

THE EFFECTS OF SUBMAXIMAL VOLUNTARY EXERCISE WITH  
ELECTROMYOSTIMULATION OF THE QUADRICEPS FEMORIS  
IN POST-POLIOMYELITIS SURVIVORS

BY

LORRAINE ROBERTSON

A Thesis  
Submitted to the Faculty of Graduate Studies  
in Partial Fulfillment of the Requirements  
for the Degree of

MASTER OF PHYSICAL THERAPY

Department of Physical Therapy  
University of Manitoba  
Winnipeg, Manitoba

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ISBN 0-315-77857-1

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## ACKNOWLEDGMENTS

First and foremost I would like to thank all the participants in the study who dedicated time and the limited energy that they had in contributing to the pursuit of science in further our understanding of exercise intervention with muscles weakened by poliomyelitis.

I would then like extend thanks to my committee; Dr. Stephen Vallentyne and Dr. Joseph Kaufert and Dr. Dean Kriellaars for their guidance and inspiration through every stage of this study.

It was Dr. Vallentyne that sparked my interest in the problems that were facing the poliomyelitis survivors. His clinical guidance was invaluable to this project. Steve may find this hard to believe, but as a clinician he is a role model for me. I admire his holistic approach to patient care, the genuine interest that he has for his patients and the excellent rapport that he is able to develop. Steve, I thank you from the bottom of my left ventricle.

It was through Dr. Kaufert that I came to appreciate the importance of the psychosocial aspects of the poliomyelitis survivors. He assisted me with the qualitative analysis and was a wealth of information in methodological research design.

It was simply fascinating to watch Dean at the computer as he instructed me through every stage of the data analysis. Initially he thought "that I was biting off a little more than I was going to be able to chew" when I first started this study, but he did put up with my persistence. I will admit that it could have been a little bit overwhelming if he hadn't broke down the data into manageable bite size pieces for me!! I thank you Dean for pulling me back into reality at those times that I was "bogged down" from venturing off tangent on minor technicalities.

Lee Gregoire, a good friend and colleague, devoted her time and energy to assist me with some of the data collection and Kelsey Nichole was born the day after the final subject was measured. Lee, don't you think that this is funny, because waiting isn't presently one of her strongest attributes. Thanks Lee!

I'd like to thank Staodyn Inc. for the use of their EMS Plus units and also to say sorry about the mailing problem that we encountered.

It also should be acknowledged that this research was supported by the Princess Elizabeth Foundation - grant #034.

Last, but certainly not least, I have to thank my family, Gary and Alicia that had to endure many sacrifices to enable me to complete this project. You were there along side me the whole way! Thanks for your understanding and perpetual encouragement. This thesis is dedicated to you.

## ABSTRACT

The development of new muscular weakness, fatigue, pain and a decline in functional ability decades following the acute insult of poliomyelitis is a well documented phenomenon. The specific pathophysiological cause of the underlying etiology remains unknown. The use of a strengthening program for poliomyelitis survivors has been controversial in the literature because of the potentially harmful effects of exercise on the motor unit.

A controlled multidimensional study was conducted to analyze the effects of a submaximal exercise/ electromyostimulation (EMS) intervention to the quadriceps femoris weakened by poliomyelitis. The creatine kinase (CK) levels were monitored to detect changes in muscle biochemistry of the subjects engaged in the exercise program. The study followed an AB design, with subjects serving as their own controls. In phase A (initial 4 weeks) of the study, there was no intervention. In the phase B (4 weeks), the subjects received an active/ EMS intervention to the quadriceps femoris. Measurements were at the commencement of the study, at the end of the control phase A and at the completion of the active/ EMS phase B of the study.

Measurements of isometric and isovelocity torque were used to evaluate muscular strength of the quadriceps femoris in 18 poliomyelitis subjects experiencing the onset of new weakness and fatigue. Isometric torque (32%) and isovelocity torque (45%) significantly increased ( $p < .05$ ) following the active exercise/EMS intervention. The increase in strength was not associated with a significant change in the muscle cross sectional area as reflected in thigh girth and skinfold measures. Functional ability, as measured by time tests, demonstrated that transfers (28%), stairs (21%) and ambulation (10%) significantly improved ( $p < .01$ ). There were no significant changes in the Barthel Index. The subject's perceived disability due to pain or fatigue, as measured by the Pain Disability Index, was significantly reduced ( $p < .01$ ). The improvement in function may be attributable to both an increase in strength and a reduction in pain for these poliomyelitis survivors.

The findings in this study revealed that submaximal voluntary exercise with EMS resulted in a significant strength increase, a significant pain reduction and a significant functional improvement without CK elevations of these self-referred poliomyelitis survivors.

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## LIST OF ABBREVIATIONS

ADL - activities of daily living

ALS - amyotrophic lateral sclerosis

BP - blood pressure

CK - creatine kinase

CNS - central nervous system

CSF - cerebral spinal fluid

EMG - electromyography

FEV<sub>1</sub> - forced expiratory volume in 1 second

FVC - forced vital capacity

HR - heart rate

PNF - proprioceptive neuromuscular facilitation

PPMA - post-poliomyelitis progressive muscular atrophy

PPS - post-poliomyelitis syndrome

TENS - transcutaneous electrical nerve stimulation

VO<sub>2</sub> - oxygen consumption

## **LIST OF APPENDICES**

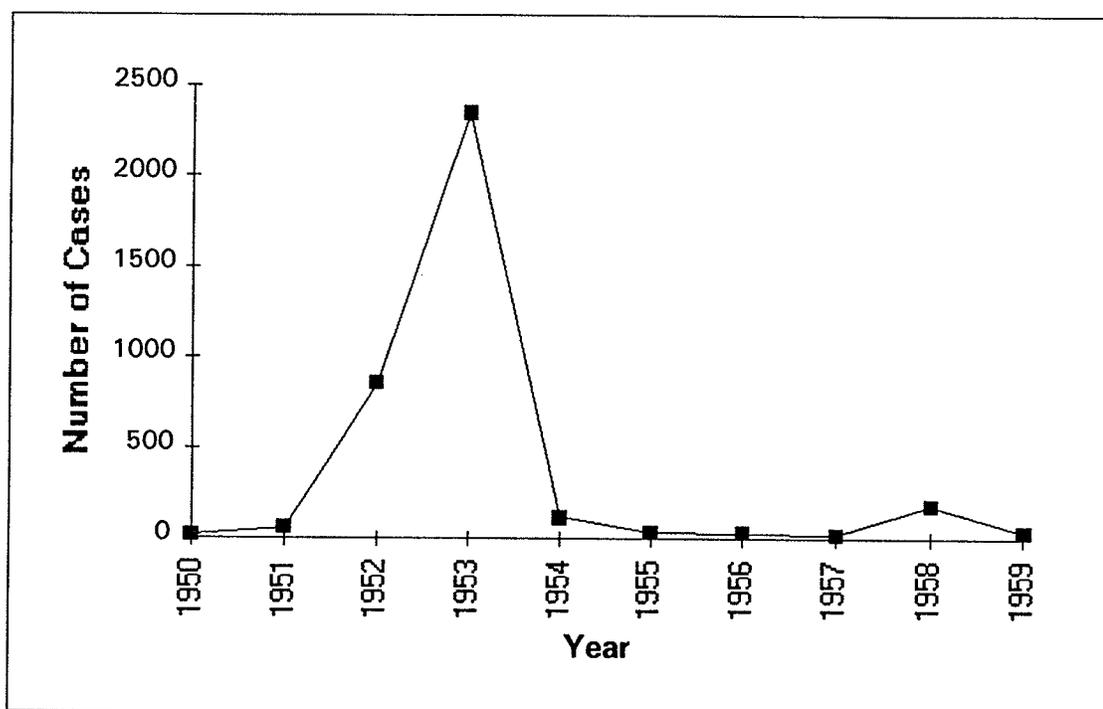
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## LITERATURE REVIEW

### INCIDENCE OF ACUTE POLIOMYELITIS

Acute poliomyelitis was once the most common viral infection of the nervous system (1). In the United States, there were approximately 25,000 - 50,000 cases of acute poliomyelitis reported annually between 1950 and 1956 (Holman 1986).

In the province of Manitoba, there were a total of 3,645 cases reported from 1950 to 1959. Figure 1 illustrates the annual incidence of acute poliomyelitis for this period (Alcock 1980).

