relationship between range of motion limitations and functional gait limitations as compared to tone and gait relationships, thus continued research related to prevention of contracture as well as treatment methods would also be suggested.

Conclusions

Hamstring range of motion intertester and intratester measurements (popliteal angle) were reliable, and the relationship between popliteal angle and selected gait parameters was significant. Spasticity ratings using the modified Ashworth scale in standing on the hamstrings were not reliable (intertester or intratester) and the relationship between spasticity and gait parameters was inconsistent.

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TABLE ONE

Modified Ashworth Scale

0 No increase in muscle tone

1 Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of the range of motion when the affected part(s) is moved in flexion or extension

1+ Slight increase in muscle tone, manifested by a catch followed by minimal resistance throughout the remainder (less than half) of the range of motion

2 More marked increase in muscle tone through most of the range of motion, but affected part(s) easily moved

3 Considerable increase in muscle tone, passive movement difficult

4 Affected parts rigid in flexion or extension

From Bohannon RW, Smith MB. Interrater of a modified ashworth scale of muscle spasticity. Phys Ther. 1986:67;206-207.

TABLE 2

Subject Demographics

Subject	Sex	Age	Assistive device	Surgical history	Height	Etiology
1	F	13.10	B canes B AFO	B add R B HS R x2 B heel R	58"	prem
2	M	6.4	B AFO	none	43"	Russell-Silver
3	M	3.3	R AFO	none	37"	prenatal stroke
4	M	13.4	Bfootplate B loft	Bheel R	60"	prem
5	F	3.10	B AFO twistercable walker	none	35"	prem
6	M	5.6	B AFO	B heel R	45"	prem
7	M	9.0	B loft B footplate	Rhizotomy	50"	prem
8	M	5.0	none	B hip fl R B TAL B HS R	47"	prem
9	F	3.9	none	B heel R B add R	36"	emerg c sec
10	M	15.0	walker	Badd Rx3 B HS x2 B derot ost	60"	prem
11	M	9.10	B AFO	B HS R B heel R B hip flex R	58"	prem

KEY: B=bilateral, R=release, HS=hamstring, prem=prematurity, AFO=ankle foot orthosis, derot ost= derotational osteotomy, hip flex= hip flexors, TAL= tendoachilles lengthening, lofts= loftstrand crutches, add= adductor, heel= heelcord

TABLE 3

Spearman Rank Correlation Coefficient (r_s)

Ashworth scores correlated with velocity, stride length and step length

	Velocity(ft/s)*	Step length(cm)*	Stride length(cm)*
Ashworth score (R)	-.305	-.417	-.431
Ashworth score (L)	-.569	-.618	-.877
Critical value of r_s	.564	.536	.536

alpha= .05
*n= 10
#n= 11

TABLE 4

Pearson Product Moment Correlation Coefficient (r)

Popliteal angle measurements correlated with velocity, stride length and step length

	Velocity(ft/s)*	Stride length(cm)*	Step length (cm)*
Popliteal angle (R)	.685	.822	.777
Popliteal angle (L)	.671	.772	.840
Critical value of r	.549	.521	.521

alpha= .05
*n= 10
#n= 11

TABLE 5

Intraclass Correlation Coefficient

Ashworth scores intertester and intratester reliability (hamstrings)

	Intertester	Intratester Rater 1	Intratester Rater 2
Ashworth score (R)	.242	.690	.941
Ashworth score (L)	.613	.487	.672

TABLE 6

Percent Agreement

Ashworth scores intratester and intertester reliability (hamstrings)

	Intertester	Intratester Rater 1	Rater2
	exact/ within 1	exact/ within 1	exact/ within 1
Ashworth score (R)	0%/ 28%	75%/ 75%	0%/ 100%
Ashworth score (L)	14%/ 57%	25%/ 50%	25%/ 75%

