

**ASSESSMENT OF SPASTIC PATIENTS  
BEFORE AND AFTER  
SELECTIVE POSTERIOR LUMBAR  
RHIZOTOMY**

**By**

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## ***ABSTRACT***

*For many decades people working with spastic patients have searched for better methods of alleviating spasticity and its debilitating effects. Although many methods have been used, no single method has been completely successful.*

*It was not until the nineteen seventies that Selective Posterior Lumbar Rhizotomy (SPLR) was used successfully with spastic patients. In 1980 Peacock started doing this procedure on a large number of spastic patients in South Africa.*

*I became interested in SPLR after seeing the dramatic reduction in spasticity and the improvement in function in my patients who had undergone the procedure.*

*Since observations of improvement up to this point had been subjective, I decided to measure and document the physical status of patients before and after surgery.*

*Twenty nine patients who underwent Selective Posterior Lumbar Rhizotomy in 1985 were studied. In order to be as objective as possible, crawling and gait analyses were done in addition.*

*All the patients were assessed clinically two days prior to surgery and then between four and fourteen months after surgery. For both preoperative and postoperative assessments, resistance to passive movement, degree of joint stiffness, ability to initiate and inhibit voluntary movement, and the degree of abnormal function in the developmental positions (rolling, sitting, kneeling, crawling, standing and walking) were graded on rating scales. Analyses of gait and crawling were done using a digital camera system.*

*Results of the study indicated that SPLR produced a statistically significant reduction in muscle tone and in joint stiffness. Improvements in voluntary movement and functional movement (rolling, sitting, kneeling, crawling, standing and walking) were also statistically significant.*

*It was concluded that Selective Posterior Lumbar Rhizotomy is a useful method of reducing spasticity and results in improvement of the quality of life in patients with spastic cerebral palsy.*

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## LIST OF ABBREVIATIONS

ADD	adductor
ABM	ambulator
A S Q	athetoid spastic quadriplegic
BI	bilateral
BI PLANT FASC	bilateral plantar fasciotomy
BI TP LEN	bilateral tibialis posterior lengthening
DI	diplegic
DIFF	difference
EVERT	eversion
F	female
FLEX	flexor
HAMS	hamstrings
INIT	initial
INVERT	inversion
I.Q.	intelligence quotients
M	male
MAX	maximum
MIN	minimum
ORTHO	orthopaedic surgery
PAT	patient
PL FASC	plantar fasciotomy
POSTOP	postoperative

<b>PREOP</b>	<b>preoperative</b>
<b>Q</b>	<b>quadriplegic</b>
<b>SD</b>	<b>standard deviation</b>
<b>SPLR</b>	<b>selective posterior lumbar rhizotomy</b>
<b>SUP</b>	<b>supinated</b>
<b>TA</b>	<b>tendon Achilles</b>
<b>vs</b>	<b>versus</b>

# CHAPTER 1

## INTRODUCTION

Spasticity and its treatment have baffled and frustrated the medical and paramedical professions from the time that an interest was first shown in the rehabilitation of cerebral palsied patients.

Often cerebral palsy is not diagnosed early. It is only when abnormal development becomes very obvious that doctors are prepared to label the condition. However with new specialized techniques for the diagnosis of brain damage in premature infants it is possible for doctors to predict which children are likely to have problems and early treatment can be prescribed.

The spastic type of cerebral palsy, the most common type, is caused by damage to the motor cortex of the immature brain. This damage occurs before, during or shortly after birth. The damage is permanent but not progressive. The main characteristic of spasticity is resistance to passive stretching of the muscle with resulting difficulty in performing voluntary movements. Other symptoms are: increased deep tendon reflexes, clonus and the retention of

primitive reflexes. Righting and equilibrium reactions are delayed or absent.

As the baby develops, spasticity and the problems associated with it affect the development of normal movements. Developmental milestones are not only delayed but when achieved, are abnormal. In order to achieve the most basic movements, compensations have to be made. These abnormal movements further impair the progression of normal motor development.

This combination of abnormal movement and the use of compensatory movements in order to function, causes deformities. This is a vicious cycle, as the compensations which are made, make movements even less effective and further deformities develop with resulting decrease in function. These deformities and inefficient movements are very difficult to treat.

Not only are there physical problems, but psychological problems develop as a result of them.

Many of these patients experience serious perceptual and learning problems once they enter school. When spasticity is present, the patient is unable to experience the tactile, proprioceptive, kinaesthetic and other sensations necessary for learning. What he experiences is often distorted and inaccurate.

Some of the complex problems related to spasticity which therapists have to deal with are that:

- \* Normal movement can only be elicited once spasticity is inhibited.
- \* This inhibition must be maintained to allow the patient to function (very often the spasticity can only be inhibited as long as the hands of the therapist are on the patient or for very short periods thereafter).
- \* Despite the spasticity the therapist must strive to devise ways of assisting the patient to become independent in mobility and self-care activities.
- \* Positions in which the patient is placed, must enable him to learn the things that a normal child learns at play and at school.
- \* Parents have to be educated in various handling techniques so that more normal movement is promoted.

Apart from these physical problems, psychological and emotional problems must also be considered in therapy. The stress on the mother, father and siblings is tremendous, and just as spasticity increases with age, so do stresses in coping with it. Parents of spastic patients seek help from

many different sources and try many different "remedies" only to continue feeling disheartened when the treatments do not meet their expectations.

For many years, specialists in different fields have used their expertise to try to solve the problems of spasticity. Although some relief of spasticity was obtained to a greater or lesser degree, it was not totally alleviated, and the battle to achieve movement closer to normal continued.

Although orthopaedic surgery corrects deformities to a certain extent, the pain and suffering involved is not rewarded with big improvements in function.

Dr. W.J. Peacock, who was the paediatric neurosurgeon at the Red Cross Children's Hospital in Cape Town, became interested in the management of spasticity and began using the surgical procedure of Selective Posterior Lumbar Rhizotomy (SPLR) on patients in 1980. Soon patients were selected for this procedure from many different parts of the country and travelled to Cape Town for surgery.

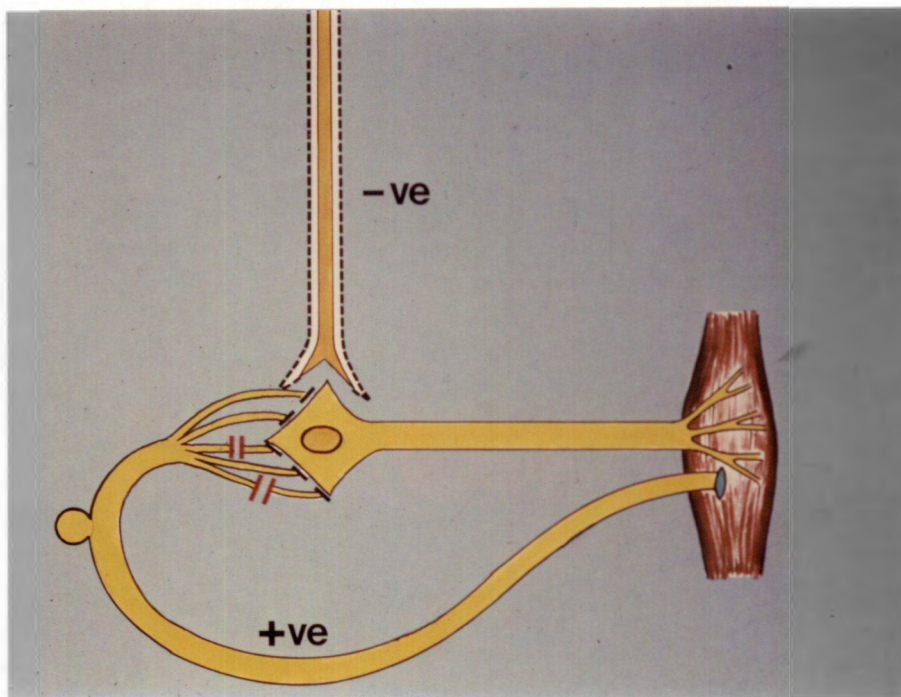
The objective of SPLR is to get to the "root" of the problem by permanently removing the spasticity, rather than treating the consequences of it. When there is spasticity there is too little inhibition from the cortex and therefore less dampening effect on the sensory input which occurs when a muscle is stretched. By selectively cutting the posterior

nerve rootlets of the spinal nerves, a better balance is created between the incoming sensory impulses and the damaged descending fibres (Fig. 1, page 6). This balance allows the patient to utilize his remaining abilities without spasticity. In other words, he is able to use the voluntary movement more effectively, and deformities are not likely to develop due to spasticity. This is not a cure for the brain damage, but if normal movement is facilitated, excellent results are possible and the quality of living can be improved substantially.

The initial results of SPLR were very exciting. Therapists and parents saw dramatic changes. For the first time, therapists did not need to "fight" spasticity. They were able to facilitate normal movement patterns and to concentrate on participation in activities. Patients also became aware of the improvement and made comments such as "I have new legs now". They moved with greater freedom and through wider ranges. Their independence and concentration improved and they cooperated better in therapy.

FIGURE 1

A graphic representation of two posterior nerve rootlets which have been cut in order to balance the incoming sensory impulses with the limited inhibition from the damaged descending tracts



As an occupational therapist concerned with both the physical and psychological well-being of my patients, I was excited by these changes.

Since the observations of improvement were subjective and from varying sources, it seemed important from both the doctor's and therapist's points of view to measure the results of SPLR objectively. This could lead to more effective selection of patients. It could also facilitate planning and implementation of the occupational therapy program following surgery.

With the encouragement of Dr. Peacock, a formal prospective clinical study was planned and undertaken in 1985.

The objective of this study was to determine the effects of Selective Posterior Lumbar Rhizotomy on muscle tone, voluntary movement, joint stiffness and functional movement and to determine the relationships between these parameters. In order to do so, objective assessment scales had to be developed.

## CHAPTER 2

### LITERATURE REVIEW

The literature will be reviewed in the following sequence: cerebral palsy, spasticity, treatment (conservative and surgical) and methods of clinical evaluation of spasticity.

#### CEREBRAL PALSY

##### Definition

The International Dictionary of Medicine and Biology (Landau, 1986) states: Cerebral Palsy is "any number of chronic, non progressive disorders of the brain which impair motor function, occurring in young children. The brain lesion may be developmental or acquired as a result of perinatal infection, birth injury or asphyxia, or kernicterus, often due to Rh factor incompatibility. Occasionally it results from postnatal infection or trauma. The typical picture is that of spastic tetraplegia, diplegia or hemiplegia, often combined with athetosis or ataxia. In addition both convulsions and mental retardation are common".

In cerebral palsy, the state of muscle tone and abnormal patterns of movement interfere with the orderly sequence of motor development (Semans, 1965).

### Aetiology

Any abnormalities which damage the immature brain can lead to cerebral palsy. The most frequent abnormalities include hypoxia, periventricular haemorrhage, injury due to mechanical trauma, intrauterine infection and kernicterus (Erenberg, 1984).

The aetiological factors, location of the lesions and consequent manifestations are diverse (Milner-Brown & Penn, 1979). Nelson and Ellenberg (1986), in a study examining prenatal and perinatal factors predicting cerebral palsy, concluded that:

- \* No single intervention is likely to prevent a large proportion of cerebral palsy if the causes are unknown, numerous or arise early in development.
  
- \* Labour and delivery have a relatively small role in accounting for cerebral palsy. In a large proportion of cases the outcome may have been related partly or wholly to defects intrinsic to the fetus. Pre-existing abnormalities suggest that brain damage was prenatal rather than intrapartum.

These authors feel that the causes of cerebral palsy may need re-evaluation.

In a recent epidemiological study (Hagberg & Hagberg in Galjaard et al. 1987) the authors point out that cerebral palsy is most often related to negative events during the perinatal period.

#### Types of Cerebral Palsy

There are several types of cerebral palsy but the most common type is the spastic variety which comprises approximately 65% of cerebral palsied patients. In this type there is a lesion in the motor portion of the cerebral cortex. The other main types are athetosis (25%) where the damage is in the basal ganglia, ataxia (10%), where involvement is in the cerebellum and various combinations of the above. All types present with varying degrees of severity and associated disabilities. Associated disabilities include for example, mental retardation, dysphasia, apraxia, hemianopia, feeding and other problems (Moore, 1984). The clinical neurological lesion has variable distribution: monoplegia, hemiplegia, paraplegia, quadriplegia and diplegia (Scherzer & Tscharnuter, 1982).

## SPASTICITY

The motor symptoms and topographic involvement of cerebral palsy have long been recorded in the history of man but it was not until 1853 when medical attention was called to the clinical condition by an English surgeon, William J. Little (Wolf, 1969).

Dorland's Illustrated Medical Dictionary (Friel, 1981) defines spasticity as "a state of hypertonicity, or increase over the normal tone of a muscle, with heightened deep tendon reflexes". Clasp-knife spasticity is "the phenomenon occurring when a hypertonic muscle is passively stretched: after an initial increased resistance there is a sudden relaxation".

This state of neuromuscular dysfunction is characterized by predictable, constant distribution of hypertonus, greatest in the flexors of the upper extremities and extensors of the lower extremities (Bleck, 1979).

### Aetiology of Spasticity

The exact neurophysiological mechanisms underlying spasticity are not fully known. Sherrington (1913) and Magoun and Rhines (1946) have shown that decerebration of a cat or dog sets free centres of inhibition and facilitation within the reticular substance of the brainstem, which influence postural tone by modifying the spinal reflex mechanism of muscular coordination. Decerebration also sets free the various tonic reflexes which produce the typical patterns of posture and movement associated with hypertonus.

Although research has thus shown that spasticity is caused by release of the gamma system from higher inhibitory control, lack of presynaptic inhibition and other abnormalities are also possible causes (Barlot-Romana, 1980).

### Characteristics

Spasticity consists of a marked increase in muscle tone, an increase in deep tendon reflex activity and results in resistance to active and passive movements. In the spastic individual, an increase in resistance, which appears to be dependent on the velocity of passive movement, is detectable with an intensity that reflects the degree of involvement (Otis et al. 1983).

Gillette (1966) describes the following characteristics:

- \* clonus
- \* short stretch augments tone
- \* long stretch inhibits tone
- \* dominant flexor and extensor synergies
- \* contractures which are common and appear early in hypertonic muscle groups
- \* imbalance in power due to constant imbalanced tone, leads to contractures and weakness from disuse
- \* perceptual disturbances.

### Effects

The spastic type of cerebral palsy is commonly characterized by increasing spasticity and deformity as the child grows (Perry in Feldman et al. 1980).

Normal efficient movement is dependent on normal muscle tone and on postural alignment (Campbell, 1984).

In patients with spasticity, pliability of the limbs is decreased and voluntary movement is slow and hence there is generalized instability of posture and movement. When they are brought into use the opposing muscles are weak.

All these factors affect the development of normal motor milestones.

Poor gait is due to failure of development of power to attain and maintain simultaneous abduction, external rotation and extension of the thigh with dorsiflexion of the foot (Collis et al. 1956).

## TREATMENT

Many methods have been used to reduce spasticity. These methods differ widely but the main emphasis is to enable the patient to become as functional as possible in his interaction with his environment (Levitt in Galjaard et al. 1987).

Each method is useful, and produces some change, but no method produces the desired effects quickly, and none effects a cure (Cardwell, 1956).

### Conservative Treatment

These methods are, among others, described by Phelps, Fay, Deaver, Kabat, Bobath, Swartz, Pohl, Doman and Rood (Cardwell, 1956; Scherzer, 1986). The rationale behind most physiotherapy procedures is to interrupt abnormal patterns and to prevent secondary deformities. Patients must be taught new movement patterns that will become automatic (Bishop, 1977).

### Drug Therapy

Other non-surgical means of treatment include the use of centrally-acting and peripherally acting drugs, nerve block at the neuromuscular junction, and chemical rhizotomy. Most drugs work in various ways on the synapses (Davidoff, 1985). The most commonly used drugs are diazepam (Valium), dantrolene (Dantrium) and baclofen (Lioresal). Varying degrees of success have been claimed with certain drugs (Penn & Kroin, 1987; Carpenter, 1980). However, the reduction of spasticity is not permanent and there are often undesirable side effects. The major side effects are sedation and weakness which occur with the high doses often needed to achieve the desired effects on spasticity (van Hemert in Feldman et al. 1980; Erenberg, 1984).

### Bracing

A trial has shown that bracing does not prevent the development of deformity (Sharrard, 1984). Bracing produces a shift of spasticity to the nearest free joint and with prolonged use of braces the balance between opposing spastic muscle groups will even out, but at the expense of mobility (Bryce, 1976).

Fatigue occurs 50% sooner when walking in braces than out of them and produces no permanent change in, or cure of, the abnormal stance (Frost, 1971).

In a prospective study to determine the benefits of inhibitive casts as an adjunct to neurodevelopmental therapy, Watt et al. (1986) concluded that orthoses failed to maintain the improvement observed immediately after cast removal.

Normalization of tone by the above methods is typically short term and has minimal effect on attaining either postural alignment or improving coordination of patterns of movement (Campbell, 1984).

Braces and orthopaedic operations can do more harm than good since "the basic abnormality resides in the nervous system, not in the limbs, and braces, and orthopaedic operations have no effect whatsoever on the abnormal neural mechanisms" (Frost, 1971).

#### Orthopaedic Procedures

Extensive work has been done in the field of orthopaedic surgery. Many new procedures for example tendon lengthenings, releases and peripheral neurectomies have been reported and recommended.

Although the results of surgical techniques have improved, spasticity is not removed. Perry in Feldman et al. (1980) reports that surgical results have been inconsistent.

Since spasticity is coordinated into patterns, the release of one group of muscles will not effect the pattern itself but will allow the effect of spasticity in opposing muscle groups to be much stronger (Bryce, 1976; Gillette, 1966).

Gillette (1966) points out that no matter how one re-establishes the length of contracted hamstrings, the central nervous system lesion is irreparable and life long: the spasticity continues. Keats (1970) emphasises that recurrences of deformity following orthopaedic surgery develop due to alteration of muscle length and bone growth in the presence of muscle imbalance. If no efforts are made to prevent recurrence with night support and exercises, especially during the growing period, any procedure will fail (Banks, 1972).

There are many specific corrective procedures at each joint. The choice of technique is based on whether one wishes to remove the offending muscle action or only weaken it (Perry in Feldman et al. 1980). It is easier to predict the results of orthopaedic operations with regard to deformities than with regard to functional activity (Holt, 1966).

## Specific Lower Extremity Problems

### The Hip

Flexion, adduction and internal rotation deformities are common. Hip dislocation occurs in the most severely involved spastic cerebral palsied patients. There is no single appropriate surgical procedure. Samilson (1981) emphasises that careful analysis of the exact cause of the problem is important in planning treatment. Procedures for distal rectus femoris transfer, adductor transfer and iliopsoas recession, in combination with other procedures, have been described by Perry (1987), Root & Spero (1981) and Bleck (1971a) respectively. These authors report improvement in function.

Patients under 8 years of age benefit from advancement of the insertion of gluteus medius and minimus to correct internal rotation gait (Steel, 1980). For patients older than 7 years, secondary skeletal changes seem to preclude success by muscle release alone. In these cases, varus derotation femoral osteotomy to correct the associated excessive femoral anteversion and acetabular reconstruction appear to have been successful in preventing further subluxation and progression to dislocation. Muscle surgery, including iliopsoas tenotomy or elongation, is not expected to benefit older patients (Bleck, 1971a; Bleck, 1979).

Hoffer (1986) writes that the aim of using soft tissue procedures and osteotomies is to prolong the ability to walk and to decrease pain.

### The Knee

Knee flexion deformity, recurvatum, valgus, patellar alta, patellar dislocation and chondromalacia patella are common knee problems in cerebral palsy (Samilson, 1981). Various procedures are described to manage the problems (Bleck, 1979).

If adequate correction of hip flexion deformity and equinus deformity is made before it becomes severe, the incidence of knee flexion deformity diminishes considerably (Sharrard, 1984).

### The Ankle and Foot

Equinus or plantar flexed deformity is a common occurrence in spastic cerebral palsy. Optimal management of equinus deformity depends on accurate diagnosis (Makley & Kim in Thompson et al. 1983). A clinical assessment of equinus in patients before and after surgery was made over a 20 year period by Truscelli, Lespargota & Tardieu (1979), who concluded that the results did not depend on the surgical technique but on the differences in pathophysiology.

Recurrence is the most common problem in the surgical correction of the equinus deformity resulting from spastic paralysis (Lee & Bleck, 1978).

Other problems of the foot include calcaneus deformity, valgus deformity, hallux valgus and toe flexion deformities. These are treated orthopaedically by various techniques (Samilson, 1981).

#### Cerebellar Stimulators

Cerebellar stimulation is another method which appears to have beneficial effects on spasticity (Penn, Gottlieb & Agarwal, 1978). However, there does not appear to be an objective improvement in function as reported in a well designed study by Gahm, Russman, Cereciello et al. (1981).

#### Selective Posterior Lumbar Rhizotomy

There has been considerable disagreement as to the most appropriate methods of managing spasticity (Wolf, 1969) and research to evaluate the effectiveness of different methods has been limited. Since the late seventies, however, a neurosurgical technique has been used successfully to reduce muscle tone: this method is called Selective Posterior Lumbar Rhizotomy Procedure (SPLR).

Sherrington transected the midbrains of cats and produced spasticity. The posterior nerve roots of the spinal nerves were then cut and spasticity disappeared (Sherrington, 1898; 1913).

Frazier (1910) in a preliminary report, mentioned early results of SPLR performed by Tietze, Gottstein, Spiller, Clark and Taylor, as well as his personal experience with the procedure which was limited to three cases. Foerster (1913) reported good results following posterior rhizotomy but noted sensation loss which impaired function. These side effects, caused by destruction of the proprioceptive afferent nerves, probably caused the operative procedure to fall into disuse.

In 1967 Gros et al. modified this technique, sparing one quarter or one fifth of the rootlets (those bearing small arteries). In these patients sensory loss was slight and trophic ulcers were avoided. This technique was further refined by using electrophysiological stimulation, muscle testing and electromyographic methods by which rootlets subserving "useful" spasticity were preserved (Privat, 1976).

Fasano et al. (1979) reported a significant reduction of spasticity following selective sectioning of posterior nerve rootlets depending on intraoperative electrode stimulation. In 1983, Laitinen et al. published results using a technique similar to that of Fasano in the treatment of 8 patients with spasticity in the legs and one patient with spasticity in an arm. All fascicles of posterior nerve roots T12 through to S1 and C6 through to C8 were stimulated electrically during surgery. Rootlets that failed to suppress stimulation at high frequencies were sectioned. All patients showed marked reduction of spasticity, mobility of the limbs improved but cutaneous and joint sensation remained unchanged.

Peacock has used a modification of Fasano's approach in a variety of patients most of whom had cerebral palsy (Peacock & Eastman, 1981). The results were positive: spasticity was markedly reduced, mass patterns of movements were reduced and the quality of functional abilities for example sitting, standing and walking improved. Nursing care was made easier in cases of severe spasticity (Peacock & Arens, 1982).

## METHODS OF CLINICAL EVALUATION

Many methods of clinical evaluation have been used to determine the developmental level and degree of motor handicap of patients with delayed motor development.

### Developmental Assessments

Assessments have been based on the research of many practitioners including Baley, (1958); Bobath, (1966, 1975); Capute, (1973); Dubowitz, (1968); Frankenberg, (1971); Gesell, (1947); Illingworth, (1966); Paine, (1961) and others.

The most commonly used procedure is a general clinical assessment. This is based on the "normal" sequence of neuromotor development; its weakness is that data from this method cannot be transferred to a developmentally impaired patient (Scherzer & Tscharnuter, 1982). Comparison with the normal child does not provide an indication of rate and quality of change. Changes in the patient's function may occur but these have not yet become part of functional development, that is, they do not use these movements efficiently.

There are also diverse ways of reporting and the content is not uniform. This diversity and lack of uniformity makes analysis of change in a patient and the judgement of results

of treatment difficult, often biased and unreliable (Scherzer & Tscharnuter, 1982).

Semans (1965) reviews various developmental evaluations and points out that although the tests are based on the achievement of milestones the processes involved in bringing about the latter are lacking. Assessments do not indicate the nature and extent of the interference caused by brain injury.

#### Electromyography

Electromyography provides valuable information (Bleck, 1971) and orthopaedic surgeons are making use of it to assist them in making accurate assessments when planning operative procedures (Bennet, Jones & Rang, 1981). Perry, Giovan, Antonelli & Greenberg (1974) have shown that it is possible to distinguish dynamic posturing from static contracture using electromyography. Csongradi, Bleck & Ford (1979) reported the use of electromyographic analysis of quadriceps femoris and the medial hamstrings in spastic patients to determine alterations from the normal as well as the relationships between these muscles.

#### Video Recording

Therapists and physicians have also been using video recordings (Peacock, Arens & Berman, 1987) but, although the

latter provide good visual images before and after treatment, the changes cannot be quantified.

#### Evaluation of Muscle Tone

Investigators have attempted to quantify the degree of increased muscle tone (Halpern, Patterson, Mackie, Runck & Eyler, 1979). These methods involved the study of one or of a few muscle groups, and are complicated and impractical if many muscle groups are to be tested. Clinical methods, based on the memory of the examiner are often criticised as being unreliable (Scherzer & Tscharnuter, 1982).

#### Evaluation of Voluntary Movement

There are no documented methods of evaluating voluntary movement other than looking at movement in relation to function (Bobath, 1963). Voluntary movement is usually divided into range of movement and muscle power and assessed as these separate components.

#### Evaluation of Joint Stiffness

"Joint Stiffness" or contracture of a joint is examined by testing whether the limb can passively be moved through its full range. It is often very difficult to determine whether any fixed deformity exists.

Different methods are used to determine the degree of contracture (Bartlett, Wolf, Shurtleff & Stahell, 1985).

Bleck (1971b) has described more accurate methods to determine fixed deformities. He feels that the Thomas test for determining the degree of hip flexion contracture only provides one with an estimate of the degree of hip flexion deformity. In addition, he describes roentgenographic measurements to determine the degree of hip flexion contracture in patients.

Roosth (1971) mentions that with spastic cerebral palsy, a knee flexion contracture which is secondary to a hip flexion contracture may or may not be a fixed deformity.

Banks and Panagakos (1966), Holt (1966) and Pollock (1975) agree that the outcome of orthopaedic surgery is difficult to predict because the root of the problem lies in the central nervous system. In many cases the apparent contracture may not be structural and can be reversed by counterbalancing the pattern of spasticity responsible (Bryce, 1976). The structural component is secondary to the dynamic component (Frost, 1971). Tscharnuter (1934) distinguishes between true contractures and correctable movement limitations caused by abnormal postural tone.

### Evaluation of Functional Movement

Numerous investigators have developed charts to determine the developmental levels of patients, among others Gesell (1947); Bayley (1958); Illingworth (1966); Bobath (1975); Capute & Biehl (1973).

Little has however been done to examine the degree of abnormality. Bly (1980) discusses "blocks" as "milestones" in the abnormal development of the cerebral palsied patient. When the antigravity flexor activity does not balance the antigravity extensor activity, as is the case when muscle tone is abnormal, the patient has difficulty stabilizing one part of his body so that another can move. Because the patient cannot stabilize himself normally, he "learns" to "fix" and this fixing prevents further movement of that segment. From the point of the "block", the patient develops compensations in order to function and abnormal motor development progresses.

### Evaluation of Gait

Gait is defined by the International Dictionary of Medicine and Biology (Landau, 1986) as "the way in which an individual walks". Spastic gait is "the gait which results from spasticity". When both lower limbs are spastic, the patient walks stiffly, scraping the feet along the ground.

With severe spasticity, as in cerebral diplegia, the legs may cross each other alternately while walking ("scissors gait"). Spastic equinus gait is "gait characterized by spastic and uncoordinated movements of the lower limbs, with weight borne primarily on the forefoot, since the ankles are plantar flexed. There is often associated adduction and internal rotation of the hips causing the knees and feet to turn inward" (Landau, 1986).

In patients with spasticity the imbalance of muscle activity causes inappropriate muscle action. In analysing spastic gait patterns, the problems are diverse (Hoffer & Perry, 1983).

Basic information on developing walking patterns seems a prerequisite to the analysis of pathologic problems (Burnett & Johnson, 1971). Sutherland (1984) points out the most important indicators of mature gait are:

- \* duration of single limb stance
- \* walking velocity
- \* cadence
- \* step length
- \* the ratio of the pelvic span to the ankle spread

Many methods of gait analysis used today are costly and not easily accessible to the clinician. The methods used by various investigators were reviewed by Sutherland (1978).

The gait cycle is defined as the period from heelstrike of one extremity to heelstrike of the same extremity and includes a stance and a swing phase (Burnett & Johnson, 1971). Only by precise preoperative and postoperative studies can treatment for locomotor problems be reliably assessed. Progress in cerebral palsied patients cannot be achieved without such objective assessment (Sutherland, 1978).

Variations in normal walking include changes in length and frequency, range of motion and speed. Walking for a child with spasticity is made difficult by the following factors:

- \* it is hard for him to achieve balance due to the internally rotated thighs and overactive adductors.
- \* spasticity and contractures leave him a poor base of support.
- \* abnormal compensations occur in order to allow him to maintain the upright position.
- \* weight distribution and balance of his trunk and pelvis on his legs are poor due to the decrease of the width of stance. Scissoring may occur.
- \* his inability to contract and relax leg and knee muscles makes it impossible for him to achieve normal rhythmic walking movements.

\* pathological foot posture: equinus, varus, valgus and calcaneus may be present (Cardwell, 1956; Hoffer & Perry, 1983).

Hoffer and Perry (1983) state that analysis of these various problems enables the clinician to plan the most appropriate treatment in order to improve the patient's gait.

While a substantial amount of work has been done on gait analysis of spastic patients to assist in the planning and evaluation of orthopaedic surgery (Riso & Marsolais in Thompson et al. 1983), no work had been done, prior to this study, to investigate the effects of SPLR on gait.

## CHAPTER 3

### METHODOLOGY

An ideal design would have involved a comparison of the results of the experimental treatment against a control group which had a sham operation. As mentioned in a later chapter, I decided against this design.

The null hypothesis was assumed (i.e. that there was no difference between the preoperative and postoperative groups) and the following were examined:

#### Hypothesis 1

SPLR does not lead to a reduction in muscle tone of all spastic muscle groups of upper and lower limbs.

#### Hypothesis 2

SPLR does not lead to an improvement in voluntary movement of all spastic muscle groups of upper and lower limbs.

#### Hypothesis 3

SPLR does not lead to an improvement of joint stiffness of upper and lower limb joints.

#### Hypothesis 4

SPLR does not lead to an improvement in functional movement (rolling, sitting, kneeling, crawling, standing and walking).

In order to test these null hypotheses, a prospective study was done using each patient as his own control. This involved a comparison of the clinical condition of each patient before and after Selective Posterior Lumbar Rhizotomy.

#### SAMPLE SELECTION

All patients who underwent surgery for SPLR during 1985 were examined before and after surgery.

Patients were referred to Dr. Peacock by hospitals and schools for the cerebral palsied from all parts of the Republic of South Africa. For the purposes of the study, they were assessed in Cape Town and followed up in the towns from which they came.

Patients varied in age from 2 to 35 years and were selected from all races and socio-economic groups. They varied with regard to intelligence and associated handicaps. There were 18 males and 11 females.

### Eligibility Criteria

All patients were cerebral palsied due to prenatal, perinatal or postnatal aetiology with the exception of three who had spasticity due to cerebral trauma. The presence of spasticity in the limbs was the main criterion for selection for surgery regardless of the patient's motor abilities.

Dr. Peacock selected all patients for SPLR according to the following criteria:

- \* Realistic goals were set, for example whether the SPLR would be performed to improve functional movement or to facilitate caretaking.
- \* Patients had to have spasticity.
- \* Athetoids were excluded, although if spasticity was present with athetosis, patients were evaluated on an individual basis. If spasticity was the main problem, they were considered for surgery.
- \* Patients who had severe underlying muscle weakness were excluded.
- \* The patients all had to have the facility for postoperative therapy.

Even patients who were less able to cooperate due to low intelligence were not excluded from this study, since it was possible to complete most of the tests required.

## VARIABLES

The following variables were identified:

### Independent Variable

The neurosurgical procedure (SPLR) was the only independent variable.

### Dependent Variables

The following dependent variables were identified:

Hypothesis 1 - The state of muscle tone.

Hypothesis 2 - The underlying voluntary movement.

Hypothesis 3 - The degree of joint stiffness.

Hypothesis 4 - The state of functional movement  
(rolling, sitting, kneeling, crawling,  
standing and walking).

### Confounding Variables

The following confounding variables were noted:

Age

This varied from 2 to 35 years. Twenty seven of

the 29 patients were 15 years and under; 2 were over 21 years of age.

#### **Sex**

There were 11 females and 18 males.

#### **Socio-economic status**

This varied widely with some patients coming from wealthy families and others from very poor ones.

#### **Intelligence Quotients**

These varied widely from very low to above average.

#### **Therapy**

Patients came from many different parts of the country and some received therapy more regularly than others.

#### **Previous Orthopaedic Surgery**

Some patients had undergone various types of orthopaedic procedures whereas others had not had any surgery.

#### **Reassessment**

Many patients lived in other parts of the country (Kimberley, Johannesburg, Durban and Port Elizabeth) and they were reassessed in their home environments no less than four months postoperatively.

### Severity of spasticity

Although all patients were spastic, the severity and distribution of spasticity varied widely from patient to patient.

### TESTING PROCEDURE

Data was collected over a period of 14 months and all testing and retesting was done under similar conditions. The formal clinical evaluation was identical in all 29 patients before and after the surgery.

Procedures were designed to test the muscle tone, voluntary movement, joint stiffness of upper and lower limbs and functional movement of the patients in certain developmental positions. Patients were assessed two days prior to undergoing SPLR, and after a period of at least 4 months they were reassessed. All the assessments and reassessments were done by the same therapist in order to minimise inconsistency. The preoperative scores of the assessments were not seen while the postoperative tests were being done.

Patients were assessed before surgery in the hospital on a firm surface in a quiet, warm room. After surgery, they were reassessed at the schools they attended or at a hospital, under similar conditions.

Analysis of gait and crawling was done in conjunction with Dr. C.L. Vaughan (Department of Biomedical Engineering, University of Cape Town) in a room attached to the hospital or at the patient's school.

For logistical reasons, all patients living outside Cape Town were assessed during one week and therefore the postoperative assessment was done up to 14 months after surgery in some patients.

#### SCORING PROCEDURE

Prior to using the method of assessment in this study, the grading system was distributed among twenty therapists who were trained in Neurodevelopmental Therapy (Bobath) and comments were invited. Improvements were made according to their suggestions and assessment forms were prepared for this study (Appendix C, page 197).

The following were quantified according to the degree of limitation and scales were designed by which changes could be measured:

1. Muscle Tone
2. Voluntary Movement
3. Joint Stiffness
4. Functional Movement

In the case of voluntary movement and functional movement, 1 represented "normal" and 5 the severest limitation. In the case of joint stiffness, 1 represented "normal" and 4 indicated the severest limitation. In the case of muscle tone, 2 represented "normal", 1 "low" and 5 the severest spasticity.