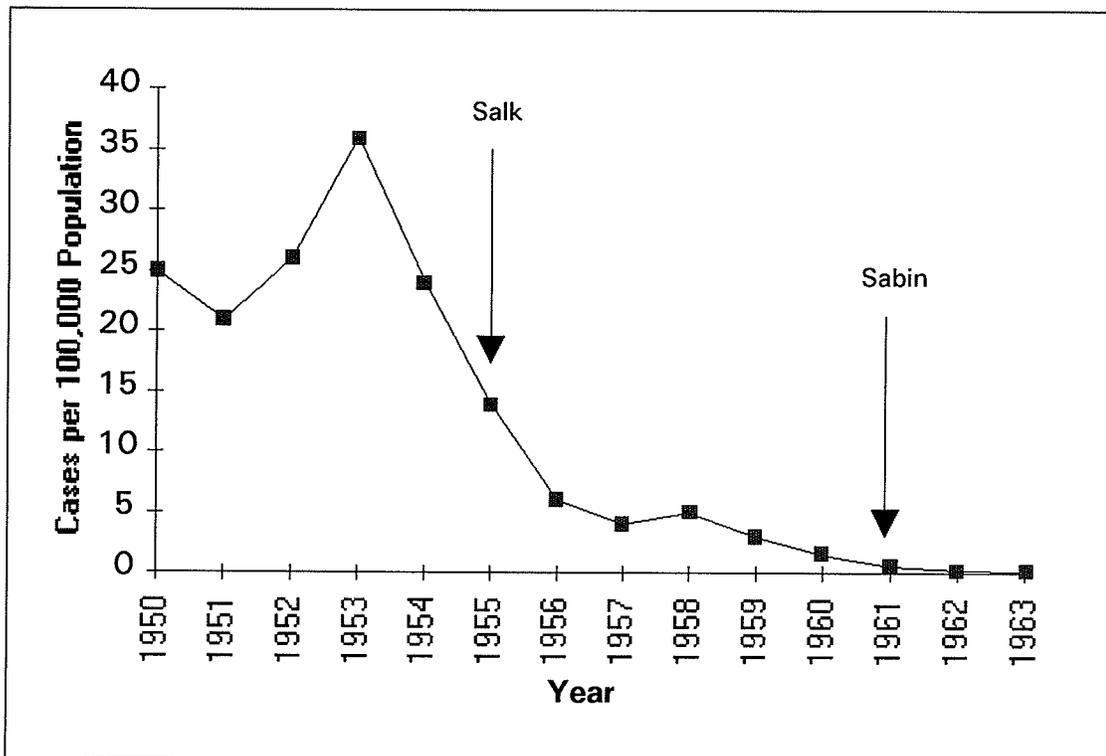


**Figure 1:** The annual incidence rates of acute poliomyelitis in the province of Manitoba from 1950 to 1959 (taken from Alcock 1980).

The incidence of poliomyelitis has decreased dramatically since the introduction of the poliomyelitis vaccines and have virtually eliminated the incidence of poliomyelitis in North America (Paul 1971). In 1955, the Salk inactivated vaccine was found to produce circulatory IgG, IgM, and IgA, but not alimentary tract immunoglobins. It was in 1961, that the Sabin oral vaccine was reported to stimulate gastrointestinal productions of secretory

IgA, which appeared to prevent the poliomyelitis viruses from multiplying. There have been a few exceptional cases reported with relation to the live attenuated strains used in the oral vaccine. The incidence of poliomyelitis, however, has remained low to date.

The poliomyelitis surveillance reports, as illustrated in Figure 2, shows the magnitude of the decrease in incidence of reported cases of poliomyelitis in the United States in the years between 1950 and 1963.



**Figure 2:** Annual incidence rates of acute poliomyelitis in the United States from 1950 to 1963 (taken from the United States Public Health Service).

Acute poliomyelitis is by no means eliminated in the Third World countries (Chaukar 1986). The National Institute for Allergy and Infectious Diseases estimates one out of every 200 children born into the world will suffer paralytic poliomyelitis (Spencer 1986). It has been estimated that there may be around five million new cases of poliomyelitis annually in the Third World countries (Spencer 1986). His statistics are based on an extrapolation from various sources including the World Health Organization. It has been difficult to project the incidence of poliomyelitis in the Third World because it is endemic. The magnitude of the disease in the Third World is understated. There are no health records kept, a high

proportion of cases are unrecognized and the majority of new cases are not seen by doctors (Spencer 1986). There are efforts to eradicate the disease in the Third World countries by the turn of the century, however there have been many psychosocial obstacles in the education of immunization within the lower classes. There is a lack of understanding within the lower classes regarding the seriousness of the disease (Chaukar 1986). There are also economic restraints in purchasing, distributing, and administering the vaccine.

### **PATHOLOGY OF ACUTE POLIOMYELITIS**

Acute poliomyelitis is a viral infection. The three polioviruses are among the smallest of known viruses. They are spherical particles approximately 20mu in size containing a core of ribonucleic acid (RNA) enclosed with a protein outer coat (Johnson 1982).

It has been postulated that only 1-5% of the patients infected experienced the clinical symptoms of poliomyelitis (Holman 1986). Thus, approximately 95-99% infected individuals remain asymptomatic.

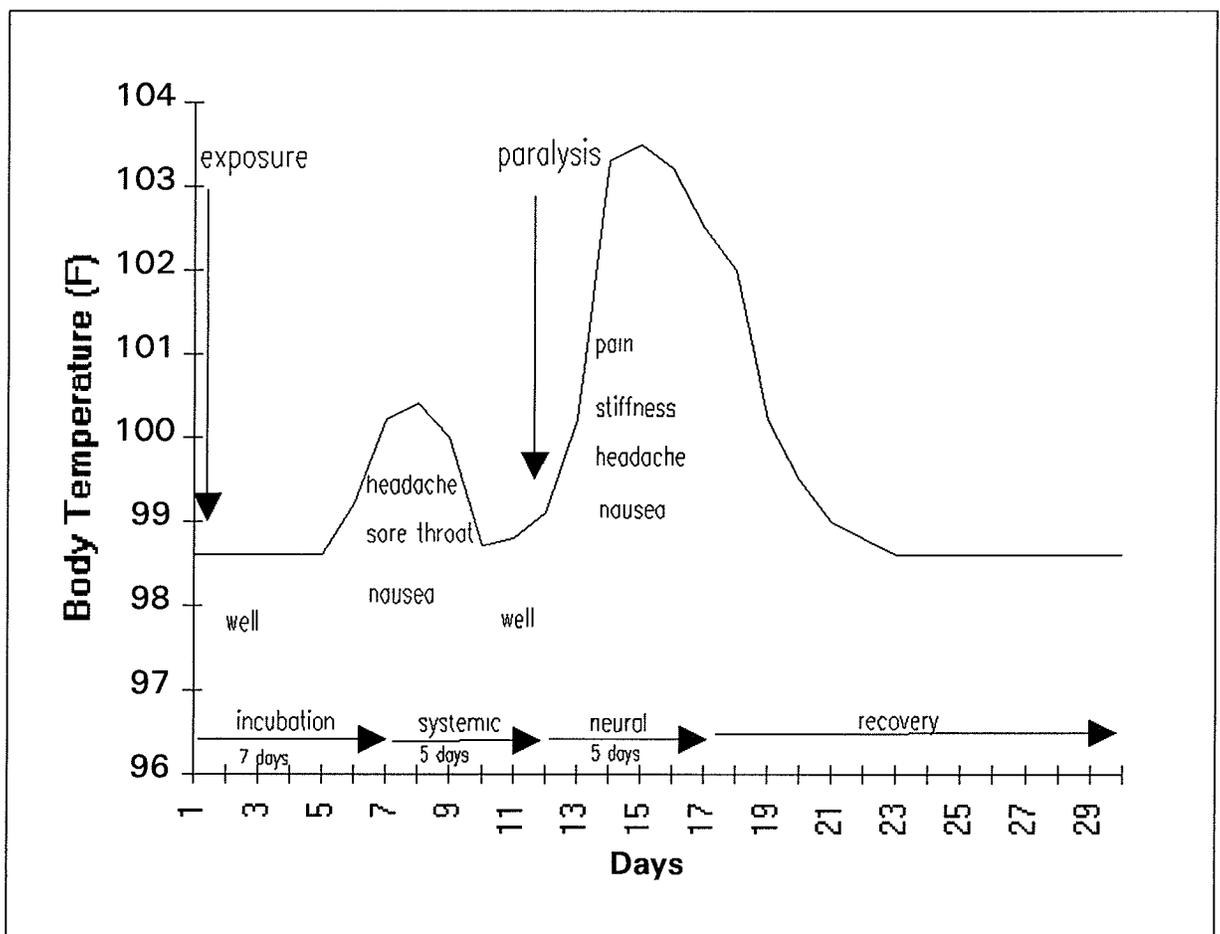
The poliomyelitis viral infection route is by way of oral ingestion. The poliovirus initially infects the mucosa of the respiratory and the gastrointestinal tracts. In abortive poliomyelitis, a brief febrile illness occurs with nonspecific flu-like symptoms of general malaise, diarrhea and loss of appetite (Bodian 1982).

In nonparalytic poliomyelitis, more severe systemic symptoms develop with headaches, rigidity, muscle pains and bowel/bladder dysfunction. This typically occurred within a week following the infection. The poliovirus later invaded the nervous system via the bloodstream at vulnerable areas through the blood-brain barrier (Bodian 1982).