TABLE 7

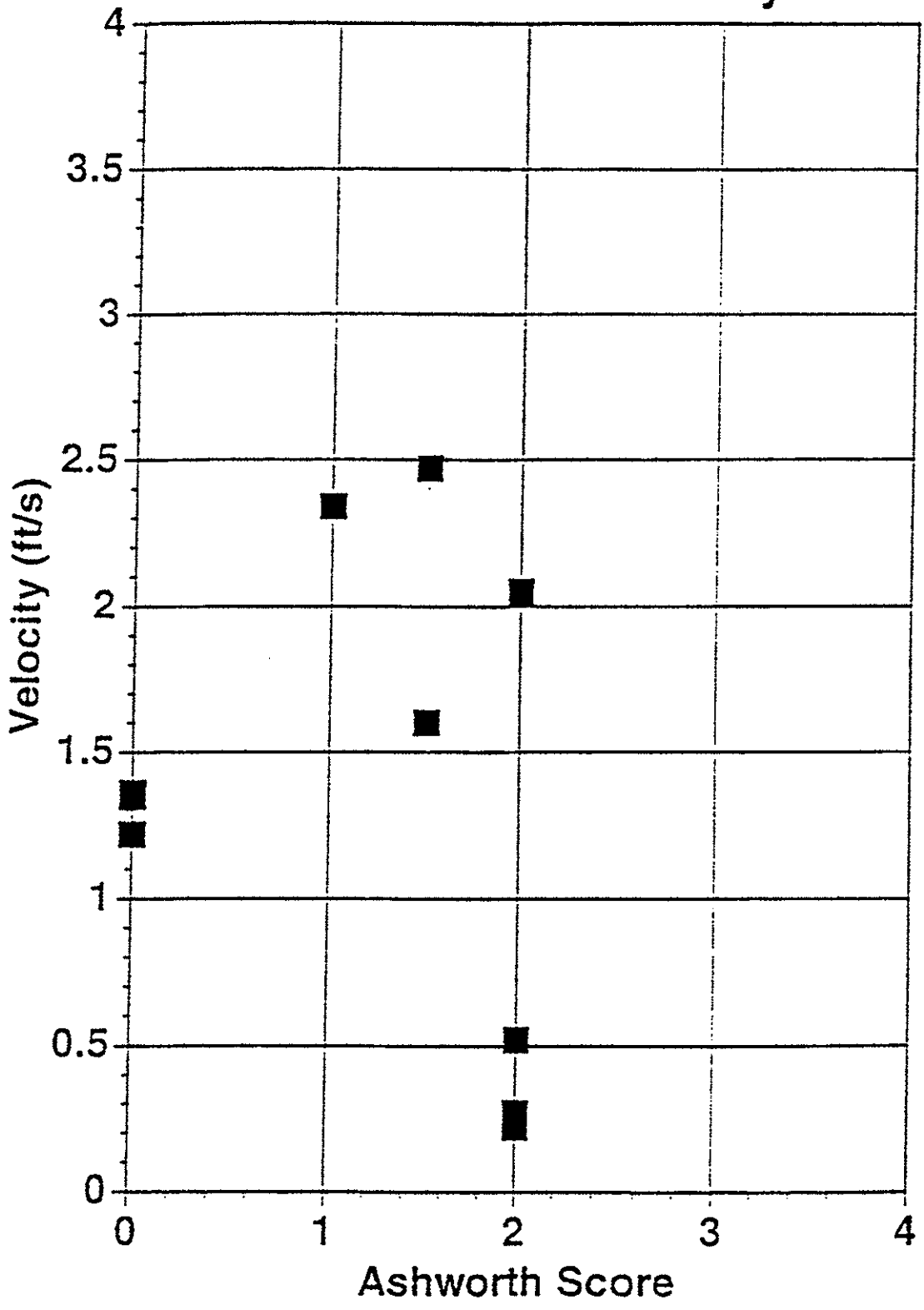
Intraclass Correlation Coefficient

Popliteal angle intertester and intratester reliability

	Intertester	Intratester Rater 1	Intratester Rater 2
Popliteal angle (R)	.884	.933	.909
Popliteal angle (L)	.957	.962	.920