#### ASSESSMENT OF MUSCLE TONE

Increased muscle tone is influenced by many factors including anxiety, temperature, rate of stretch, effort and different body positions. Pathological reflexes also influence muscle tone. Clinicians who treat cerebral palsied patients and are experienced in handling them will be able to rate the degree of predominant tone that is present on testing according to the scale described here.

## Rating Scale For Muscle Tone

### 1 = HYPOTONIA

The limb feels floppy and is unable to resist gravity.

### 2 = NORMAL

A good balance between agonist and antagonist. Movement feels controlled. No resistance to passive movement.

### 3 = MILD INCREASE

There is slight resistance to passive stretch and there is a slight decrease in joint mobility. The stretch reflex occurs when the muscle is in a lengthened position.

### 4 = MODERATE INCREASE

There is greater resistance to passive movement than in 3 above and a greater decrease in mobility. The stretch reflex occurs in the middle of the range.

### 5 = SEVERE INCREASE

There is a severe increase in resistance to passive stretch and a severe decrease in mobility. The stretch reflex occurs when the muscle is in a shortened position.

## TECHNIQUES AND PATTERNS USED FOR TESTING MUSCLE TONE

In order to test the tone in any muscle group, the limb was passively moved in a direction opposite to the action of the muscle group being tested.

### Lower Limbs

Hip flexors (Iliopsoas, Sartorius, Tensor fascia latae, Rectus femoris) (Fig. 2a, page 45)

Shortening of the hip flexors including the rectus femoris and iliopsoas muscles was determined using the rectus femoris (Roosth, 1971) and Thomas tests (Thomas, 1876) prior to testing the tone in these muscles. Since one cannot isolate individual hip flexors, the prone rectus test can be positive for any of the hip flexors (Hoffer, 1986).

### Test of muscle tone

The patient was placed in a side lying position with the thigh in line with the trunk. Alternate hips were passively extended. Resistance to extension indicated increased tone in the hip flexors.

Hip Extensors (Gluteus maximus) (Fig. 2b, page 45)

The hip extensors of spastic cerebral palsied patients are usually weak due to disuse from the muscle imbalance caused by the spasticity.

Test of muscle tone

The patient was placed in side lying. Alternate legs were flexed at the hips starting with the thigh in line with the trunk. The degree of resistance to passive movement was rated.

Hip Abductors (Gluteus medius, Gluteus minimus, Tensor fasciae latae) (Fig. 2c, page 45)

Test of muscle tone

The patient's legs were passively adducted from a position of abduction with the patient in the supine position. (As with the hip extensors, this muscle group is usually weak due to adductor spasticity).

Hip Adductors (Pectineus, Adductor magnus, Gracilis, Adductor brevis, Adductor longus) (Fig. 2d, page 45)

Test of muscle tone

The patient was positioned in supine and the legs were abducted from a position of adduction, first with extended knees and then with flexed knees. The degree of muscle tone was rated.

Knee Flexors (Hamstrings: Semitendinosus, Semimembranosus) (medial), Biceps femoris (lateral) (Fig. 2e, page 46)

Test of muscle tone

Resistance to knee extension was tested with the patient in a supine position. This was done starting with the knees flexed.

Knee Extensors (Quadriceps femoris) (Fig. 2f, page 46)

Test of muscle tone

With the patient in the supine position, starting with the knees in extension, the resistance to knee flexion was rated.

Plantar flexors (Gastrocnemius, Soleus, Tibialis posterior, Peroneus longus, Peronius brevis, Flexor hallucis longus, Flexor digitorum brevis) (Fig. 2g, page 46)

Test of muscle tone

With the patient in the supine position, and with the feet plantar flexed, alternate feet were dorsiflexed. This was done with first with the knees extended and then with the knees flexed. The degree of resistance to dorsiflexion was rated.

Dorsiflexors (Tibialis anterior, Extensor hallucis longus)  
(Fig. 2h, page 46)

Test of muscle tone

The feet were alternately plantar flexed starting with the feet in a dorsiflexed position. The degree of resistance to plantar flexion was rated. The patient was tested in supine. (This group of muscles is usually weak due to the overactivity of the plantar flexors).

Invertors (Tibialis anterior, Extensor hallucis longus, Tibialis posterior, Flexor digitorum longus, Flexor hallucis longus) (Fig. 2i, page 47)

Test of muscle tone

The resistance to passive movement into eversion was tested and the muscle tone rated. The patient remained supine.

Evertors (Extensor digitorus longus, Peroneus tertius, Peroneus longus and brevis) (Fig. 2j, page 47)

Test of muscle tone

The patient's feet were inverted starting with the feet in a neutral position and the muscle tone was rated by the degree of resistance to inversion.

## Upper Limbs

The reason for examining muscle tone in the upper limbs was to determine whether there was any significant change postoperatively in those muscles. Although the posterior nerve rootlets are selectively cut in the lumbar region only, many collateral nerve fibres which may influence different levels, traverse the spinal cord.

The assessment was done with the patient in the supine and sitting positions. As when testing muscle tone in the lower limbs, the joint was passively moved in the direction opposite to the action of the muscle group being tested. Resistance to shoulder abduction, adduction, internal and external rotation, flexion, extension, elbow flexion and extension, forearm supination and pronation, wrist flexion and extension, finger flexion and extension, and thumb abduction were tested and rated on the 5 point scale.

FIGURES 2a - 2d

TESTS OF MUSCLE TONE

To test the muscle tone in any muscle group, the limb was passively moved in a direction opposite to the action of the muscle group being tested (the arrow indicates the direction of the passive movement of the limb).

Fig. 2a. Hip flexors

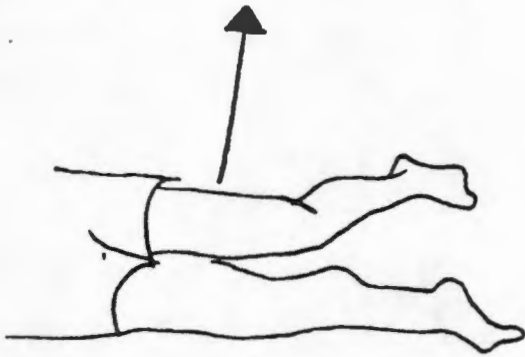


Fig. 2b. Hip extensors

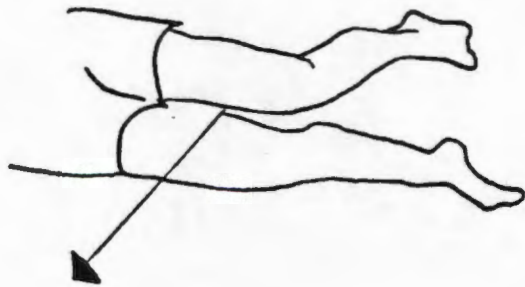


Fig. 2c. Hip abductors

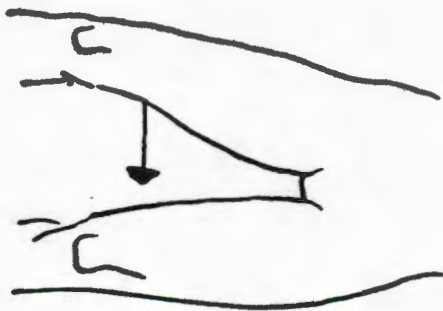
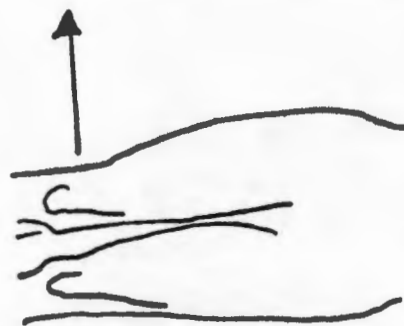


Fig. 2d. Hip adductors



FIGURES 2e - 2h

TESTS OF MUSCLE TONE

Fig. 2e. Knee flexors

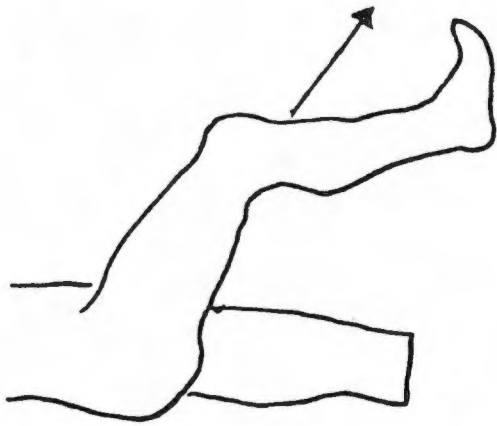


Fig. 2f. Knee extensors

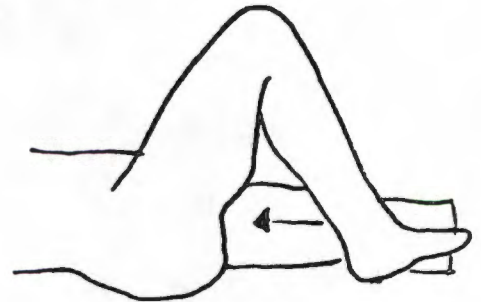
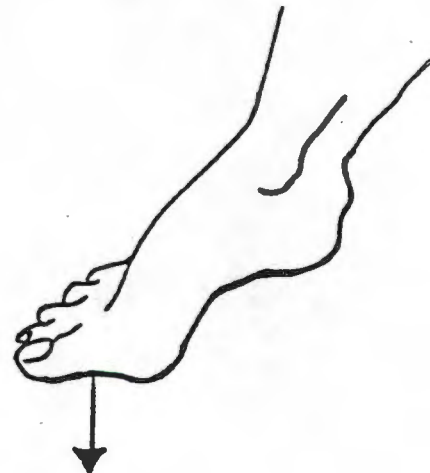


Fig. 2g. Plantar flexors



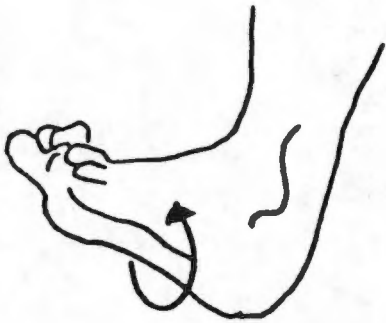
Fig. 2h. Dorsiflexors



**FIGURES 2i - 2j**

**TESTS OF MUSCLE TONE**

**Fig. 2i. Invertors**



**Fig. 2j. Evertors**



## ASSESSMENT OF VOLUNTARY MOVEMENT

A 5 point scale was devised and the quality of voluntary movement at the joints of the upper and lower limbs was rated. The patient was asked to start and stop a movement 8 times within his full available range of movement, counting with each movement. The joints proximal to the movement were stabilized in a position so as to inhibit mass patterns as much as possible.

## Rating Scale For Voluntary Movement

### 1 = NORMAL

Able to initiate and inhibit (ie. stop and start) a movement 7 to 8 times during one full range of movement. The movement is smooth.

### 2 = MILD LIMITATION

Able to initiate and inhibit the movement 5 to 6 times. Can achieve full range but movement is jerky and mass patterns are present.

### 3 = MODERATE LIMITATION

Able to initiate and inhibit movement 3 to 4 times. Can achieve middle or full range but movement is not smooth.

### 4 = MODERATELY SEVERE LIMITATION

Able to initiate and inhibit movement 1 to 2 times. Movement is jerky or overshoots, or there is total movement and patient uses compensations in attempting the movement.

### 5 = SEVERE LIMITATION

Unable to initiate and inhibit movement. Range is totally limited or there is a totally abnormal pattern.

## TECHNIQUES AND PATTERNS USED FOR TESTING VOLUNTARY MOVEMENT

Patients were all tested in a position to eliminate the influence of gravity. This was a supine or side lying position.

*The patient was asked to move the limb in the direction of the action of the muscle group being tested.*

In the case of uncooperative or young children, movements were observed rather than being formally tested (this was the case with three patients); caretakers and parents were questioned with regard to the child's ease of movement before and after surgery but the results were not included in the statistical analysis.

### Lower Limbs

#### Hip flexors (Fig. 3a, page 53)

In the side lying position, the patient was asked to initiate and inhibit hip flexion through the full available range starting from 15 degrees of hip extension.

#### Hip extensors (Fig. 3b, page 53)

In the side lying position, the legs were alternately placed in flexion at the hips and the patient was asked to extend his hips by voluntarily initiating and inhibiting

the movement. Ten degrees beyond the neutral position is expected for a score of 1 (the score for normal).

Hip abductors (Fig. 3c, page 53)

In the supine position the patient was asked to abduct each leg starting from a position with both legs together. This movement was to be initiated and inhibited 8 times. It was important to note whether the patient used his hip flexors in order to achieve abduction.

Hip adductors (Fig. 3d, page 53)

In the supine position the patient's legs were abducted and he was required to initiate and inhibit adduction 8 times.

Knee Flexors (Fig. 3e, page 54)

In the side lying position, the knees were extended and the patient was asked to initiate and inhibit knee flexion. Hips were in the neutral position, i.e. in line with the trunk.

Knee Extensors (Fig. 3f, page 54)

In the side lying position, the knees were flexed and the patient was asked to initiate and inhibit knee extension. Hips were in a neutral position.

Plantar flexors (Fig. 3g, page 54)

With the patient lying in the supine position, the foot was placed in a dorsiflexed position and he was asked to plantar flex his foot by initiating and inhibiting the movement 8 times.

Dorsiflexors (Fig. 3h, page 54)

The patient remained supine and the foot was placed in plantar flexion. The patient was asked to initiate and inhibit the movement into dorsiflexion 8 times.

Invertors (Fig. 3i, page 55)

The test was done in the supine position. The patient was asked to initiate and inhibit inversion from the everted position 8 times.

Evertors (Fig. 3j, page 55)

The patient was asked to evert the foot from the inverted position, 8 times. The patient remained supine.

Upper Limbs

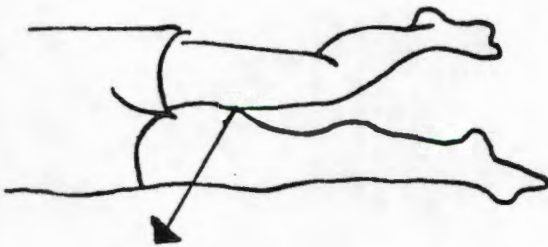
Upper limb movements were assessed in the same way as lower limbs movements. The patient was assessed in the supine and sitting positions and all movements were rated according to the rating scale.

**FIGURES 3a - 3d**

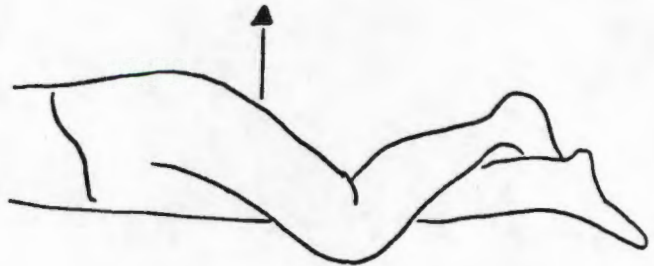
**TESTS OF VOLUNTARY MOVEMENT**

To test the voluntary movement in any muscle group, the patient was asked to move the limb in the direction of the action of the muscle group being tested (the arrow indicates the direction of the active movement of the limb).

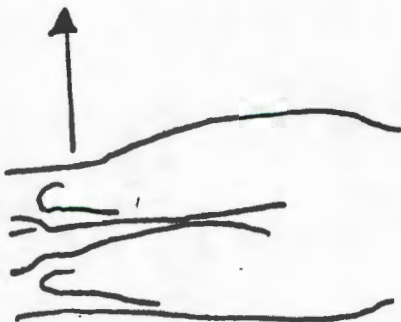
**Fig. 3a. Hip flexors**



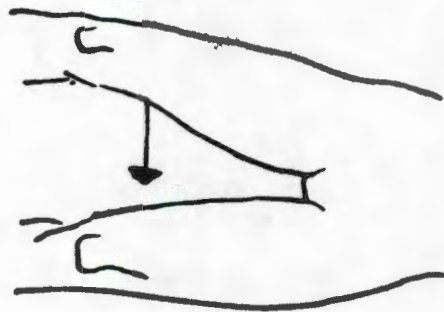
**Fig. 3b. Hip extensors**



**Fig. 3c. Hip abductors**



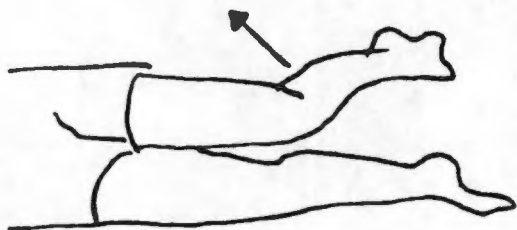
**Fig. 3d. Hip adductors**



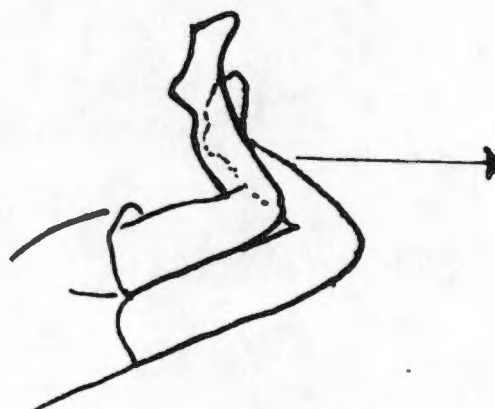
**FIGURES 3e - 3h**

**TESTS OF VOLUNTARY MOVEMENT**

**Fig. 3e. Knee flexors**



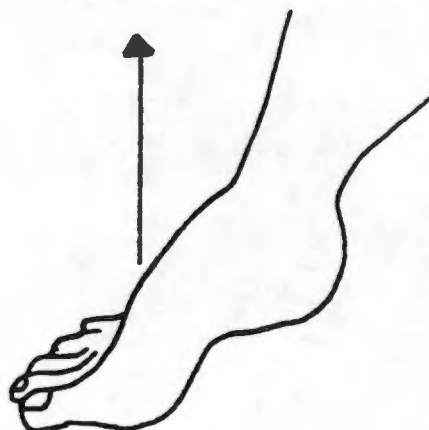
**Fig. 3f. Knee extensors**



**Fig. 3g. Plantar flexors**



**Fig. 3h. Dorsiflexors**



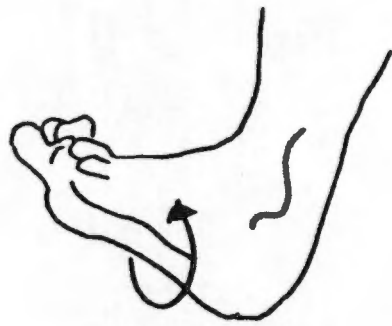
**FIGURES 3i - 3j**

**TESTS OF VOLUNTARY MOVEMENT**

**Fig. 3i. Invertors**



**Fig. 3j. Evertors**



## ASSESSMENT OF JOINT STIFFNESS

It is difficult to distinguish clinically between fixed contractures and apparent contractures, due to excessively high muscle tone in some patients. It was therefore necessary to develop a rating scale to record the degree of joint stiffness. In this way joint stiffness before and after surgery could be compared.

Rating Scale For Joint Stiffness

1 = NORMAL

No contracture. The joint can be moved freely and full range is easily attained.

2 = MILD LIMITATION

There is SOME STIFFNESS. This can easily be released by passive movement.

3 = MODERATE LIMITATION

There is a GREAT DEAL OF STIFFNESS but this can be released with difficulty on passive movement. Full range can be attained.

4 = TOTAL LIMITATION

APPARENT FIXED CONTRACTURE or FIXED CONTRACTURE. Great limitation to passive movement. Full range of movement of the joint cannot be achieved before surgery but may be achieved after surgery.

## TECHNIQUES AND PATTERNS USED FOR TESTING JOINT STIFFNESS

The patient was positioned in the same position as for testing muscle tone. Each joint was rated on a 4 point scale according to the degree of joint stiffness.

*To test joint stiffness in any joint, the limb was moved passively through the maximum range of movement of the joint for that patient and compared with the normal full range of joint movement.*

### Lower Limbs

#### Hip flexion (Fig. 4a, page 61)

Rectus femoris is a hip flexor and knee extensor. A contracture of rectus femoris is tested by placing the patient in a prone position and flexing the knees. If the hips flex and the pelvis lifts off the surface, an apparent or fixed contracture may be present. Pressure should be put on the pelvis in a downward direction. The degree of joint stiffness is rated according to the ease with which the pelvis comes to rest on the surface.

Hip extension (Fig. 4b, page 61)

The legs were alternately moved in the direction of hip extension and the degree of limitation noted.

Hip abduction (Fig. 4c, page 61)

The legs were abducted and any stiffness was noted.

Hip adduction (Fig. 4d, page 61)

Legs were alternately adducted and the degree of limitation was rated.

Knee flexion (Fig. 4e, page 62)

The knees were alternately flexed. The degree of limitation of joint movement was rated.

Knee extension (Fig. 4f, page 62)

With the patient in the supine position, first one and then the other leg was extended at the knee and flexed at the hip. The degree of joint stiffness and limitation to full extension was rated.

Plantar flexion (Fig. 4g, page 62)

The feet were alternately plantar flexed. Any joint stiffness was rated.

Dorsiflexion (Fig. 4h, page 62)

The feet were alternately dorsiflexed. The degree of limitation of movement into dorsiflexion denoted the degree of joint stiffness. Normally, the foot should be correctible to 10 degrees beyond 90 degrees if no contracture is present. When testing, the foot should be well aligned and the calcaneus should be supported by the tester's hand.

Inversion (Fig. 4i, page 63)

The feet were alternately inverted and the degree of joint stiffness rated.

Eversion (Fig. 4j, page 63)

The feet were alternately everted and the degree of limitation was rated.

Upper Limbs

The upper limb joints were tested and rated in the same way as the lower limbs.

FIGURES 4a - 4d

TESTS OF JOINT STIFFNESS

To test joint stiffness in any joint, the limb was moved passively through the maximum range of movement of the joint for that patient and compared with the normal full range (the arrow indicates the direction of the passive movement of the limb).

Fig. 4a. Hip flexion

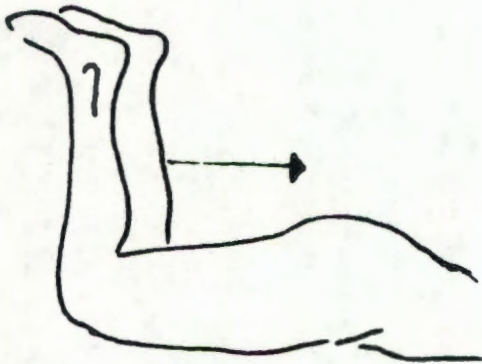


Fig. 4b. Hip extension

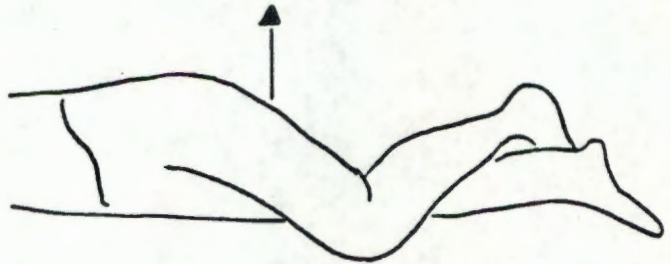
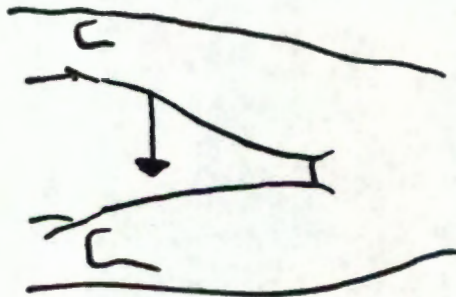


Fig. 4c. Hip abduction



Fig. 4d. Hip adduction



FIGURES 4e - 4h

TESTS OF JOINT STIFFNESS

Fig. 4e. Knee flexion

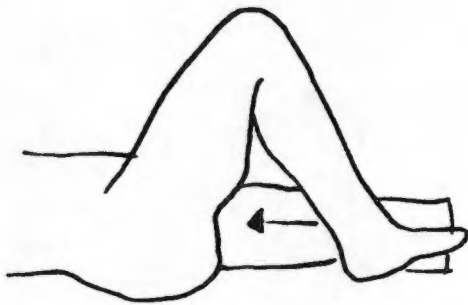


Fig. 4f. Knee extension

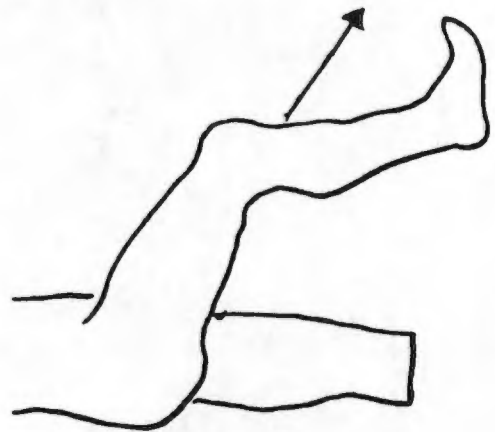
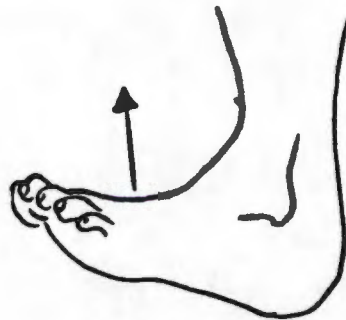


Fig. 4g. Plantar flexion



Fig. 4h. Dorsiflexion



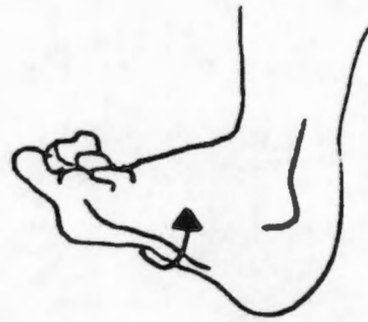
**FIGURES 4i - 4j**

**TESTS OF JOINT STIFFNESS**

**Fig. 4i. Inversion**



**Fig. 4j. Eversion**



## ASSESSMENT OF SELECTED FUNCTIONAL MOVEMENT

The degree of abnormal movement was rated on a 5 point scale. This scale was developed according to the degree of progression of abnormal functional movement patterns in the spastic patient.

### Rolling

From a supine position on the mat, the patient was asked to roll to the left and to the right. The quality was rated on a 5 point scale.

#### Normal rolling (Fig. 5a, page 66)

As the normal baby develops and the righting and equilibrium reactions become refined, the ability to roll from supine to prone and prone to supine develops. This ability is developed by about 6 months of age. In rolling, there is some rotation between the shoulders and the pelvis and separation between the legs.

#### Abnormal rolling (Fig. 5b, page 66)

There are many variations of abnormal rolling. In attempting to roll, the spastic cerebral palsied patient uses abnormal compensatory patterns, as spasticity blocks his ability to achieve this in the normal way. The most common way to roll is to "use" the abnormal predominant extensor tone and push back and roll over en bloc. The spastic patient often achieves rolling without rotation.

## Rating Scale for Rolling

### 1 = NORMAL

The patient can roll with good separation between the shoulders and the pelvis. In moving from prone to supine, the pelvis leads and from supine to prone, the shoulders lead. There is good separation between the legs.

### 2 = MILD LIMITATION

The patient can roll with some separation between the shoulders and the pelvis. There is some separation of the legs and some pelvic mobility.

### 3 = MODERATE LIMITATION

The patient rolls without separation between the shoulders and the pelvis but with dissociation between the legs or with some degree of separation between the shoulders and the pelvis but without dissociation between the legs.

### 4 = MODERATELY SEVERE LIMITATION

The patient rolls en bloc without separation between the legs and without separation between the shoulders and the pelvis.

### 5 = SEVERE LIMITATION

The patient cannot roll independently or may roll only with facilitation by the therapist.

**FIGURES 5a - 5b**

**Fig. 5a. Normal Rolling**



**Fig. 5b. Abnormal Rolling**



### Long Sitting

This is sitting on a mat with the hips flexed and knees extended, and the back straight. The patient was asked to assume this position or was placed in this position. The quality of long sitting is an excellent indicator of improved function following surgery.

#### Normal Long Sitting (Fig. 6a, page 69)

In order to long sit on the floor with the legs extended, the patient must be able to break up his pattern i.e. sit with his hips flexed and the knees extended, maintaining a straight back. He should be able to free his hands for reaching. The normal baby should achieve this ability by about 11 months of age.

#### Abnormal Long Sitting (Fig. 6b, page 69)

The patient is unable to flex the hips and to extend the knees i.e. to break up a total pattern. To compensate for this inability and to maintain this position, the back rounds and the patient fixes his body position with hip adduction and internal rotation, knee flexion and plantar flexion of the feet.

## Rating Scale For Long Sitting

### 1 = NORMAL

Patient can long sit independently. No rounding of the back. Good hip flexion and knee extension. He does not require his arms to "hang on".

### 2 = MILD LIMITATION

The patient can long sit and uses arm support but can let go momentarily. He long sits with some rounding of the back.

### 3 = MODERATE LIMITATION

The patient long sits with some rounding of the back, poor hip flexion and leg extension and some internal rotation of the legs. He can support himself on the side.

### 4 = MODERATELY SEVERE LIMITATION

The patient long sits but uses forward arm support. There is a lot of back rounding, poor hip flexion and knee extension and marked internal rotation of the hips.

### 5 = SEVERE LIMITATION

The patient can long sit only with assistance from the therapist.

**FIGURE 6a - 6b**

**Fig. 6a. Normal Long Sitting**



**Fig. 6b. Abnormal Long Sitting**



### Side Sitting

The patient was asked to assume side sitting independently and to free his arms from the supporting surface. If he was unable to do so, he was placed in this position. The ability to assume this position was a good indicator of improved function.

#### Normal Side Sitting (Fig. 7a, page 72)

As the normal baby learns to sit up from the prone position, his righting reactions become more refined and he is better able to shift his weight and elongate the weight bearing side. He is able to free his hands in this position. The normal baby achieves this ability by about 9 months of age.

#### Abnormal Side Sitting (Fig. 7b, page 72)

In the presence of spasticity, the patient still attempts to achieve side sitting although he is less able to transfer his weight and elongate the weight bearing side. However, in the absence of well developed righting reactions, a variety of compensations are possible to enable him to free his arms in this position.

## Rating Scale For Side Sitting

### 1 = NORMAL

The patient can assume side sitting and sits adequately i.e. protraction of the weight bearing hip. Lateral weight shift and elongation of the weight bearing side facilitates lateral righting of the trunk on that side. He is able to free his arms from the support surface.

### 2 = MILD LIMITATION

The patient can assume side sitting with inadequate weight shift onto the weight bearing side (ie. he sits on both buttocks) due to inability to protract the weight bearing side. He lacks mobility at the pelvis. He can maintain this position. He can free his arms.

### 3 = MODERATE LIMITATION

The patient can assume the position as in 2 above but is unstable and cannot maintain it. He may or may not be able to free his arms.

### 4 = MODERATELY SEVERE LIMITATION

The patient cannot assume the side sitting position but can maintain it if the therapist provides compression through the shoulder and elbow of the arm on the weight bearing side and assists with weight transference onto the weight bearing side.

### 5 = SEVERE LIMITATION

The patient can be placed in a side sitting position but needs a lot of external support and he cannot maintain this position.

**FIGURE 7a - 7b**

**Fig. 7a. Normal Side Sitting**



**Fig. 7b. Abnormal Side Sitting**



### Prone Kneeling (quadruped)

The patient was asked to assume a quadruped position.

#### Normal (Fig. 8a, page 75)

The normal baby learns to assume prone kneeling with good weight bearing on the arms, a well aligned trunk and the lower extremities in a position with the hips at 90 degrees to the trunk and knees at an angle of 90 degrees to the thighs. This ability is developed by about 8 months of age.

#### Abnormal (Fig. 8b, page 75)

In the patient with spasticity, this position will vary, depending on the degree of spasticity. It is difficult to assess small and subtle differences as this position is influenced mainly by the quality of the patient's arm support, the trunk control and the degree of internal rotation of the hips.

## Rating Scale For Prone Kneeling

### 1 = NORMAL

The quadruped position is assumed and maintained without a lordosis and with good arm support. The thighs are held at an angle of 90 degrees to the trunk while the knees are kept at an angle of 90 degrees to the thighs.

### 2 = MILD LIMITATION

The patient can assume and maintain a quadruped position but he has a lordosis with some lower leg abduction. He stabilizes his pelvis with active hip flexion. Arm support is good.

### 3 = MODERATE LIMITATION

The patient can assume the quadruped position. He maintains hip flexion, adduction and internal rotation and knee flexion. The lower legs are slightly abducted to stabilize the base. The feet are in a mass dorsiflexion pattern or rest on the medial borders. Arm support is fair or good.

### 4 = MODERATELY SEVERE LIMITATION

The patient can assume the quadruped position only with difficulty. The hips remain semi-extended and internally rotated with adduction or abduction. Mass dorsiflexion of feet may be present. Arm support is poor.

### 5 = SEVERE LIMITATION

The patient cannot assume this position and needs assistance to maintain it. The arms collapse as arm support may be poor.

**FIGURE 8a - 8b**

**Fig. 8a. Normal Prone Kneeling**



**Fig. 8b. Abnormal Prone Kneeling**



### Kneel Standing

The patient was asked to rise up against gravity in an upright kneeling position. This position was a good indicator of improved function as a good balance between the hip flexors and hip extensors is required in order to assume and maintain this upright kneeling position.

#### Normal (Fig. 9a, page 78)

The patient with normal movement patterns can rise against gravity and can break up his movement patterns in order to achieve hip extension with knee flexion and plantar flexion of the feet. He has good pelvic control ie. a good balance between his abdominal muscles, hip extensors, abductors and adductors which is necessary to maintain this position. This ability is developed in the normal baby by about 10 months of age.

#### Abnormal (Fig. 9b, page 79)

The spastic patient is often hampered by his lack of development of good pelvic control due to the imbalance caused by the spastic muscles. An anterior pelvic tilt and compensatory lumbar lordosis results when the patient attempts to assume this position. The degree of difficulty experienced depends on the degree of increased muscle tone.

## Rating Scale For Kneel Standing

### 1 = NORMAL

The patient can assume and maintain the kneel standing position and maintain a neutral pelvic tilt.

### 2 = MILD LIMITATION

The patient can rise against gravity and may or may not have difficulty maintaining this position. He maintains an anterior pelvic tilt, a lordosis and hip flexion with lower leg abduction. He has great difficulty shifting weight. He can perform an activity in this position.

### 3 = MODERATE LIMITATION

As in 2 above but the hips are adducted and internally rotated and there is lower leg abduction. He can maintain this position for a few seconds with little support. He is unable to shift weight and has difficulty freeing his arms.