In paralytic poliomyelitis, signs of weakness were evident in one or more of the spinal and or cranial nerves. The cellular changes that resulted from the viral multiplication produced neuronal damage. In spinal paralytic poliomyelitis, the virus had a selective affinity to the large motoneurons located in the anterior horn of the spinal cord. This caused chromatolysis with acidophilic inclusions and necrosis of the motoneurons resulting in muscular paralysis (Bodian 1982). Flaccid limb weakness with eventual atrophy as well as restrictive lung disease due to diaphragmatic and intercostal weakness are the clinical markers of spinal poliomyelitis.

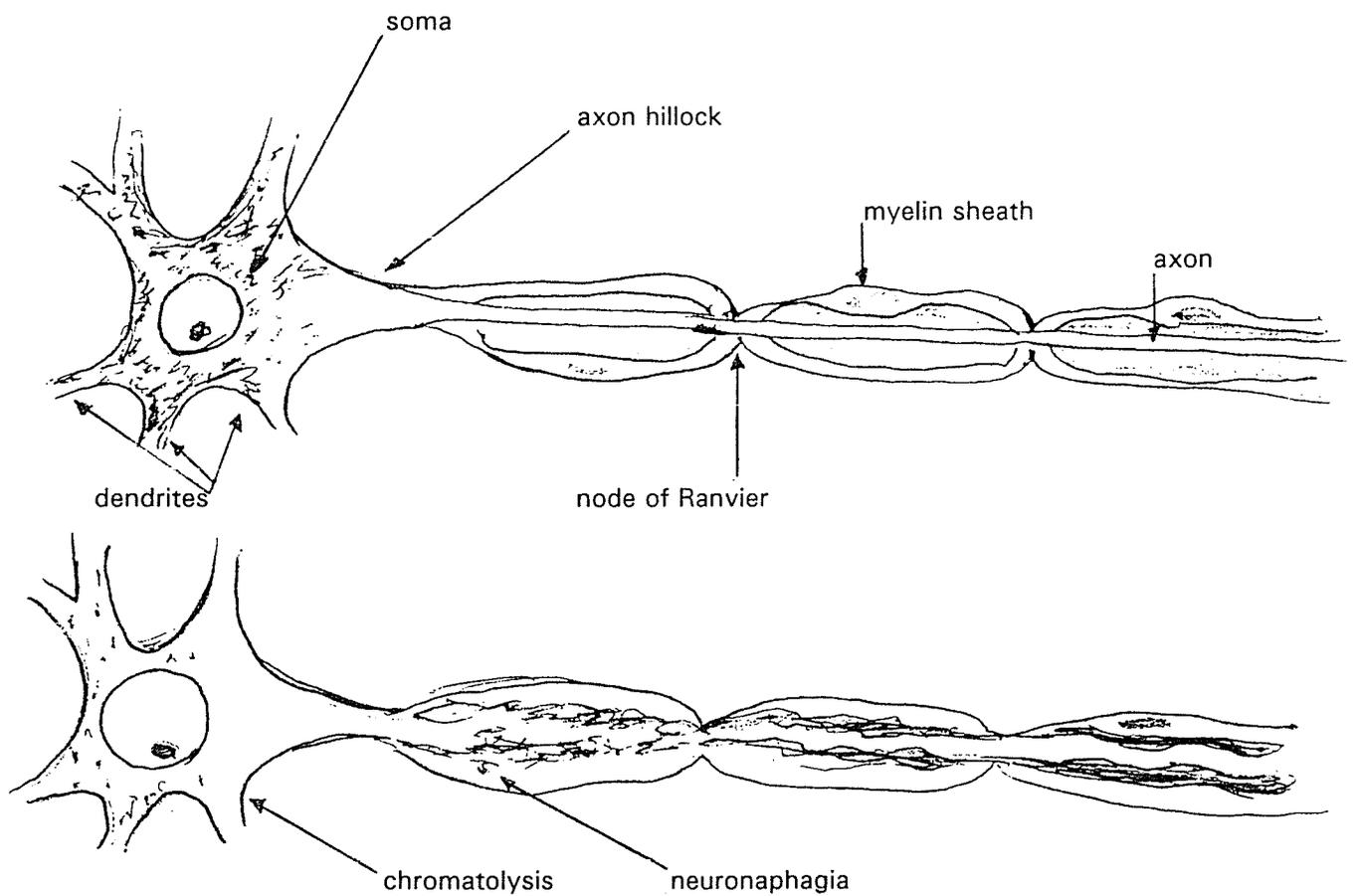
Bulbar poliomyelitis refers to the involvement of brainstem motor nuclei with clinical evidence of dysphagia and spinal apnea.

The acute clinical findings were flaccid paralysis, areflexia, and fasciculations, with no sensory changes. The cerebrospinal fluid revealed an elevated lymphocyte count (Bodian 1982). Independent of the extent of paralysis, the virus typically infected over 95% of the motoneurons with evidence of widespread dissemination throughout the central nervous system (Bodian 1982). The course of the paralytic poliomyelitis infection (taken from Bodian 1982) is summarized in Figure 3.



**Figure 3:** Schematic representation of the course of the paralytic poliomyelitis infection, showing time relations of clinical and pathogenetic events (from Bodian 1982).

The destruction of the soma of the motoneurone resulted in degeneration of the axon, as illustrated in Figure 4, that was accompanied by an inflammatory response, chromatolysis, neuronophagia, and an active gliosis (Bodian 1982).



**Figure 4:** Degeneration of the motoneurone.

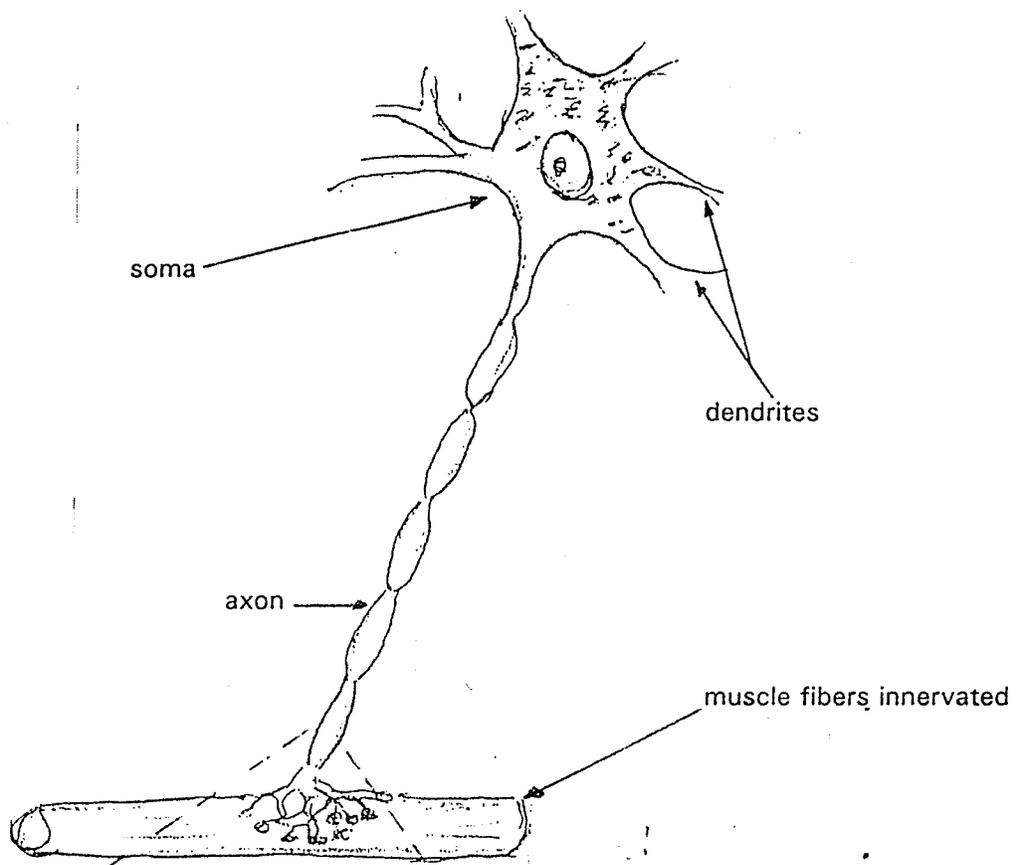
## PHYSIOLOGY OF THE NORMAL MOTOR UNIT

A review of the basic physiology of the normal motor unit will provide a context for the impact of the poliomyelitis virus on the motoneurone.