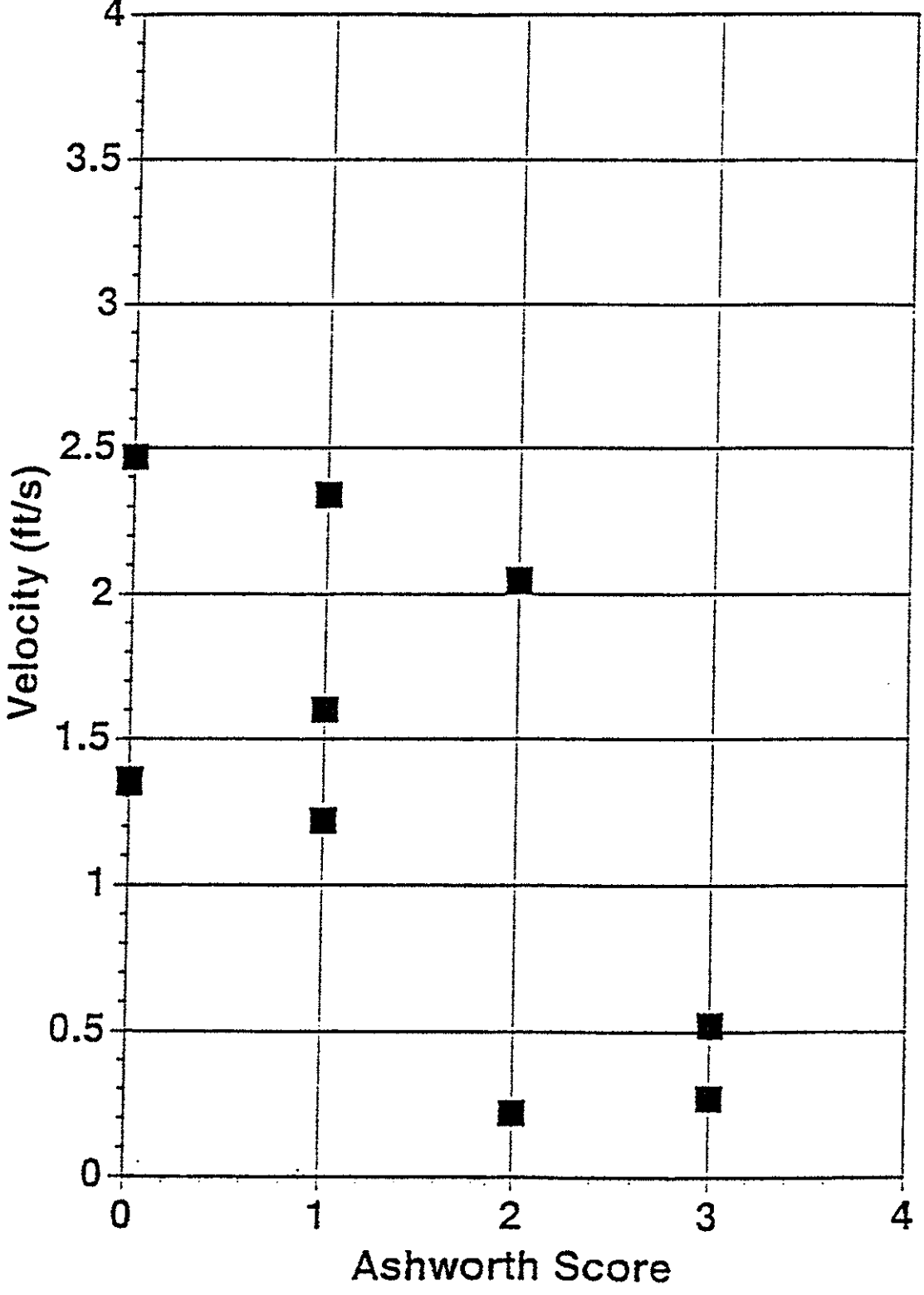
Graph 1

Scatterplot: Right Ashworth Scores and Velocity



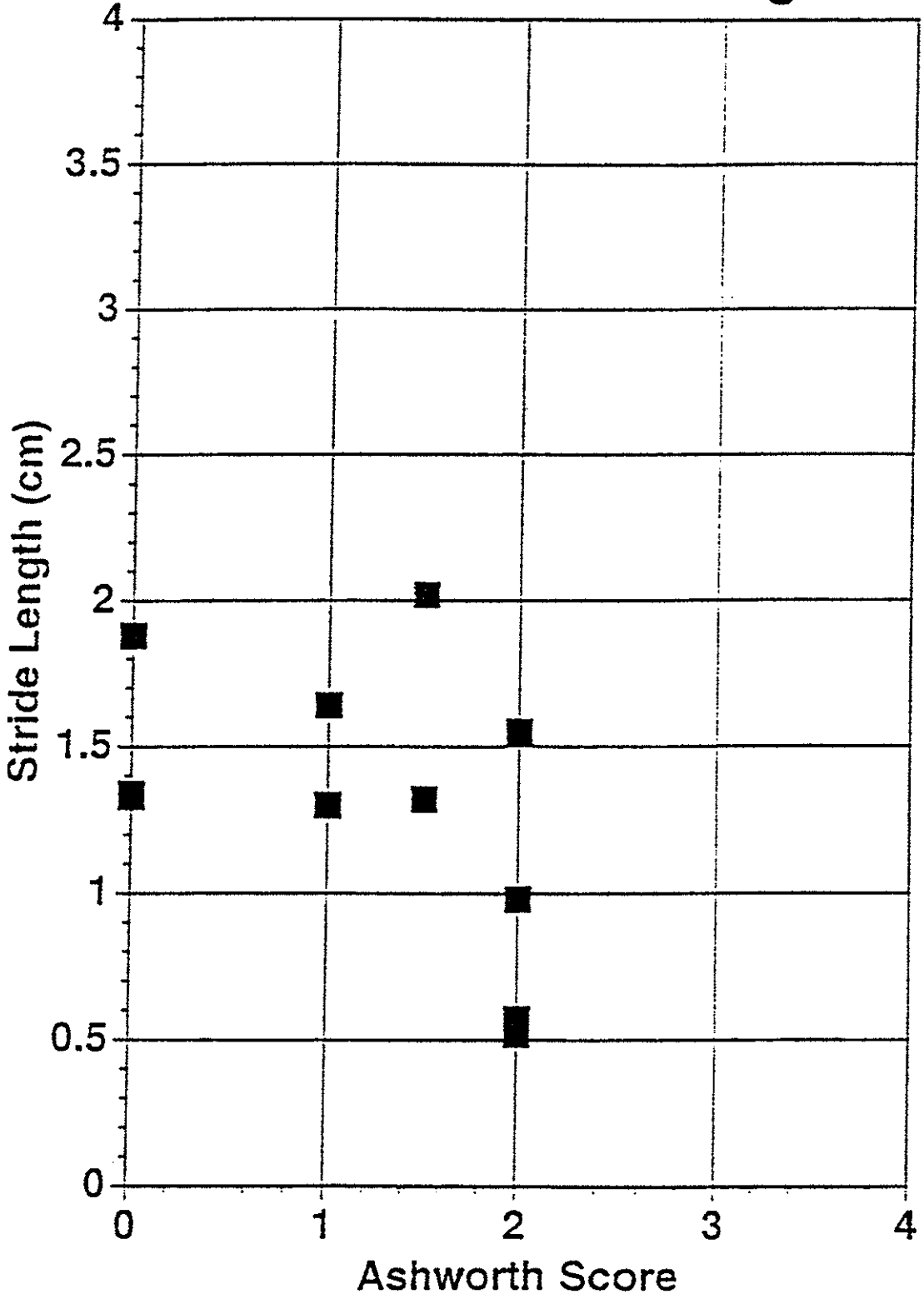
Graph 2

Scatterplot: Left Ashworth Scores and Velocity



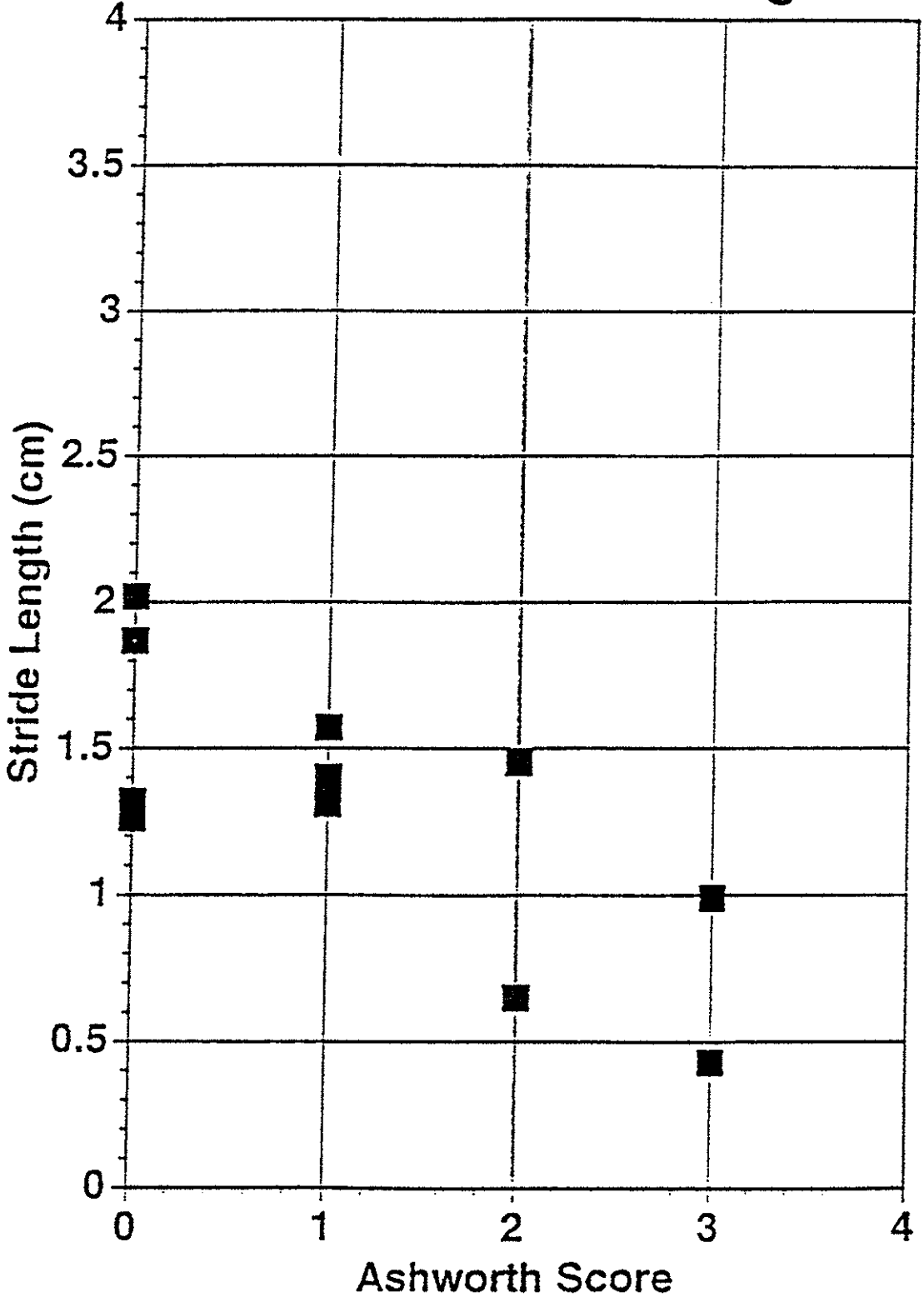