### 4 = MODERATELY SEVERE LIMITATION

The patient assumes or is assisted to assume kneel standing. He has an anterior pelvic tilt, a lordosis, hip adduction, internal rotation and flexion. He can maintain this position but is unable to free his hands.

### 5 = SEVERE LIMITATION

He cannot assume this position but needs to be placed in the position and given full external support.

**FIGURE 9a**

**Normal Kneel Standing**



**FIGURE 9b**

**Abnormal Kneel Standing**



### Half Kneeling

The patient was asked to bear weight on alternate knees while bringing the opposite leg forward. This was an excellent indicator of improved functional movement, as the patient required a good balance between hip flexors and extensors as well as an ability to dissociate his legs in order to assume and maintain this position.

### Normal (Fig. 10a, page 82)

To assume and maintain this position, the patient must have good trunk and pelvic control as well as the ability to dissociate his legs ie. the ability to "break up movement patterns". This requires refined control and well integrated balance and equilibrium reactions. This ability is achieved in the normal baby by about 11 months of age.

### Abnormal (Fig. 10b, page 83)

Due to increased muscle tone, the patient is unable to separate the legs and shift his weight in order to maintain his balance. Righting and equilibrium reactions are often poorly developed.

## Rating Scale For Half Kneeling

### 1 = NORMAL

The patient can assume and maintain a good half kneeling position. He is able to free his arms for an activity.

### 2 = MILD LIMITATION

The patient can assume and maintain the half kneeling position but internally rotates the forward leg and flexes the hip of the weight bearing leg. Legs separate easily and the patient can free his arms.

### 3 = MODERATE LIMITATION

The patient can assume or is helped to assume half kneeling. The legs separate but the patient needs support ie. protraction of the pelvis on the weight bearing side and traction of the forward leg. He cannot free his arms or can do so for only a short while.

### 4 = MODERATELY SEVERE LIMITATION

The patient can assume this position but can separate his legs with difficulty and he cannot free his hands. If he cannot assume this position and is placed in it, he can only maintain it with difficulty.

### 5 = SEVERE LIMITATION

The patient cannot assume this position. There is great difficulty in separating the legs when he is placed in this position. The therapist must maintain full external support and the child collapses after a few seconds.

FIGURE 10a

Normal Half Kneeling



FIGURE 10b

Abnormal Half Kneeling



## Crawling

Clinical observations were supported by analysis of crawling using a digital camera system.

There are numerous individual variations in crawling.

### Normal (Fig. 11a, page 86)

Normal crawling is possible when the patient is able to bear weight evenly through the arms and the knees and maintain a good alignment of the head and trunk in this position. Reciprocal movement occurs when the patient is able to transfer weight and elongate the weight bearing side while propelling himself forward. Crawling is developed by about 9 months of age in the normal baby.

### Abnormal (Fig. 11b, page 86)

Spasticity prevents the patient from developing the ability to bear weight evenly in the crawling position and various attempts at forward propulsion result. The patient is unable to dissociate his legs adequately, and in attempting to propel himself forward, he either moves in a mass flexion pattern ("bunny hopping"), or manages some degree of dissociation. He is however, not able to transfer weight adequately. In order to gain momentum he often shoots the legs into extension.

## Rating Scale For Crawling

### 1 = NORMAL

The patient crawls reciprocally with well integrated righting and equilibrium reactions. His legs separate with ease. The weight is well distributed and arm support is good.

### 2 = MILD LIMITATION

The patient crawls with some reciprocation but uses side flexion instead of elongation of the weight bearing side. Arm support is good.

### 3 = MODERATE LIMITATION

As in 2 above but the legs shoot into extension on the non-weight bearing side or he crawls with a mass flexion pattern with legs widely abducted at the hips and with flexion at the knees. Arm support may vary.

### 4 = MODERATELY SEVERE LIMITATION

The patient bunny-hops, moving both lower extremities together. Arm support may vary.

### 5 = SEVERE LIMITATION

The patient is unable to assume the four point kneeling position. He "mermaid" crawls by pulling his body forward with his arms while the legs remain in extension. There is very poor dissociation of the legs. Arm support is usually poor.

FIGURE 11a - 11b

Fig. 11a. Normal Crawling



Fig. 11b. Abnormal Crawling



### Standing

There are a number of individual variations in assuming and maintaining this position.

#### Normal (Fig. 12a, page 89)

The child is able to rise against gravity. The head, neck, trunk, pelvis and feet are well aligned and there is no asymmetry. There is a neutral pelvic tilt and the weight is evenly distributed. The normal baby achieves this ability by about 11 months of age.

#### Abnormal (Fig. 12b, page 90)

There are many variations especially when the patient is asymmetrically affected. The scale broadly attempts to rate the degree of disability.

## Rating Scale for Standing

### 1 = NORMAL

The patient is able to assume and maintain a good standing posture.

### 2 = MILD LIMITATION

The patient can assume the standing position but stands with an anterior pelvic tilt. He can shift his weight using side flexion. He stands independently. Feet may or may not be in a valgus position.

### 3 = MODERATE LIMITATION

As in 2 above but he needs to hold on due to lack of stability. The patient has an anterior pelvic tilt, some internal rotation at the hips with slight flexion and some knee flexion. The feet may be in a valgus position.

### 4 = MODERATELY SEVERE LIMITATION

Contractures and deformities are developing at the hips, knees and ankles. The patient can only assume standing for a few seconds. Feet may be in a valgus position.

### 5 = SEVERE LIMITATION

As in 4 above. The patient cannot assume standing. The patient requires full external support to stand. Hips, knees and ankles may have fixed deformities. Feet may be in a valgus position.

**FIGURE 12a**

**Normal Standing**



FIGURE 12b

Abnormal Standing



## Walking

### Normal (Fig. 13a, page 94)

Walking requires the ability to maintain the upright posture against gravity and the ability to control and coordinate many muscle actions. Normal walking usually occurs by about the age of 12 months.

The normal walking cycle describes the events that occur during ambulation. It begins and ends with heel strike of the same limb. It is divided into two phases; the first is the stance phase when the foot is on the ground and the second is the swing phase when the foot is swinging off the ground.

### Abnormal (Fig. 13b, page 95)

In spastic cerebral palsy there are many alterations in the walking cycle, depending on the degree of cerebral involvement.

The initial contact may not be a heel strike but rather a toe strike. An equinus gait often results due to spasticity of the plantar flexors. This may result in hyperextension at the knee joint or compensatory flexion at the hip and knee joints. This lack of dorsiflexion results in a decreased step length.

Spasticity in the knee flexors often results in compensatory flexion at the hip and dorsiflexion at the ankle, depending on the severity of the spasticity. This also results in a shortened step length.

When a patient has spastic or contracted hip flexors, he may need to compensate for it by increasing his lumbar lordosis or by flexion of the knees. This combination results in a shortened step length.

"Scissor gait" in which one leg over adducts so as to cross over the other as the patient attempts to walk is common.

## Rating Scale For Walking

### 1 = NORMAL

The patient walks reciprocally; Starting from a stable standing posture the body is propelled forward in a smoothly coordinated way. The gait cycle consists of a stance and a swing phase. The stance is a 5 phased activity involving contact, loading response, mid-stance, terminal stance and pre-swing. The swing is divided into initial, mid and terminal thirds (Hoffer & Perry, 1983).

### 2 = MILD LIMITATION

The patient walks with an anterior pelvic tilt but has the necessary break-up of patterns.

### 3 = MODERATE LIMITATION

The patient can walk, has the necessary break-up of patterns but lacks rotation and has poor weight-shift. He uses upper trunk compensations to assist with gait.

### 4 = MODERATELY SEVERE LIMITATION

The patient has poor break-up of patterns and cannot take a few steps independently or has very poor balance.

### 5 = SEVERE LIMITATION

No reciprocal gait, no break-up of patterns and no weight-shift. The patient needs full external support.

FIGURE 13a

Normal Walking



FIGURE 13b

Abnormal Walking



## COMPUTER ANALYSIS OF CRAWLING AND GAIT

Where possible, the crawling and walking patterns were assessed in conjunction with Dr. C.L. Vaughan using a Microneye digital camera. Objective measurements of crawling and walking before and after surgery could therefore be compared. The measurements were done in the sagittal plane from left to right and right to left. Patients who were unable to walk independently were allowed to walk with the aid of walkers or walking frames.

### Objectives

- a. to investigate the change in velocity.
- b. to investigate the change in cadence (the number of steps per second or the number of steps per minutes i.e. step frequency).
- c. to investigate the change in cycle time (i.e. the time taken to complete the cycle which is from one heel strike to the next heel-strike).
- d. to compare the range of movement before and after surgery at the hip and knee joints through the gait cycle. Stick figures could be plotted thus providing an easy visual picture of joint positions.
- e. to plot angle-angle diagrams for the thigh and knee angles, to examine the interaction of these two, and left/right symmetry.

## System Description

The following system was used to evaluate gait and crawling patterns before and after surgery. The study was restricted to two dimensions and specifically to the sagittal plane.

## Components

### The Digital Camera (Fig. 14, page 98)

The camera used was the Microneye Camera, made by:  
Micron Technology, Division Systems Group,  
2805 East Columbia Rd,  
Boise, Idaho 83706 U.S.A.

This camera is similar to a standard camera except that instead of photographic film, it uses an electronic integrated circuit (IC) which is sensitive to changes in light. This IC is connected directly to a computer (Vaughan, Smith & du Toit, 1987). Its operative part is a dynamic random access memory (RAM) chip. The markers on the patient's hip, knee and ankle reflect light directly back to the source.

FIGURE 14

Digital Camera and Microcomputer



The Markers (Fig. 15, page 100)

These are table tennis balls covered with retro-reflective material. The special reflective properties of the markers allow for filming to take place in normal ambient lighting conditions, provided that direct sunlight is shut out.

For collecting data for crawling and walking in the sagittal plane, these markers are placed on the greater trochanter of the femur (this is palpated and located by internally and externally rotating the non weight-bearing femur), the lateral epicondyle of the femur and the lateral malleolus. They adhere to the skin by means of double-sided tape.

FIGURE 15

The retroreflective markers  
on a patient's hip, knee and ankle



The Microcomputer (Fig. 14, page 98)

This takes the information from the digital camera and then calculates the position in space (in the saggital plane) of the three markers on the patient's hip, knee and ankle joints. The computer is then used to display and calculate the results in a number of different ways. The data was represented by plots of the two dimensional positions of the hip, knee and ankle markers as a function of the distance crawled or walked while in the viewing field of the camera. From this raw data (Fig. 16, page 103), stick figures were constructed by merely connecting the coordinates of the hip, knee and ankle markers which appeared in each frame (Fig. 17, page 104). The same was done for crawling (Fig. 18, page 105). This was done by means of a special routine within the display program. The stick figure diagrams were used to determine the positions of consecutive heel strikes of the ipsilateral leg. The number of frames in between heel strikes was used to calculate the amount of time which had lapsed. Using the distance and temporal information provided by the stick figures, the stride length and speed of walking and crawling could be calculated.

It was also possible to extract angular displacement from the raw data using some simple trigonometry in the display program. Flexion at the hip was so defined as to result in a positive thigh angle while extension caused the thigh angle to become negative (Fig. 19, page 106). These two angles were measured for each frame and then plotted against one another following the technique of Grieve (1968). The plots are called thigh-knee angle-angle diagrams, with the thigh angle as the x coordinate and the knee angle as the y coordinate. One cylindrical loop of such a diagram represented exactly one stride. Figures 20 and 21 (pages 107 and 108) are examples of such angle-angle diagrams for walking and crawling respectively.

From this data it was possible to calculate cadence (steps/minute), stride length (metre), and average speed (metre/second). In addition, the range (in degrees) at the thigh and knee, as well as the mid-range points (half the sum of the minimum and maximum values) for the thigh and knee were derived.

FIGURE 16

Fig. 16. Raw data generated by the digital camera. From the top the dots represent the loci of the hip, knee and ankle markers.

DATA

BRY•RWB  
11 APR 85 PRE - OP  
RIGHT WALK  
X RANGE = 2000 MM Y RANGE = 1000 MM

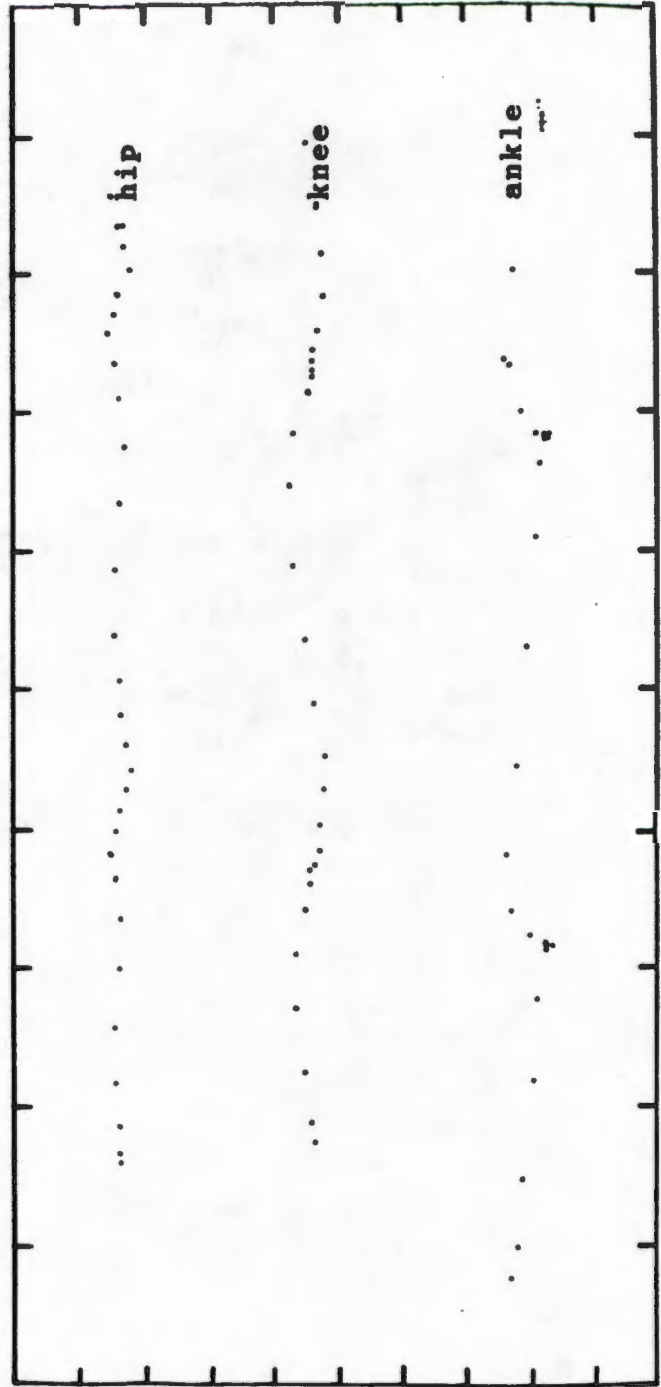


FIGURE 17

Fig. 17. Raw data converted to stick figures with consecutive heel strikes. The data shows stick figures for walking for one patient before and after surgery.

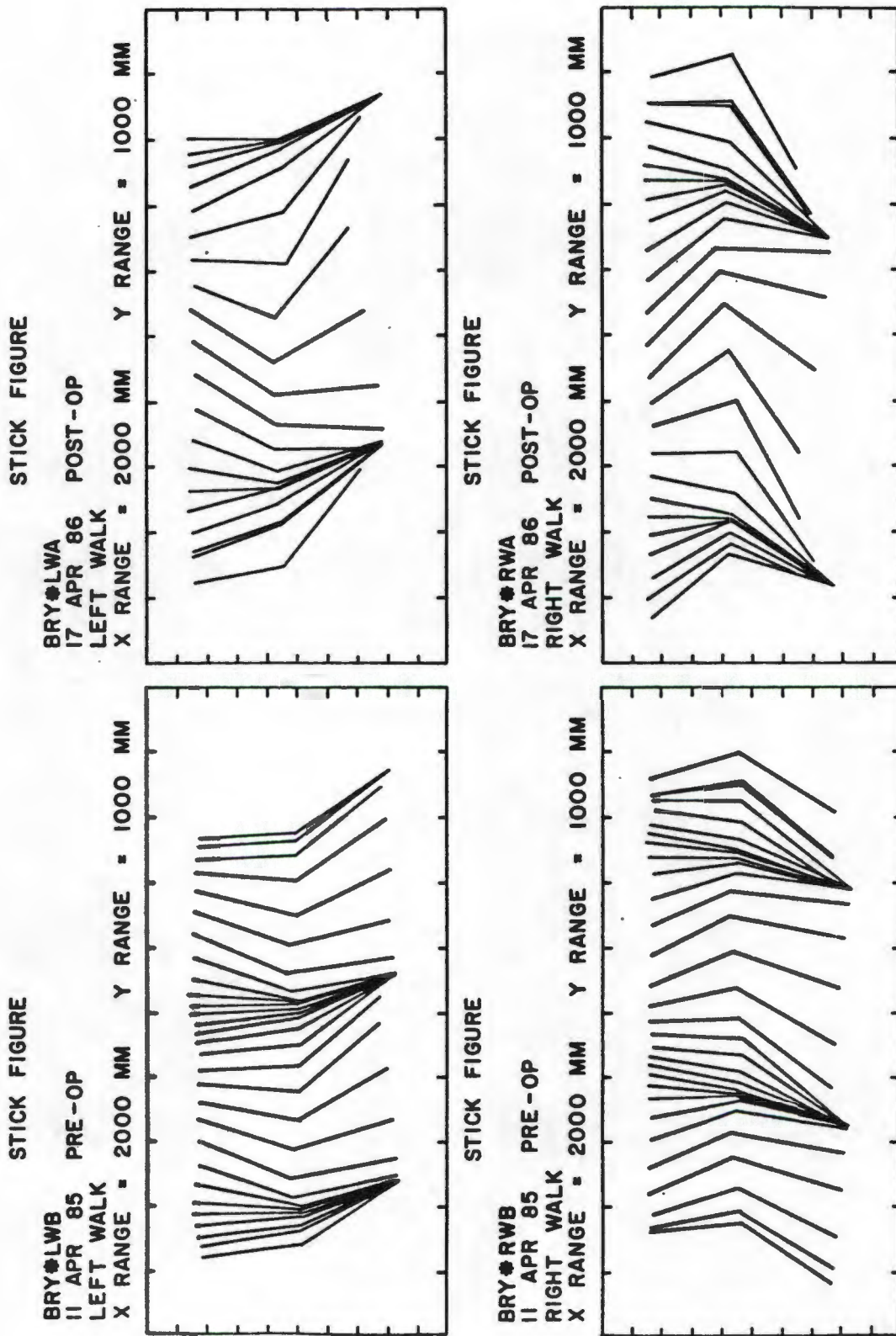
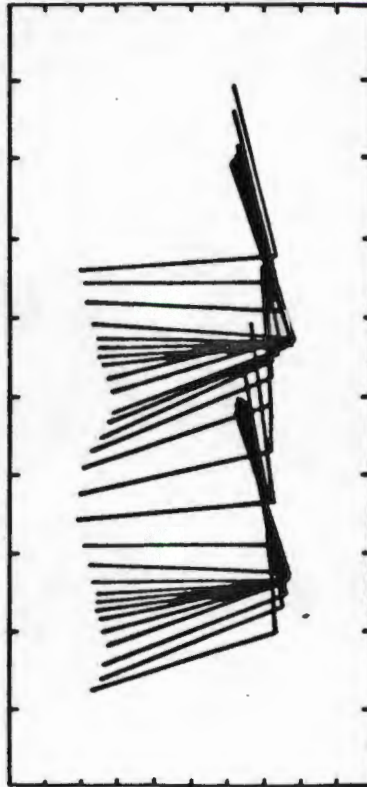


Fig. 18. Raw data converted to stick figures for crawling for one patient before and after surgery.

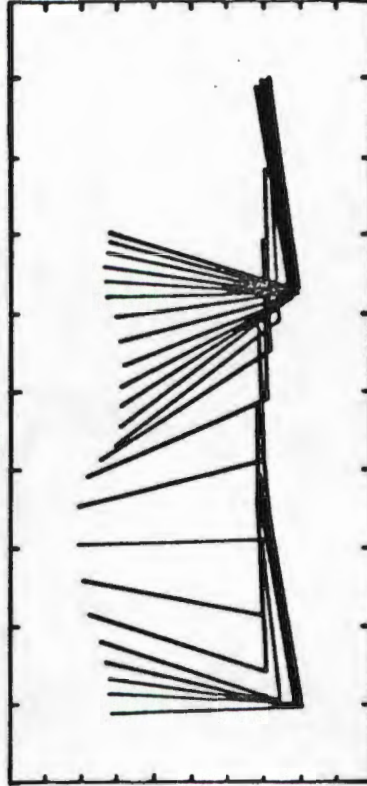
STICK FIGURE

BRY\*LCB  
11 APR 85 PRE-OP  
LEFT CRAWL  
X RANGE = 1500 MM Y RANGE = 600 MM



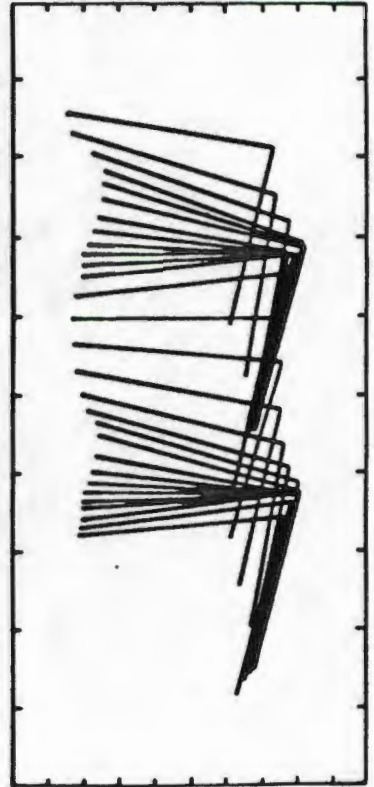
STICK FIGURE

BRY\*LCA  
17 APR 86 POST-OP  
LEFT CRAWL  
X RANGE = 1500 MM Y RANGE = 600 MM



STICK FIGURE

BRY\*RCB  
11 APR 85 PRE-OP  
RIGHT CRAWL  
X RANGE = 1500 MM Y RANGE = 600 MM



STICK FIGURE

BRY\*RCA  
17 APR 86 POST-OP  
RIGHT CRAWL  
X RANGE = 1500 MM Y RANGE = 600 MM

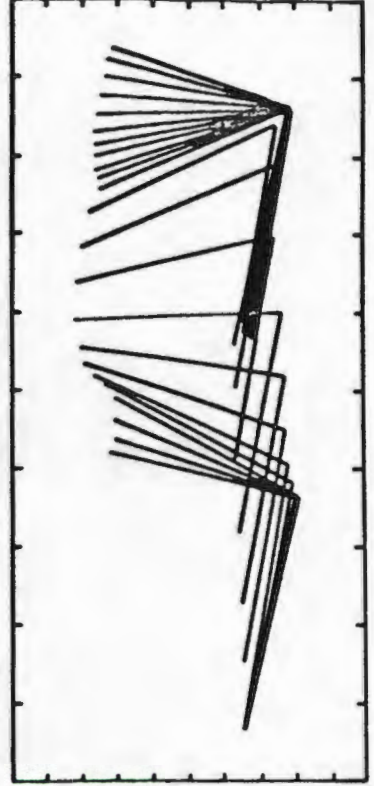


FIGURE 18

FIGURE 19

A sagittal view showing marker position and definition of thigh and knee angles

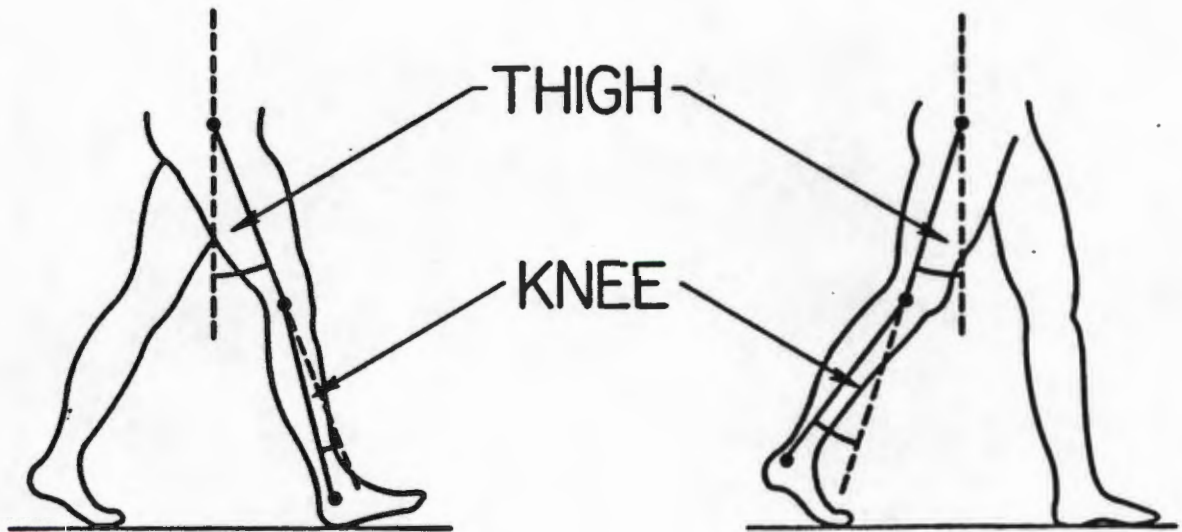


FIGURE 20

Fig. 20. Thigh-knee angle-angle diagrams of walking for one patient before after surgery.

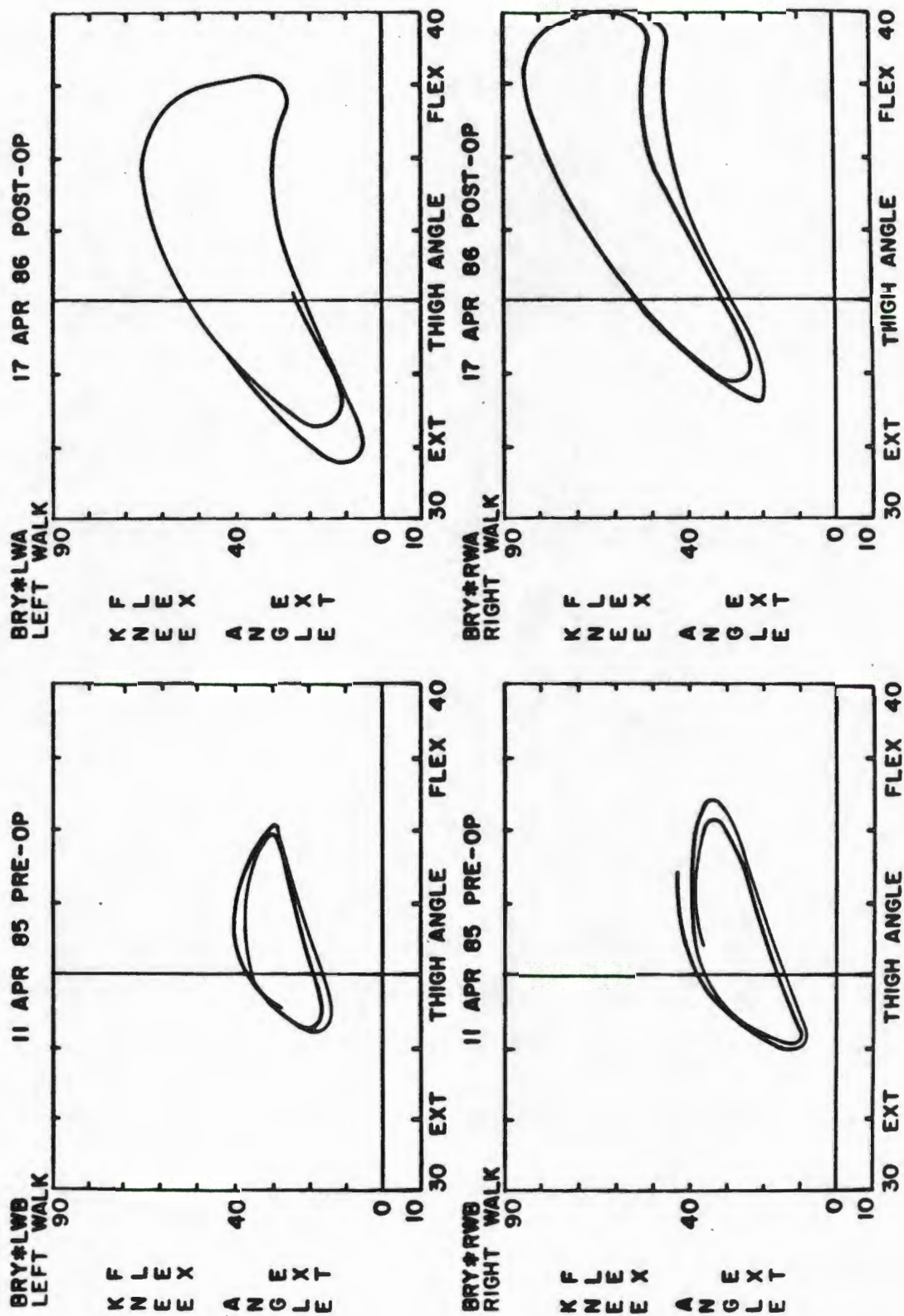
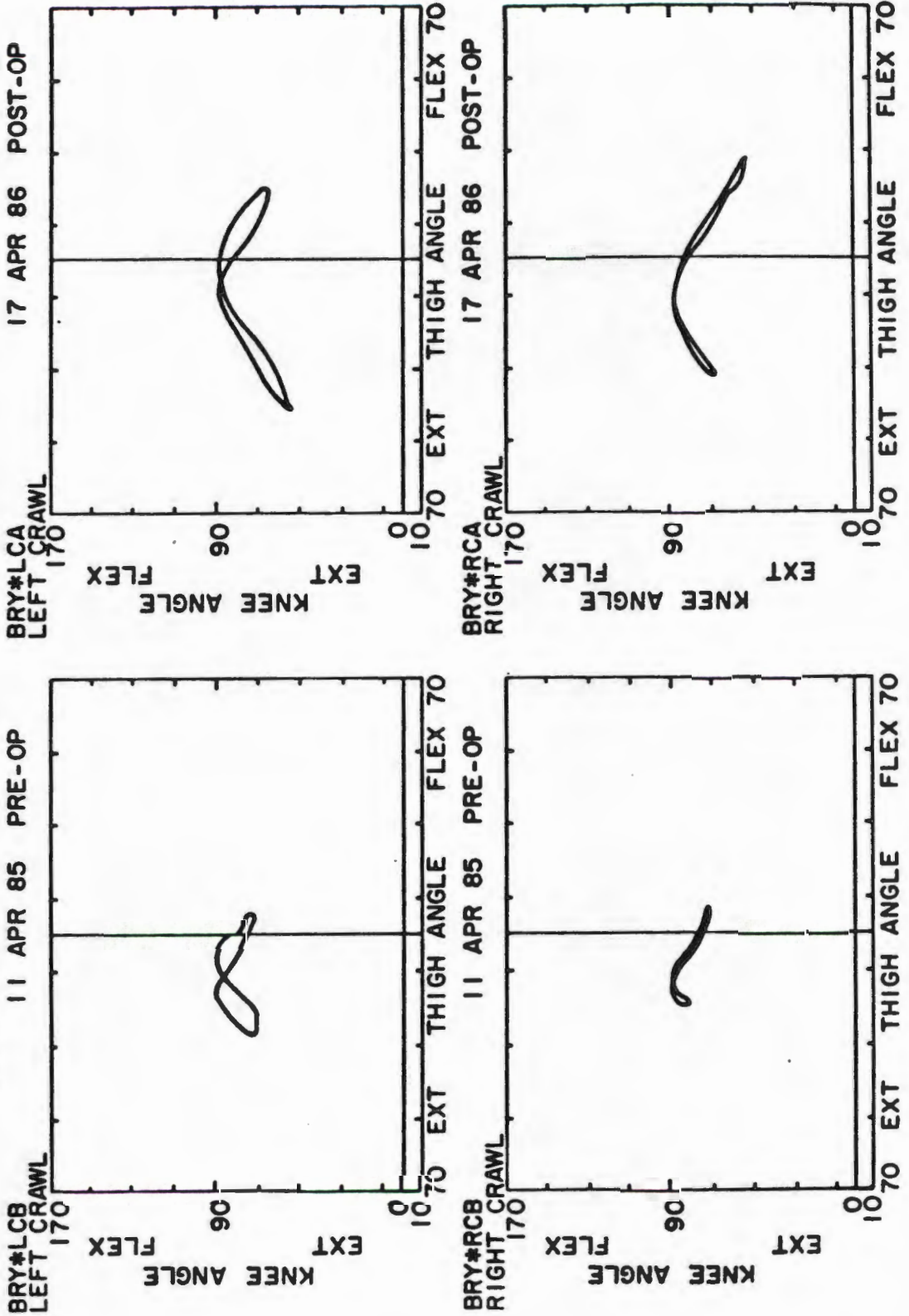


FIGURE 21

Fig. 21. Thigh-knee angle-angle diagrams of crawling for one patient before and after surgery.



## Procedure

The digital camera was positioned between 150 watt light bulbs which provided the necessary illumination of the markers. The camera and lights were mounted on a custom-built trolley and directly linked to an Apple compatible microcomputer, also on the trolley (Fig. 14, page 98).

During the filming, the data on the 2D positions of the retroreflective markers were sent from the camera to the microcomputer. Special purpose computer programs, written in assembler language and BASIC, were developed for an Apple II microcomputer to capture and process the raw two dimensional data. This data could be displayed or printed out.

With markers correctly positioned, the patients were asked to crawl and then to walk first in one direction and then, with the markers placed on the contralateral side, in the opposite direction. The system has a sampling rate of 13Hz and between one and a half and four cycles of data were always captured.

## SELECTIVE POSTERIOR LUMBAR RHIZOTOMY PROCEDURE

Following induction of endotracheal general anaesthesia, the patient was placed prone on the operating table with the pelvis and chest supported on bolsters, allowing the anterior abdominal wall to hang freely (Fig. 22, page 113). No long-acting muscle relaxants were used. The incision was marked on the skin of the back from the spine of L1 to the spine of S2. After preparing and draping the skin, a midline lumbosacral incision was made and limited laminectomies performed from the second lumbar vertebra to the first sacral segment. The dura was opened, exposing the cauda equina. With the surgeon standing at the patient's left side, the second lumbar spinal nerve on the right was identified (Fig. 23, page 114) at its point of exit from the dura and the posterior root separated from the anterior root (Fig. 24, page 115).

Using two insulated angled probes which were attached via electrical leads to the electromyograph, the posterior root was stimulated. A single stimulus was applied, using increasing voltages from 5 to 50 volts (Peacock, Arens & Berman, 1988). The response was noted on the electromyograph from electrodes placed on the appropriate muscles (Fig. 25, page 116). A response was considered normal if the action potential was below 50, if the muscle contraction was brief, was produced by a stimulus of 20 volts and remained confined

to the muscle groups innervated by that spinal segment (Fig. 26a, page 117). The response was considered abnormal if the muscular contraction was sustained or diffused to other muscle groups (Fig. 26b, page 118).