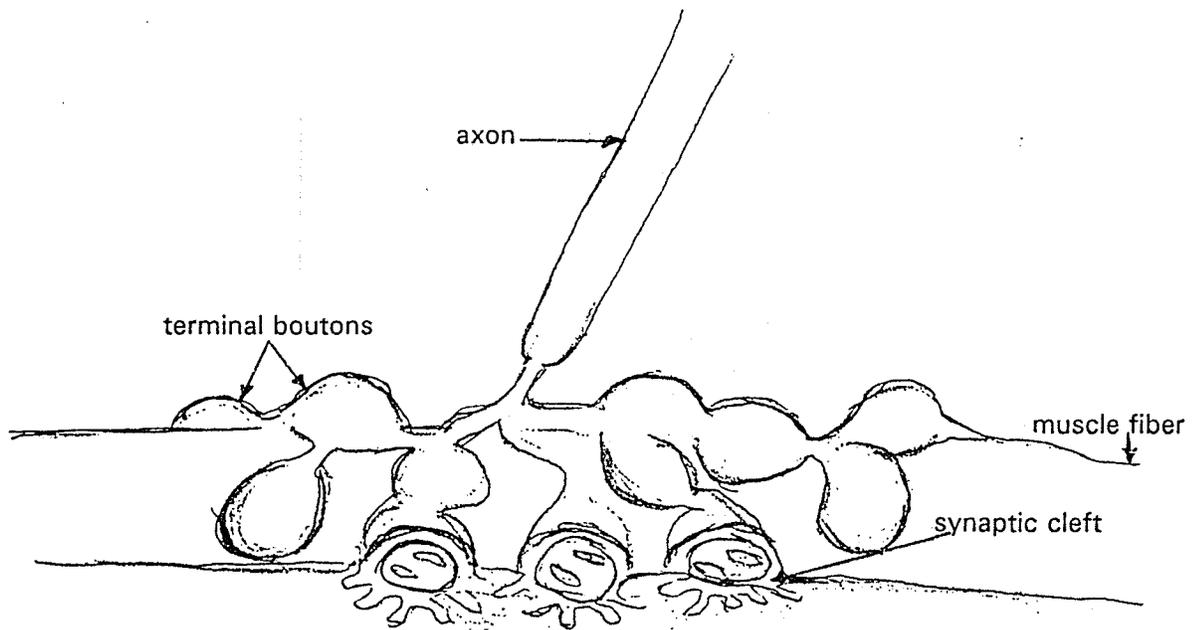
A motor unit as defined by Sherrington consists of a motoneurone soma, the axon, the dendrites and all the muscle fibers that it innervates (Kandel 1985) as illustrated in Figure 5. An innervation ratio refers to the number of muscle fibers innervated per motoneurone (Kandel 1985). A motor unit will innervate one type of muscle fiber (Harrison 1983). The types of muscle fibers are classified based on the enzymes, the twitch characteristics and their fatigability. The type I (slow twitch) muscle fibers have high oxidative capacity, are fatigue resistant (Kandel 1985). Type II (fast twitch) muscle fibers have low oxidative capacity, poor fatigue resistance, and high tension development (Kandel 1985). There is an even distribution of these fiber types in normal human muscle (Engel 1986).

During a simple isometric muscle contraction, type I fibers innervated by smaller motoneurons are recruited first, followed by progressively larger motoneurons innervating type II fibers of larger motor units (Edstrom 1986). This order of recruitment is called the size order recruitment principle (Henneman 1965). Less fatigable type I fibers permit sustained activities of low grade resistance, whereas type II fibers are summoned for high intensity level requirements of a short duration.

Excitation of a motoneurone produces an action potential that is propagated along the axon to the terminal branches at the neuromuscular junction. It is at this site that synaptic communication occurs between the motoneurone and the muscle fibers. The space between these two structures is the synaptic cleft as illustrated in Figure 6. The terminal boutons of the depolarized motoneurone release acetylcholine into the synaptic cleft and this acetylcholine then binds to specific receptors on the muscle membrane at the motor end plate. The sarcolemma of the muscle fiber will generate an end plate potential, the size of which is determined by the amount of acetylcholine. If the endplate potential is sufficient in size (above threshold), a muscle action potential is produced. The muscle action potential propagation down the t-tubules permits calcium release intracellularly from the sarcoplasmic reticulum which permits a contraction of the myofilaments. The endplate repolarizes and the acetylcholine in the synaptic cleft is hydrolyzed by the enzyme acetylcholinesterase.



**Figure 5:** *The normal motor unit.*



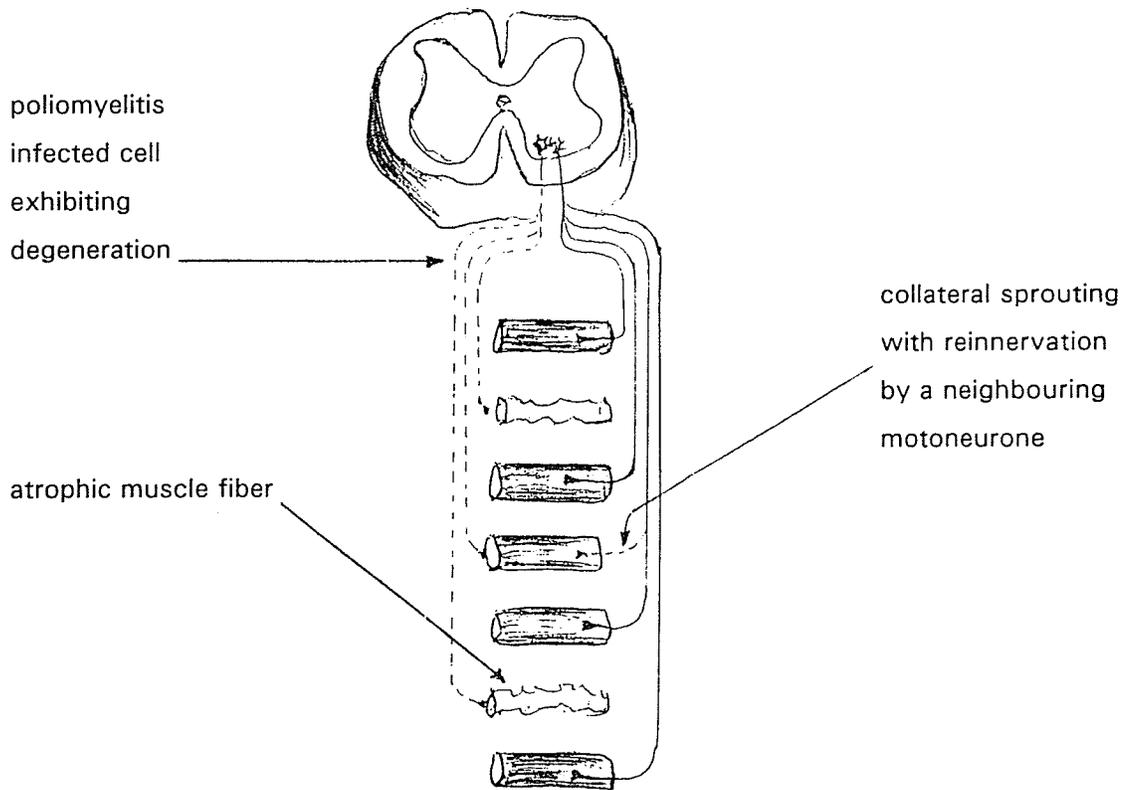
**Figure 6:** *The neuromuscular junction.*

## PHYSIOLOGY OF THE MOTOR UNIT AFFECTED BY POLIOMYELITIS

During the acute poliomyelitis, the invasion of the soma of the motoneurons resulted in the death or injury of these neurons with partial or complete neuromuscular recovery (Dalakas 1991).

The severity of the disease during the first weeks of the illness, is correlated with a greater functional impairment (Halstead 1991). For injured motoneurons there was a resultant loss of voluntary activation of the muscle fibers caused by the denervation process of their motor fibres. The extent of paralytic involvement from the motoneurone death or injury could not be determined before a period of three months had lapsed. Recovery could occur in the partially damaged cells, but the necrotic cells would be phagocytized by leukocytes and removed (Bodian 1982).

The poliomyelitis survivors would regain muscular strength and function as their denervated muscle fibers were reinnervated by neighbouring surviving motoneurons that would extend axonal sprouts to the orphaned muscle fibers (Roland 1985) as illustrated in Figure 7.



**Figure 7:** The motor unit affected by poliomyelitis with terminal axon sprouting and reinnervation of neighbouring denervated muscle fibers (taken from Roland 1985).

Muscle biopsy studies revealed that this terminal axon sprouting would enable an uninvolved or a recovered motoneurone to adopt up to 7 additional muscle fibers for every muscle cell innervated originally (Coers 1959). Therefore, a single motoneurone that originally innervated 100 muscle fibers, could conceivably innervate 700 fibers. Dalakas (1991) has speculated that this magnitude of innervation ratio would result in excessive metabolic demands, threatening an "overstressed" state on the motoneurone.

Dalakas speculated that the extent of neurological and functional recovery would be determined by the type and number of motoneurones that remained (Dalakas 1991).



1. Unaffected by poliomyelitis and were distant from the area of neuronal loss and were consequently "non-stressed".



2. Unaffected by poliomyelitis but were close to areas of destroyed neurons and are thus "overstressed" as they try to oversprout and compensate for the motoneurones that were phagocytized.