Graph 3

Scatterplot: Right Ashworth Scores and Stride Length



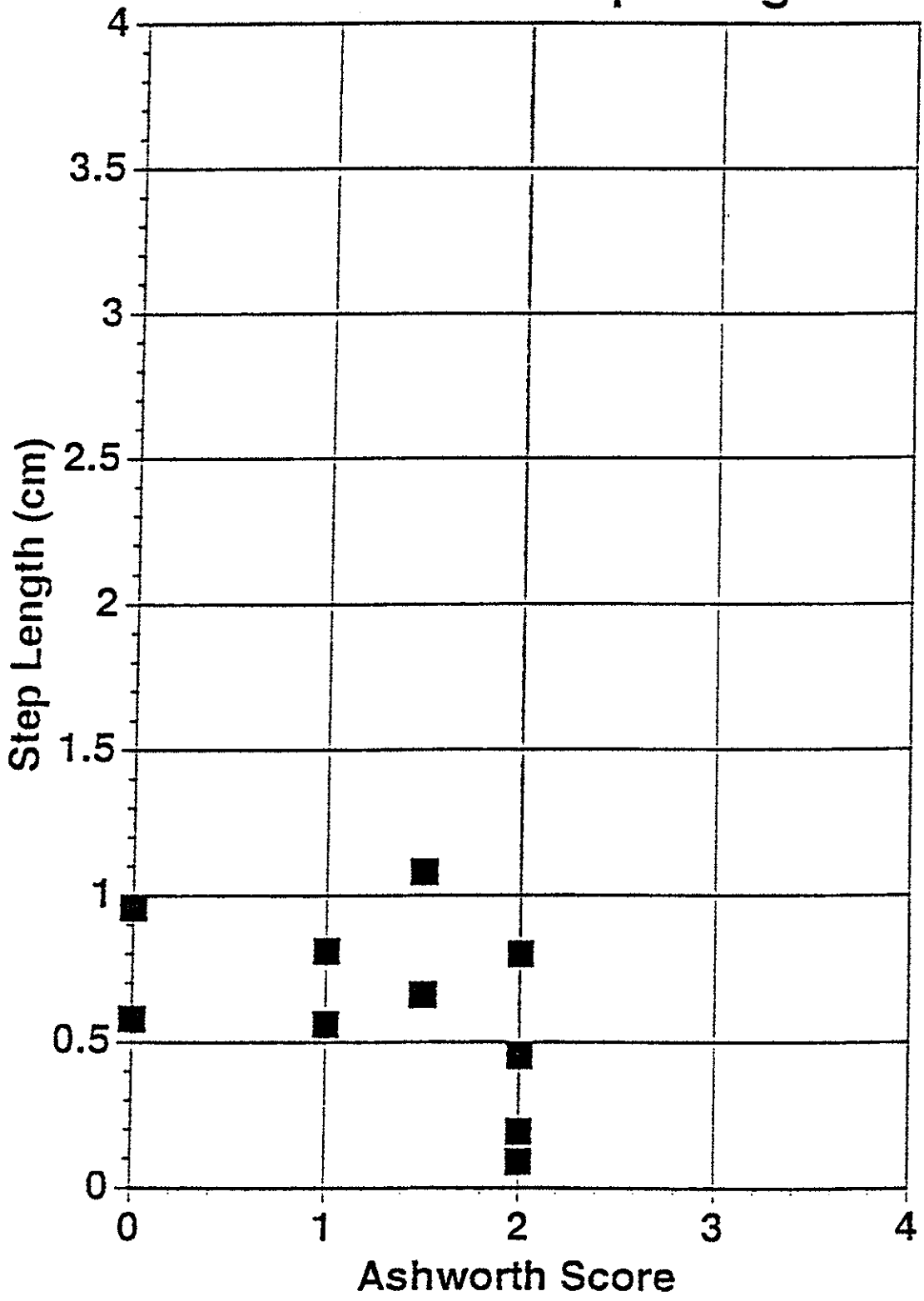
Graph 4

Scatterplot: Left Ashworth Scores and Stride Length

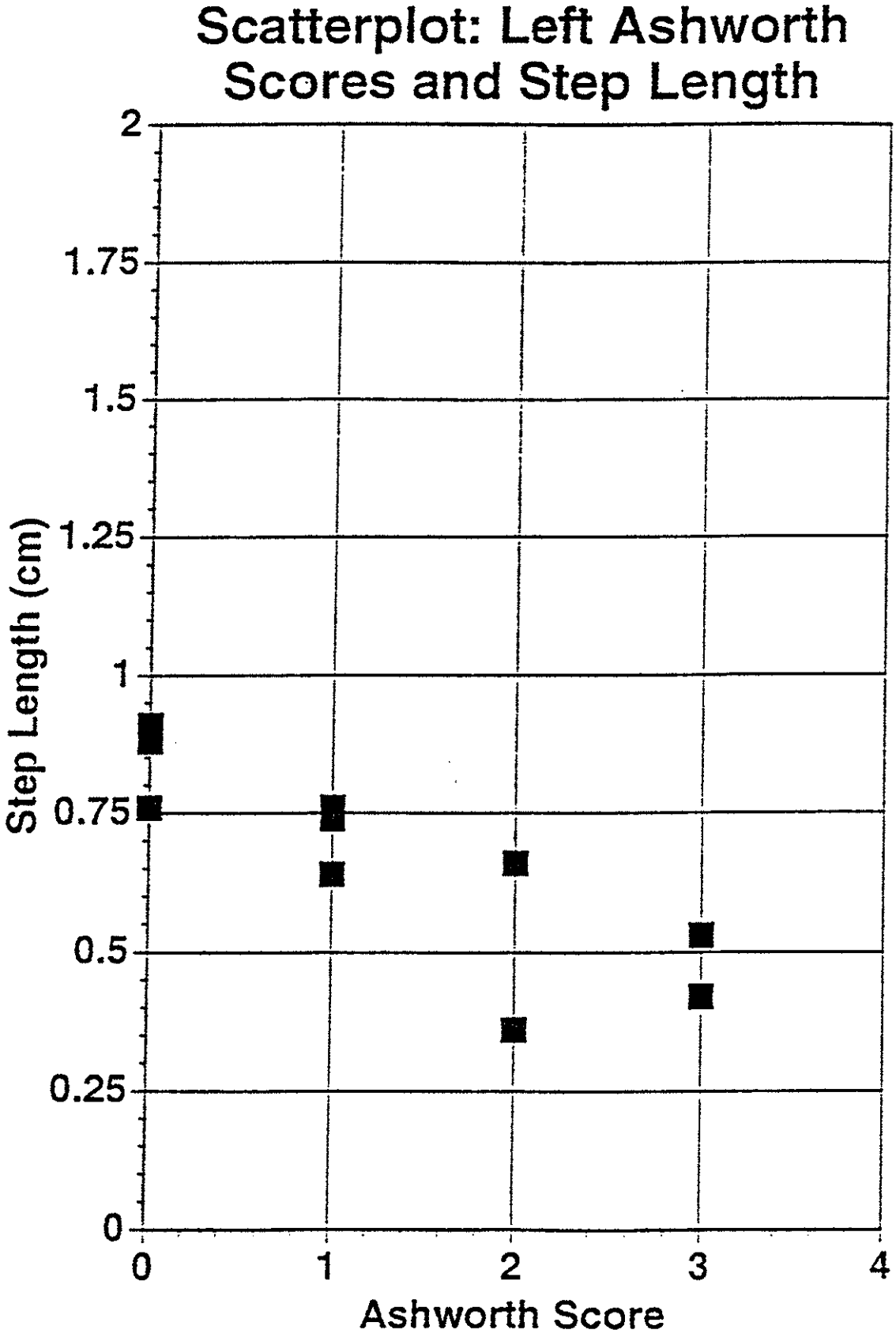


Graph 5