A tetanic stimulus was then used giving between 20 and 50 stimuli per second. The normal response to this was a brief muscle contraction confined to the relevant muscle groups with a small action potential, whereas an abnormal response was indicated by a sustained contraction with large action potentials with or without diffusion of contraction to other muscle groups.

If the whole posterior nerve root was associated with normal responses it was allowed to fall back into its normal position, but if the responses were abnormal, the posterior root was split up into its component rootlets and each posterior rootlet was then stimulated in turn in the same manner as the whole root. If a rootlet was associated with an abnormal response, it was divided with microscissors (Fig. 27, page 119). Those associated with normal responses were left intact. Each root and its component rootlets was dealt with in this way, progressing from L2 through to S1 on the right hand side. The surgeon then moved to the patient's right side and repeated the procedure, stimulating the posterior roots from L2 to S1. It is of great importance to ascertain with great accuracy the levels of nerve roots to

be stimulated so that those roots (S3, S4 and possibly S2) which innervate the bladder are not divided. This can be done with intraoperative radiological assistance. The dura was then closed in a watertight fashion and the muscles and the supraspinous ligament closely approximated before the skin was closed.

FIGURE 22

Patient prone on the operating table



FIGURE 23

A spinal nerve exposed

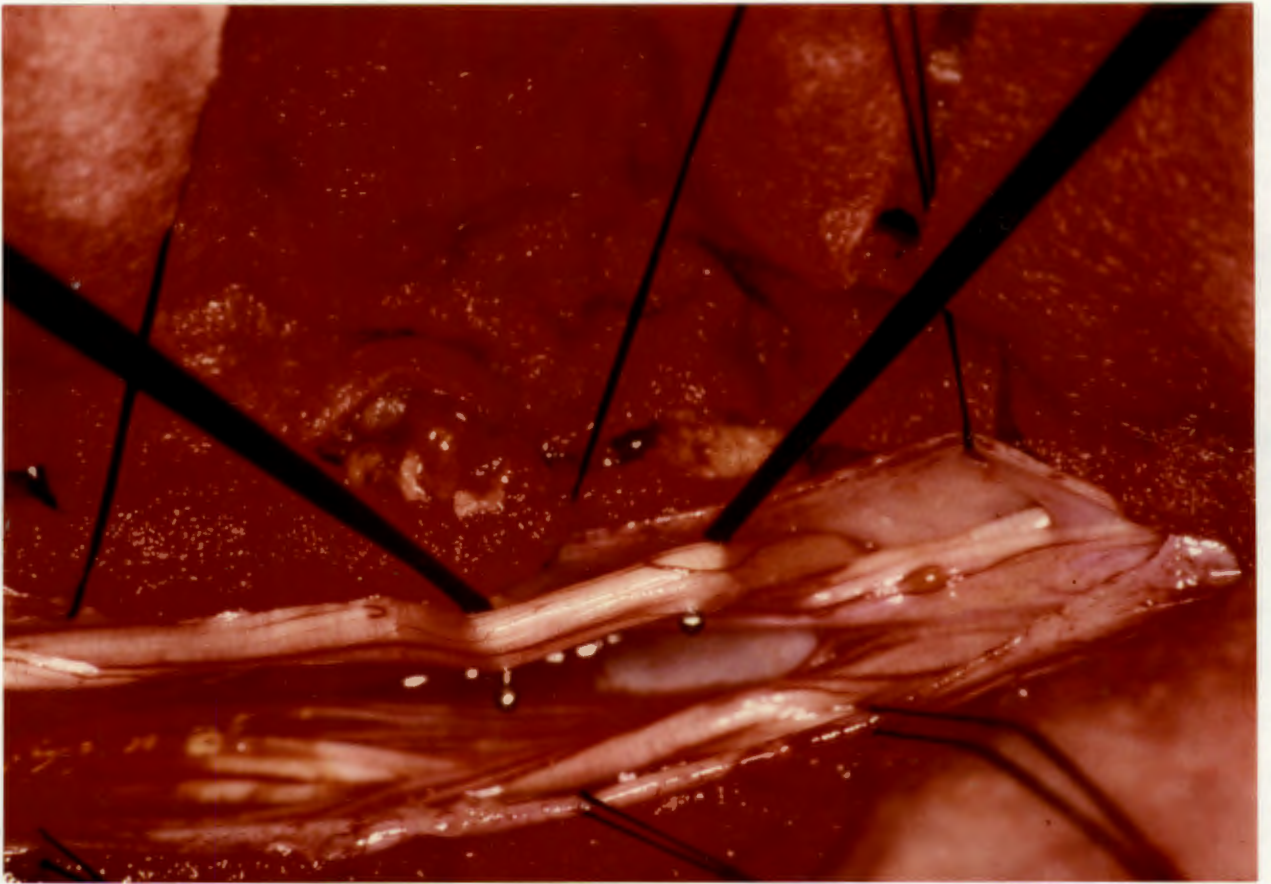


FIGURE 24

A posterior nerve root separated from an anterior root and stimulated with a probe attached by electrical leads to the electromyograph

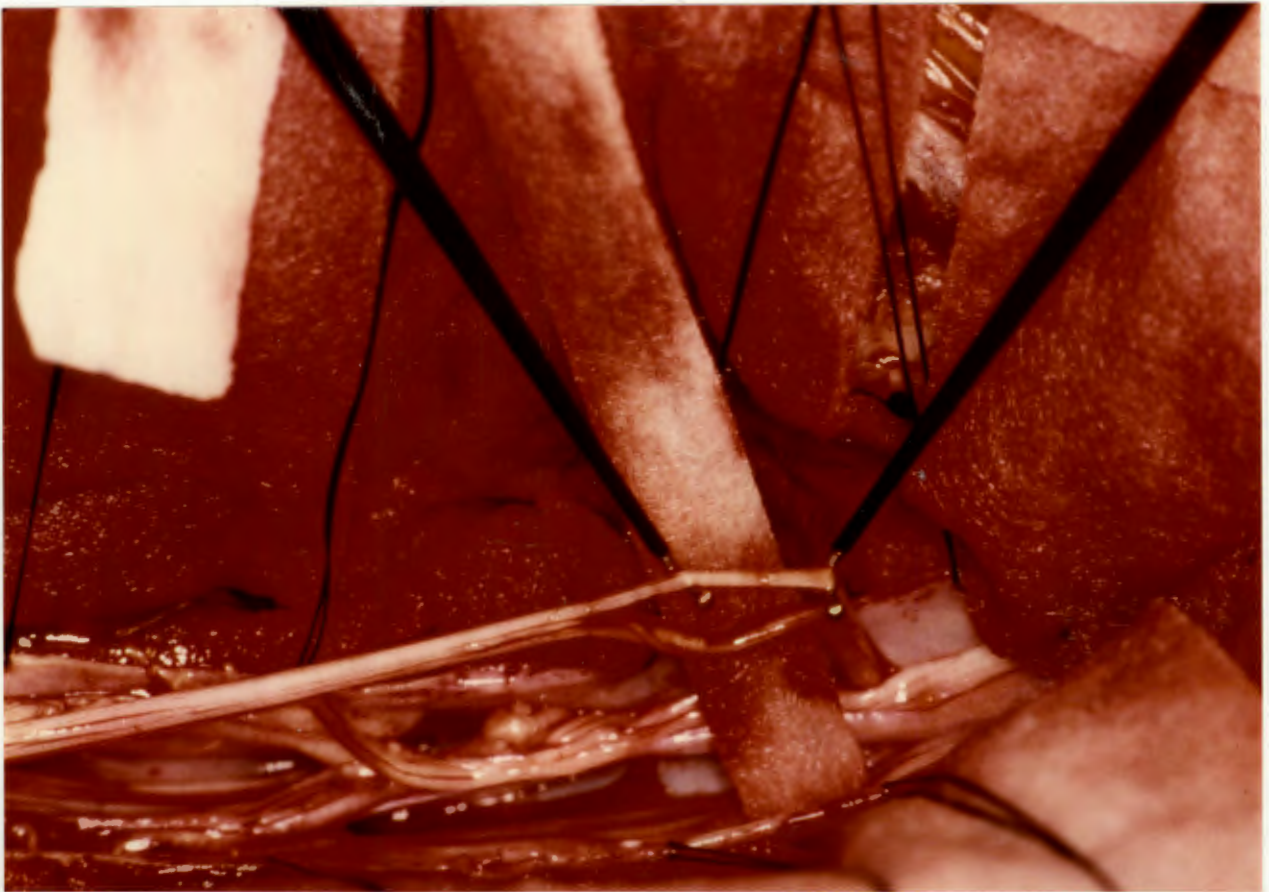


FIGURE 25

Electrodes (attached to the electromyograph) placed on the muscle groups of the lower limbs



FIGURE 26a

A normal response on the electromyograph to electrical stimulation

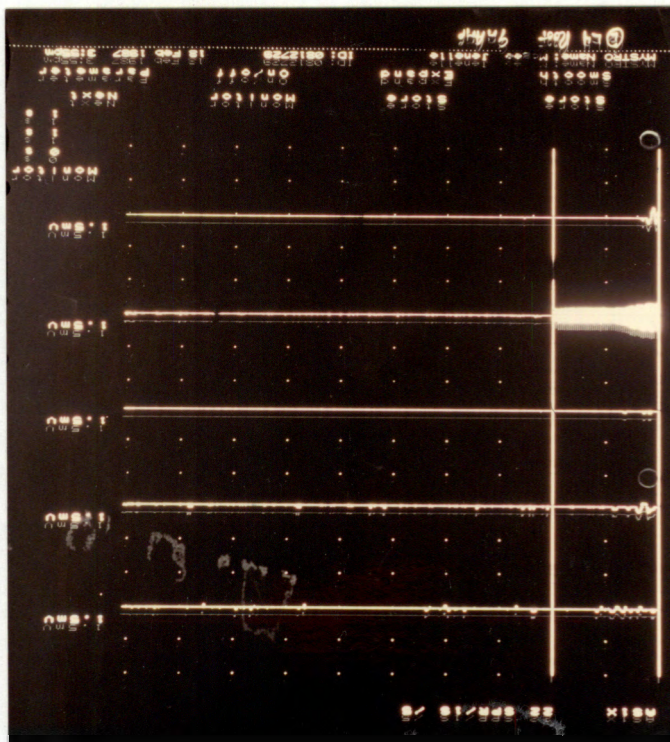


FIGURE 26b

An abnormal response on the electromyograph to electrical stimulation

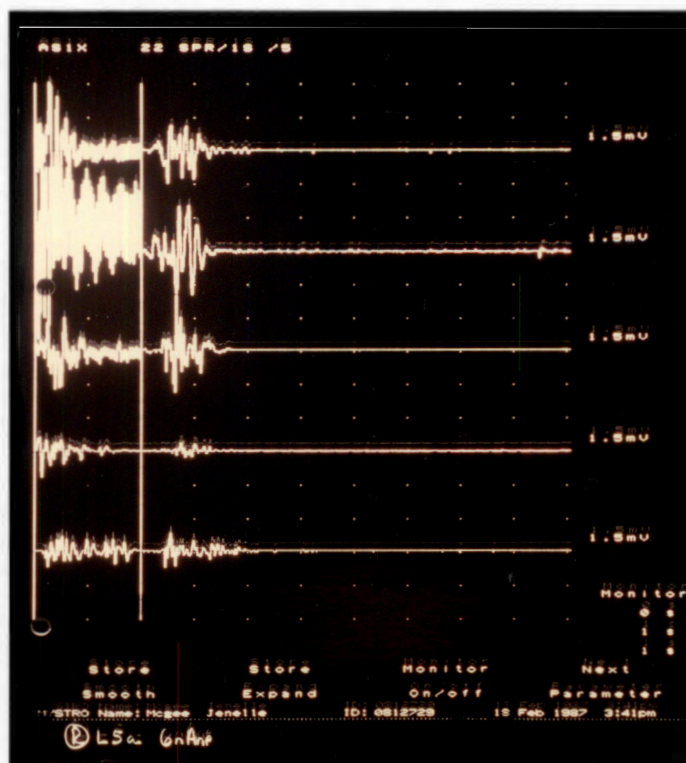


FIGURE 27

A rootlet (which gave an abnormal response to electrical stimulation) has been cut



## METHODS OF PROCESSING DATA

The data for muscle tone, voluntary movement, joint stiffness and functional movement was analysed with the assistance of Mr. R.S. Bridger, of the Department of Biomedical Engineering at the University of Cape Town. The Wilcoxon Signed-Ranks Test (Wilcoxon, 1945) and the Student paired t-test were the statistical tests used to test for the significance of differences between the measured results before and after surgery.

The computer program "Reflex" (Reflex, 1985) was used in an IBM AT compatible computer to produce the graphs based on the patient data.

Special Assembler and Basic programs (for the Apple II microcomputer) to do the analysis of gait and crawling, were developed at the Department of Biomedical Engineering, University of Cape Town.

## CHAPTER 4

### RESULTS

The results of the study reported here confirm the favourable impressions of the effects of Selective Posterior Lumbar Rhizotomy (SPLR) on cerebral palsied patients described by Peacock and Arens (1982). These results also confirm the usefulness of the methodology which was developed specifically for evaluating these patients.

### DEMOGRAPHICS

In this study 29 patients were examined before and after surgery (Tables 8 and 9, pages 174 and 175). There were 18 males and 11 females. Most of the patients were children with a mean age of 9.3 years. Twenty seven patients were 15 years of age or below at the time of surgery, and two were adults, one of 21 years and another of 35 years of age.

The causes of the cerebral palsy were prenatal and perinatal in 26 cases and post-traumatic in 3. Eighteen patients were diagnosed as spastic diplegics, 9 quadriplegics and 2 were spastic with athetosis. The severity of involvement varied. Eleven patients were non-ambulatory, 7 were household ambulators and 11 were independent ambulators. Twelve

patients had had various types of orthopaedic surgery prior to undergoing SPLR.

The Intelligence Quotients (I.Q.) of the patients varied considerably. Nineteen patients were educable and had estimated I.Q. ranging between 90 and 110, 8 patients were probably in the trainable category with estimated I.Q. between 50 and 90 and 2 patients were untrainable with I.Q. below 50.

Results are presented in the following order:

1. Muscle Tone
2. Voluntary Movement
3. Joint Stiffness
4. Functional Movement
5. Interactions

## MUSCLE TONE

### Hypothesis 1

*SPLR does not lead to a reduction in muscle tone of spastic muscle groups of the upper and lower limbs.*

TABLE 1

### MUSCLE TONE BEFORE AND AFTER SURGERY

(Mean Values and Standard Deviations, n=29)

	Preoperative	Postoperative	Difference
Upper Limbs	*2.5 (0.768)	2.4 (0.621)	0.1 (0.303)
Lower Limbs	3.5 (0.514)	2.1 (0.229)	1.4 (0.494)

\*1 = low tone, 2 = NORMAL TONE

3 = mildly increased tone, 4 = moderately increased tone

5 = severely increased tone

The Wilcoxon Signed-Ranks test was used to test the significance of differences in muscle tone before and after surgery.

For the lower limbs the difference between the mean tone scores was statistically significant ( $z=4.69$ ,  $p<0.01$ ). This was also the case for the upper limbs ( $z=2.45$ ,  $p<0.05$ ).

*The results above show that the hypothesis was disproved.*

The most remarkable feature of this study was that, no matter what the preoperative tone was, tone became normal or near normal in the lower limbs in all patients after surgery (Fig. 28, page 125). In the case of the upper limbs, there was a small but significant reduction in spasticity.

There was a positive correlation between tone before surgery and the degree of improvement in muscle tone after surgery (Kendalls tau=0.86, z=6.30, p<0.01) (Fig. 29, page 126).

This result suggests that patients with highest tone preoperatively experience the greatest reduction in tone. Tone changed to within the normal range after surgery no matter what the starting level.

FIGURE 28

Fig. 28. Muscle tone for lower limbs before and after surgery for each patient.

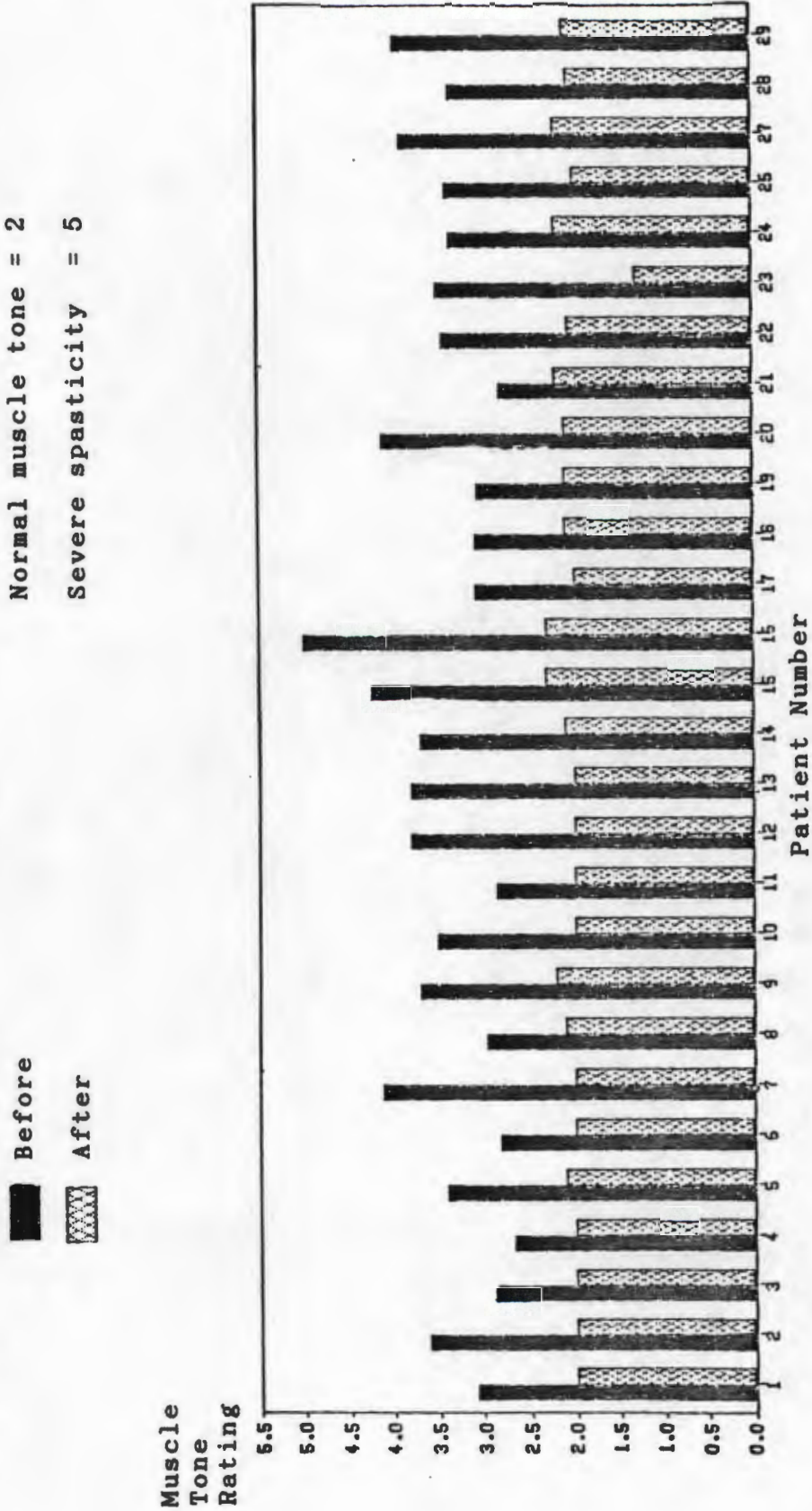
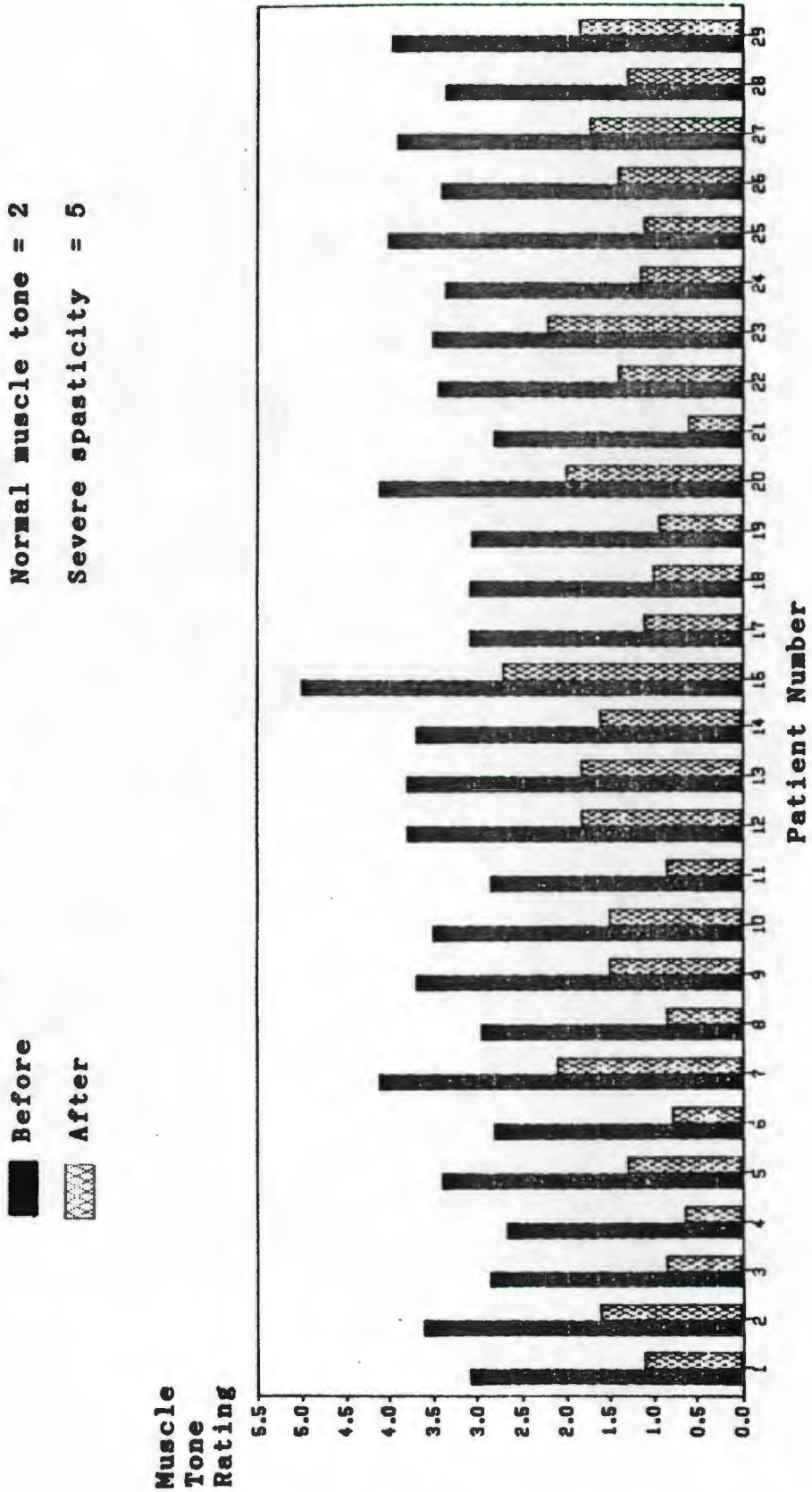


FIGURE 29

Fig. 29. Muscle tone before surgery and improvement in muscle tone after surgery for each patient.



## VOLUNTARY MOVEMENT

### Hypothesis 2

*SPLR does not lead to an improvement in voluntary movement of all spastic muscle groups of the upper and lower limbs.*

Cooperation of the patient is essential if one is to examine voluntary movement and get reproducible results. Since three patients were unable to concentrate well enough to cooperate adequately (two had low I.Q. and one was too young to cooperate), no results for voluntary movement are included for them (patients 4, 13 and 25).

TABLE 2

VOLUNTARY MOVEMENT BEFORE AND AFTER SURGERY  
(mean values and standard deviations, n=26)

---

	Preoperative	Postoperative	Difference
Upper Limbs	*1.80 (1.185)	1.75 (1.117)	0.05 (0.131)
Lower Limbs	3.96 (0.735)	2.54 (0.675)	1.42 (0.734)

---

\*1 = NORMAL, 5 = very poor

The Wilcoxon Signed-Ranks test was used to test for the significance of differences in voluntary movement before and after surgery. There was a significant difference in voluntary movement of individual muscle groups of the lower limbs ( $z=4.3$ ,  $p<0.01$ ). The improvement in the voluntary movement of the upper limbs was small and, although clinically noticeable, was not statistically significant.

*Hypothesis 2 was therefore rejected in the case of the lower limbs but not in the case of the upper limbs.*

## JOINT STIFFNESS

### Hypothesis 3

*SPLR does not lead to an improvement of joint stiffness of upper and lower limb joints.*

TABLE 3

JOINT STIFFNESS BEFORE AND AFTER SURGERY  
(mean values and standard deviations, n=29)

	Preoperative	Postoperative	Difference
Upper Limbs	*1.03 (0.077)	1.02 (0.056)	0.01 (0.037)
Lower Limbs	1.82 (0.41)	1.30 (0.284)	0.52 (0.435)

\*1=NORMAL, 4=apparent fixed contracture

Joint movement was considerably freer after surgery compared to before surgery. The differences in joint stiffness before and after surgery were statistically significant for the joints of the lower limbs but not for the upper limb joints, although some improvement in upper limb joint movement was noted.

On analysing the data about individual joints, it is apparent that the joints that improved most were the hip

followed by the knee and the ankle (see Appendix B, Tables 13A to 13I, pages 179 to 187).

*Hypothesis 3 was therefore rejected in the case of the lower limb joints but not in the case of the upper limb joints.*

#### FUNCTIONAL MOVEMENT

#### Hypothesis 4

*SPLR does not lead to an improvement in functional movement (rolling, sitting, kneeling, crawling, standing and walking).*

TABLE 4

#### FUNCTIONAL MOVEMENT BEFORE AND AFTER SURGERY

(mean values of all functions and standard deviations, n=29)

---

Preoperative	Postoperative	Difference
*3.71 (0.927)	2.79 (1.151)	0.92 (0.491)

---

\*1=NORMAL, 5=grossly abnormal

Mean preoperative and postoperative functional movement scores for all patients and all functions were compared using the Wilcoxon Signed-Ranks test. There was measurable improvement in one or more of the measured functions in 28 of the 29 patients after surgery. Patient 25 did not show

any measurable improvement in functional movement; her mother reported, however, that caretaking became easier.

The differences in functional movement before and after surgery were statistically significant ( $z=4.61$ ,  $p<0.01$ ).

*Hypothesis 4 was therefore disproved.*

**TABLE 5**  
**INDIVIDUAL FUNCTIONS BEFORE AND AFTER SURGERY**  
**(mean values, differences and P values, n=29)**

	Preop	Postop	Diff	P value
ROLLING	3.8	2.7	1.1	<0.001
LONG SITTING	2.9	2.7	0.2	<0.001
SIDE SITTING	3.5	2.7	0.8	<0.001
PRONE KNEEL	3.3	2.6	0.9	<0.001
KNEEL STAND	3.7	2.7	1.0	<0.001
HALF KNEEL	4.2	2.8	1.4	<0.001
CRAWLING	3.3	2.8	0.5	<0.001
STANDING	3.7	3.1	0.6	<0.001
WALKING	3.9	3.3	0.6	<0.001

The Student paired t-test was used to test for the significance of differences between the means of the values before and after surgery. The results indicated statistically highly significant differences as shown above.

TABLE 6  
 BIOMECHANICAL PARAMETERS FOR CRAWLING PATTERNS BEFORE AND  
 AFTER SURGERY

(means and standard deviations, n=11)

Parameter	Preop	Postop	Diff	P Value
Cadence	101	99	-2	0.878
(steps/min)	(36)	(26)		
Stride length	0.47	0.65	0.18	0.008
(m)	(0.11)	(0.24)		
Average speed	0.39	0.53	0.14	0.043
(m/s)	(0.16)	(0.22)		
Thigh range	41	59	18	0.001
(degrees)	(11)	(17)		
Knee range	29	37	8	0.005
(degree)	(8)	(9)		
Thigh midrange	-9	-3	6	0.058
(degrees)	(8)	(9)		
Knee Mid-range	74	68	-6	0.001
(degrees)	(4)	(5)		

The above table indicates that, with the exception of cadence, there were significant changes in all parameters after surgery, although the changes were marginal in the case of thigh midrange.

## GAIT

From the raw data, the results were calculated, and stick figures and angular displacement data were printed for each patient.

**TABLE 7**  
**BIOMECHANICAL PARAMETERS OF WALKING PATTERNS**  
**BEFORE AND AFTER SURGERY**  
 (means and standard deviations, n=14)

Parameter	Before	After	Diff	P Value
Stride length (m)	0.72 (0.24)	0.87 (0.33)	0.15	0.049
Average Speed (m/sec)	0.67 (0.30)	0.79 (0.36)	0.12	0.059
Cadence (steps/min)	109 (34)	105 (28)	-4	0.595
Thigh Range (degrees)	39 (10)	53 (13)	14	<0.0001
Knee Range (degrees)	39 (16)	50 (20)	11	0.029
Thigh Mid-Range Point (degrees)	15 (9)	18 (9)	3	0.154
Knee Mid-Range Point (degrees)	40 (13)	56 (11)	16	<0.0001
Area (degree <sup>2</sup> )	819 (40)	1300 (47)	481	0.002
Perimeter (degrees)	133 (40)	173 (47)	40	0.001
Ratio	0.21 (0.03)	0.20 (0.03)	-0.01	0.246

A statistical comparison of the results before and after surgery was performed using Student's paired t-test. These results show that cadence was effectively unchanged after surgery but that stride length increased significantly. This led to an increase in walking speed (although not statistically significant at the 0.05 level). The increased stride length was brought about by the large improvement in thigh and knee ranges of movement. These increases in the joint ranges of movement therefore yielded marked increases in area and perimeter although the ratio was essentially unchanged.

RELATIONSHIPS BETWEEN MUSCLE TONE, VOLUNTARY MOVEMENT,  
JOINT STIFFNESS AND FUNCTIONAL MOVEMENT

The data was analysed to determine the relationships between muscle tone, voluntary movement, joint stiffness and functional movement.

Preoperative Mean Voluntary Movement vs. Preoperative Mean Muscle Tone (Fig. 30, page 137).

There was a positive correlation between tone and voluntary movement (Kendalls tau=0.38, z=2.58, p<0.01). This fits the clinical observation that the patients who were the most spastic were the most handicapped in voluntary movement.

FIGURE 30

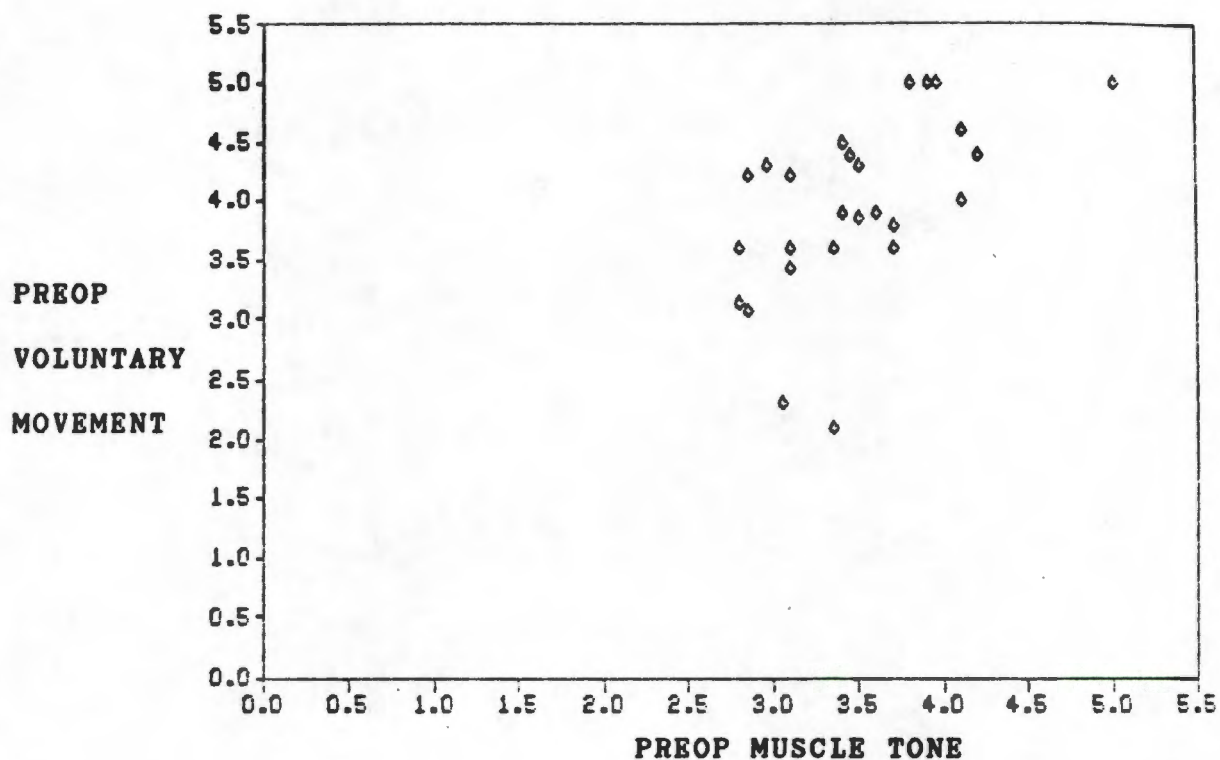


Fig. 30. Preoperative mean voluntary movement vs. preoperative mean muscle tone.

VOLUNTARY MOVEMENT Normal = 1 Very poor = 5

MUSCLE TONE Normal = 2 Severely increased = 5

Postoperative Mean Voluntary Movement vs. Postoperative Mean Muscle Tone (Fig. 31, page 139).

No correlation between voluntary movement and muscle tone was found (Kendalls tau=0.15, z=0.88, p>.05). Muscle tone decreased to normal or near normal in everyone, in the most severely handicapped as well as in those with relatively little handicap, but voluntary movement improved more selectively and improved little in the severely handicapped patients.