3. Partially affected by poliomyelitis, but they survived, are smaller and thus are "overstressed" for their normal workload.



4. Severely affected by poliomyelitis, but incompletely recovered and are consequently "scarred" and "overstressed".

**Figure 8:** The speculated types of motoneurones after recovery from acute poliomyelitis (taken from Dalakas 1991).

Muscle fiber hypertrophy was also a contributing factor for strength gains attained during the rehabilitation/recovery phase from acute poliomyelitis (Borg 1988).

It was speculated that another three or four years had to lapse before the poliomyelitis survivors felt that they had plateaued in their functional recovery. They could be functioning with significantly fewer motor units with higher innervation ratios. Manual muscle tests are useful to show substantial deficits relative to 'normal', but they are inadequate to differentiate 'normal' muscle strength. Years ago, Sharrard reported that muscles weakened by poliomyelitis could obtain a grade 5 on a manual muscle test, even with a 50% reduction in original motoneurons (Sharrard 1953).

Clinically, Beasley quantified this 'normal' muscle strength of grade 5 in the poliomyelitis population. He found that the poliomyelitis population that had grade 5 or 4 strength in the quadriceps femoris could exert an average force of 75% or 40%, respectively, when compared to the control subjects with grade 5 or 4 quadriceps femoris strength on a manual muscle test (Beasley 1961). A grade 5 manual muscle test score for a poliomyelitis survivor could realistically have a 25% to 60% strength decrement relative to age-matched normals.

The checkerboard pattern distribution of the fiber types I and II was not evident within the muscles of the poliomyelitis survivors (Kelly 1991). The muscle fiber phenotypes are related to the type of activities performed by the muscles (Salmons 1969). There appears to be a certain degree of plasticity in the muscles that were previously affected by poliomyelitis. Borg found that in the weak, yet functional muscles of the poliomyelitis survivors, there was a transformation from type II to type I fibers without a selective loss of the larger motoneurons (Borg 1988, Borg 1991). The poliomyelitis subjects with 'excessive overuse' exhibited marked hypertrophy with primarily the type I muscle fibers (Borg 1988). The increased physical demands of exercise has a potentially positive effect, due to the plasticity adaptation of the poliomyelitis affected muscles.

## **POST-POLIOMYELITIS EPIDEMIOLOGY**

The 1987 National Health Interview Survey estimated that there are 1.63 million individuals in the United States that contracted the poliovirus prior to the introduction of the Salk and Sabin vaccines (Parsons 1989). Approximately 50% of these poliomyelitis survivors that

were surveyed, reported an onset of new health problems (Parsons 1989). These individuals survived the acute stage of poliomyelitis and have enjoyed many years of functional stability. Recently however, they find themselves confronted with a combination of systemic symptoms, musculoskeletal problems and neurological manifestations.

During the 1980's, increased public awareness stimulated medical interest which promoted our understanding of these new health problems confronting the poliomyelitis population. Halstead distributed questionnaires in 1983 and 1984 to more than 500 members of the poliomyelitis communities in Dayton Ohio, St. Louis Missouri, Houston Texas, and Minneapolis Minnesota (Halstead 1985). The response rate was uncertain because the questionnaire was circulated by the poliomyelitis networks across the country. The 201 respondents were considered to be a self-referred group that probably reflect the symptomatic portion of the poliomyelitis population. They did, however, provide the first set of valuable information on the general types of new health problems experienced by some poliomyelitis survivors. The majority of the respondents were female (70%), with a median age of 49 (range of 28-80 years), who noted the onset of problems approximately 34 years after the onset of poliomyelitis. More than 50% of these subjects had contracted poliomyelitis after 1949. Of this group, 87% complained of increasing fatigue, 81% reported weakness in previously affected muscles, 71% had weakness in previously unaffected muscles, 75% complained of joint or muscle pain, and 82% were experiencing difficulties in their activities of daily living (ADL) (Halstead 1985).

In an attempt to avoid selecting a self-referred symptomatic population, Codd reviewed the Mayo Clinic records of poliomyelitis cases in Olmsted County, Minnesota from 1935 to 1955. They were able to obtain 128 respondents (41%) to their questionnaire out of the 316 cases of poliomyelitis reported in this period. There were 28 (22%) of the respondents that reported new health problems at an onset of 25 years following the acute poliomyelitis (range 12 - 35 years). Of this group, 19 (71%) had new muscle weakness, 16 (59%) reported increased fatigue, 13 (48%) had muscle pain and 7 (25%) found that their functional capabilities were decreasing (Codd 1985).

Speier accessed the poliomyelitis records from the Sister Kenny Institute, Sheltering Arms Hospital and the University of Minnesota hospitals in Minneapolis, Minnesota as their sampling frame (Speier 1987). They were able to locate 327 (43%) of the poliomyelitis survivors from their 1952 - 1953 admissions. There was a response rate of 49% which represented only a very small portion of the original epidemic population (21%). New

problems were reported by 41% of the respondents; 47% experienced muscular pain, 42% with fatigue and 40% complained of increasing weakness.

<b>New health problems</b>	<b>Halstead (1985a) N = 201</b>	<b>Codd (1985) N = 128</b>	<b>Speier (1987) N = 327</b>
fatigue	87%	13%	42%
weakness in previously affected muscles	81%	15%	51%
muscle pain	75%	11%	51%
joint pain	75%	16%	47%
weakness in previously unaffected muscles	71%	3%	16%
<b>New ADL problems</b>			
walking	82%	6%	
climbing stairs	81%	---	
transfers	51%	6%	
dressing	---	3%	

***Table 1:** New health and ADL problems experienced by the poliomyelitis survivors following years of stable recovery.*

#### **POST-POLIOMYELITIS SYNDROME vs. POST-POLIOMYELITIS PROGRESSIVE MUSCULAR ATROPHY**

The symptoms experienced by the poliomyelitis survivors are relatively common and may present a challenge for the clinician to decipher the complex interplay of their many symptoms. There appear to be a characteristic cluster of new health and ADL problems (Halstead 1985a, Codd 1985, Speier 1987), that tend to develop in the poliomyelitis survivors.

There has not been a consensus in the literature regarding the terminology to use as a diagnostic label for the poliomyelitis survivors experiencing new health problems; terms include the late effects of poliomyelitis, post-poliomyelitis sequelae, post-poliomyelitis syndrome (PPS), post-poliomyelitis progressive muscular atrophy (PPMA) and post-poliomyelitis motor neuron disease (PPMND). The difficulty with nomenclature reflects the

fact that there is no test available to specify the diagnosis. The ambiguity is due in part to our limited understanding of the underlying pathophysiology of these new complaints from the poliomyelitis survivors.

The specific criteria for the diagnosis of post-poliomyelitis syndrome (PPS) as identified by Halstead, are identified in Table 2.

1. A prior episode of acute paralytic poliomyelitis confirmed by history and physical examination;.
2. Standard EMG evaluation demonstrates changes consistent with prior poliomyelitis (fibrillations, positive sharp waves, fasciculations, giant potentials of long duration, high fiber density, the presence of neuromuscular jitter and blocking).
3. A history with a period of neurologic recovery followed by an extended interval of functional stability preceding the onset of new problems.
4. The gradual or abrupt onset of new weakness in previously affected and/or unaffected muscles. This may or may not be accompanied by other new health problems such as excessive fatigue, muscle pain, joint pain, decreased endurance, decreased function and muscular atrophy.
5. Exclusion of other medical conditions that might cause the health problems listed in #4 above.

***Table 2: Diagnostic criteria for post-poliomyelitis syndrome (from Halstead 1991).***

Post-poliomyelitis progressive muscular atrophy (PPMA) has the most specific diagnostic criteria, including the five items as identified for PPS (Table 2) and also the presence of new muscular atrophy as verified by the presence of scattered angulated fibers in muscle biopsies that indicates an active denervation process (Dalakas 1991).

PPMA is a poor term to describe the weakness that is associated with the symptomology of the poliomyelitis survivors. In PPMA, 'muscular atrophy' means a decrease in the cross sectional area of the muscle, but to the poliomyelitis survivor, it is the increasing weakness that is much more apparent than the new atrophy (Cashman 1987, Codd 1985). According to Cashman, all of the PPS patients with muscular atrophy concurrently reported that they experienced new weakness, however only 50% of those complaining of new weakness

acknowledged the presence of new muscular atrophy (Cashman 1987). 'Progressive' is to imply there is new weakness that is to be differentiated from the original muscular atrophy as a residual weakness from the acute poliomyelitis. Dalakas observed a steady decline in strength averaging 1% per year (Dalakas 1986) which is comparable to age-matched normal subjects. In contrast, there was another longitudinal study by Munin that revealed the isometric strength of the poliomyelitis survivor actually was improving by 29% per year in the affected quadriceps and by 14% per year in the nonaffected quadriceps (Munin 1991).