Scatterplot: Right Ashworth Scores and Step Length

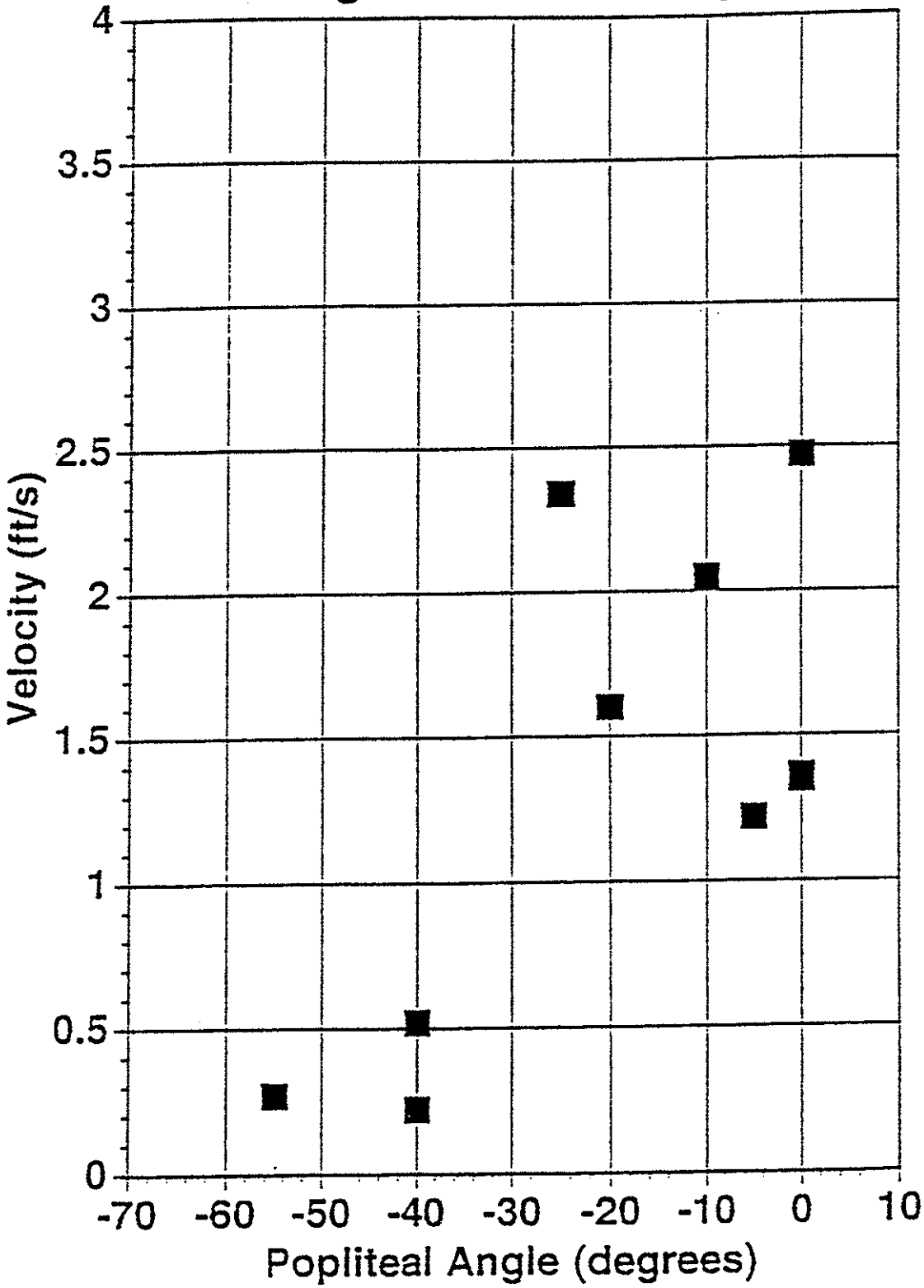


Graph 6


Scatterplot: Left Ashworth Scores and Step Length
Step Length (cm)
2
1.75
1.5
1.25
1
0.75
0.5
0.25
0
0
1
2
3
4
Ashworth Score