FIGURE 31

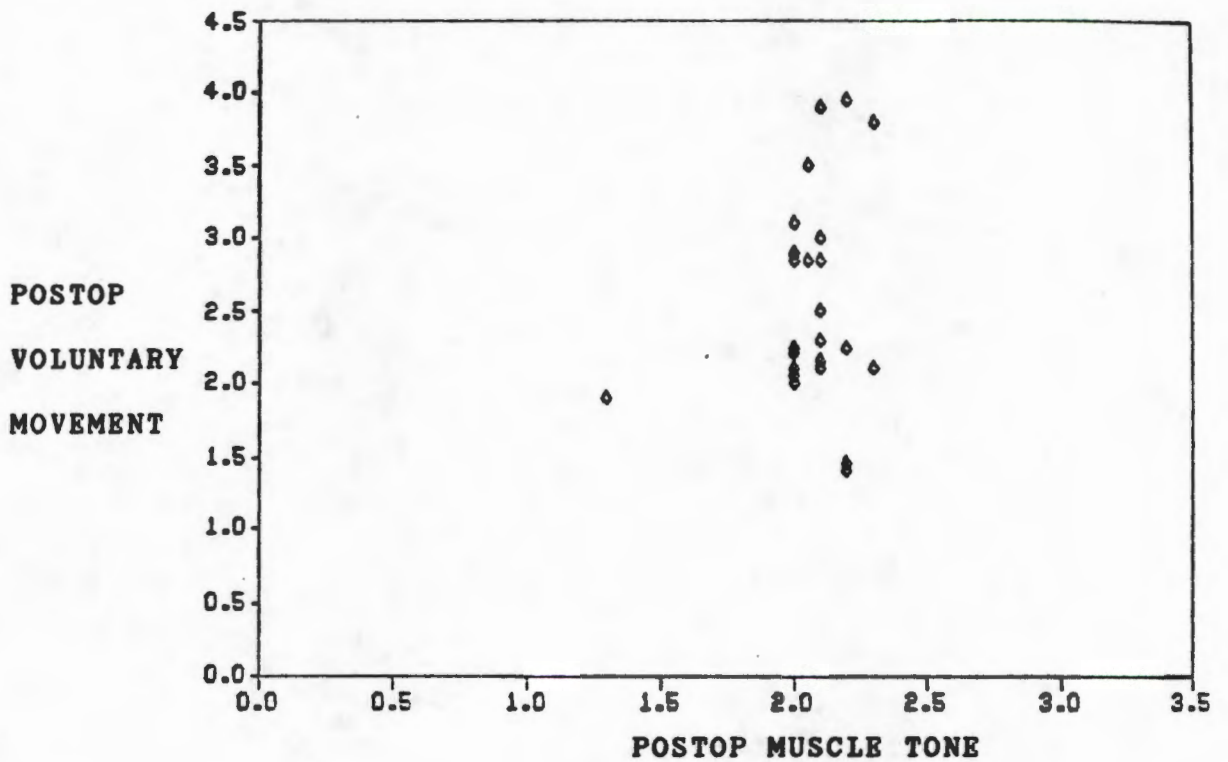


Fig. 31. Postoperative mean voluntary movement vs. postoperative mean muscle tone.

VOLUNTARY MOVEMENT Normal = 1 Very poor = 5

MUSCLE TONE Normal = 2 Severely increased = 5

Difference in Mean Voluntary Movement Before and After Surgery vs. Preoperative Mean Tone (Fig. 32, page 141).

There was a significant negative correlation between these parameters (Kendalls tau=0.29, z=1.98, p<0.05). This means that the most handicapped patients had the least improvement.

FIGURE 32

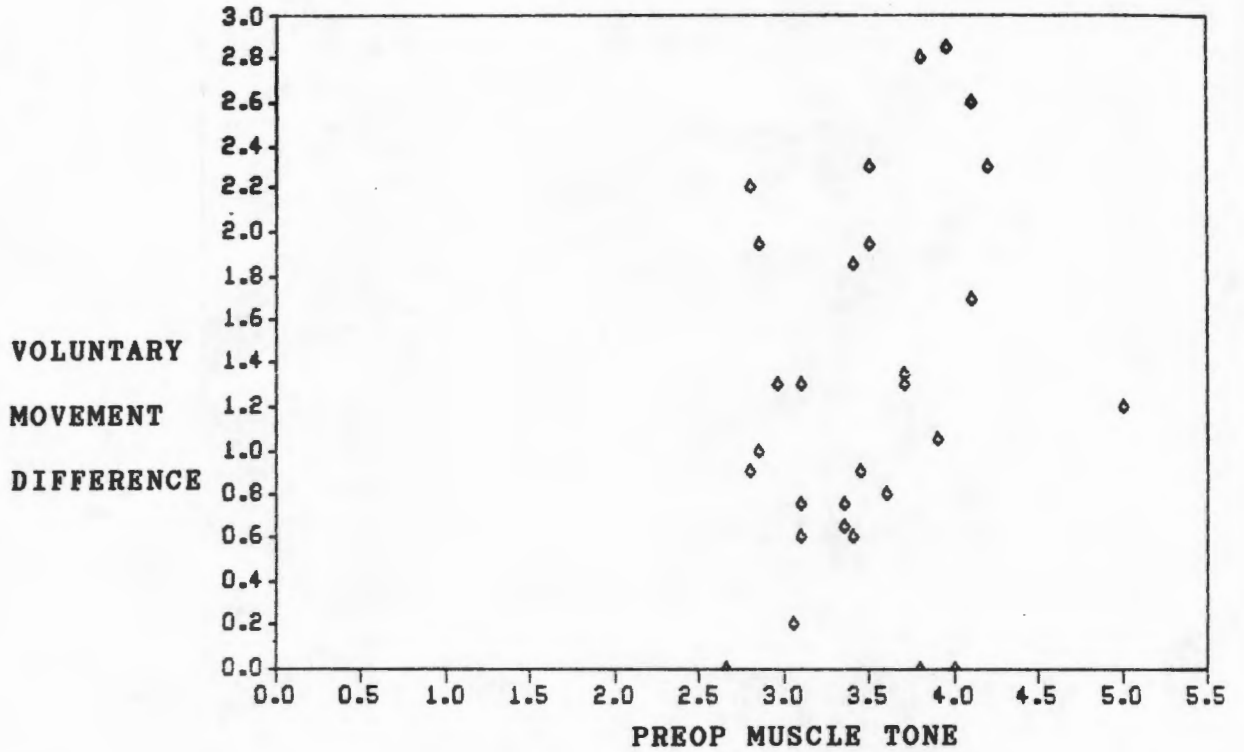


Fig. 32. Difference in voluntary movement vs. preoperative mean muscle tone.

VOLUNTARY MOVEMENT Normal = 1 Very poor = 5

MUSCLE TONE Normal = 2 Severely increased = 5

Functional Movement Difference vs. Preoperative Mean Muscle Tone (Fig. 33, page 143).

There was a negative correlation (Kendalls tau=0.32, z=2.38, p<0.05). This means that patients with less spasticity preoperatively had the greatest improvement in functional movement while those with severe spasticity had the least improvement in functional movement.

FIGURE 33

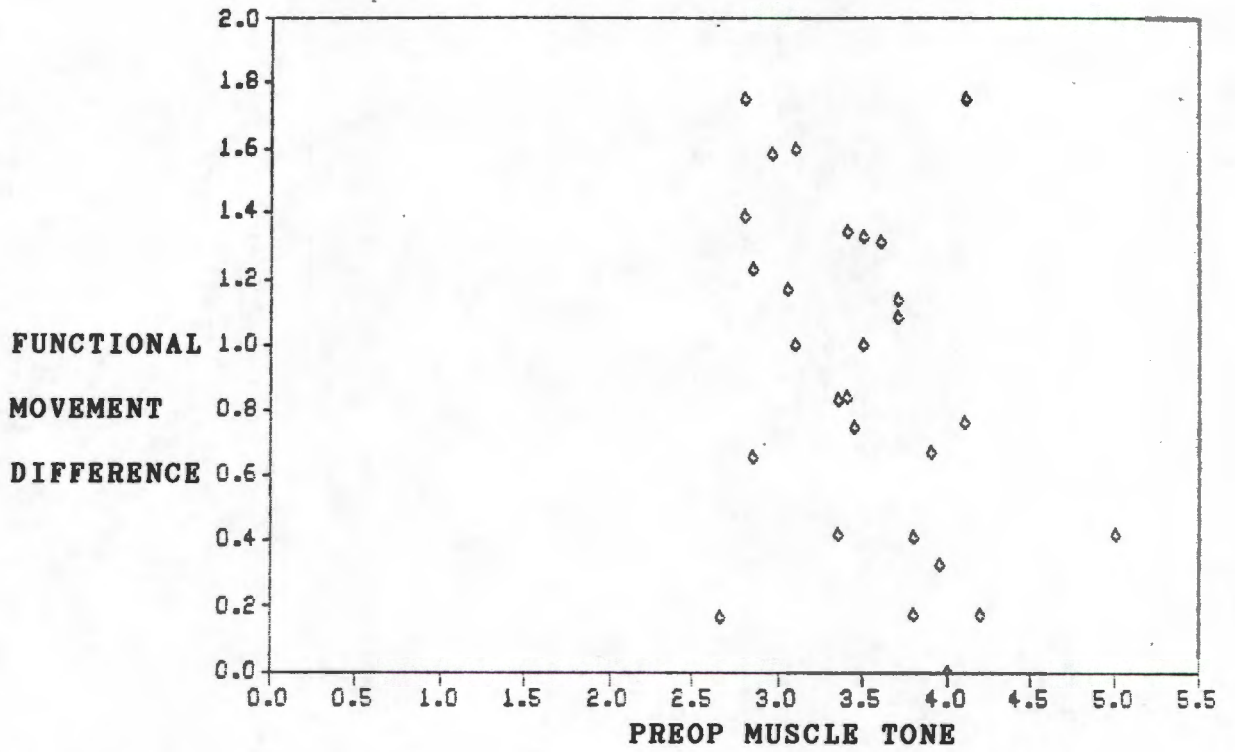


Fig. 33. Functional movement difference vs. preoperative mean muscle tone.

FUNCTIONAL MOVEMENT    Normal = 1    Very poor = 5  
MUSCLE TONE    Normal = 2    Severely increased = 5

Preoperative Mean Functional Movement vs. Preoperative Mean Muscle Tone (Fig. 34, page 145).

A positive correlation was found. (Kendalls tau=0.61, z=4.48, p<0.01).

This agrees with the clinical observation that the patients who had the most spasticity preoperatively were the most limited in functional movement.

FIGURE 34

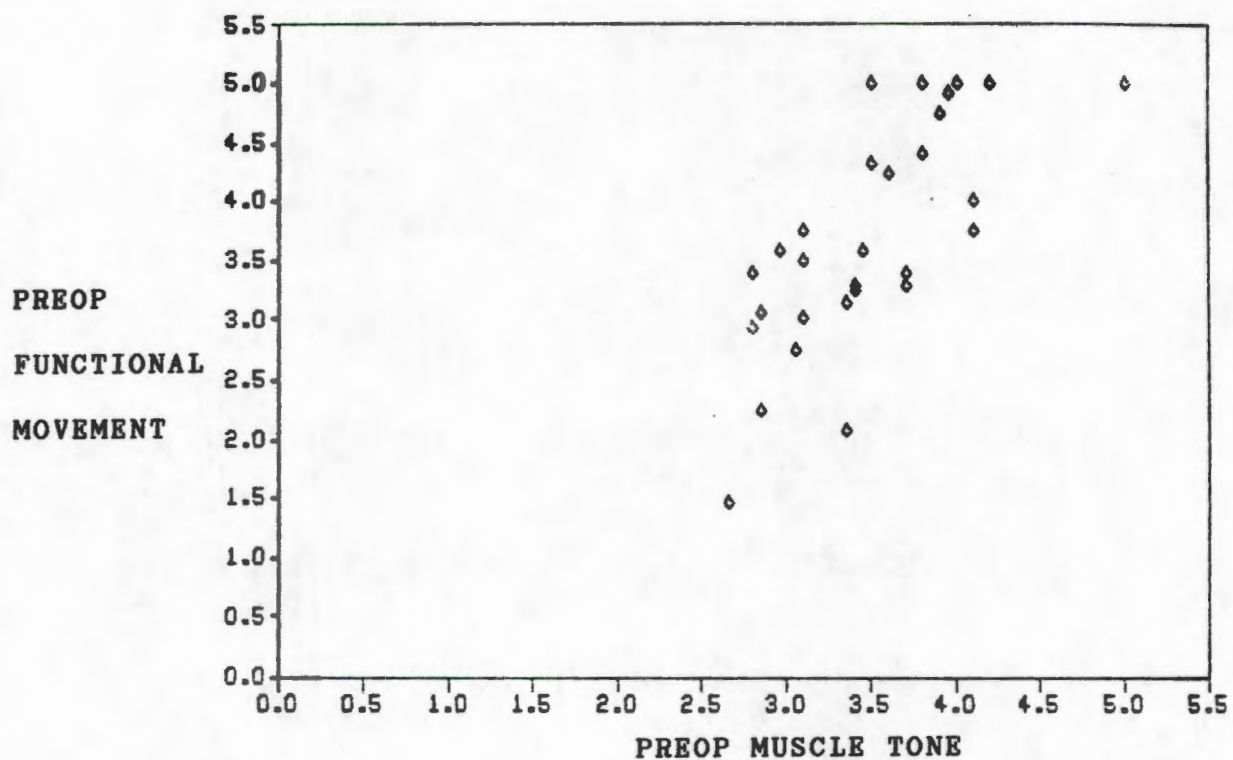


Fig. 34. Preoperative mean functional movement vs. preoperative mean muscle tone.

FUNCTIONAL MOVEMENT    Normal = 1    Very poor = 5  
MUSCLE TONE    Normal = 2    Severely increased = 5

Mean Muscle Tone, Mean Voluntary Movement, Mean Joint Stiffness And Means of Functions For All Patients.

(Fig. 35, page 147).

This figure summarizes the results. Mean muscle tone became nearly normal; mean joint stiffness showed great improvement. Mean voluntary movement improved a great deal although it still had a long way to go to normal limits. The means for all functions across all patients showed improvement although considerably less than expected from the improvement in muscle tone.

FIGURE 35

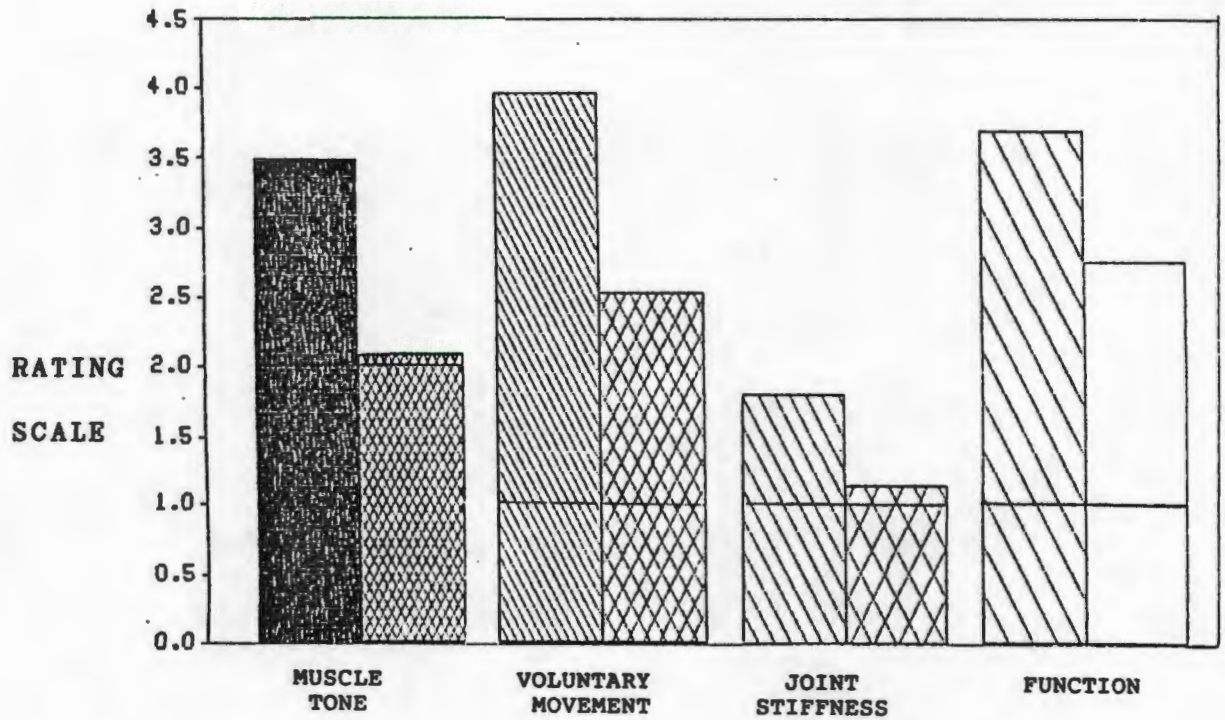


Fig. 35. Mean muscle tone, mean voluntary movement, mean joint stiffness and means of functional movement scores for all patients before and after surgery.

The left hand vertical bar for each parameter indicates the preoperative status and the right hand vertical bar the postoperative status. The horizontal line across each parameter indicates the normal value.

MUSCLE TONE Normal = 2 Severely increased = 5

VOLUNTARY MOVEMENT Normal = 1 Very poor = 5

FUNCTIONAL MOVEMENT Normal = 1 Very poor = 5

JOINT STIFFNESS Normal = 1 Apparent fixed contracture = 4

## CHAPTER 5

### DISCUSSION

Since results of an uncontrolled pilot study of Selective Posterior Lumbar Rhizotomy (SPLR) in spastic cerebral palsy patients were promising (Peacock & Arens, 1982), it was decided to do a formal prospective study.

Severing the posterior nerve rootlets during SPLR reduced the spasticity and retained sensory function, whereas earlier methods had caused a loss of sensation with resultant trophic changes and sensory ataxia.

Controlled studies on spastic patients with cerebral palsy have not been done before since many investigators regard this as impossible because of the nature of cerebral palsy and the complicated problems of research design (Wolf, 1969; Scherzer & Tscharnuter, 1984). In patients with cerebral palsy there are many variables that make it difficult to evaluate results quantitatively (Keats, 1970).

The aim of the study reported here was to study objectively the effects of SPLR on muscle tone, voluntary movement, joint stiffness and functional movement.

An ideal design for a study would be to compare two groups of patients allocated on a random basis, one group to have SPLR and the other to have a "sham" operation. The sham operation should be identical in every respect to the surgery in the first group except that the posterior rootlets are not sectioned. One problem that arises immediately is an ethical one, but this might be overcome by offering the procedure to the sham operated patients after the benefits of the procedure have been proved. Another objection would be that the therapist would know immediately after the procedure which patients have not had the surgery because of the unchanged spasticity. One could anticipate that patients who have had SRLR would be more likely to receive intensive therapy after surgery and therefore bias the study.

Another possible objection is the ethical concern of withholding possibly effective treatment from the patients who form the control group. Since such ethical questions arise when one considers a research project which involves either withholding treatment or performing sham operations on a random selection of patients, the most practical solution was to use each patient as his own control, and to compare the preoperative state with the postoperative one.

The study reported here investigated the effects of SPLR on muscle tone, voluntary movement, joint stiffness and functional movement, using each patient as his own control. As there is no standardized clinical method to quantify changes in the quality of movement (Tscharnuter, 1984), I developed the methods reported here for assessing the above parameters.

#### THE ASSESSMENT SCALES

Rating scales were devised to quantify the various parameters that were measured. For practical reasons, these scales were divided into points for easy clinical differentiation.

The development of abnormal movement in the cerebral palsied patient was used as a guide in developing these assessment scales (Bly, 1980). The scales for the various functional movements were developed by observing patients closely during treatment and by comparing their quality of movement with the normal.

An ideal assessment would be one which would measure muscle tone and simultaneously show how it affects voluntary movement, functional abilities and independence. Although quantification of aspects of movement being studied was difficult, frequent and methodical examinations of many

patients by the same examiner using the above methods led to findings which were consistent from one examination to the next.

## MUSCLE TONE

### *Hypothesis 1*

*SPLR does not lead to a reduction in muscle tone of spastic muscle groups of the upper and lower limbs.*

In all patients, the spasticity was reduced to normal or near normal, no matter what degree of tone the patient had initially (Table 10, page 176).

The most remarkable result was the consistent reduction in spasticity in all patients and spastic muscle groups innervated from L4 to S1.

There was a statistically significant reduction of muscle tone in both upper and lower limbs. This was surprising as rhizotomy was done in the lumbar region only. At this stage the exact physiological mechanisms for this carry over effect are unknown. It is speculated by Dr. Peacock that there are complicated synapses at different levels including the cervical region and that these may be affected when

posterior rootlets are severed in the lumbar region (Peacock, 1987).

The patients who had the highest tone preoperatively had the greatest reduction in muscle tone. In the case of patient 23 (Table 10, page 176), muscle tone was slightly below normal after surgery. It is speculated that this patient had not yet built up sufficient muscle strength in previously spastic muscles. Although he was last examined 6 months after surgery, further improvement can be expected with therapy.

While bearing in mind that overall function does not depend on isolated muscle groups and that postural patterns should be observed rather than single muscle groups, it was necessary to examine muscle groups systematically in order to compare preoperative status with postoperative status. It is also important to note that tone in supine and prone is functionally less important than tone in sitting, and standing as well as tone changes during movement.

Spasticity masks muscle power to a considerable degree. A pyramidal lesion may produce a disability resulting primarily in loss of power or in increased tone with little or no loss of power (Hattab in Feldman et al. 1980).

One cannot therefore predict with accuracy how much power and restoration of function there will be in the limb after the spasticity has been relieved (Frazier, 1910).

## VOLUNTARY MOVEMENT

### HYPOTHESIS 2

*SPLR does not lead to an improvement in voluntary movement in all spastic muscle groups of the upper and lower limbs.*

There was a significant change in voluntary movement of the lower limbs but there was not a statistically significant change in voluntary movement in the upper limbs after surgery. In most patients the voluntary movement in the upper limbs was good (1.8) and there was therefore less room for improvement (Table 11, page 177).

Voluntary movement is difficult to measure as the tester requires the cooperation of the patient and therefore the method of testing must be simple enough for the patient to follow instructions.

It is very difficult to assess voluntary movement in the cerebral palsied patient, as residual movement is masked by spasticity. Voluntary movement is assessed conventionally by examining muscle power and active range of movement. Voluntary muscle control is affected by the passive range of movement. The patient's ability to start and to stop a movement in a controlled manner indicates refined voluntary movement.

The individual with serious brain damage does not have the ability to perform isolated muscle contraction (Gillette, 1966). Movements occur in synergies and it is difficult for the patient to perform isolated movements. Thus, in cerebral palsied patients, mass patterns are often present eg. they have difficulty isolating knee flexion with the hips extended. In this study, the limb was therefore stabilized in a position to avoid total mass patterns i.e. the joint proximal to the movement was held fixed in position. This was judged to be a good indicator of voluntary movement before and after surgery, since it was measurable and objective.

Treatment techniques can be more effective when spasticity is reduced. This was shown in a double blind study; the reduction of spasticity by the drug called baclofen led to better voluntary movement and coordination and the Bobath therapy approach became more effective (Van Hemert, in Feldman et al. 1980). This will possibly also be the case when spasticity is permanently reduced by rhizotomy.

There was considerable variation from patient to patient in the degree of improvement of voluntary movement following rhizotomy. This variability in response may be related to the specific pathology in the motor system of each patient.

There are pathophysiological mechanisms which inhibit voluntary movement and these depend on the location and the extent of the lesions (Moore, 1984).

In my own experience, other factors that may influence the measurement of voluntary movement are the age, the I.Q., the emotional state and the position in which the patient is tested.

Although there was great variation in voluntary control, all patients displayed better control as well as greater ease of movement postoperatively.

## JOINT STIFFNESS

### Hypothesis 3

*SPLR does not lead to an improvement of joint stiffness of upper and lower limb joints.*

There was a significant overall reduction in joint stiffness in the joints of the lower limbs, but this was not the case for the upper limb joints.

This difference in the improvement of joint stiffness between the upper and lower limbs was probably due to the fact that most patients were spastic diplegics. The reduction in spasticity in the upper limbs, although statistically significant, was small and few patients had fixed contractures in these joints.

Contractures develop when fibrous changes occur in the joints primarily due to the lack of movement occurring at the joints. This lack of movement occurs primarily as result of spasticity or as a compensatory mechanism when the patient habitually assumes a static joint position to assist function.

The assessment of passive movement in the presence of spasticity makes it difficult to differentiate between

shortening of the muscle from increased tone and true relative shortening (Sharrard, 1967). Samilson (1981) describes a dynamic deformity as one related to spasticity without structural shortening in a musculotendinous unit or in the joint or joints over which it passes. Tscharnuter (1984) also makes the distinction between true contractures and correctible movement limitations caused by abnormal postural tone. Once deformity has developed, the stronger muscle is able to act with greater mechanical advantage and the antagonist suffers mechanical disadvantage.

In evaluating the results of the study reported here, it is obvious that there was greatest reduction of contractures in the hip followed by the knee and ankle (Table 13I, page 187). Movement in these joints improved dramatically after surgery.

It is therefore important to consider that what appears to be a fixed contracture preoperatively, may in fact only be stiffness from spasticity. This factor should play a major role when considering orthopaedic releases, and should lead to use of SPLR in many cases which would otherwise undergo orthopaedic surgery. In the case of many patients, SPLR should be done prior to orthopaedic surgery.

## FUNCTIONAL MOVEMENT

### Hypothesis 4

*SPLR does not lead to an improvement in functions such as rolling, sitting, kneeling, crawling, standing and walking.*

The results of this study indicate that there was a definite improvement in most functions in 28 of the 29 patients, and in the patient who showed no measurable improvement, caretaking became much easier (Tables 14 to 22, pages 188 to 196).

Functional movement varied in each patient, probably depending on the degree of cerebral involvement. Patients with less disability made the greatest gains in terms of function while the more involved patients made smaller gains.

### Rolling (Table 14, page 188)

Due to the nature and distribution of spasticity and to the development of abnormal movement, patients with spasticity tend to roll en bloc without segmental rotation. After surgery it was noticeable that most patients were able to achieve better rotation and separation between the shoulders and pelvis and between the legs, on rolling. Nine patients

did not improve in rolling; five of these were severely impaired.

Long sitting (Table 15, page 189)

This improved in 26 patients. Patients 13 and 25 did not improve in this function because they were severely handicapped and had fixed joint contractures; it was, however, possible to place them into this position with greater ease. Patient 18 was able to long sit before surgery, but not well, and did not show any improvement.

The ability to assume long sitting is dependent on the ability to flex at the hips and simultaneously extend the knees. This function is very difficult when spasticity is present as the mass patterns that are present make it difficult to isolate movements. Once the spasticity was removed, it was possible to flex the hips and extend the knees with much greater ease.

Side sitting (Table 16, page 190)

This function improved in nineteen patients. Five of the patients who showed no improvement were severely handicapped; five were moderately handicapped. Once spasticity was reduced the patients were better able to assume and maintain the side sitting position. Due to the reduction in spasticity in muscle groups around the pelvis

and legs, the patients were able to shift their weight, elongate the weight bearing side and rotate the trunk. Arm support was achieved with greater ease as there was less need to fix in this position to maintain balance. Patients were also able to free the arms with greater ease and maintain this position with better weight distribution.

Prone kneeling (Table 17, page 191)

After surgery, patients were able to assume or be placed in this position with greater ease due to the ability of the patients to distribute their weight better. Nine patients who were severely handicapped, one moderately and three mildly handicapped patients did not show any improvement.

When spasticity is present there is a tendency for the patient not to bear weight on all four limbs but rather to "fix" in this position with excessive hip flexion and a lumbar lordosis. Once spasticity was reduced this position could be assumed with better symmetry and less need to "fix" to maintain the position.

Kneel standing (Table 18, page 192)

This function improved after surgery in patients who were able to assume this position preoperatively. Two mildly handicapped and 5 very handicapped patients did not show any

change. Twenty two patients showed an improvement in this function.

Patients used less hip flexion when assuming this position. Once spasticity was removed there was a reduction in joint stiffness and less need to "fix", using hip flexion to maintain balance. Without spasticity the legs could provide a better base for support and the patients were able to "break" up the total pattern. The lordosis improved as less compensation was needed to balance the degree of hip flexion. Although this function improved, it was necessary through therapy to improve the equilibrium reactions to help the patient to maintain the position.

#### Half kneeling (Table 19, page 193)

The ability to separate the legs during half kneeling improved dramatically after the surgical reduction in spasticity. The reduction in muscle tone in the hip flexors, adductors, internal rotators, knee flexors and plantar flexors of the feet made it possible for patients to separate the legs and to maintain this position with more stability. This enabled patients to use their arms more effectively.

Most patients showed marked improvement. Three patients who were severely impaired, did not improve. Two of these patients had multiple fixed contractures. Another patient

who did not show any change, was very mildly affected, and one other was moderately affected.

Crawling (Table 20, page 194)

Thirteen patients improved clinically while sixteen did not show a measurable improvement. Once the spasticity was reduced, patients were able to distribute weight better and crawl with better reciprocation. It was easier to shift weight and to elongate the weight bearing side. There was also a marked reduction in mass patterns that were previously used.

Standing (Table 21, page 195)

Fourteen patients did not show any measurable change; eight were non-ambulators; two were household ambulators and four were independent ambulators. Fifteen of the 29 patients in the study improved. Standing improved because the patients used less of a lordosis since there was less need to compensate by using hip flexion. Reduction of spasticity in the muscle groups of the feet resulted in a more stable base of support.

In comparison with other functions, gains were smaller for standing and for crawling. The fact that many patients were severely handicapped and had not yet developed adequate

righting and equilibrium reactions, may explain these results.

Walking (Table 22, page 196)

The quality of walking improved in fifteen patients. Eight of nine patients who were graded as 4 before surgery improved by one or two grades - a clinically significant difference since they became more functional walkers. Twelve patients who were not functional walkers before surgery, were still unable to do so after surgery; all were severely impaired.

Once spasticity is reduced, it is necessary to elicit balance reactions through therapy, as balance is not directly affected by the SPLR.

COMPUTER ANALYSIS OF CRAWLING AND WALKING DATA

Figures 36 to 50 in Appendix D, pages 202 to 216 present data derived from the computer analysis of crawling and walking. In a number of instances, the data is incomplete because of the unavailability or malfunctioning of equipment at the time of testing, lack of patient cooperation and therefore inability to capture a full cycle of data.

Crawling (Computer Analysis) (Table 6, page 133)

From the data presented, a number of trends emerge: cadence tends to be quite variable but does not appear to have been altered much by the surgery. However there is a marked increase in stride length (over 36%) and this leads to an increased average speed as would be expected (since speed = cadence x stride length). This increased stride length is brought about by an increase in the range of joint movement, primarily where the thigh's range increases by almost 44%. This is due to the reduction in spasticity of the hip flexors which consequently allows freer voluntary movement. Postoperatively the patients would appear to adopt a midrange position of the thigh which is slightly more flexed while the knee is more extended. This finding indicates that the hamstring muscle group, which tends to extend the hip and flex the knee, is not playing such a dominant role postoperatively. Both these factors (increased joint ranges of movement, and shifting of the midrange points) can be attributed to the reduction in spasticity and consequent improvement in voluntary movement.

The influence of maturation and segmental growth could play a role in the increased stride length and speed. However, it is felt that the significant increases in the range of joint movement played a far more dominant role. This is an important point, especially in the light of Sutherland's

work (1984), in which it was shown that while children and adults had different stride length and speed, there was almost no difference in their ranges of joint movement.

Walking (Computer Analysis) (Table 7, page 134).

As in the case of crawling, the analysis of the walking data indicates a statistically significant increase in the thigh range after surgery. The knee range also increased considerably. These changes in range resulted in an increase in stride length and speed.

Sutherland (1984) points out that as normal children get older, cadence decreases. In this study with spastic patients, cadence did not show a significant change. This may be because the four to fourteen month period between examinations was too short to show a difference or due to the fact that while relief of spasticity improves the range of movement, there is still a marked impairment of balance due to the brain damage.

RELATIONSHIPS BETWEEN MUSCLE TONE, VOLUNTARY MOVEMENT,  
JOINT STIFFNESS AND FUNCTIONAL MOVEMENT

Analysis of the data revealed that, before surgery, patients with greatly increased muscle tone had poorer voluntary movement (Fig. 30, page 137). This also applied to functional movement (Fig. 34, page 145). This is exactly what one might expect in spastic patients.

Patients with higher tone preoperatively showed less improvement in voluntary movement postoperatively than those with less severe spasticity. The more handicapped patients made smaller gains in voluntary movement postoperatively, possibly because of the degree of cerebral damage.

There was no correlation between postoperative voluntary movement and postoperative muscle tone (Fig. 31, page 139). Although muscle tone returned to normal or near normal after surgery, there was less improvement in voluntary movement. This also applied to functional movement. This may be due to the fact that, in some cases, spasticity may be associated with loss of power while in other cases there may be little or no loss of power (Hattab in Feldman et al. 1980). The results also indicate a negative correlation between preoperative tone and improvement in functional movement after surgery (Fig. 33, page 143). This means that the more spastic the patient was before surgery, the smaller the improvement in function. It is obvious that the less

impaired patient with less spasticity had the greatest improvement in function.

Patient 25 (Tables 14 to 22, pages 188 to 196) did not show any measurable improvement in functional movement. Her mother reported, however, that following the reduction of spasticity, placing her into positions and general caretaking became much easier.

Patient 4 showed a very small improvement; preoperatively he was a functional walker and the reduction in spasticity resulted in small gains in terms of voluntary movement, joint stiffness and function. He was also very young and had not developed contractures. Although measurable improvement was small, the quality of his movements improved and the probability of the need for orthopaedic surgery in the future lessened.

The patients who had the greatest improvement in voluntary movement were patients 7, 12, 15 and 29 (Table 11, page 177). Patient 7 was a severely spastic patient who had previously not had therapy. When the spasticity was reduced, she was spontaneously able to move much more freely. Patients 12 and 15 were severe athetoids with spasticity who, prior to the surgery, had very little ability to move voluntarily. One can speculate that the variability in improvement of voluntary movement is related to the amount of cerebral damage.

Patient 29 was a severely spastic quadriplegic patient who had very little movement due to the severity of his spasticity. He showed a great improvement in voluntary movement but a small change in functional movement.

Patients 12 and 15 (Tables 10, 11 and 14 to 22, pages 176, 177 and 188 to 196) were athetoids with spasticity. The reduction of spasticity in these patients allowed them much greater voluntary movement but not the same degree of improvement in functional movement. This is probably due to the fact that these patients were very handicapped by spasticity and once this was reduced, voluntary movement improved considerably. Although the spasticity was reduced, they had not yet built up righting and equilibrium reactions necessary to assume and maintain positions and therefore functional movement did not improve as much.

Patient 19 (Table 11, page 177, Tables 14 to 22, pages 188 to 196) had a very small change in voluntary movement but a big change in function. This patient was mildly handicapped initially and following the surgery, had very intensive therapy. He was a bright and well motivated patient who had parental support as well as high expectations for improvement. His improvement in function is probably partly due to perseverance on his part and the strenuous therapy program.

## SIGNIFICANCE OF THIS STUDY FOR OCCUPATIONAL THERAPISTS

The occupational therapist is concerned about the patient's ability to participate in activities in order to live a meaningful life. This study shows that the reduction of muscle tone produced by Selective Posterior Lumbar Rhizotomy in spastic patients significantly improves function.