The sequelae from the PPS does not necessarily have to be viewed as progressive. There have been reports that with lifestyle modification, protective bracing or the use of ambulatory assistive devices, the secondary muscle strength may recover (Perry 1985a, Twist 1986, Peach 1990, Packer 1991).

The epidemiological studies imply there are at least 20% (Codd 1985) of the poliomyelitis population that are experiencing new muscular weakness. This new weakness is not necessarily PPS. Musculoskeletal disuse can contribute to muscle atrophy, weakness, contracture formation and various other complications associated with a sedentary lifestyle. Halstead has presented a conceptual framework as illustrated in Figure 9, for musculoskeletal disuse, musculoskeletal overuse, or motor unit dysfunction that may act alone or in combination to produce these symptoms of muscular weakness in the poliomyelitis population (Halstead 1991).

### **Differential Diagnosis**

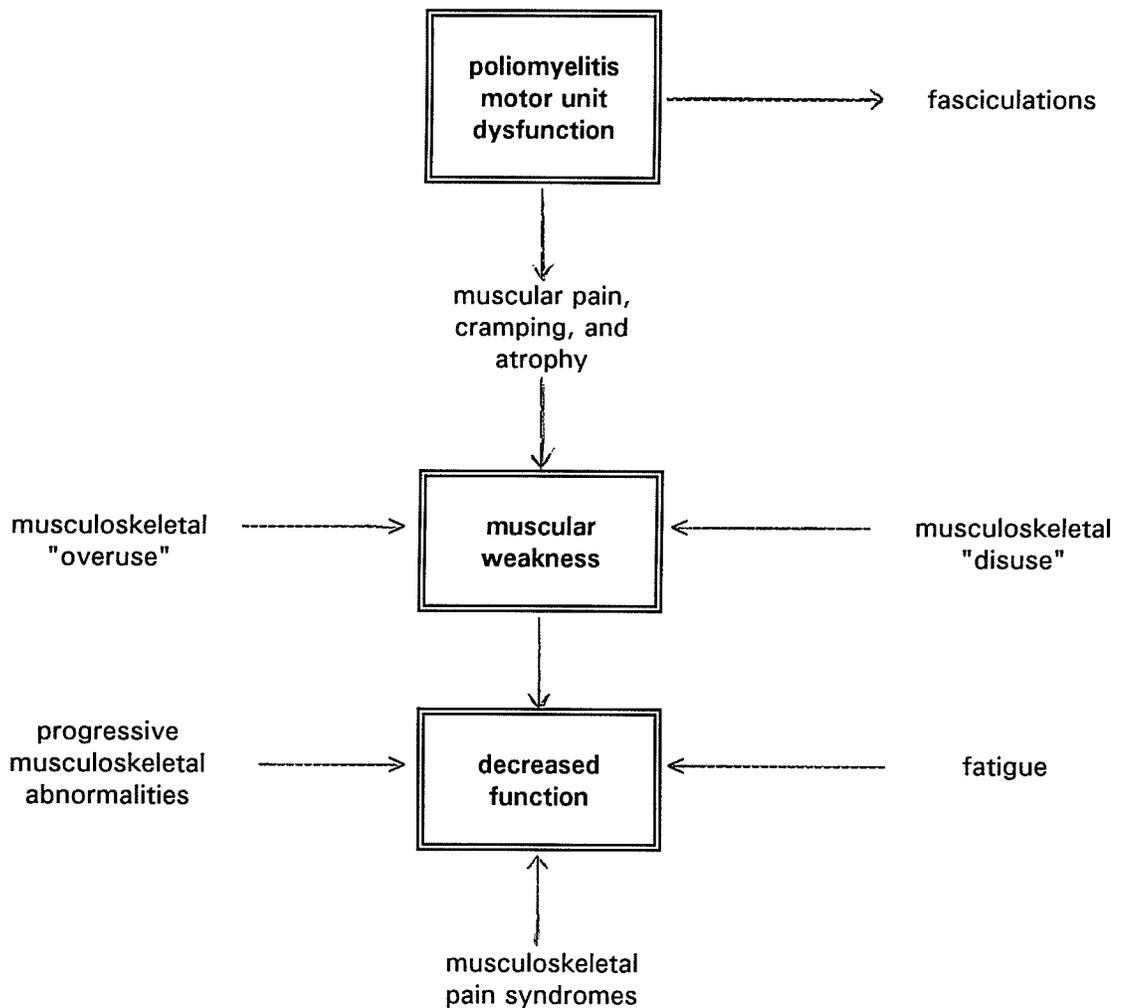
Poliomyelitis survivors may develop secondary weakness due to other physical problems such as an extremity fracture. In qualitative evaluations it has been reported that it is difficult to regain strength following a period of immobilization (Halstead 1991). A similar situation in years previous to a secondary health problem would not have caused the same decline in their health and function (Halstead 1991). This would be consistent with the role for disuse atrophy in producing increased muscle weakness for poliomyelitis survivors.

New health problems may or may not be associated with other medical conditions. Halstead claims that the diagnosis for post-poliomyelitis progressive muscular atrophy is that of exclusion (Halstead 1991).

1. **Unrelated causes** New health problems that could develop in this population that are unrelated to the history of poliomyelitis. Diseases such as amyotrophic lateral sclerosis, diabetes, heart conditions and cancer would fall into this category.

2. **Related known causes** New health problems with a known etiology could develop related to remote poliomyelitis. As a consequence of the residual neuromuscular weakness, joint deformities, arthritis, and overuse could develop.

3. **Related unknown causes** New health problems associated with remote poliomyelitis could be due to the new neuromuscular degeneration of unknown etiopathogenesis.



**Figure 9** : Conceptual framework of the interacting variables contributing to post-poliomyelitis progressive muscular atrophy (taken from Halstead 1991).

## **NEW MUSCULAR WEAKNESS ASSOCIATED WITH POST POLIOMYELITIS**

The development of new muscular weakness years after acute poliomyelitis was first documented more than 100 years ago (Raymond, 1875, Cornel 1875). It was in 1957 that Bennett and Knowlton reported an overuse weakness in muscles affected by poliomyelitis. Overuse weakness refers to a loss of maximal force following exhaustive exercise that is persistent for days, weeks, or longer (Bennett 1958, Knowlston 1957).

The late onset of weakness has been described in muscles that were previously affected by poliomyelitis. Based on four case summaries, Kayser-Gatchalian (1973) claimed that the muscles previously affected by poliomyelitis were those primarily involved with the new weakness. On the other hand, Campbell (1969), reported involvement in muscles that were not previously affected in all five cases described. The epidemiology surveys of Halstead, Codd and Speier, as identified in Table 1, report the onset of new weakness in muscles that were previously involved and also to the muscles that were previously uninvolved by the acute poliomyelitis. This would cause one to suspect that previously 'unaffected' motoneurons were either minimally or mildly involved during the acute infection, or previously 'unaffected' motoneurons are newly involved with a poliomyelitis-related sequela. The poliomyelitis survivors could be suffering from a musculoskeletal disuse or a sedentary life style. Clinically, the pathophysiological changes could have escaped unnoticed due to the scattered nature of their motor deficits and the extraordinary ability for compensation with the use of the healthier muscle fibers.

Previously affected and previously unaffected muscles may develop new weakness. The new weakness however, is more likely in muscles with severe involvement, which may be related to the diminished numbers of surviving motoneurons (Klingman 1987).

The poliomyelitis survivor that was an adult at the time of the acute infection, appears to have more extensive residual paralysis (Weinstein 1957), or due to less resiliency, or plasticity, of a mature nervous system. The extent of paralysis may be due to a rapid dissemination via rapid axonal transport of the virus within the central nervous system (CNS) of the adults (Jubelt 1986). It has also been documented that the more severe the acute illness, the more likelihood of developing the onset of new muscular weakness.

The latency period, the time of acute poliomyelitis until the onset of new muscular weakness, is summarized in Table 3. The mean latency period as identified in the literature is approximately 38 years. The peak incidence of acute poliomyelitis was in 1952. We are currently in the peak era for new weakness associated with previous poliomyelitis.

# of cases	individual mean age for onset of acute polio (years)	mean interval (years)	Reference
13	7	32.7	Geiger (1952)
6	6.5	41.2	Campbell et al. (1969)
34	5	37	Mulder et al. (1972)
4	1	61	Kayser-Gatchalian (1973)
5	5.8	38.8	Palmucci et al. (1980)
17	--	42.8	Martinez et al. (1983)
10	8	36.6	Dalakas et al. (1985)
201	10	37.4	Halstead et al. (1985)
268	12	34	Ontario March of Dimes (1985)
18	13	31	Maynard (1985)
27	11	28.8	Dalakas et al. (1986)
mean		<b>38.3</b>	

**Table 3 :** *The latency period; the interval between the onset of acute poliomyelitis and the onset of new muscular weakness.*

### FATIGUE ASSOCIATED WITH POST-POLIOMYELITIS

The poliomyelitis survivors may experience a "generalized" fatigue that is quite severe even without muscular wasting or weakness (Halstead 1991). Post-poliomyelitis fatigue usually occurs in the late morning or early afternoon (Packer 1991) and is most common in ambulatory individuals (Halstead 1991). Halstead describes this fatigue as "hitting the wall" (Halstead 1991); as indicated by the poliomyelitis survivor, a sudden onset of overwhelming exhaustion after minimal physical activity (Halstead 1985). The fatigue can affect the energy level, endurance and mental alertness of the poliomyelitis survivor (Halstead 1991).