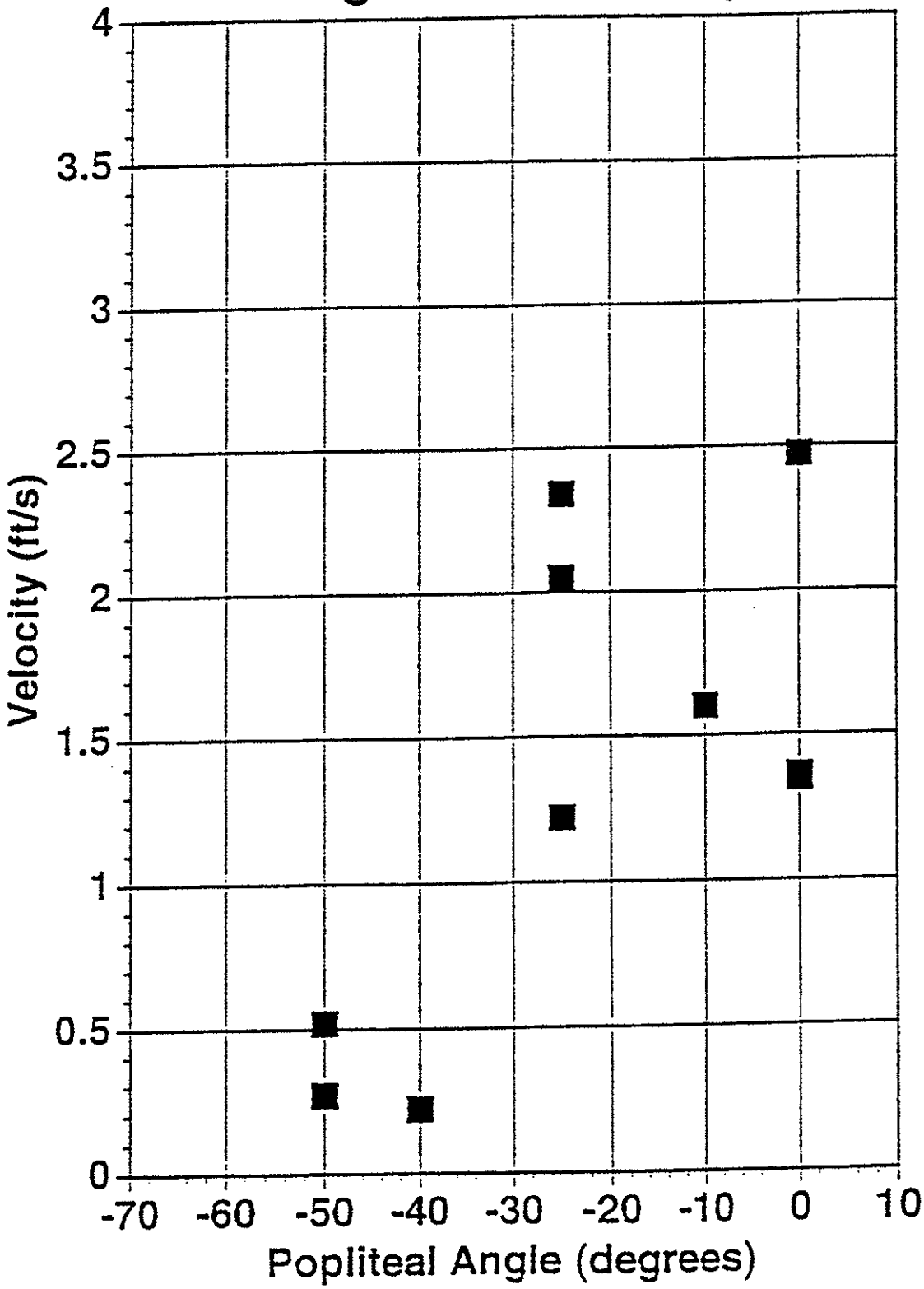
Graph 7

Scatterplot: Right Popliteal Angle and Velocity



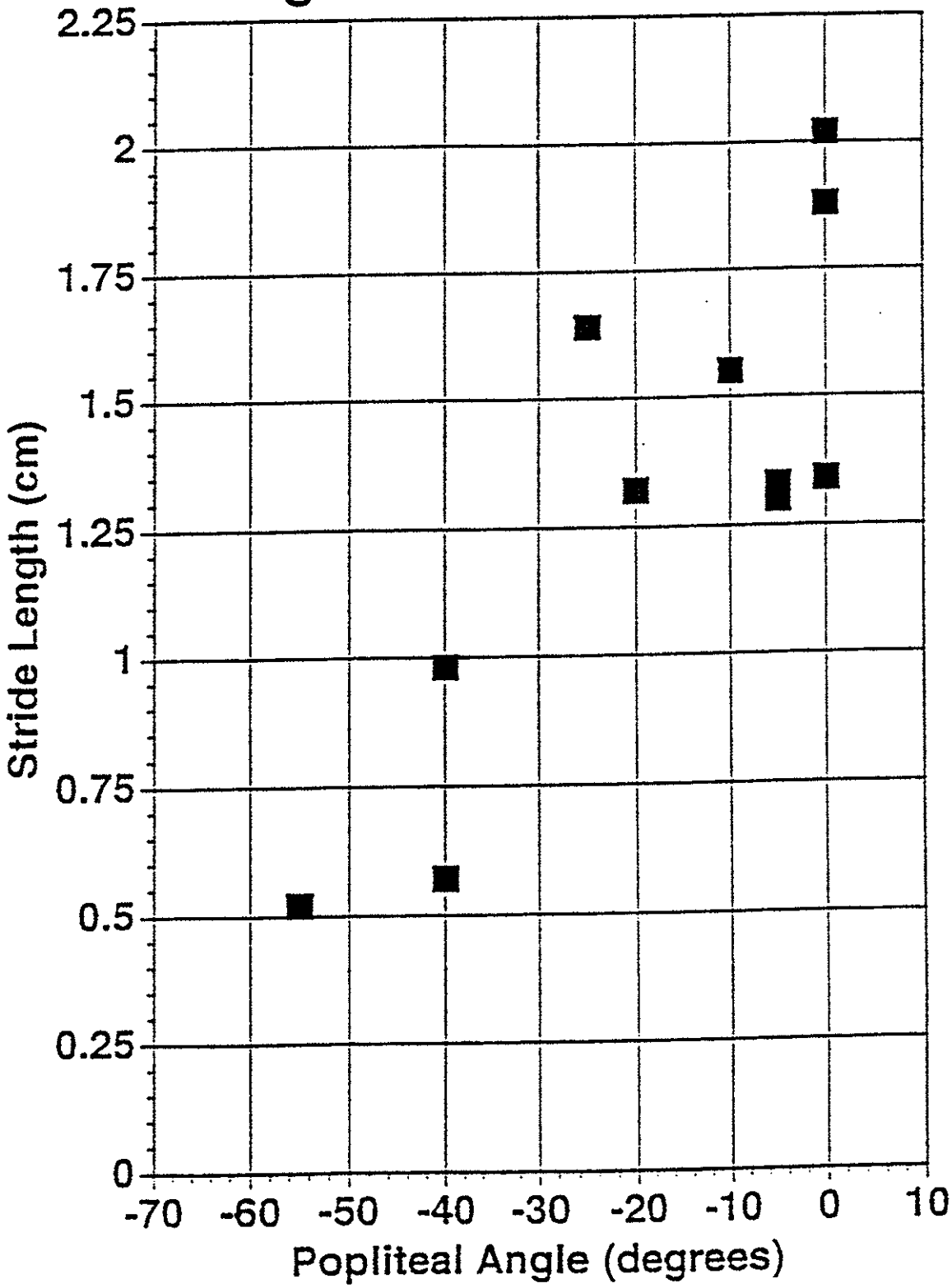
Graph 8

Scatterplot: Left Popliteal Angle and Velocity

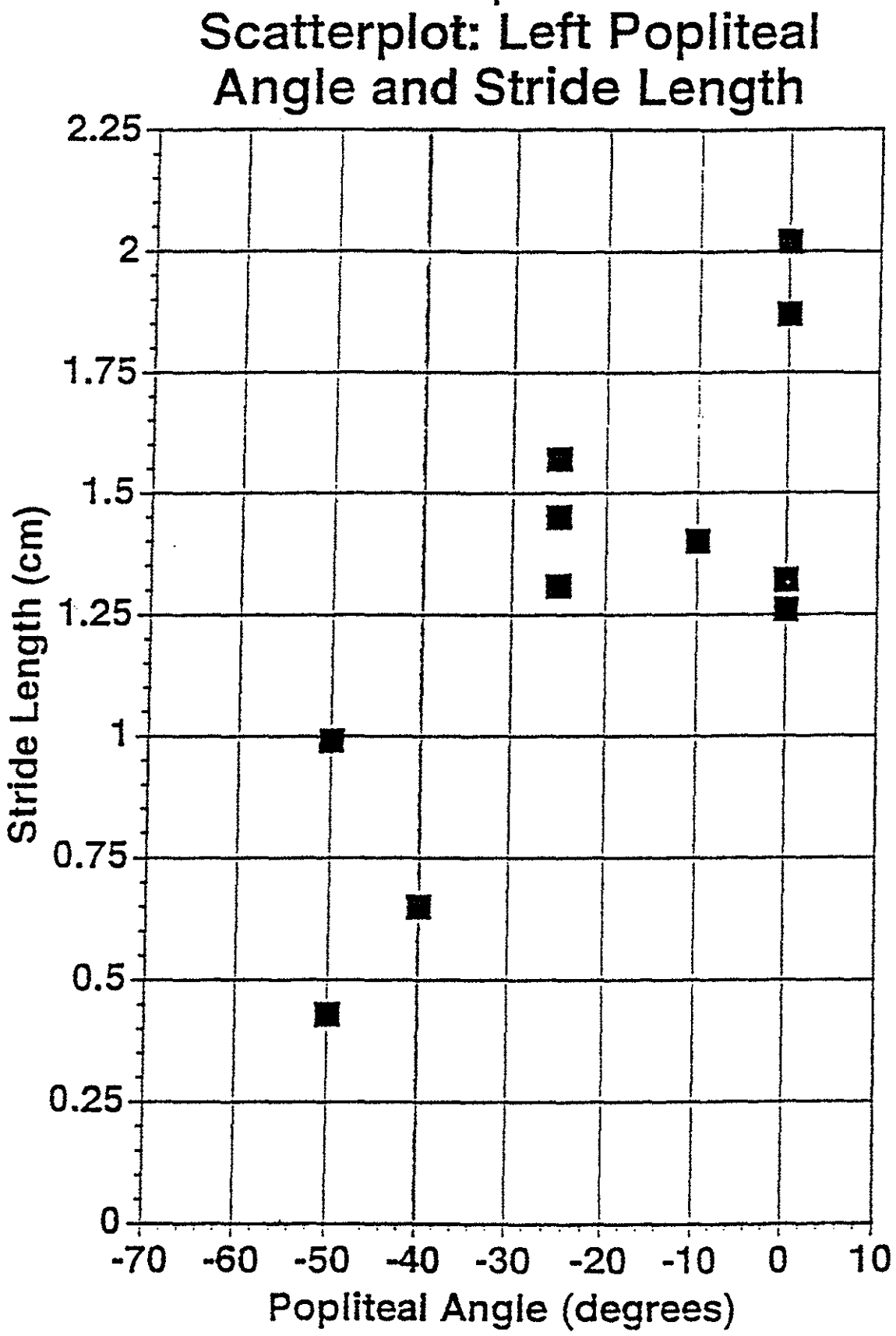


Graph 9