Once the spasticity has been reduced, the therapist has less need to inhibit spasticity and can proceed with the facilitation of normal movement patterns. This saves valuable treatment time and results in quicker progress for the patient. He is also able to move with greater ease through wider ranges. One of the greatest gains made is the patient's improved ability to assume and maintain positions. This enables him to participate better in activities. When a patient can sit more easily, his upper limb function becomes more efficient.

With an increase in the range of movement, gait and transfers from one surface to another become easier and less laboured. This helps him in his every day activities such as dressing, bathing and general mobility.

This study has shown that the cerebral palsied patient makes large gains in motor ability after surgery. Sensory-motor learning improves with improvement in motor ability which facilitates the development of social, emotional and intellectual function.

These results provide a solid basis for continuing therapy and assist the patient and therapist in the rehabilitation process.

This study has also shown the important contribution that the occupational therapist, with knowledge of and experience in the evaluation of the spastic patient, can make in the selection and treatment of patients before and after surgery.

## CONCLUSION

*Twenty nine patients with cerebral palsy who were selected for Selective Posterior Lumbar Rhizotomy were studied before and after surgery. The following conclusions were drawn from this study:*

- 1. The rating scales that were developed were useful in the preoperative and postoperative assessment of the patients who were studied.*
- 2. Surgery produced a change in muscle tone to normal or close to normal.*
- 3. There was a marked improvement in voluntary muscle movement and in functional movement after surgery.*
- 4. Joint stiffness decreased markedly after surgery even where fixed contractures were suspected in some cases.*
- 5. An indirect conclusion is that the occupational therapist becomes more effective and that the patient can make quicker progress after surgery.*
- 6. Selective Posterior Rhizotomy is a useful procedure in patients with spastic cerebral palsy and this study thus confirms earlier clinical findings.*

## SUGGESTIONS FOR FURTHER STUDIES

The following need further investigation by occupational therapists working in the field of cerebral palsy:

1. The improvement in function after SPLR with emphasis on activities of daily living.
2. The change in upper limb weightbearing and manipulative skills after SPLR.
3. The effects of SPLR on righting and equilibrium reactions.
4. Efficacy of treatment techniques after SPLR.
5. Investigation of the long term effects on patients who undergo SPLR surgery.

APPENDIX A

TABLE 8

DEMOGRAPHICS A

PAT	INIT	AGE	SEX	DIAG	ORTHO	SEVERITY
1	A.B.	13	F	Q	TA&ADD	HOUSEHOLD AMB
2	S.W.	3	M	D	NONE	NON AMB
3	J.S.	10	M	D	NONE	AMB
4	F.S.	3 1/2	M	D	NONE	AMB
5	N.M.	2 1/2	F	D	NONE	HOUSEHOLD AMB
6	M.J.	3 1/2	F	D	NONE	AMB
7	D.P.	9	F	Q	NONE	NON AMB
8	M.W.	6	F	D	NONE	AMB
9	A.C.	7	M	D	BI TA	AMB
10	J.J.	4	M	D	NONE	NON AMB
11	O.W.	10	M	D	TA	AMB
12	H.V.	4	F	A, S, Q	NONE	NON AMB
13	L.P.	4	F	Q	NONE	NON AMB
14	M.N.	35	M	Q	NONE	HOUSEHOLD AMB
15	R.O.	5	M	A, S, Q	NONE	NON AMB
16	M.K.	10	F	Q	NONE	NON AMB
17	G.B.	4	M	D	NONE	HOUSEHOLD AMB
18	L.R.	11	F	D	2X BI TA	HOUSEHOLD AMB
19	B.P.	7	M	D	BI TA	AMB
20	R.P.	14	M	D	TA & ADD	HOUSEHOLD AMB
21	T.E.	21	M	D	BI ADD	AMB
22	P.R.	12	M	D	BI TA ADD, 2X HAMS	AMB
23	Z.S.	8	M	Q	NONE	NON AMB
24	C.R.	11	F	D	BI TP LEN, BI PLANT FASC	AMB
25	J.H.	9	F	D	BI ADD	NON AMB
26	J.B.	12	M	D	BI HAMS 2X TA	AMB
27	J.S.	14	M	Q	TA & HIP FLEX	NON AMB
28	S.C.	6	M	D	NONE	HOUSEHOLD AMB
29	N.G.	3 1/2	M	Q	NONE	NON AMB

TABLE 9  
DEMOGRAPHICS B

PAT	INIT	AGE	SEX	DIAG	*INTELLECT	CAUSE
1	A.B.	13	F	Q	EDUCABLE	CONGENITAL
2	S.W.	3	M	D	EDUCABLE	CONGENITAL
3	J.S.	10	M	D	TRAINABLE	CONGENITAL
4	F.S.	3 1/2	M	D	EDUCABLE	CONGENITAL
5	N.M.	2 1/2	F	D	EDUCABLE	CONGENITAL
6	M.J.	3 1/2	F	D	EDUCABLE	CONGENITAL
7	D.P.	9	F	Q	TRAINABLE	CONGENITAL
8	M.W.	6	F	D	EDUCABLE	CONGENITAL
9	A.C.	7	M	D	TRAINABLE	CONGENITAL
10	J.J.	4	M	D	EDUCABLE	CONGENITAL
11	O.W.	10	M	D	EDUCABLE	CONGENITAL
12	H.V.	4	F	A, S, Q	TRAINABLE	CONGENITAL
13	L.P.	4	F	Q	UNTRAINABLE	POST TRAUMA
14	M.N.	35	M	Q	EDUCABLE	POST TRAUMA
15	R.O.	5	M	A, S, Q	EDUCABLE	CONGENITAL
16	M.K.	10	F	Q	EDUCABLE	POST TRAUMA
17	G.B.	4	M	D	EDUCABLE	CONGENITAL
18	L.R.	11	F	D	EDUCABLE	CONGENITAL
19	B.P.	7	M	D	EDUCABLE	CONGENITAL
20	R.P.	14	M	D	TRAINABLE	CONGENITAL
21	T.E.	21	M	D	TRAINABLE	CONGENITAL
22	P.R.	12	M	D	TRAINABLE	CONGENITAL
23	Z.S.	8	M	Q	TRAINABLE	CONGENITAL
24	C.R.	11	F	D	EDUCABLE	CONGENITAL
25	J.H.	9	F	Q	UNTRAINABLE	CONGENITAL
26	J.B.	12	M	D	EDUCABLE	CONGENITAL
27	J.S.	14	M	Q	TRAINABLE	CONGENITAL
28	S.C.	6	M	D	EDUCABLE	CONGENITAL
29	N.G.	3 1/2	M	Q	EDUCABLE	CONGENITAL

Educable I.Q. 90 and above  
 Trainable I.Q. 50-90  
 Untrainable I.Q. <50

APPENDIX B

Appendix B contains raw data of muscle tone, voluntary movement, joint stiffness and selected functions for individual patients before and after surgery.

TABLE 10

MUSCLE TONE BEFORE AND AFTER SURGERY

PAT	UPPER LIMB TONE			LOWER LIMB TONE		
	PREOP	POSTOP	DIF	PREOP	POSTOP	DIF
1	2.9	2.9	0.0	3.1	2.0	1.1
2	2.6	2.6	0.0	3.6	2.0	1.6
3	2.0	2.0	0.0	2.9	2.0	0.9
4	2.0	2.0	0.0	2.7	2.0	0.7
5	2.0	2.0	0.0	3.4	2.1	1.3
6	2.0	2.0	0.0	2.8	2.0	0.8
7	2.2	2.2	0.0	4.1	2.0	2.1
8	2.4	2.0	0.4	3.0	2.1	1.1
9	2.0	2.0	0.0	3.7	2.2	1.5
10	2.0	2.0	0.0	3.5	2.0	1.5
11	2.0	2.0	0.0	2.9	2.0	0.9
12	3.2	2.5	0.7	3.8	2.0	1.8
13	2.8	2.8	0.0	3.8	2.0	1.8
14	2.0	2.0	0.0	3.7	2.1	1.6
15	4.7	4.7	0.0	4.2	2.3	1.9
16	4.7	3.3	1.4	5.0	2.3	2.7
17	3.2	2.9	0.3	3.1	2.0	1.1
18	2.8	2.2	0.6	3.1	2.1	1.0
19	2.0	2.0	0.0	3.1	2.1	1.0
20	2.0	2.0	0.0	4.1	2.1	2.0
21	2.0	2.0	0.0	2.8	2.2	0.6
22	2.0	2.0	0.0	3.5	2.1	1.4
23	3.1	2.9	0.2	3.5	1.3	2.2
24	2.0	2.0	0.0	3.4	2.2	1.2
25	2.2	2.2	0.0	4.0	2.9	1.1
26	2.0	2.0	0.0	3.4	2.0	1.4
27	3.8	3.5	0.3	3.9	2.2	1.7
28	2.0	2.0	0.0	3.4	2.1	1.3
29	2.9	2.9	0.0	4.0	2.1	1.9
MEAN	2.53	2.40	0.13	3.50	2.09	1.41
SD	0.768	0.621	0.303	0.514	0.229	0.494

TABLE 11

## VOLUNTARY MOVEMENT BEFORE AND AFTER SURGERY

PAT	UPPER LIMB			LOWER LIMB		
	PREOP	POSTOP	DIF	PREOP	POSTOP	DIF
1	1.5	1.2	0.3	3.5	2.9	0.6
2	2.0	2.0	0.0	3.9	3.1	0.8
3	1.0	1.0	0.0	4.2	2.3	1.9
5	1.0	1.0	0.0	4.5	3.9	0.6
6	1.0	1.0	0.0	3.2	2.3	0.9
7	1.0	1.0	0.0	4.6	2.0	2.6
8	1.0	1.0	0.0	4.3	3.0	1.3
9	1.0	1.0	0.0	3.6	2.3	1.3
10	1.0	1.0	0.0	4.3	2.0	2.3
11	1.0	1.0	0.0	3.1	2.1	1.0
12	4.1	4.0	0.1	5.0	2.2	2.8
14	2.0	2.0	0.0	3.8	2.5	1.3
15	3.9	3.9	0.0	4.4	2.1	2.3
16	4.8	4.2	0.6	5.0	3.8	1.2
17	2.6	2.4	0.2	4.2	2.9	1.3
18	1.0	1.0	0.0	3.6	2.9	0.7
19	1.0	1.0	0.0	2.3	2.1	0.2
20	1.0	1.0	0.0	4.0	2.3	1.7
21	1.0	1.0	0.0	3.6	1.4	2.2
22	1.0	1.0	0.0	4.4	3.5	0.9
23	3.3	3.3	0.0	3.9	1.9	2.0
24	1.0	1.0	0.0	2.1	1.5	0.6
26	1.0	1.0	0.0	3.9	2.1	1.8
27	3.3	3.2	0.1	5.0	3.9	1.1
28	1.0	1.0	0.0	3.6	2.9	0.7
29	3.3	3.3	0.0	5.0	2.2	2.8
MEAN	1.80	1.75	0.05	3.96	2.54	1.42
SD	1.185	1.117	0.131	0.735	0.675	0.734
MIN	1.0	1.0	0.0	2.1	1.4	0.6
MAX	4.8	4.2	0.6	5.0	3.9	2.8

TABLE 12

## JOINT STIFFNESS BEFORE AND AFTER SURGERY

PAT	UPPER LIMBS			LOWER LIMBS		
	PREOP	POSTOP	DIF	PREOP	POSTOP	DIF
1	1.00	1.00	0.00	3.50	2.90	0.60
2	1.00	1.00	0.00	1.30	1.15	0.15
3	1.00	1.00	0.00	2.05	1.10	0.95
4	1.00	1.00	0.00	1.30	1.15	0.15
5	1.00	1.00	0.00	1.60	1.10	0.50
6	1.00	1.00	0.00	1.10	1.00	0.10
7	1.00	1.00	0.00	3.00	1.14	1.86
8	1.00	1.00	0.00	2.00	1.65	0.35
10	1.00	1.00	0.00	1.90	1.00	0.90
12	1.00	1.00	0.00	1.30	1.20	0.10
13	1.00	1.00	0.00	2.45	1.10	1.35
14	1.27	1.27	0.00	1.95	1.25	0.70
15	1.33	1.13	0.20	2.00	1.50	0.50
16	1.00	1.00	0.00	1.25	1.00	0.25
17	1.00	1.00	0.00	1.60	1.05	0.55
23	1.00	1.00	0.00	1.90	1.30	0.60
25	1.00	1.00	0.00	1.75	1.75	0.00
28	1.00	1.00	0.00	1.85	1.50	0.35
29	1.00	1.00	0.00	1.00	1.00	0.00
MEAN	1.04	1.02	0.01	1.74	1.22	0.52
SD	0.094	0.067	0.046	0.486	0.225	0.480
MIN	1.0	1.0	0.0	1.0	1.0	0.0
MAX	1.3	1.3	0.2	3.0	1.8	1.860

TABLE 13A

JOINT STIFFNESS OF LOWER LIMBS  
BEFORE AND AFTER SURGERY

PATIENT	1		2		3		4	
	PRE	POST	PRE	POST	PRE	POST	PRE	POST
HIP FLEXION	4.0	3.0	1.0	1.0	4.0	1.0	2.0	2.0
HIP EXTENSION	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
KNEE EXTENSION	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
KNEE FLEXION	3.5	2.5	4.0	2.5	4.0	2.0	3.0	1.5
HIP ABDUCTION	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
HIP ADDUCTION	1.0	1.0	1.0	1.0	4.0	1.0	1.0	1.0
PLANTARFLEXION	3.0	2.0	1.0	1.0	2.5	1.0	1.0	1.0
DORSIFLEXION	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
INVERSION	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
EVERSION	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
MEAN	1.8	1.5	1.3	1.2	2.1	1.1	1.3	1.2
MAX	4.0	3.0	4.0	2.5	4.0	2.0	3.0	2.0
MIN	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
SD	1.17	0.72	0.90	0.45	1.35	0.30	0.64	0.32

TABLE 13B

JOINT STIFFNESS OF LOWER LIMBS  
BEFORE AND AFTER SURGERY

PATIENT	5		6		7		8	
	PRE	POST	PRE	POST	PRE	POST	PRE	POST
HIP FLEXION	3.0	1.0	2.0	1.0	4.0	1.0	3.0	2.0
HIP EXTENSION	1.0	1.0	1.0	1.0	3.0	1.0	1.0	1.0
KNEE EXTENSION	1.0	1.0	1.0	1.0	4.0	1.0	1.0	1.0
KNEE FLEXION	3.0	2.0	1.0	1.0	4.0	4.0	4.0	2.5
HIP ABDUCTION	1.0	1.0	1.0	1.0	2.0	1.0	1.0	1.0
HIP ADDUCTION	3.0	1.0	1.0	1.0	3.0	1.0	4.0	4.0
PLANTARFLEXION	1.0	1.0	1.0	1.0	2.0	1.0	1.0	1.0
DORSIFLEXION	1.0	1.0	1.0	1.0	3.0	1.0	1.0	1.0
INVERSION	1.0	1.0	1.0	1.0	3.0	1.0	1.0	1.0
EVERSION	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
MEAN	1.6	1.1	1.1	1.0	2.9	1.3	1.8	1.6
MAX	3.0	2.0	2.0	1.0	4.0	4.0	4.0	4.0
MIN	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
SD	0.92	0.30	0.30	0.00	0.94	0.96	1.25	0.96

TABLE 13C

JOINT STIFFNESS OF LOWER LIMBS  
BEFORE AND AFTER SURGERY

PATIENT	9		10		11		12	
	PRE	POST	PRE	POST	PRE	POST	PRE	POST
HIP FLEXION	4.0	4.0	4.0	1.0	3.0	2.0	1.0	1.0
HIP EXTENSION	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
KNEE EXTENSION	1.0	1.0	1.0	1.0	2.0	1.0	1.0	1.0
KNEE FLEXION	3.0	2.0	3.0	1.0	2.0	1.5	4.0	3.0
HIP ABDUCTION	1.0	1.0	1.0	1.0	2.0	1.0	1.0	1.0
HIP ADDUCTION	4.0	4.0	4.0	1.0	3.0	1.0	1.0	1.0
PLANTARFLEXION	3.0	1.0	2.0	1.0	2.5	1.5	1.0	1.0
DORSIFLEXION	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
INVERSION	1.0	1.0	1.0	1.0	2.5	1.0	1.0	1.0
EVERSION	1.0	1.0	1.0	1.0	2.5	1.0	1.0	1.0
MEAN	2.0	1.7	1.9	1.0	2.3	1.2	1.3	1.2
MAX	4.0	4.0	4.0	1.0	3.0	2.0	4.0	3.0
MIN	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
SD	1.23	1.19	1.22	0.00	0.56	0.33	0.90	0.60

TABLE 13D

JOINT STIFFNESS OF LOWER LIMBS  
BEFORE AND AFTER SURGERY

PATIENT	13		14		15		16	
	PRE	POST	PRE	POST	PRE	POST	PRE	POST
HIP FLEXION	4.0	1.0	4.0	1.0	4.0	1.0	1.0	1.0
HIP EXTENSION	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
KNEE EXTENSION	1.0	1.0	1.0	1.0	3.0	1.0	1.0	1.0
KNEE FLEXION	4.0	2.0	4.0	1.0	4.0	3.0	1.0	1.0
HIP ABDUCTION	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
HIP ADDUCTION	3.0	1.0	1.0	1.0	3.0	4.0	1.0	1.0
PLANTARFLEXION	3.5	1.0	4.5	3.5	1.0	1.0	3.5	1.0
DORSIFLEXION	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
INVERSION	3.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
EVERSION	3.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
MEAN	2.5	1.1	2.0	1.3	2.0	1.5	1.3	1.0
MAX	4.0	2.0	4.5	3.5	4.0	3.0	3.5	1.0
MIN	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
SD	1.23	0.30	1.46	0.75	1.27	1.03	0.75	0.00

TABLE 13E

JOINT STIFFNESS OF LOWER LIMBS  
BEFORE AND AFTER SURGERY

PATIENT	17		18		19		20	
	PRE	POST	PRE	POST	PRE	POST	PRE	POST
HIP FLEXION	4.0	1.0	1.0	1.0	4.0	2.0	4.0	4.0
HIP EXTENSION	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
KNEE EXTENSION	1.0	1.0	2.0	1.0	1.0	1.0	1.0	1.0
KNEE FLEXION	4.0	1.5	2.0	1.0	4.0	1.5	4.0	4.0
HIP ABDUCTION	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
HIP ADDUCTION	1.0	1.0	1.0	1.0	3.0	1.0	4.0	4.0
PLANTARFLEXION	1.0	1.0	3.5	2.0	1.0	1.0	3.0	3.0
DORSIFLEXION	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
INVERSION	1.0	1.0	3.0	1.0	2.0	1.0	1.0	1.0
EVERSION	1.0	1.0	3.0	1.0	2.0	1.0	1.0	1.0
MEAN	1.6	1.1	1.9	1.1	2.0	1.2	2.1	2.1
MAX	4.0	1.5	3.5	2.0	4.0	2.0	4.0	4.0
MIN	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
SD	1.20	0.15	0.95	0.30	1.18	0.32	1.38	1.38

TABLE 13F

JOINT STIFFNESS OF LOWER LIMBS  
BEFORE AND AFTER SURGERY

PATIENT	21		22		23		24	
	PRE	POST	PRE	POST	PRE	POST	PRE	POST
HIP FLEXION	4.0	4.0	4.0	1.0	4.0	1.0	2.0	1.0
HIP EXTENSION	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
KNEE EXTENSION	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
KNEE FLEXION	4.0	1.0	4.0	4.0	4.0	4.0	4.0	2.0
HIP ABDUCTION	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
HIP ADDUCTION	1.0	1.0	4.0	1.0	4.0	1.0	2.0	1.0
PLANTARFLEXION	1.0	1.0	1.0	1.0	1.0	1.0	3.0	1.0
DORSIFLEXION	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
INVERSION	1.0	1.0	1.0	1.0	1.0	1.0	3.0	1.0
EVERSION	1.0	1.0	1.0	1.0	1.0	1.0	3.0	1.0
MEAN	1.6	1.3	1.9	1.3	1.9	1.3	2.1	1.1
MAX	4.0	4.0	4.0	4.0	4.0	4.0	4.0	2.0
MIN	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
SD	1.20	0.90	1.38	1.30	1.38	1.30	1.04	1.10

TABLE 13G

JOINT STIFFNESS OF LOWER LIMBS  
BEFORE AND AFTER SURGERY

PATIENT	25		26		27		28	
	PRE	POST	PRE	POST	PRE	POST	PRE	POST
HIP FLEXION	1.0	1.0	4.0	4.0	4.0	1.0	3.5	1.0
HIP EXTENSION	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
KNEE EXTENSION	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
KNEE FLEXION	1.0	1.0	4.0	4.0	4.0	4.0	4.0	4.0
HIP ABDUCTION	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
HIP ADDUCTION	1.0	1.0	4.0	4.0	1.0	1.0	1.0	1.0
PLANTARFLEXION	3.5	3.5	1.0	1.0	1.0	1.0	4.0	3.0
DORSIFLEXION	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
INVERSION	3.5	3.5	1.0	1.0	1.0	1.0	1.0	1.0
EVERSION	3.5	3.5	1.0	1.0	1.0	1.0	1.0	1.0
MEAN	1.8	1.8	1.9	1.9	1.6	1.3	1.9	1.5
MAX	3.5	3.5	4.0	4.0	4.0	4.0	4.0	4.0
MIN	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
SD	1.15	1.15	1.38	1.38	1.20	0.90	1.31	1.03

TABLE 13H

JOINT STIFFNESS OF LOWER LIMBS  
BEFORE AND AFTER SURGERY

PATIENT	29	
	PRE	POST
HIP FLEXION	1.0	1.0
HIP EXTENSION	1.0	1.0
KNEE EXTENSION	1.0	1.0
KNEE FLEXION	1.0	1.0
HIP ABDUCTION	1.0	1.0
HIP ADDUCTION	1.0	1.0
PLANTARFLEXION	1.0	1.0
DORSIFLEXION	1.0	1.0
INVERSION	1.0	1.0
EVERSION	1.0	1.0
MEAN	1.0	1.0
MAX	1.0	1.0
MIN	1.0	1.0
SD	0.00	0.00

TABLE 131

JOINT STIFFNESS OF LOWER LIMBS BEFORE AND AFTER SURGERY  
FOR ALL PATIENTS AND ALL JOINTS

	PRE	POST
HIP FLEXION	1.1	1.0
HIP EXTENSION	3.1	1.6
KNEE EXTENSION	3.3	2.3
KNEE FLEXION	1.2	1.0
HIP ABDUCTION	2.3	1.5
HIP ADDUCTION	1.1	1.0
PLANTARFLEXION	1.1	1.0
DORSIFLEXION	2.0	1.4
INVERSION	1.4	1.1
EVERSION	1.5	1.1
MEAN	1.82	1.30

TABLE 14

ROLLING LEFT AND RIGHT  
BEFORE AND AFTER SURGERY

PAT	ROLLING TO RIGHT			ROLLING TO LEFT		
	PREOP	POSTOP	DIF	PREOP	POSTOP	DIF
1	4	3	1	4	3	1
2	4	3	1	4	3	1
3	4	2	2	4	2	2
4	1	1	0	1	1	0
5	3	3	0	3	3	0
6	4	2	2	4	2	2
7	4	3	1	4	3	1
8	4	2	2	4	2	2
9	4	3	1	4	3	1
10	4	3	1	4	3	1
11	3	2	1	3	2	1
12	3	3	0	3	3	0
13	3	3	0	3	3	0
14	4	3	1	4	3	1
15	5	5	0	5	5	0
16	5	5	0	5	5	0
17	4	1	3	4	1	3
18	3	2	1	3	2	1
19	4	1	3	4	1	3
20	4	3	1	4	3	1
21	4	2	2	4	2	2
22	4	3	1	4	3	1
23	5	2	3	5	2	3
24	2	2	0	2	2	0
25	5	5	0	5	5	0
26	4	3	1	4	2	2
27	4	3	1	4	3	1
28	3	3	0	3	3	0
29	5	3	2	4	3	2
MEAN	3.79	2.83	0.97	3.66	2.69	1.00
SD	0.886	0.985	0.890	0.992	1.021	0.910
MIN	1	1	0.0	1	1	0.0
MAX	5	5	3	5	5	3

TABLE 15

## LONG SITTING BEFORE AND AFTER SURGERY

PAT	PREOP	POSTOP	DIF
1	3	2	1
2	5	3	2
3	3	2	1
4	2	1	1
5	4	2	2
6	3	1	2
7	5	3	2
8	4	1	3
9	3	2	1
10	4	2	2
11	3	2	1
12	4	3	1
13	5	5	0
14	3	2	1
15	5	4	1
16	5	4	1
17	4	2	2
18	2	2	0
19	3	2	1
20	5	3	2
21	4	2	2
22	4	3	1
23	5	4	1
24	3	2	1
25	5	5	0
26	3	2	1
27	5	4	1
28	4	3	1
29	5	4	1
MEAN	3.90	2.66	1.26
SD	0.959	1.092	0.677
MIN	2	1	0
MAX	5	5	3

TABLE 16

SIDE SITTING LEFT AND RIGHT  
BEFORE AND AFTER SURGERY

PAT	SIDE SITTING TO RIGHT			SIDE SITTING TO LEFT		
	PREOP	POSTOP	DIF	PREOP	POSTOP	DIF
1	4	2	2	4	2	2
2	5	3	2	3	2	1
3	2	1	1	3	2	1
4	3	2	1	2	2	0
5	3	2	1	3	2	1
6	2	1	1	3	2	1
7	4	2	2	4	2	2
8	3	2	1	2	2	0
9	3	2	1	3	3	0
10	4	2	2	4	2	2
11	2	2	0	2	2	0
12	4	4	0	4	4	0
13	5	5	0	5	5	0
14	3	3	0	3	3	0
15	5	5	0	5	5	0
16	5	4	1	5	4	1
17	2	2	0	3	2	0
18	2	2	0	3	3	0
19	3	2	1	3	2	1
20	4	2	1	4	2	1
21	3	2	1	3	2	1
22	3	3	0	3	3	0
23	5	4	1	5	4	1
24	3	2	1	2	2	0
25	5	5	0	5	5	0
26	2	2	0	3	2	1
27	5	3	2	5	3	2
28	2	2	0	3	3	0
29	5	4	2	4	4	2
MEAN	3.48	2.66	0.83	3.48	2.79	0.69
SD	1.133	1.123	0.746	0.969	1.030	0.748
MIN	2	1	0	2	2	0
MAX	5	5	2	5	5	2

TABLE 17

## PRONE KNEELING BEFORE AND AFTER SURGERY

PAT	PREOP	POSTOP	DIF
1	3	3	0
2	4	3	1
3	2	1	1
4	1	1	0
5	3	2	1
6	2	1	1
7	3	3	0
8	3	2	1
9	2	1	1
10	4	2	2
11	1	1	0
12	5	5	0
13	5	5	0
14	3	2	1
15	5	5	0
16	5	5	0
17	3	1	2
18	2	1	1
19	3	2	1
20	3	1	2
21	3	1	2
22	3	3	0
23	5	5	0
24	1	1	0
25	5	5	0
26	3	1	2
27	5	5	0
28	3	1	2
29	5	5	2
MEAN	3.28	2.55	0.73
SD	1.284	1.652	0.804
MIN	1	1	0.0
MAX	5	5	2

TABLE 18

## KNEEL STANDING BEFORE AND AFTER SURGERY

PAT	PREOP	POSTOP	DIF
1	4	3	1
2	4	3	1
3	4	1	3
4	2	2	0
5	3	2	1
6	3	1	2
7	5	3	2
8	3	2	1
9	3	2	1
10	4	3	1
11	2	1	1
12	5	5	0
13	5	5	0
14	4	2	2
15	5	5	0
16	5	5	0
17	4	2	2
18	3	2	1
19	3	2	1
20	3	1	2
21	3	1	2
22	3	2	1
23	5	4	1
24	2	1	1
25	5	5	0
26	3	1	2
27	5	5	0
28	3	2	1
29	5	5	2
MEAN	3.72	2.69	1.03
SD	1.014	1.488	0.803
MIN	2	1	0.0
MAX	5	5	3

TABLE 19  
 HALF KNEELING LEFT AND RIGHT  
 BEFORE AND AFTER SURGERY

PAT	HALF KNEELING RIGHT			HALF KNEELING LEFT		
	PREOP	POSTOP	DIF	PREOP	POSTOP	DIF
1	5	3	2	5	3	2
2	4	3	1	4	3	1
3	4	2	2	4	2	2
4	1	1	0	1	1	0
5	4	3	1	4	3	1
6	4	1	3	4	2	2
7	5	4	1	5	4	1
8	5	2	3	5	2	3
9	4	2	2	4	2	2
10	5	3	2	5	3	2
11	3	2	1	2	1	1
12	5	3	2	5	3	2
13	5	4	1	5	4	1
14	4	2	0	4	2	0
15	5	5	0	5	5	0
16	5	4	1	5	4	1
17	4	3	1	4	3	0
18	4	2	2	4	2	2
19	2	1	1	2	1	1
20	5	2	1	5	2	1
21	5	2	3	4	2	2
22	5	3	2	5	3	2
23	5	4	1	5	4	1
24	2	1	1	2	2	0
25	5	5	0	5	5	0
26	5	2	3	5	2	3
27	5	5	0	5	5	0
28	4	2	2	4	2	2
29	5	4	2	5	4	2
MEAN	4.28	2.76	1.48	4.21	2.79	1.34
SD	1.047	1.194	0.895	1.095	1.156	0.882
MIN	1	1	0.0	1	1	0.0
MAX	5	5	3	5	5	3

TABLE 20

## CRAWLING BEFORE AND AFTER SURGERY

PAT	PREOP	POSTOP	DIF
1	4	2	2
2	4	3	1
3	2	2	0
4	1	1	0
5	3	2	1
6	2	1	1
7	5	2	3
8	3	2	1
9	3	2	1
10	4	3	1
11	2	1	1
12	5	5	0
13	5	5	0
14	2	2	0
15	5	5	0
16	5	5	0
17	3	2	1
18	2	2	0
19	2	2	0
20	3	2	1
21	3	1	2
22	3	3	0
23	5	5	0
24	2	2	0
25	5	5	0
26	2	2	0
27	5	5	0
28	2	2	0
29	5	5	0
MEAN	3.34	2.79	0.62
SD	1.294	1.447	0.806
MIN	1	1	0
MAX	5	5	3

TABLE 21

## STANDING BEFORE AND AFTER SURGERY

PAT	PREOP	POSTOP	DIF
1	4	4	0
2	5	4	1
3	3	2	1
4	2	2	0
5	3	3	0
6	2	2	0
7	5	4	1
8	3	2	1
9	3	2	1
10	5	5	0
11	2	2	0
12	5	5	0
13	5	5	0
14	4	2	2
15	5	5	0
16	5	5	0
17	3	2	1
18	4	2	2
19	2	1	1
20	4	3	1
21	2	1	1
22	4	3	1
23	5	5	0
24	2	1	1
25	5	5	0
26	2	2	0
27	5	5	0
28	3	2	1
29	5	5	2
MEAN	3.69	3.14	0.55
SD	1.206	1.456	0.665
MIN	2	1	0
MAX	5	5	2

TABLE 22

## WALKING BEFORE AND AFTER SURGERY

PAT	PREOP	POSTOP	DIF
1	4	4	0
2	5	4	1
3	3	3	0
4	1	1	0
5	4	3	1
6	3	2	1
7	5	4	1
8	4	3	1
9	4	2	2
10	5	5	0
11	2	2	0
12	5	5	0
13	5	5	0
14	3	2	1
15	5	5	0
16	5	5	0
17	4	2	2
18	4	2	2
19	2	2	0
20	4	3	1
21	3	2	1
22	4	3	1
23	5	5	0
24	2	1	1
25	5	5	0
26	3	2	1
27	5	5	0
28	4	3	1
29	5	5	0
MEAN	3.90	3.28	0.62
SD	1.125	1.362	0.665
MIN	1	1	0
MAX	5	5	2

APPENDIX C

TABLE 23A

ASSESSMENT FOR SELECTIVE POSTERIOR LUMBAR RHIZOTOMY

NAME \_\_\_\_\_

ADDRESS \_\_\_\_\_

\_\_\_\_\_

DATE OF BIRTH \_\_\_\_\_

DATE OF ASSESSMENT \_\_\_\_\_

DATES OF REASSESSMENT \_\_\_\_\_

\_\_\_\_\_

I.Q. \_\_\_\_\_

MOTIVATION \_\_\_\_\_

PREVIOUS ORTHOPAEDIC  
SURGERY \_\_\_\_\_

\_\_\_\_\_

HIP X-RAYS \_\_\_\_\_

TABLE 23B

ASSESSMENT FORM

FUNCTIONS

	PREOP		POSTOP	
	R	L	R	L
ROLLING				
LONG SITTING				
SIDE SITTING				
PRONE KNEELING				
KNEEL STANDING				
HALF KNEELING				
CRAWLING				
STANDING				
WALKING				

TABLE 23C  
ASSESSMENT FORM  
UPPER LIMBS

	MUSCLE TONE				VOL MOVE				JOINT STIFF			
	PRE		POST		PRE		POST		PRE		POST	
	R	L	R	L	R	L	R	L	R	L	R	L
SHOULDER ABD FIRST 90 DEG												
SHOULDER ABD SECOND 90 DEG												
SHOULDER EXT ROTATION												
SHOULDER INT ROTATION												
ELBOW FLEXION (FOREARM SUP)												
ELBOW FLEXION (HALF SUP)												
ELBOW EXTENSION												
FOREARM SUPINATION												
FOREARM PRONATION												
WRIST FLEXION												
FINGER EXTENSION												
FINGER FLEXION												

TABLE 23D  
ASSESSMENT FORM  
LOWER LIMBS

	MUSCLE TONE				VOL. MOVE.				JOINT STIFF.				
	PRE		POST		PRE		POST		PRE		POST		
	R	L	R	L	R	L	R	L	R	L	R	L	
HIP FLEXION													
HIP EXTENSION													
KNEE EXTENSION													
KNEE FLEXION													
HIP ABDUCTION													
HIP ADDUCTION													
PLANTAR FLEXION													
DORSIFLEXION													
INVERSION													
EVERSION													

## APPENDIX D

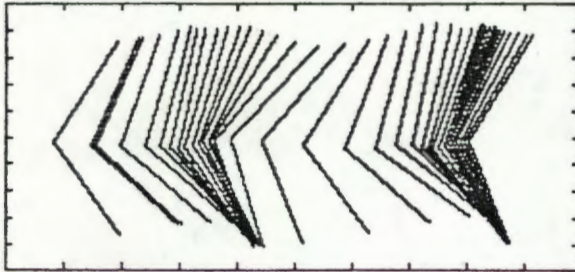
The following figures (Figs. 36-50) contain the stick figures for crawling and walking before and after surgery