Berly (1991) conducted a survey to determine how people with PPS define their fatigue and to explore the effects of fatigue on their lifestyle, employment, and quality of life. The subjects with PPS described their fatigue as occurring daily and increased in severity as the

day progressed. Both the subjects with PPS and the controls described the fatigue as a tiredness with a lack of energy, but physical weakness was only reported only with the PPS group. "Minimal physical exercise", or work, exacerbated the fatigue in 48% of the PPS group, but decreased fatigue in 70% of the controls (and in 15% of the PPS group). There were 27% of the PPS group and no controls that reported mild to moderate depressive symptoms. The difference in prevalence and description of their chronic fatigue was not significantly affected (Berlly 1991). Fatigue that tends to last all day is not typical in the poliomyelitis survivor. Differential diagnosis is therefore required. Fatigue that is evident upon awakening is also atypical and may be due to sleep disturbances associated with musculoskeletal discomfort, or possibly sleep apnea (Halstead 1991). Fatigue may be alleviated for the poliomyelitis survivor by balancing the overall physical activity and by scheduling intermittent rest periods throughout the day (Packer 1991).

Halstead (1991) and Berlly (1991) associate fatigue in the poliomyelitis survivors with "minimal physical activity". In actuality, "minimal physical activity" could be "maximal" for the work output of the poliomyelitis survivor. The work output of the activites should be relative to the subject group because the poliomyelitis survivors already have a compromised strength capacity. For instance, vacuuming the living room might be equivalent to 42 kilojoules of work (10 Kcal), a small percentage of the total work capacity for normal individuals, but relatively much greater for PPS individuals.

There should be a distinction made between fatigue in weakened muscle and the general fatigue associated with cardiopulmonary deconditioning. Pulmonary function testing, or other screening procedures, should be done prior to the commencement of an exercise program (Owen 1985).

#### **PAIN ASSOCIATED WITH POST-POLIOMYELITIS**

The pain associated with poliomyelitis may occur in muscles, joints or both. Superficial muscle pain may present as hypersensitivity or a "crawling" sensation (Halstead 1991). Deep muscular pain is described as "cramping" (Glasber 1978), or "aching" similar to the acute episode or "soreness" that usually follows vigorous exercise. The poliomyelitis survivor however, experience symptoms following only light physical activity (Halstead 1991). These muscle pains can be aggravated by physical activity, stress and cold temperatures (Halstead 1991).

Joint pain may be associated with physical activities such as weight bearing, but is not necessarily associated with swelling or inflammation of the structures (Halstead 1991). Yarnell (1986) claims the primary cause of pain for the poliomyelitis survivor is that of degenerative arthritis. Muscle imbalances may be due to joint laxity or muscular tightness. Altered biomechanical factors may contribute to a breakdown of the cartilage and some erosion of the underlying bone which could lead to bony thickening and finally bony spur formation. Yarnell also claimed that 66% of his poliomyelitis group had joint laxity or joint contracture.

Yarnell (1986) reports that in 50 poliomyelitis survivors, 100% had some degree of scoliosis curvature. Degenerative disc disease may be associated with the scoliosis resulting in excessive loading of the facet joints. If the scoliosis is associated with a leg length discrepancy, there could be a resultant pelvic obliquity that could produce sacroiliac joint pain. Orthotic devices and shoe lifts could potentially reduce biomechanical strains (Maynard 1985, Smith 1987). Longstanding weakness predisposes to joint deformities as a consequence of biomechanical imbalances. Genu recurvatum, for example, could occur secondarily to increasing weakness of the quadriceps femoris. Loss of ambulation could result, often necessitating the use of ambulatory assistive devices (Anderson 1972). Perry used EMG to compare the gait patterns of symptomatic and asymptomatic poliomyelitis survivors with approximately the same amount of residual loss (Perry 1987). They found that the symptomatic patients had a less efficient gait pattern and the intensity and contraction duration of the quadriceps femoris were increased compared to the asymptomatic poliomyelitis patients (Perry 1987). Gait training with biofeedback techniques or selective surgery to alter the muscle biomechanics could make the gait pattern more efficient. Muscular strain, ligamentous sprain, and spondylosis could be related to overuse or misuse and, consequently, could contribute to pain experienced by poliomyelitis survivors (Smith 1987).

There is usually a reduction of pain with the use of conservative measures such as energy conservation techniques similar to that described by Young (1991), effective protection and stabilization of the abnormal joint movement. Smith focuses intervention upon restoring the erect posture in sitting, standing and walking (Smith 1987). There are many avenues, such as lumbar rolls, ergonomic chairs, or corsets, for the restoration of lumbar and cervical curves in symptomatic poliomyelitis survivors. Pain can be reduced by "respecting the intricate balance" between ligaments and muscles (Maynard 1985a, Maynard 1985b).

## **FUNCTIONAL DETERIORATION ASSOCIATED WITH POST POLIOMYELITIS**

Functional deterioration tends to parallel the progression of muscle weakness and can be quite pronounced if the 'functional reserve' is limited. There may be increased difficulty in ambulation, standing, climbing stairs or other endurance activities that can compromise the activities of daily living (ADL) (Halstead 1991). It was suspected that these routinely performed functions, with repetition or sustained muscle contractions, are susceptible to developing new muscular weakness (Maynard 1986). For example, the progressive weakness appears to occur more often in weight bearing muscles of the legs than in non-weight bearing muscles of the arms (Windebank 1987). This observation would be consistent with the pathological process of new motor unit dysfunction associated with musculoskeletal "overuse" (Dalakas 1991) or, conversely, weakness limiting mobility may be more salient to the poliomyelitis survivors. Maynard describes his clinical observation of functional muscular overuse in poliomyelitis survivors:

"The muscles that are used most vigorously in normal activities are the ones most likely to develop new weakness. This opinion is not based on scientific evidence, but on experience."

"The best example of this is walking. Normal walking relies heavily on the quadriceps muscle of the anterior thigh to stabilize the knee joint during stance. Several studies suggest that one only uses about 15% of the maximal strength in that muscle to stabilize the knee during normal walking. Some post-polios with a weakened quadriceps muscle as their acute polio residual are only able to walk using 80-100% of the maximal strength of that muscle. Therefore when they walk everyday for varying distances, they are in fact, producing maximum contractions of that muscle repeatedly on a long term basis" (Maynard 1986).

Education about self-awareness of physical limitations is a key ingredient in successful lifestyle modification at work or in the home environment (Packer 1991). Work simplification and energy conservation may be by way of orthoses, adaptive equipment, and mobility aids, or it may entail changing jobs or terminating employment (Perry 1985).

## **DIAGNOSTIC EMG CHANGES IN THE MUSCLES AFFECTED BY POLIOMYELITIS**

The first electromyographic abnormality during the acute phase of poliomyelitis is a reduced recruitment pattern with fibrillations potentials within the denervated muscle fibres that developed as the axons of the motoneurons degenerated. Without adequate reinnervation, fibrillation potentials may persist many years after the acute episode (Dalakas 1991). The

process of reinnervation is associated with a reduction of spontaneous discharges and the appearance of large amplitude and long duration motor unit potentials (Wiechers 1985).

Fasciculation and spontaneous activity are common in acute poliomyelitis, and are seen in chronic stages of poliomyelitis survivors following fatigue (Dalakas 1987).

Hayward and Seaton (1979) reported that the electromyographic evidence of partial denervation was more widespread than clinically suspected. Mean amplitude of motor unit action potential was increased in both weak muscles affected by poliomyelitis and the apparently unaffected muscles (that occurred most commonly contralateral to the spinal segment involved). The number of motor units reduced approximately in proportion to the degree of muscle atrophy (Hayward 1979).

Wiechers and Hubbell (1981) have shown that poliomyelitis survivors demonstrate an increased neuromuscular jitter that represents variability in the time interval between depolarizations of two single muscle fibers of the same motor unit. As reinnervation continues and myelination proceeds, the neuromuscular junction matures and neuromuscular jitter decreases.

Neurogenic jitter on the other hand, is absent in poliomyelitis and frequently found in the amyotrophic lateral sclerosis (ALS). This poliomyelitis abnormality suggests that the individual axonal terminals are affected. With ALS, the whole nerve is affected (Dalakas 1991).