Scatterplot: Right Popliteal Angle and Stride Length

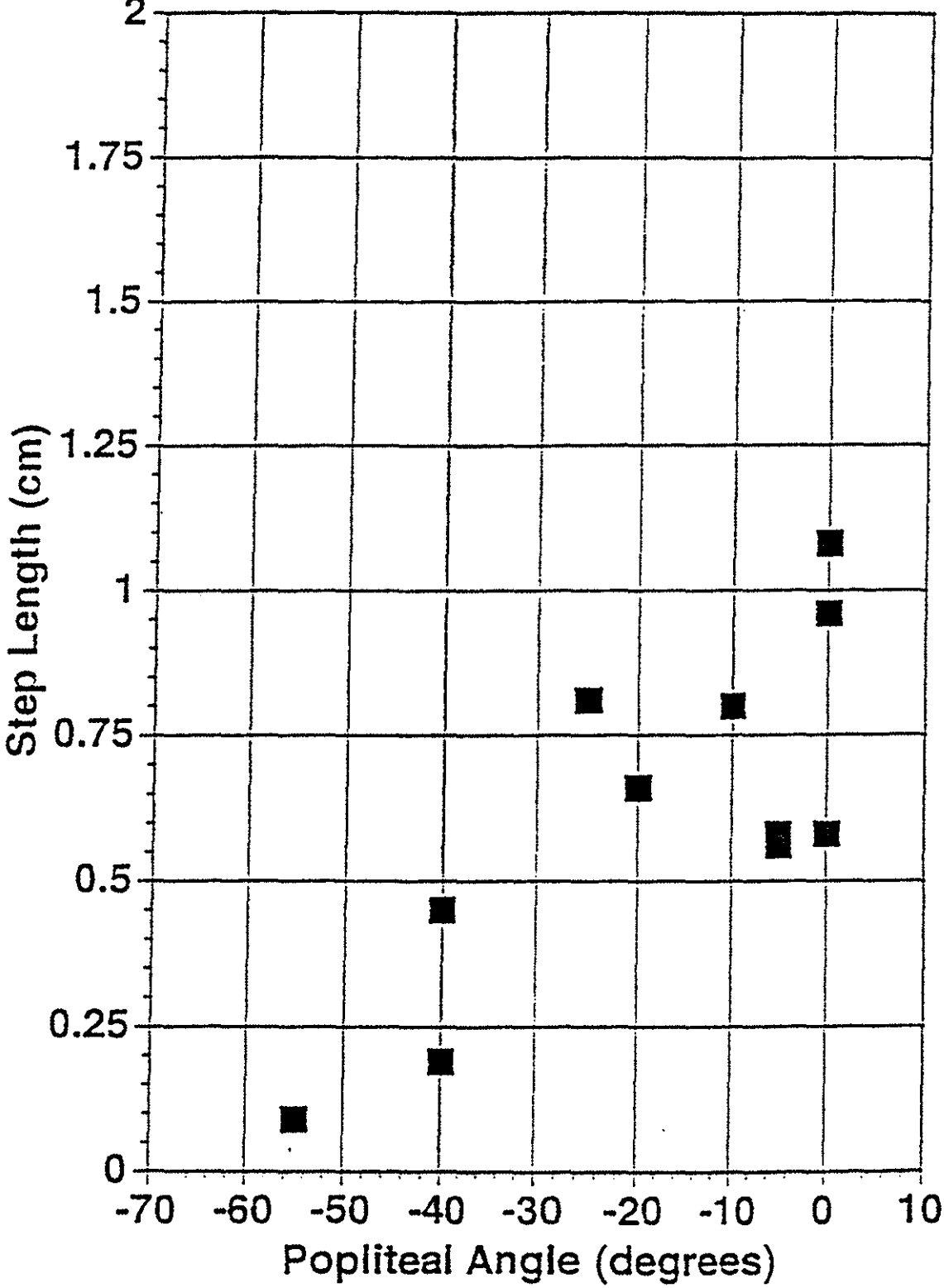


Graph 10


Scatterplot: Left Popliteal Angle and Stride Length
Stride Length (cm)
2.25
2
1.75
1.5
1.25
1
0.75
0.5
0.25
0
-70
-60
-50
-40
-30
-20
-10
0
10
Popliteal Angle (degrees)