FIGURE 36

Patient 1

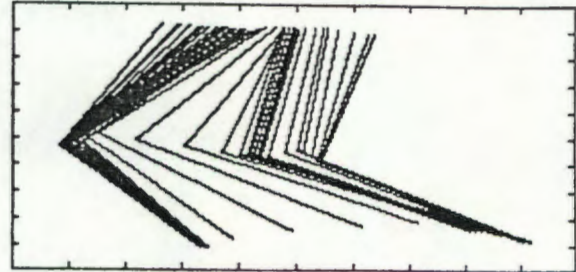
STICK FIGURE

BOR\*LWB  
14 AUG 85 PRE-OP  
LEFT WALK WITH WALKER  
X RANGE = 1676 MM Y RANGE = 865 MM



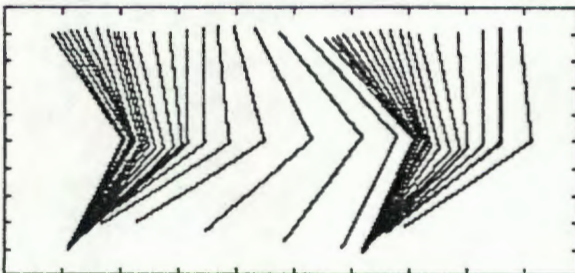
STICK FIGURE

BOR\*LWA  
16 JUN 86 POST-OP  
LEFT WALK WITH FRAME AND PHYSIO  
X RANGE = 785 MM Y RANGE = 838 MM



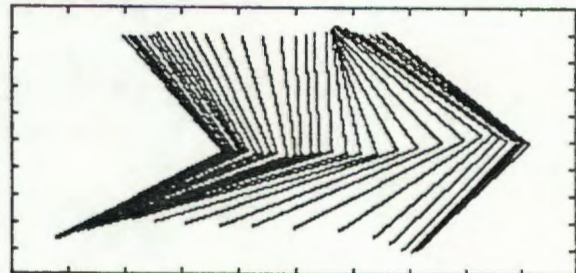
STICK FIGURE

BOR\*RWB  
14 AUG 85 PRE-OP  
RIGHT WALK WITH WALKER  
X RANGE = 1191 MM Y RANGE = 855 MM



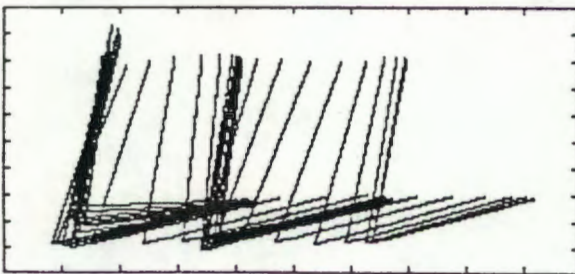
STICK FIGURE

BOR\*RWA  
16 JUN 86 POST-OP  
RIGHT WALK WITH FRAME AND PHYSIO  
X RANGE = 785 MM Y RANGE = 824 MM



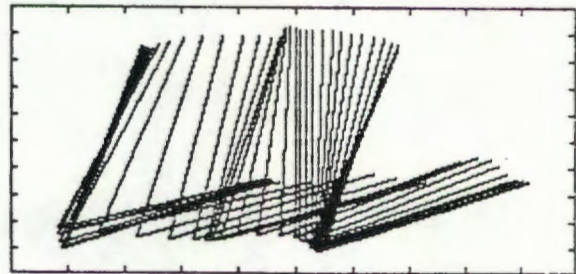
STICK FIGURE

BOR\*LCB  
14 AUG 85 PRE-OP  
LEFT CRAWL  
X RANGE = 1050 MM Y RANGE = 519 MM



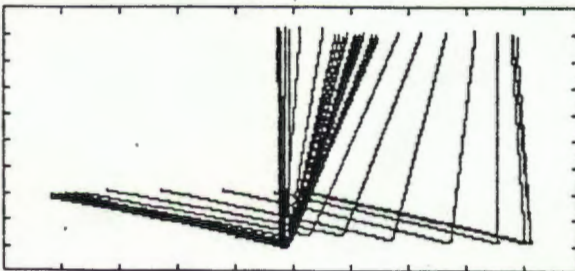
STICK FIGURE

BOR\*LCA  
16 JUN 86 POST-OP  
LEFT CRAWL  
X RANGE = 912 MM Y RANGE = 513 MM



STICK FIGURE

BOR\*RCB  
14 AUG 85 PRE-OP  
RIGHT CRAWL  
X RANGE = 892 MM Y RANGE = 484 MM



STICK FIGURE

BOR\*RCA  
16 JUN 86 POST-OP  
RIGHT CRAWL  
X RANGE = 856 MM Y RANGE = 472 MM

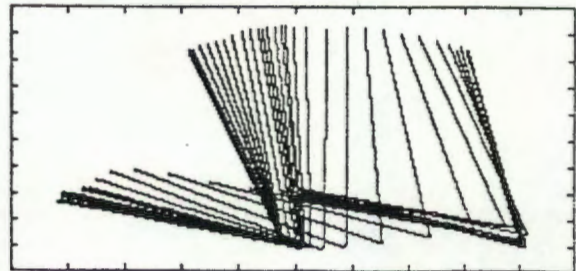
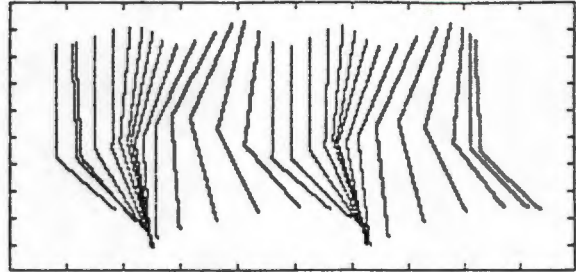


FIGURE 37

Patient 3

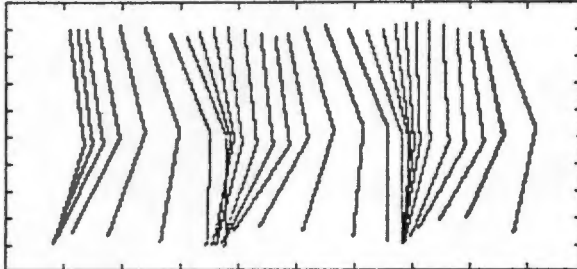
STICK FIGURE

SMI\*LWA  
06 JUN 86 POST-OP  
LEFT WALK  
X RANGE = 2503 MM Y RANGE = 520 MM



STICK FIGURE

SMI\*EWE  
08 AUG 85 PRE-OP  
RIGHT WALK  
X RANGE = 1998 MM Y RANGE = 622 MM



STICK FIGURE

SMI\*RWA  
06 JUN 86 POST-OP  
RIGHT WALK  
X RANGE = 2613 MM Y RANGE = 507 MM

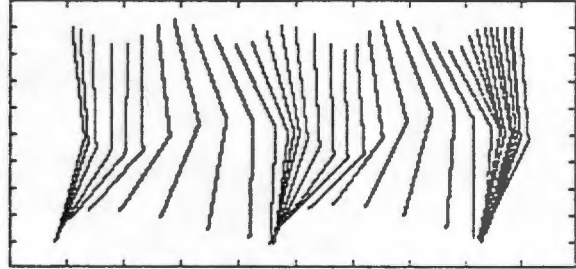
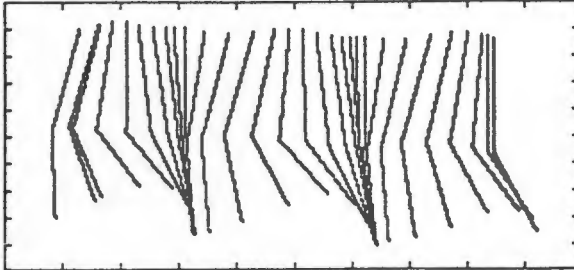


FIGURE 38

Patient 4

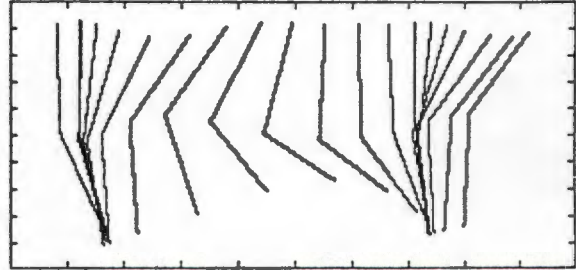
STICK FIGURE

SON\*LWE  
22 OCT 88 PRE-OP  
LEFT WALK  
X RANGE = 1737 MM Y RANGE = 493 MM



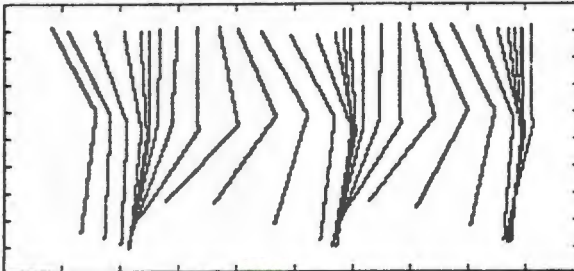
STICK FIGURE

SON\*LWA  
28 MAY 88 POST-OP  
LEFT WALK  
X RANGE = 1666 MM Y RANGE = 472 MM



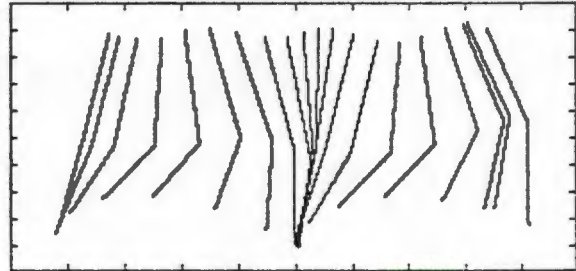
STICK FIGURE

SON\*RWE  
22 OCT 88 PRE-OP  
RIGHT WALK  
X RANGE = 1344 MM Y RANGE = 447 MM



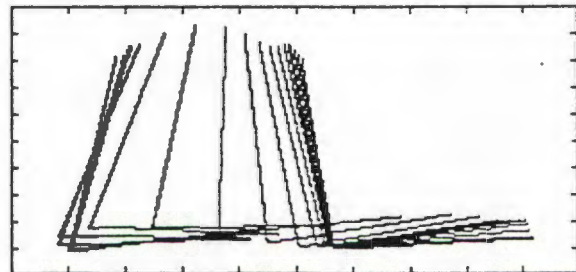
STICK FIGURE

SON\*RWA  
28 MAY 88 POST-OP  
RIGHT WALK  
X RANGE = 2065 MM Y RANGE = 483 MM



STICK FIGURE

SON\*LCA  
28 MAY 88 POST-OP  
LEFT CRAWL  
X RANGE = 784 MM Y RANGE = 265 MM



STICK FIGURE

SON\*RCA  
28 MAY 88 POST-OP  
RIGHT CRAWL  
X RANGE = 1035 MM Y RANGE = 258 MM

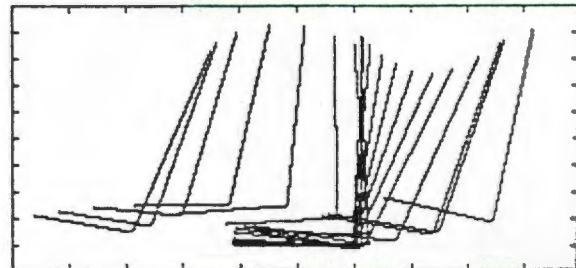
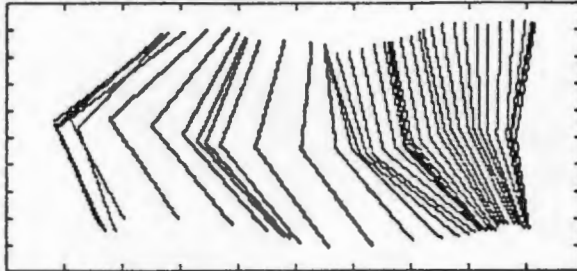


FIGURE 39

Patient 5

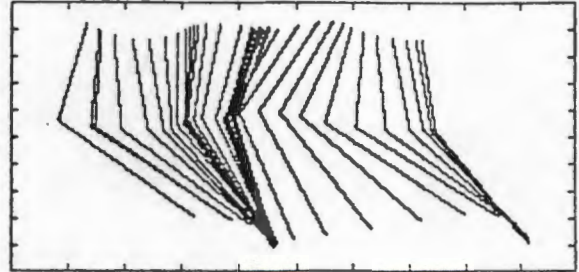
STICK FIGURE

MAR\*LWB  
30 OCT 85 PRE-OP  
LEFT WALK  
X RANGE = 522 MM Y RANGE = 447 MM



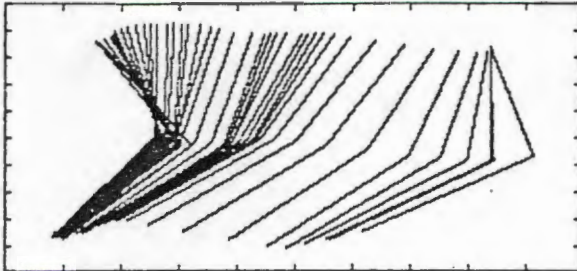
STICK FIGURE

MAR\*LWA  
18 JUN 86 POST-OP  
LEFT WALK  
X RANGE = 826 MM Y RANGE = 445 MM



STICK FIGURE

MAR\*RMB  
30 OCT 85 PRE-OP  
RIGHT WALK  
X RANGE = 502 MM Y RANGE = 435 MM



STICK FIGURE

MAR\*RWA  
18 JUN 86 POST-OP  
RIGHT WALK  
X RANGE = 840 MM Y RANGE = 434 MM

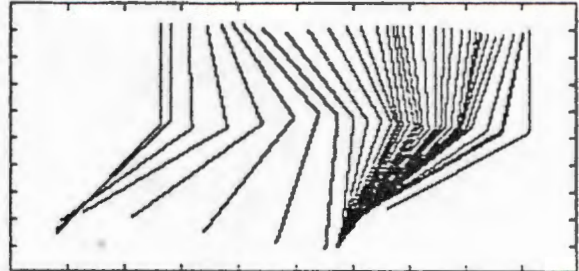
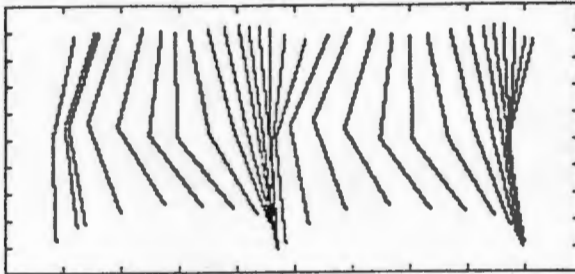


FIGURE 40

Patient 6

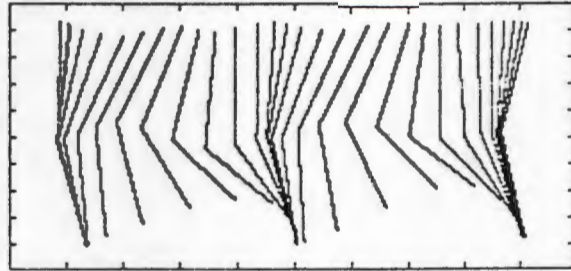
STICK FIGURE

JOR\*LWB  
10 APR 85 PRE-OP  
LEFT WALK  
X RANGE = 1970 MM Y RANGE = 574 MM



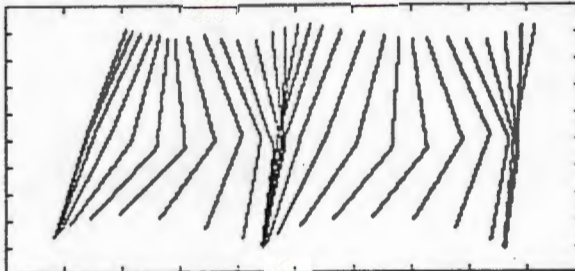
STICK FIGURE

JOR\*LWA  
06 JUN 86 POST-OP  
LEFT WALK  
X RANGE = 1838 MM Y RANGE = 439 MM



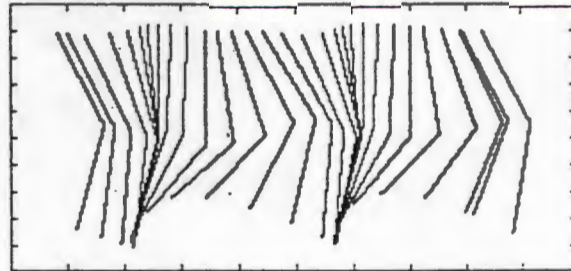
STICK FIGURE

JOR\*RWB  
10 APR 85 PRE-OP  
RIGHT WALK  
X RANGE = 1494 MM Y RANGE = 536 MM



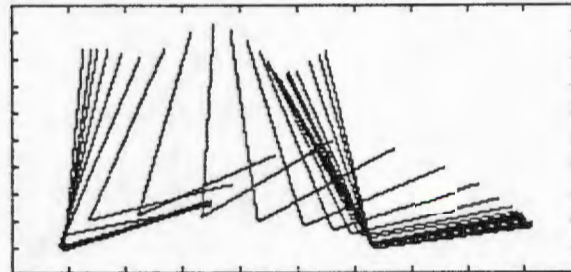
STICK FIGURE

JOR\*RWA  
06 JUN 86 POST-OP  
RIGHT WALK  
X RANGE = 1934 MM Y RANGE = 439 MM



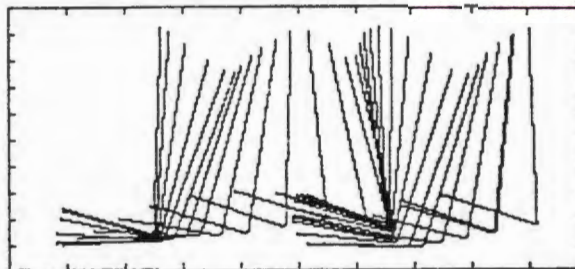
STICK FIGURE

JOR\*LCA  
06 JUN 86 POST-OP  
LEFT CRAWL  
X RANGE = 984 MM Y RANGE = 249 MM



STICK FIGURE

JOR\*RCA  
10 APR 85 PRE-OP  
RIGHT CRAWL  
X RANGE = 1484 MM Y RANGE = 313 MM



STICK FIGURE

JOR\*RCA  
06 JUN 86 POST-OP  
RIGHT CRAWL  
X RANGE = 787 MM Y RANGE = 250 MM

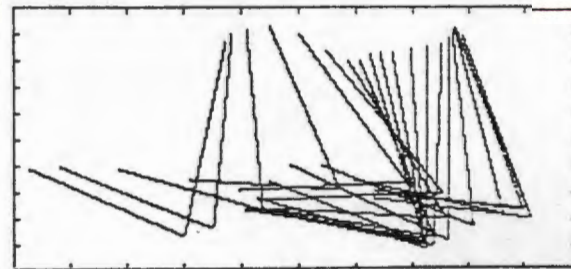
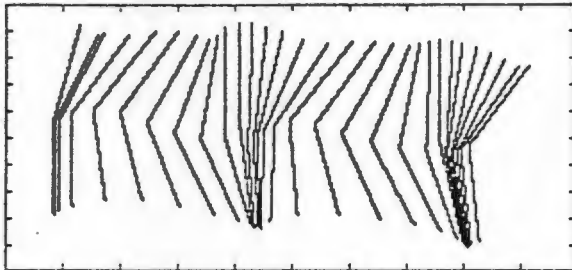


FIGURE 41

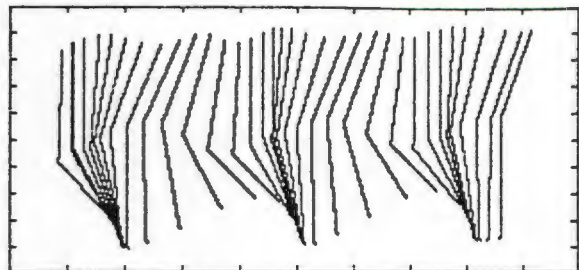
STICK FIGURE Patient 8

WIL\*LWB  
23 APR 85 PRE-OP  
LEFT WALK  
X RANGE = 1498 MM Y RANGE = 666 MM



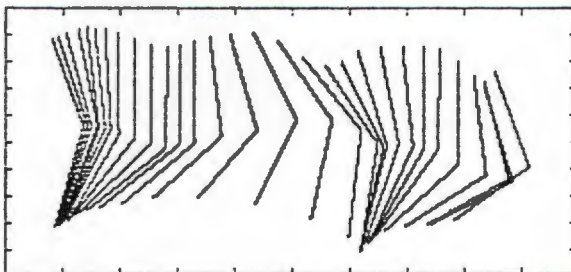
STICK FIGURE

WIL\*LWA  
06 JUN 86 POST-OP  
LEFT WALK  
X RANGE = 2613 MM Y RANGE = 416 MM



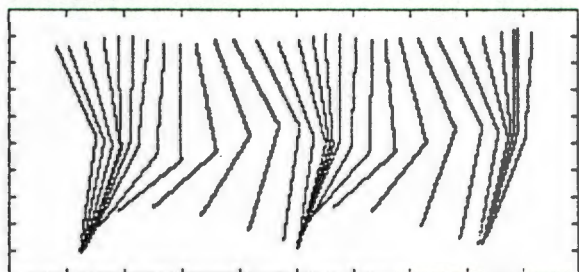
STICK FIGURE

WIL\*RWB  
23 APR 85 PRE-OP  
RIGHT WALK  
X RANGE = 1546 MM Y RANGE = 619 MM



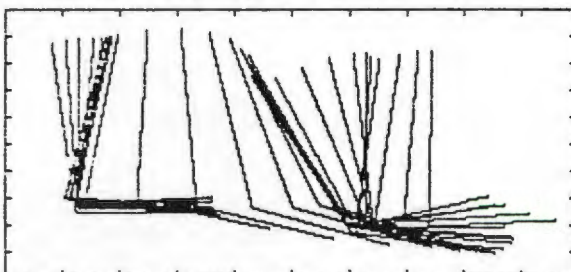
STICK FIGURE

WIL\*RWA  
06 JUN 86 POST-OP  
RIGHT WALK  
X RANGE = 2124 MM Y RANGE = 446 MM



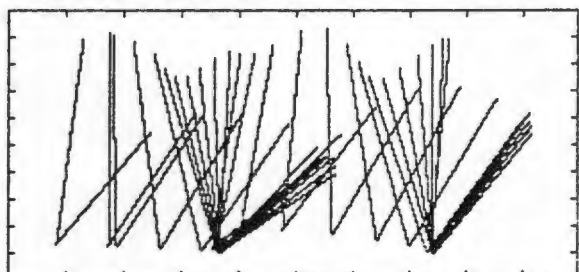
STICK FIGURE

WIL\*LCB  
23 APR 85 PRE-OP  
LEFT CRAWL  
X RANGE = 982 MM Y RANGE = 378 MM



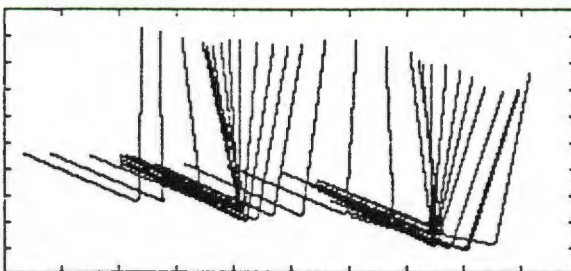
STICK FIGURE

WIL\*LCA  
06 JUN 86 POST-OP  
LEFT CRAWL  
X RANGE = 1148 MM Y RANGE = 234 MM



STICK FIGURE

WIL\*RCB  
23 APR 85 PRE-OP  
RIGHT CRAWL  
X RANGE = 1119 MM Y RANGE = 328 MM



WIL\*RCA  
06 JUN 86 POST-OP  
RIGHT CRAWL  
X RANGE = 1057 MM Y RANGE = 237 MM

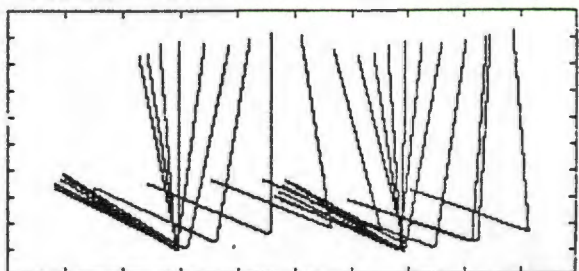
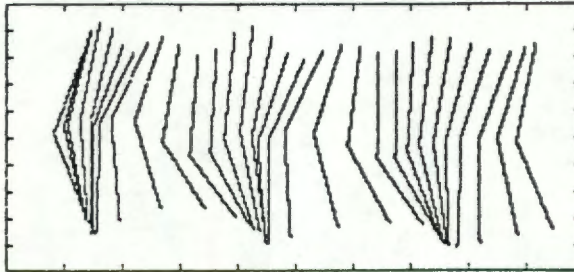


FIGURE 42

Patient 9

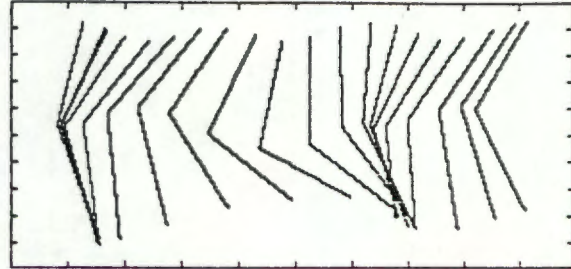
STICK FIGURE

CAL\*LWB  
12 NOV 85 PRE-OP  
LEFT WALK  
X RANGE = 3141 MM Y RANGE = 734 MM



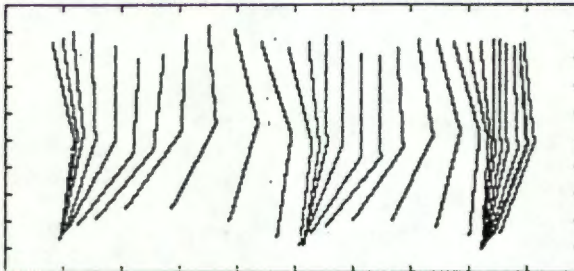
STICK FIGURE

CAL\*LWA  
28 MAY 86 POST-OP  
LEFT WALK  
X RANGE = 2370 MM Y RANGE = 672 MM



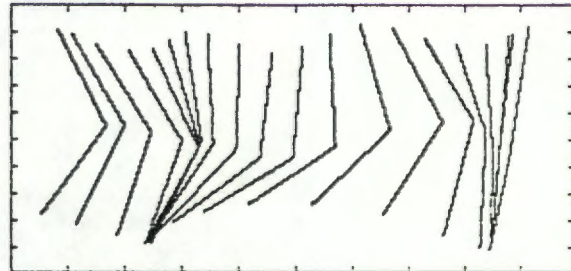
STICK FIGURE

CAL\*REB  
12 NOV 85 PRE-OP  
RIGHT WALK  
X RANGE = 2634 MM Y RANGE = 812 MM



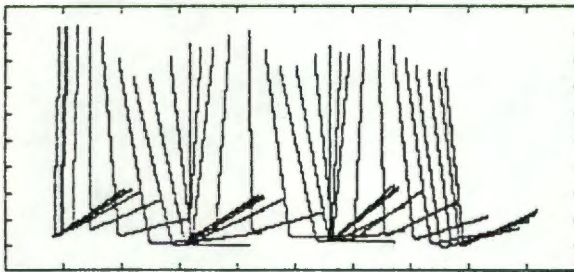
STICK FIGURE

CAL\*RNA  
28 MAY 86 POST-OP  
RIGHT WALK  
X RANGE = 2116 MM Y RANGE = 679 MM



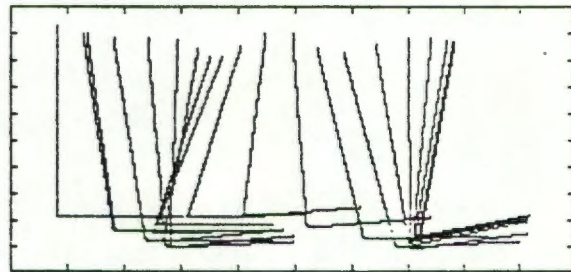
STICK FIGURE

CAL\*LCE  
12 NOV 85 PRE-OP  
LEFT CRAWL  
X RANGE = 2488 MM Y RANGE = 369 MM



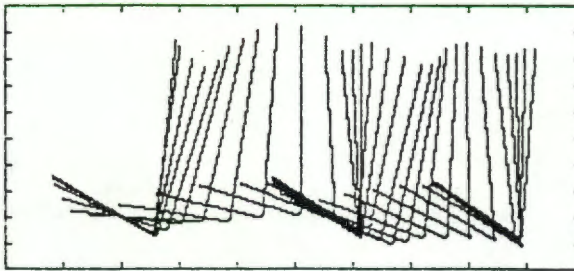
STICK FIGURE

CAL\*LCA  
28 MAY 86 POST-OP  
LEFT CRAWL  
X RANGE = 1759 MM Y RANGE = 343 MM



STICK FIGURE

CAL\*RCB  
12 NOV 85 PRE-OP  
RIGHT CRAWL  
X RANGE = 1654 MM Y RANGE = 376 MM



STICK FIGURE

CAL\*RCA  
28 MAY 86 POST-OP  
RIGHT CRAWL  
X RANGE = 1626 MM Y RANGE = 392 MM

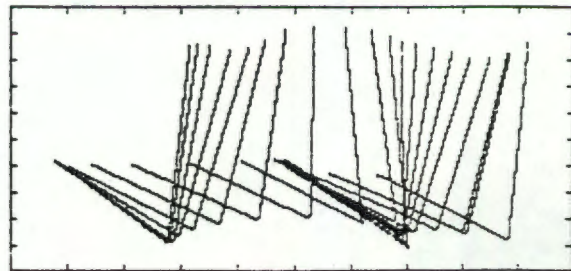
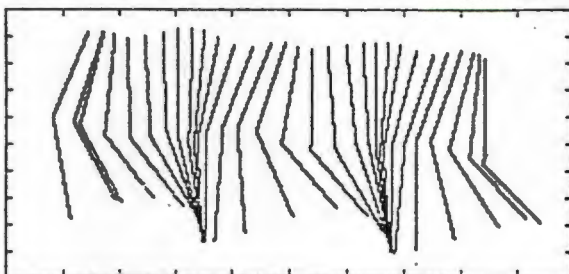


FIGURE 43

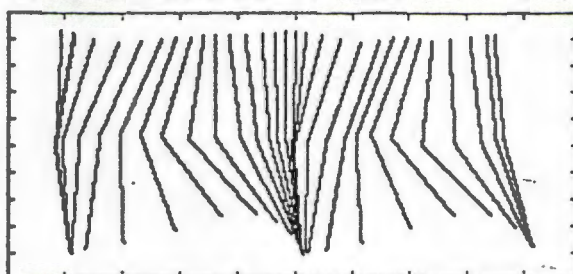
STICK FIGURE

WAT\*LWB  
29 MAY 85 PRE-OP  
LEFT WALK  
X RANGE = 2878 MM Y RANGE = 813 MM



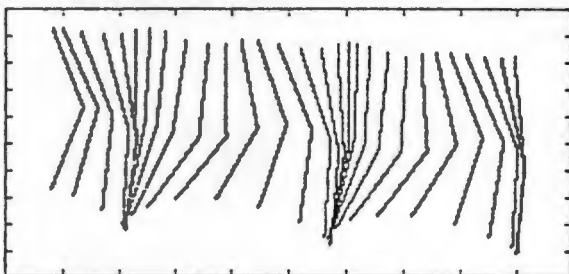
STICK FIGURE

WAT\*LWA  
29 OCT 85 POST-OP  
LEFT WALK  
X RANGE = 1896 MM Y RANGE = 747 MM



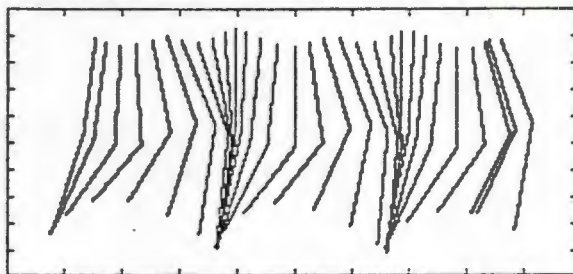
STICK FIGURE

WAT\*RWB  
29 MAY 85 PRE-OP  
RIGHT WALK  
X RANGE = 3613 MM Y RANGE = 828 MM



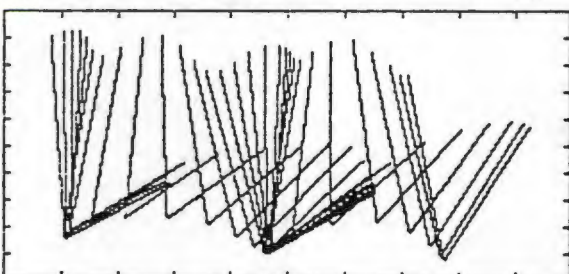
STICK FIGURE

WAT\*RWA  
29 OCT 85 POST-OP  
RIGHT WALK  
X RANGE = 3163 MM Y RANGE = 778 MM



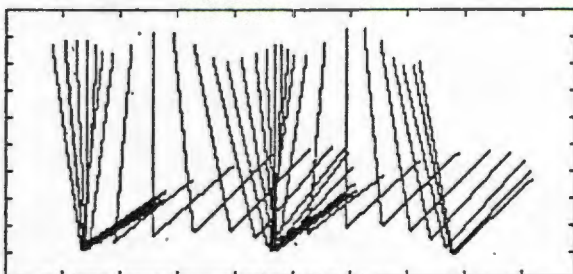
STICK FIGURE

WAT\*LCB  
29 MAY 85 PRE-OP  
LEFT CRAWL  
X RANGE = 1794 MM Y RANGE = 418 MM



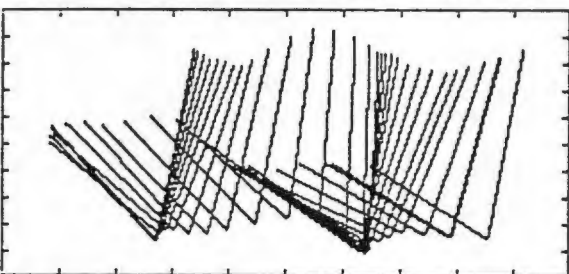
STICK FIGURE

WAT\*LCA  
29 OCT 85 POST-OP  
LEFT CRAWL  
X RANGE = 2823 MM Y RANGE = 361 MM



STICK FIGURE

WAT\*RCB  
29 MAY 85 PRE-OP  
RIGHT CRAWL  
X RANGE = 1515 MM Y RANGE = 368 MM



STICK FIGURE

WAT\*RCA  
29 OCT 85 POST-OP  
RIGHT CRAWL  
X RANGE = 1814 MM Y RANGE = 379 MM

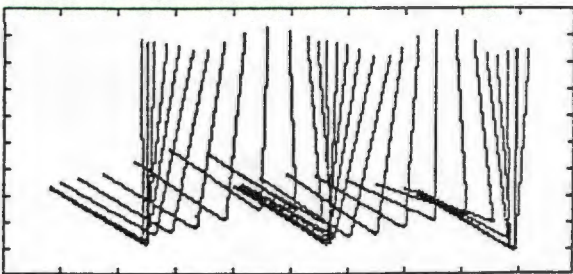
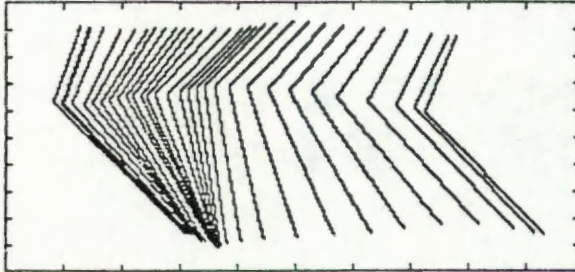


FIGURE 44

Patient 14

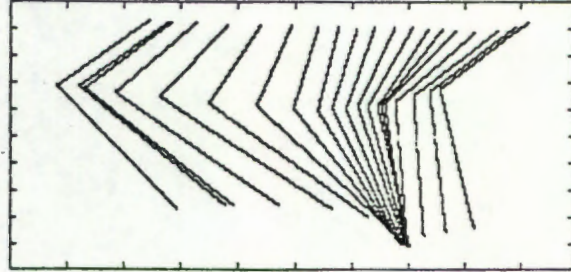
STICK FIGURE

NGO\*LWB  
20 JUN 85 PRE-OP  
LEFT WALK  
X RANGE = 1243 MM Y RANGE = 813 MM



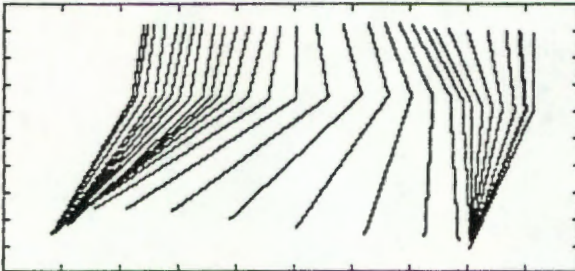
STICK FIGURE

NGO\*LWA  
28 MAY 86 POST-OP  
LEFT WALK  
X RANGE = 1118 MM Y RANGE = 755 MM



STICK FIGURE

NGO\*RWB  
20 JUN 85 PRE-OP  
RIGHT WALK  
X RANGE = 982 MM Y RANGE = 920 MM



STICK FIGURE

NGO\*RWA  
28 MAY 86 POST-OP  
RIGHT WALK  
X RANGE = 2079 MM Y RANGE = 754 MM

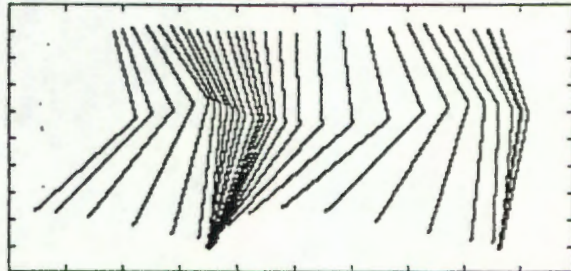
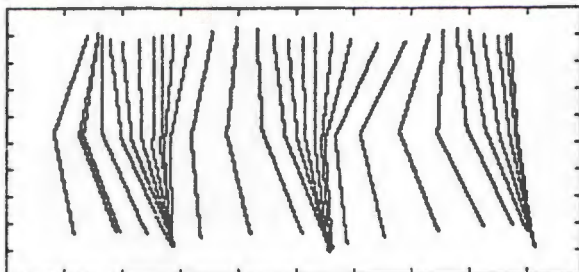


FIGURE 45

Patient 18

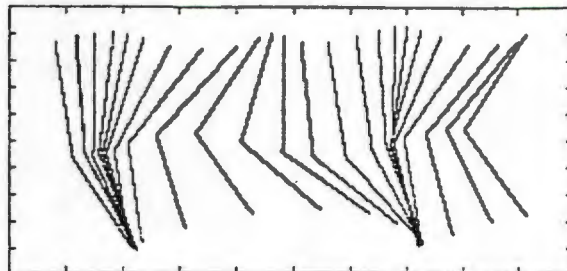
STICK FIGURE

RED\*LWE  
02 OCT 85 PRE-OP  
LEFT WALK  
X RANGE = 1572 MM Y RANGE = 717 MM



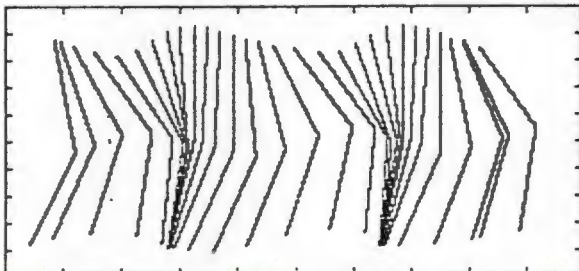
STICK FIGURE

RED\*LWA  
15 APR 86 POST-OP  
LEFT WALK  
X RANGE = 1711 MM Y RANGE = 531 MM



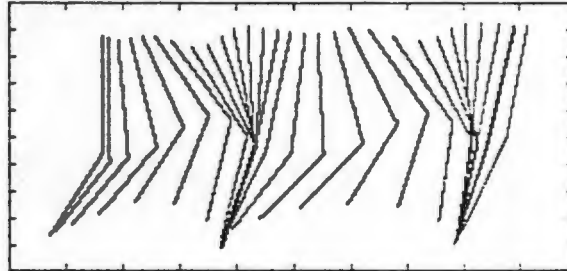
STICK FIGURE

RED\*RWB  
02 OCT 85 PRE-OP  
RIGHT WALK  
X RANGE = 1968 MM Y RANGE = 669 MM



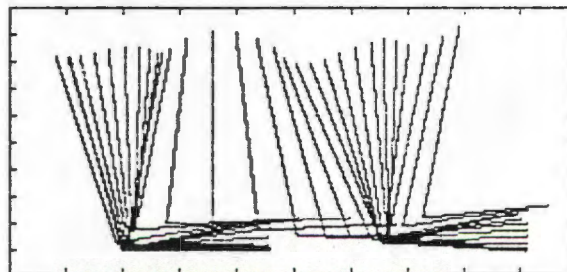
STICK FIGURE

RED\*RWA  
15 APR 86 POST-OP  
RIGHT WALK  
X RANGE = 2416 MM Y RANGE = 676 MM



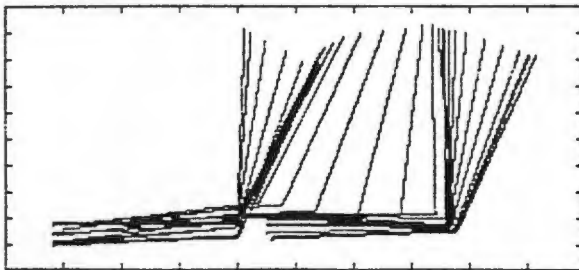
STICK FIGURE

RED\*LCA  
15 APR 86 POST-OP  
LEFT CRAWL  
X RANGE = 1285 MM Y RANGE = 373 MM



STICK FIGURE

RED\*RCB  
20 OCT 85 PRE-OP  
RIGHT CRAWL  
X RANGE = 938 MM Y RANGE = 361 MM



STICK FIGURE

RED\*RCA  
15 APR 86 POST-OP  
RIGHT CRAWL  
X RANGE = 1485 MM Y RANGE = 353 MM

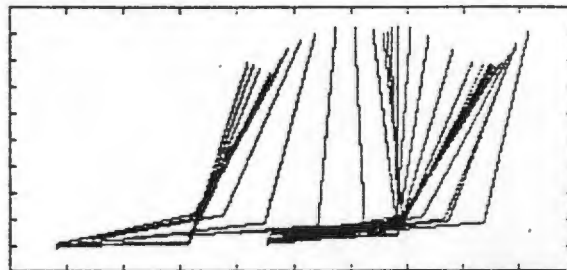
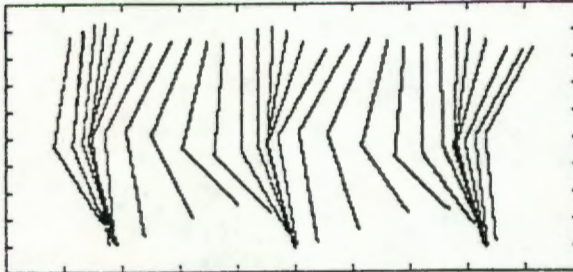


FIGURE 46

Patient 19

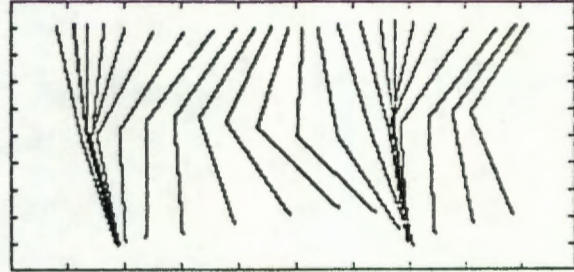
STICK FIGURE

PEI\*LWE  
03 DEC 85 PRE-OP  
LEFT WALK  
X RANGE = 2461 MM Y RANGE = 633 MM



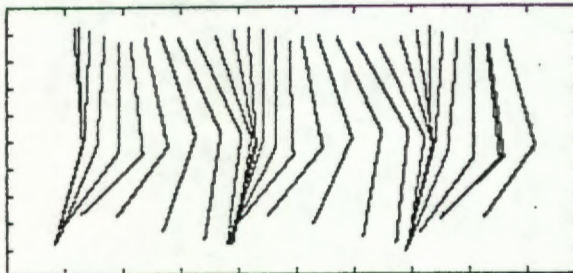
STICK FIGURE

PEI\*LWA  
15 APR 86 POST-OP  
LEFT WALK  
X RANGE = 1733 MM Y RANGE = 536 MM



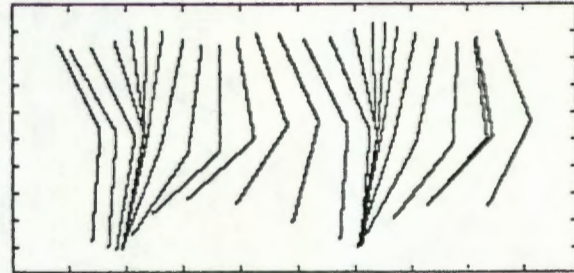
STICK FIGURE

PEI\*RWE  
03 DEC 85 PRE-OP  
RIGHT WALK  
X RANGE = 2329 MM Y RANGE = 685 MM



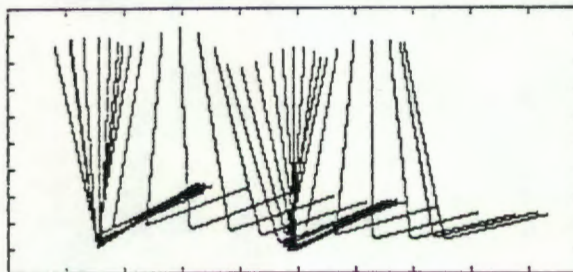
STICK FIGURE

PEI\*RWA  
15 APR 86 POST-OP  
RIGHT WALK  
X RANGE = 2605 MM Y RANGE = 676 MM



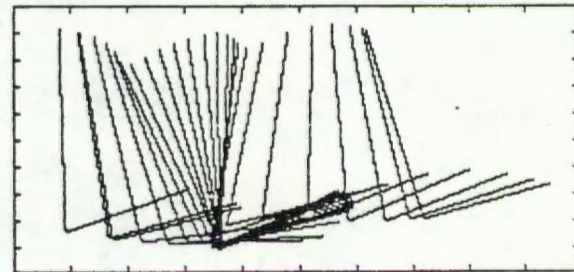
STICK FIGURE

PEI\*LCE  
03 DEC 85 PRE-OP  
LEFT CRAWL  
X RANGE = 1512 MM Y RANGE = 382 MM



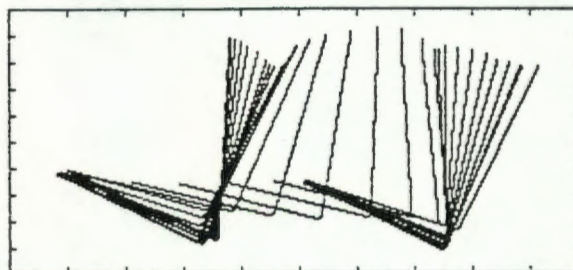
STICK FIGURE

PEI\*LCA  
15 APR 86 POST-OP  
LEFT CRAWL  
X RANGE = 1369 MM Y RANGE = 345 MM



STICK FIGURE

PEI\*RCE  
03 DEC 85 PRE-OP  
RIGHT CRAWL  
X RANGE = 1078 MM Y RANGE = 366 MM



STICK FIGURE

PEI\*RCA  
15 APR 86 POST-OP  
RIGHT CRAWL  
X RANGE = 1130 MM Y RANGE = 338 MM

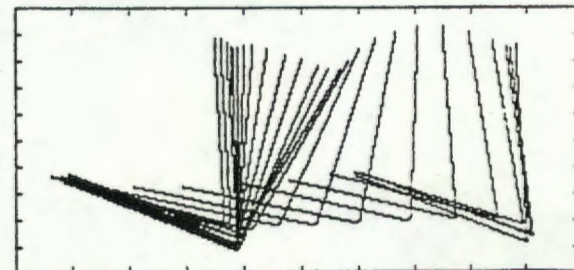
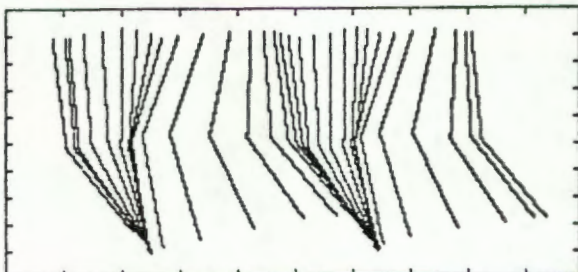


FIGURE 47

Patient 22

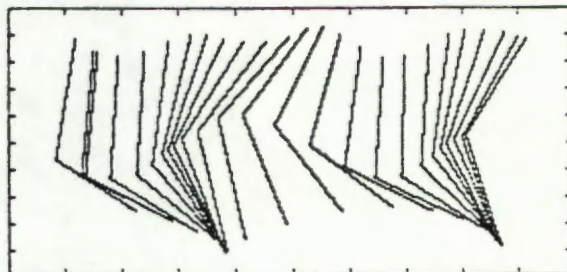
STICK FIGURE

LER\*LWB  
01 MAY 85 PRE-OP  
LEFT WALK  
X RANGE = 2410 MM Y RANGE = 872 MM



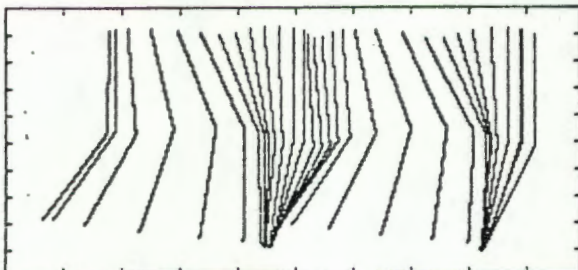
STICK FIGURE

LER\*LWA  
18 APR 86 POST-OP  
LEFT WALK  
X RANGE = 2628 MM Y RANGE = 750 MM



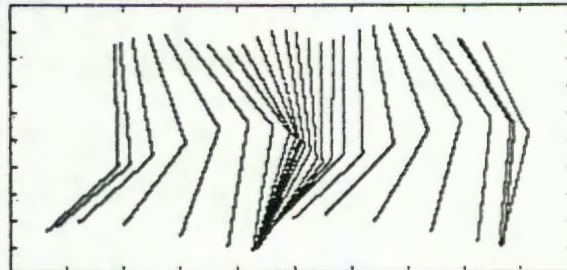
STICK FIGURE

LER\*RWB  
01 MAY 85 PRE-OP  
RIGHT WALK  
X RANGE = 2197 MM Y RANGE = 853 MM



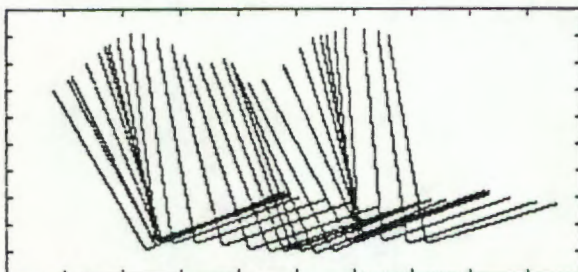
STICK FIGURE

LER\*RWA  
18 APR 86 POST-OP  
RIGHT WALK  
X RANGE = 2875 MM Y RANGE = 714 MM



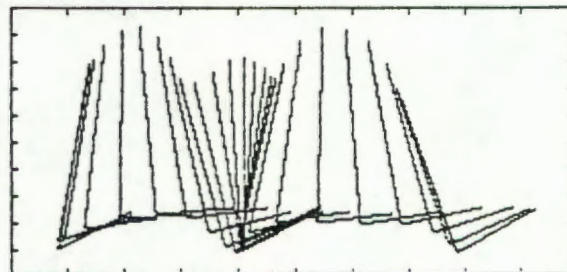
STICK FIGURE

LER\*LCB  
01 MAY 85 PRE-OP  
LEFT CRAWL  
X RANGE = 1526 MM Y RANGE = 403 MM



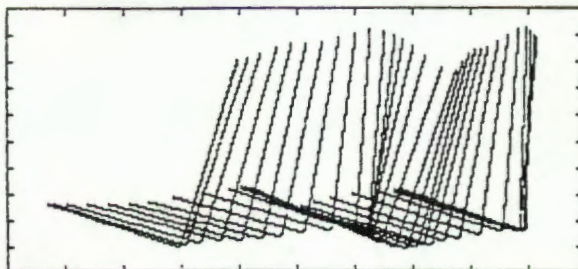
STICK FIGURE

LER\*LCA  
18 APR 86 POST-OP  
LEFT CRAWL  
X RANGE = 3056 MM Y RANGE = 423 MM



STICK FIGURE

LER\*RCB  
01 MAY 85 PRE-OP  
RIGHT CRAWL  
X RANGE = 1544 MM Y RANGE = 405 MM



STICK FIGURE

LER\*RCA  
18 APR 86 POST-OP  
RIGHT CRAWL  
X RANGE = 2067 MM Y RANGE = 412 MM

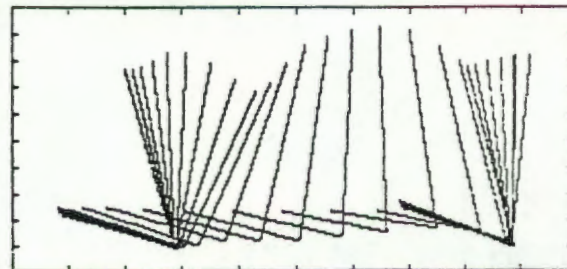
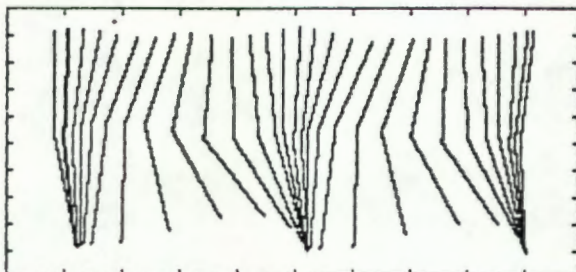


FIGURE 48

Patient 24

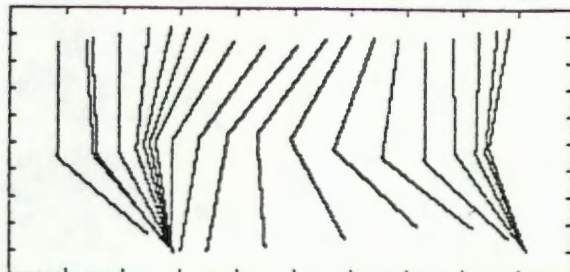
STICK FIGURE

RAS\*LMB  
24 SEP 85 PRE-OP  
LEFT WALK  
X RANGE = 2333 MM Y RANGE = 355 MM



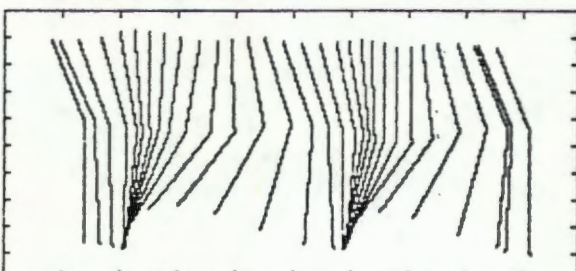
STICK FIGURE

RAS\*LWA  
16 APR 86 POST-OP  
LEFT WALK  
X RANGE = 2577 MM Y RANGE = 355 MM



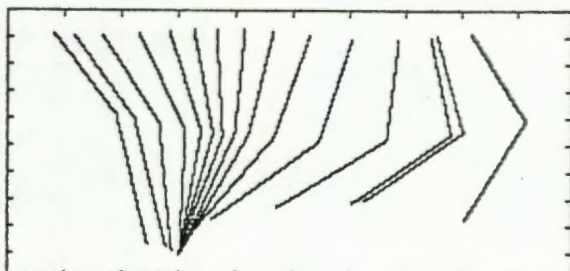
STICK FIGURE

RAS\*RMB  
24 SEP 85 PRE-OP  
RIGHT WALK  
X RANGE = 2830 MM Y RANGE = 317 MM



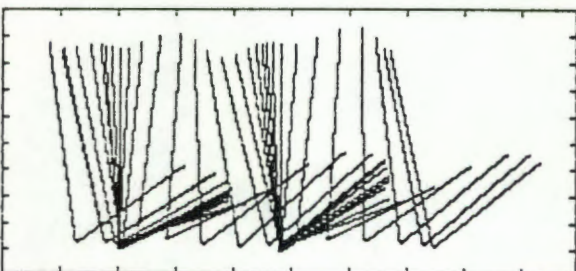
STICK FIGURE

RAS\*RWA  
16 APR 86 POST-OP  
RIGHT WALK  
X RANGE = 2047 MM Y RANGE = 325 MM



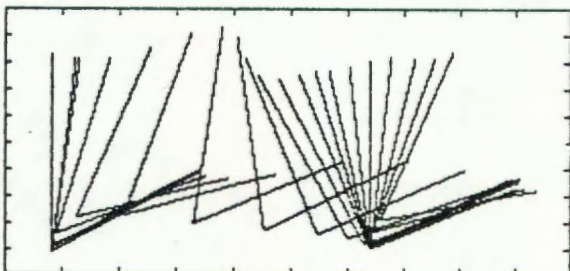
STICK FIGURE

RAS\*LCB  
24 SEP 85 PRE-OP  
LEFT CRAWL  
X RANGE = 1914 MM Y RANGE = 360 MM



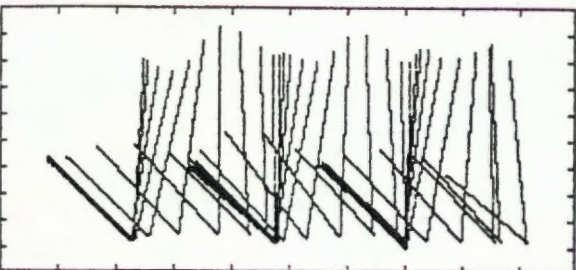
STICK FIGURE

RAS\*LCA  
16 APR 86 POST-OP  
LEFT CRAWL  
X RANGE = 1809 MM Y RANGE = 531 MM



STICK FIGURE

RAS\*RCB  
24 SEP 85 PRE-OP  
RIGHT CRAWL  
X RANGE = 2415 MM Y RANGE = 334 MM



STICK FIGURE

RAS\*RCA  
16 APR 86 POST-OP  
RIGHT CRAWL  
X RANGE = 1735 MM Y RANGE = 424 MM

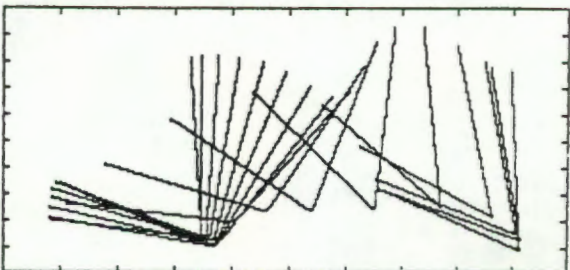
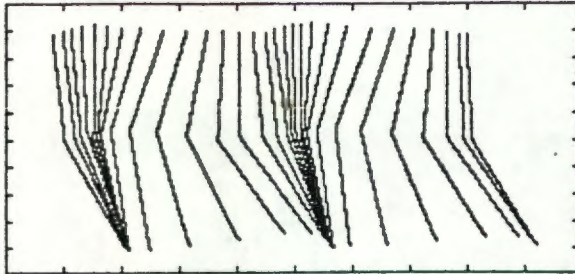


FIGURE 49

Patient 26

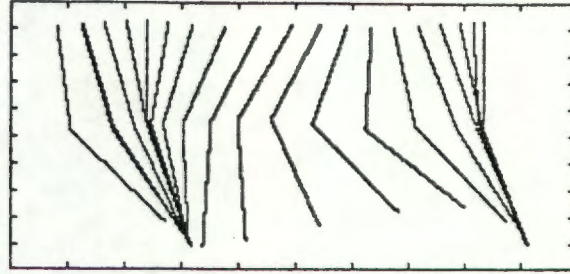
STICK FIGURE

ERY\*LWB  
11 APR 88 PRE-OP  
LEFT WALK  
X RANGE = 1792 MM Y RANGE = 814 MM



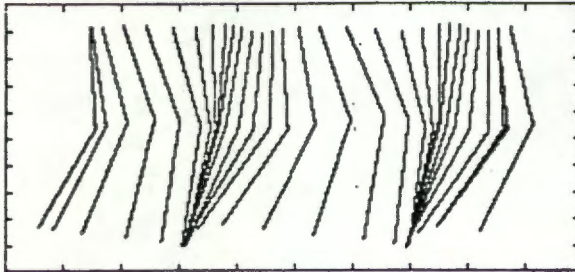
STICK FIGURE

ERY\*LWA  
17 APR 86 POST-OP  
LEFT WALK  
X RANGE = 1785 MM Y RANGE = 770 MM



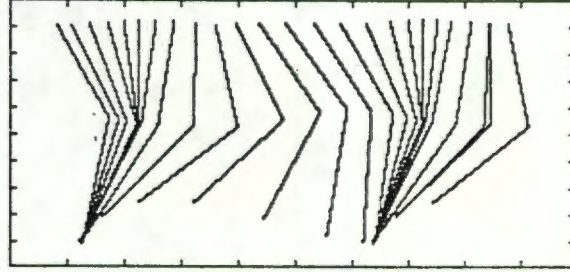
STICK FIGURE

ERY\*RWB  
11 APR 88 PRE-OP  
RIGHT WALK  
X RANGE = 1903 MM Y RANGE = 817 MM



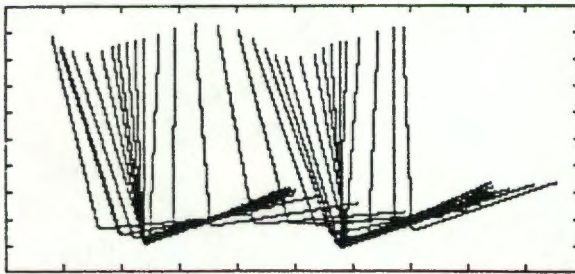
STICK FIGURE

ERY\*RWA  
17 APR 86 POST-OP  
RIGHT WALK  
X RANGE = 2047 MM Y RANGE = 742 MM



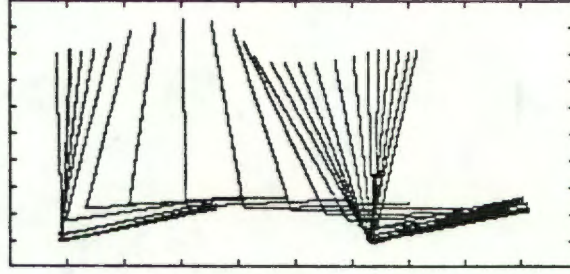
STICK FIGURE

ERY\*LCB  
11 APR 88 PRE-OP  
LEFT CRAWL  
X RANGE = 1330 MM Y RANGE = 422 MM



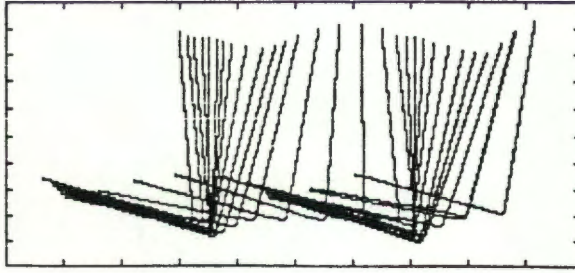
STICK FIGURE

ERY\*LCA  
17 APR 86 POST-OP  
LEFT CRAWL  
X RANGE = 1462 MM Y RANGE = 441 MM



STICK FIGURE

ERY\*RCB  
11 APR 88 PRE-OP  
RIGHT CRAWL  
X RANGE = 1309 MM Y RANGE = 482 MM



STICK FIGURE

ERY\*RCA  
17 APR 86 POST-OP  
RIGHT CRAWL  
X RANGE = 1562 MM Y RANGE = 451 MM

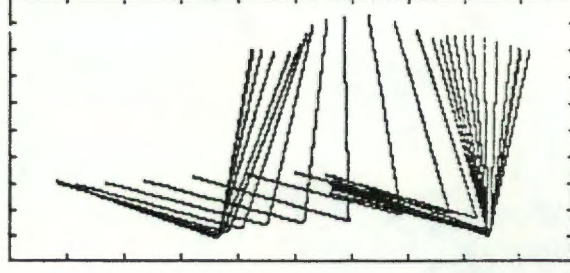
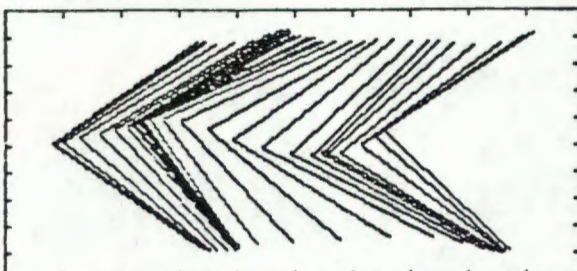


FIGURE 50

Patient 28

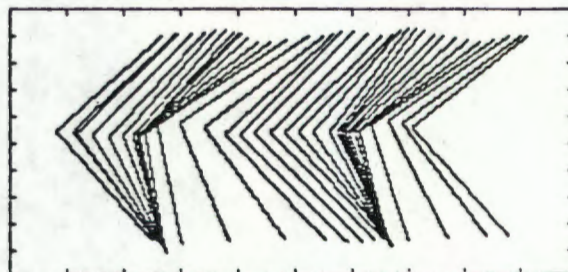
STICK FIGURE

CLA\*LWB  
31 JUL 85 PRE-OP  
LEFT WALK  
X RANGE = 748 MM Y RANGE = 542 MM



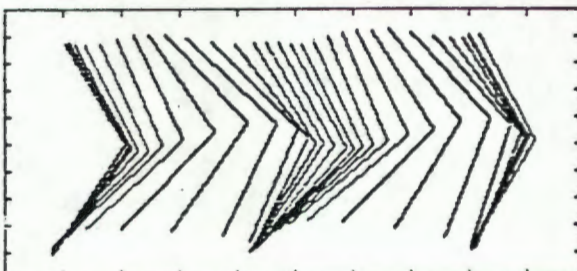
STICK FIGURE

CLA\*LWA  
18 APR 86 POST-OP  
LEFT WALK  
X RANGE = 939 MM Y RANGE = 604 MM



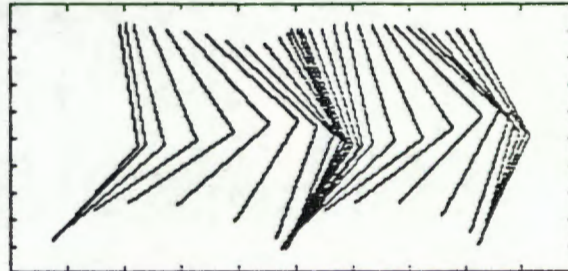
STICK FIGURE

CLA\*RWB  
31 JUL 85 PRE-OP  
RIGHT WALK  
X RANGE = 1215 MM Y RANGE = 624 MM



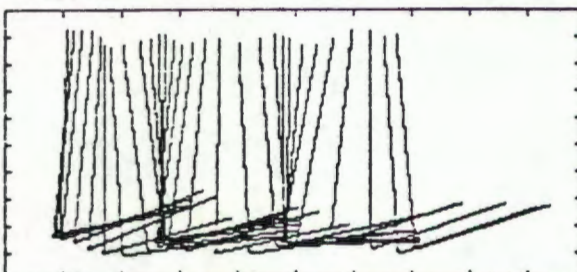
STICK FIGURE

CLA\*RWA  
18 APR 86 POST-OP  
RIGHT WALK  
X RANGE = 1422 MM Y RANGE = 555 MM



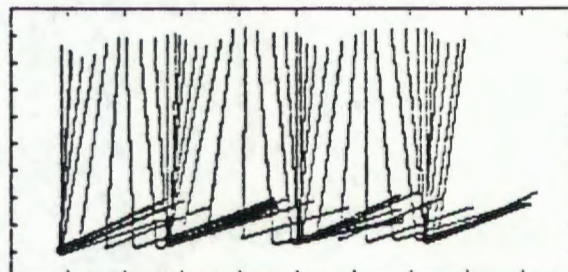
STICK FIGURE

CLA\*LCP  
31 JUL 85 PRE-OP  
LEFT CRAWL  
X RANGE = 1300 MM Y RANGE = 322 MM



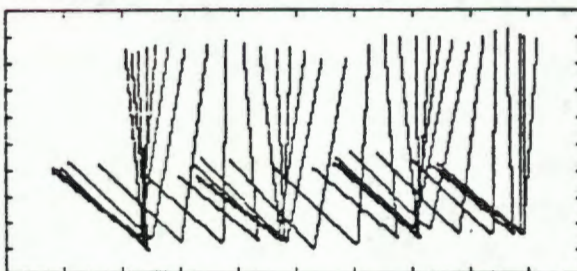
STICK FIGURE

CLA\*LCA  
18 APR 86 POST-OP  
LEFT CRAWL  
X RANGE = 1725 MM Y RANGE = 321 MM



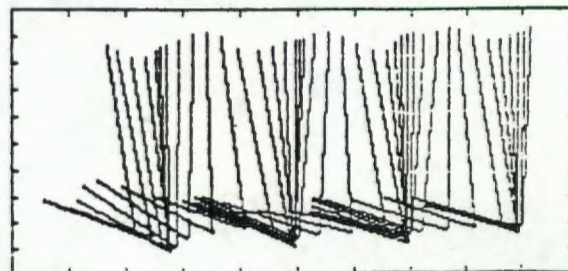
STICK FIGURE

CLA\*RCB  
31 JUL 85 PRE-OP  
RIGHT CRAWL  
X RANGE = 1804 MM Y RANGE = 349 MM



STICK FIGURE

CLA\*RCA  
18 APR 86 POST-OP  
RIGHT CRAWL  
X RANGE = 1993 MM Y RANGE = 314 MM



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