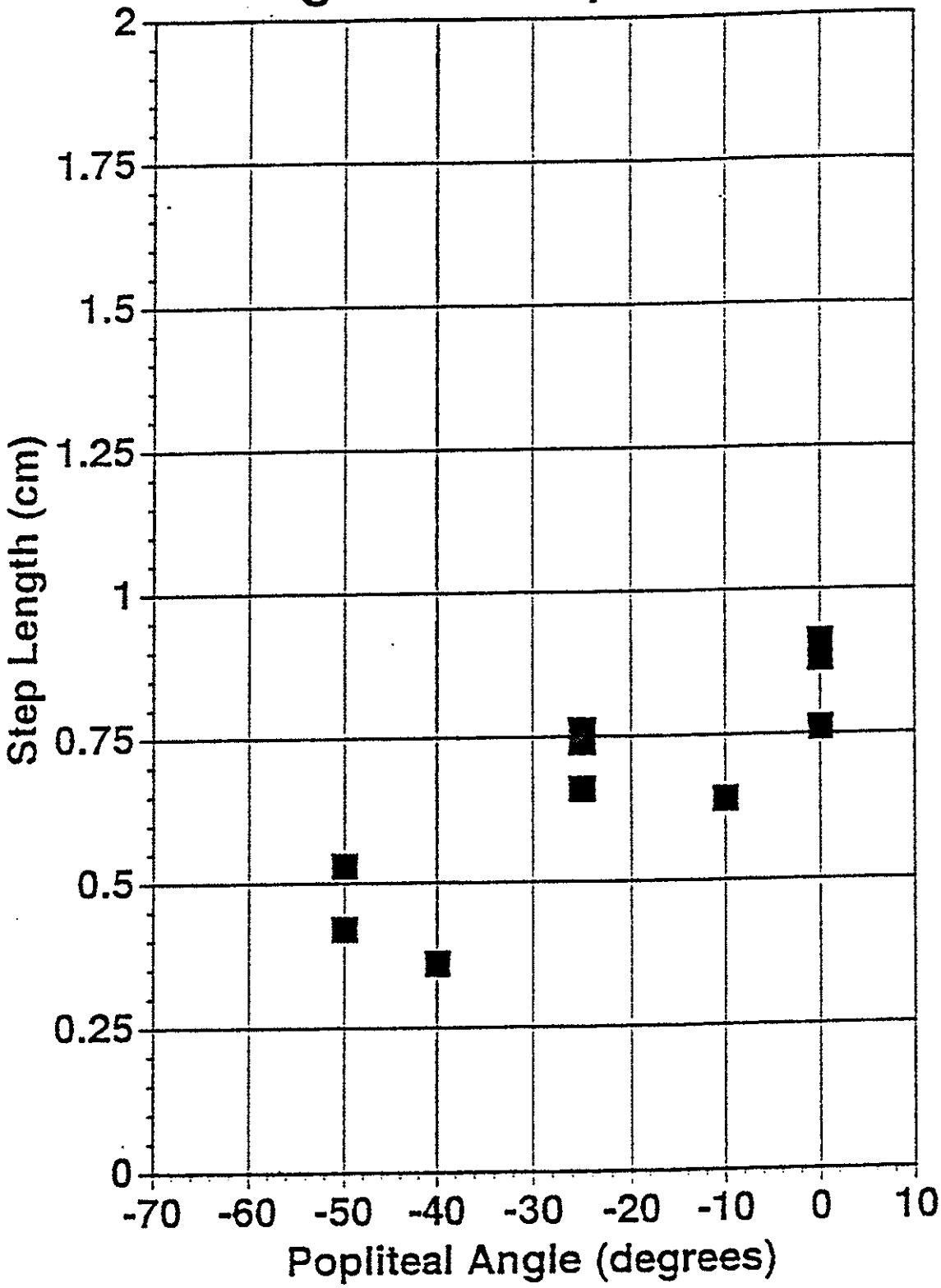
Graph 11

Scatterplot: Right Popliteal Angle and Step Length



Graph 12

Scatterplot: Left Popliteal Angle and Step Length



APPENDIX A

August 25, 1993

Dear Colleague,

I am writing to request your assistance in obtaining subjects for research that I am conducting to complete my master's thesis. The study will examine the correlations between hamstring length, hamstring spasticity and selected gait parameters in children with spastic cerebral palsy. I have enclosed portions of my research proposal and a copy of the evaluation for your information. I have also included a letter to parents providing information to introduce the study.

I would greatly appreciate your referring to me children that meet the following criteria:

* age 1-21 years
* primary diagnosis of spastic diplegia
* ability to ambulate at least five feet (may use walkers, canes, splints and/or braces)
* ability to follow simple commands.

I will be evaluating bilateral lower extremity R.O.M., tone (using the Ashworth scale), strength, and gait parameters by simple ink/paint and paper technique. There is minimal risk involved as there is nothing invasive about the evaluation. The entire evaluation should last approximately 45 minutes to one hour and will take place at Old Dominion University during September and October 1993. Some children will be asked to come back a second time for reliability studies.

Ideally, I would like the child's therapist/ physician to introduce the study, give parents the information sheet and ask if I may contact them by phone. If the child's parent prefers, though, they may contact me. I will make every attempt to make the evaluation enjoyable for the children.

Again, I would appreciate any help that you can offer to me. I will be happy to share the research results with anyone interested. Please call me with any questions or concerns (work- 622-2208, home- 436-7141). I will be in touch.

Thank you,

Erin M. Glace. P.T.

APPENDIX B

CLINICAL EVALUATION

SUBJECT NAME: _____ DOB: _____

DATE OF TESTING: _____ TESTER: _____

DIAGNOSIS: _____

_____

MEDICATIONS _____

ORTHOPEDIC AND RELATED SURGERY: _____

_____

ORTHOSES/ASSISTIVE DEVICES: _____

HEIGHT: _____ WEIGHT: _____ PT: _____

POSITION: WALKING DATA		
	LEFT	RIGHT
STEP LENGTH		
STRIDE LENGTH		
TIME		
NO. OF STEPS		
POSITION: STANDING WITH HIP FLEXED 30°		
HAMSTRING TONE		
POPLITEAL ANGLE		
QUADRICEPS TONE		
HAMSTRING STRENGTH		
QUADRICEPS STRENGTH		
POSITION: SUPINE WITH LEGS OVER EDGE		
OF TABLE		
HAMSTRING TONE (h 90)		
HAMSTRING TONE (h 0)		
KNEE EXTENSION ROM		
POPLITEAL ANGLE		
QUADRICEPS TONE		
QUADRICEPS STRENGTH		
POSITION: PRONE		