If the conduction deficit in the terminal sprouts is severe enough, its passage may be blocked. Neuromuscular blocking is also frequent in the individuals with poliomyelitis (Dalakas 1991).

In normals, it is unusual for two adjacent muscle fibers to be innervated by the same motoneurone. Many studies have reported increased fiber density characterized by clusters of three, four or even more adjacent fibers innervated by the same neuron in the poliomyelitis population (Cashman 1987, Dalakas 1991, Wiechers 1985).

It is important to confirm the presence of these characteristic electromyographic abnormalities in the symptomatic muscles because this is a useful objective measure. Electrodiagnosis, however, cannot distinguish between symptomatic and asymptomatic

poliomyelitis survivors as both groups may manifest increased neuromuscular jitter and fiber density. Recognized also that EMG is unable to identify disuse atrophy.

Some investigators thought the symptoms of post-poliomyelitis were a form of ALS (Mulder 1972). The EMG may assist with the differential diagnosis of other motor unit diseases as summarized in Table 4. Amyotrophic lateral sclerosis (ALS) that has a history of rapid loss of muscle strength, has upper motoneurone signs and EMG changes that are more widespread involving the whole motoneurone.

EMG findings	Post-poliomyelitis
fibrillations and positive sharp waves	present (+)
fasciculations	frequently present (+)
giant potentials of long duration. polyphasic	often above 10mV in amplitude
fiber density	very high
neuromuscular jitter and blocking	increased, even in "stable" muscles suggestive of ongoing reinnervation (very unstable in weak muscles)
neurogenic jitter	none - suggestive of instability of distal sprouts and failure of reinnervation of groups
macro EMG	amplitude increased, but drops with progressive weakness

**Table 4** : Electromyographic findings in post-poliomyelitis (taken from Dalakas 1991).

## HISTOLOGICAL CHANGES IN MUSCLES AFFECTED BY POLIOMYELITIS

Muscle biopsy studies show data that is consistent with a possible abnormality at the axon terminals. Cashman (1987) and Dalakas (1986) have reported isolated and scattered angular fibers in muscle biopsies from patients with prior poliomyelitis, consistent with an active process of degeneration of the motor unit. Fiber density is increased in poliomyelitis survivors and appears to correlate with the amount of original reinnervation and aging (Martinez 1983, Weicher 1985).

Muscle biopsy findings	Post-poliomyelitis
fiber-type grouping of normal-size fibers	always present even in asymptomatic muscles (large groups, up to 200 fibers per group)
scattered angulated fibers	present in PPMA but absent in asymptomatic post polio
group atrophy	rare in the newly weakening and previously healthy muscles
inflammation	up to 40% of biopsies
hypertrophy, moth-eaten and targetoid fibers	often present due to long-standing partial denervation

**Table 5** :*Histological characteristics of the muscle biopsies in post-poliomyelitis (taken from Dalakas 1991).*

In 1988, Pezeshkpour and Dalakas examined the spinal cords of 8 poliomyelitis individuals who died from non-neurological diseases (5 asymptomatic and 3 symptomatic). They found that an inflammatory reaction was present in the spinal cord for as long as 44 years after the acute poliomyelitis infection. These changes were not correlated with symptomatic or asymptomatic status. The spinal cords of symptomatic cases, however, did reveal that motoneurons were undergoing atrophy and chromatolysis (Pezeshkpour 1988). This finding is consistent with the theory of post-poliomyelitis in that motoneurons have lost their capacity for further reinnervation and become dysfunctional in their ability to support metabolic needs of the distal sprouts (Dalakas 1988).

## BIOCHEMICAL CHANGES IN POLIOMYELITIS SURVIVORS

In the assessment and differential diagnosis of the poliomyelitis survivor, the standard battery of biochemical screening tests, such as an SMA 24, thyroid panel, fasting glucose, etc. have not proven to be helpful or cost effective, except for creatine kinase (CK) (Halstead 1991).

Creatine kinase is a muscle enzyme that is a component of the sarcolemma. The normal range is 33 - 186 U/L and elevations of the CK levels are correlated with acute muscle damage.

Normal subjects may experience an increase in CK levels following exercise, and the elevation could directly relate to the duration and intensity of the exercise performed (Newham 1983).

Waring (1989) found that CK levels were abnormally elevated in 25 (40%) of the post-poliomyelitis subjects with clinical weakness (N=62) compared to the 8% without clinical weakness (N=13). They also report a correlation between the CK levels and the self-reports of strenuous work performed (Waring 1989).

In a Mayo Clinic study (Windebank 1991), 10 of 32 symptomatic subjects (31%) were found to have mild to moderately elevated CK levels, while none of the 18 asymptomatic subjects had abnormal levels.

A clinical case presentation by Peach (1990) demonstrated that a symptomatic poliomyelitis survivor could reduce an elevated CK level following treatment intervention of protective bracing that reduced the severity of symptoms experienced.

The role of monitoring CK levels on a regular basis to assist in clinical management of the poliomyelitis survivor is not very clear in the literature. Serial analyses, however, could serve as an indicator of the muscle response to an exercise program.

## **POSSIBLE ETIOLOGIES FOR THE ONSET OF NEW SYMPTOMS ASSOCIATED WITH POST-POLIOMYELITIS**

The cause of PPS is not known, but the numerous etiologic hypotheses that have been suggested in the literature will now be described.

### **1. Chronic poliovirus infection**

Viruses have been latent and later reactivate to cause disease (Jubelt 1987). The poliovirus has caused persistent asymptomatic infection in animals (Miller 1981), and in immunosuppressed humans (Davis 1977). Recently, Sharief proposed there is a persistent poliovirus in human subjects (Sharief 1991). He hypothesized that the poliovirus, located intrathecally, is protected against immunologic attack. There were 58% of 36 subjects with PPS that evidenced the poliovirus and there was no evidence in 13 poliomyelitis survivors without PPS. He suggests that for decades, the poliovirus "escaped surveillance of the immune system" and that the "persistent poliovirus infection might then have a gradual but progressive cytopathic effect that would eventually lead to either neuronal-cell lysis or alterations of specialized cellular functions" (Sharief 1991).

Bruno presents the question, "if the poliovirus has been residing unnoticed outside of the motoneurone, how and when did it enter the neuron, how does it produce its 'cytopathic effect', and why has this effect taken decades to become evident?" (Bruno 1991).

### **2. Death of remaining motoneurons with normal aging**

Some studies indicate that people naturally start to lose motoneurons as they age, however this loss is not apparent until after age 60 (McComas 1973, Tomlinson 1977). Poliomyelitis survivors who originally had fewer motoneurons, may consequently suffer a disproportionate loss of function. The increased giant motor units that are heavily relied upon for the poliomyelitis survivor, die off with the normal aging process. Wiechers and Hubbell (1981) claim that EMG changes of abnormal neuromuscular jitter and blocking correlate with the time since the attack of acute poliomyelitis and not with the chronological age of the poliomyelitis survivor. No study has demonstrated an earlier onset of the normal aging process in the post-poliomyelitis survivors.

### **3. Premature aging of cells permanently damaged by the poliovirus**

Abiotrophy (Lewis 1977) is a gradual loss of vitality of cells or an exhaustion of the motoneurons. Any cell that was invaded by the poliomyelitis virus is likely to be permanently damaged (Jubelt 1987). The viral attack may leave some motoneurons functional but impaired, making them vulnerable to dysfunction over time (Mulder 1972).

### **4. Premature aging of remaining normal motoneurons due to an increased metabolic demand**

The remaining motoneurons that have reinnervated denervated muscle fibers are supplying a larger than normal number of fibers. Collateral sprouting of nerves has increased the motor unit territory and there is consequently an increased metabolic demand from the terminal sprouts of these enlarged motor units. Collateral innervation is greater in the previously involved muscles (Martinez 1983) and, it is speculated that previously involved muscles are more likely to develop new weakness and progressive atrophy. This could then result in premature exhaustion of the motoneuron (McComas 1973), however there is no experimental data to confirm this speculation (Bradley 1987).

### **5. Immune-mediated syndrome**

Dalakas (1986) presented evidence of a lymphocytic response in muscle biopsies and IgG oligoclonal bands in the cerebrospinal fluid (CSF) in 7 of 13 symptomatic poliomyelitis patients, whereas the remaining 6 asymptomatic patients had no oligoclonal bands in their CSF. Lymphocytic infiltrates were reported in 40% of the muscle biopsy specimens (Dalakas 1986). The earlier work of Dalakas (1985) identified various abnormalities in T-cell substrates of the poliomyelitis survivors, however there were no consistent changes in the ratios.

The work of Sharief (1991) may cause one to suspect an immunopathological basis for post-poliomyelitis syndrome. The immunoglobulins might be directed at the motoneurons, nerve terminals, or postsynaptic antigens. These patients however, have not responded to immunosuppressant therapy and immunological involvement precipitating the weakness associated with post-poliomyelitis syndrome remains unclear.

## 6. Loss of individual muscle fibers per reinnervated motor unit over time

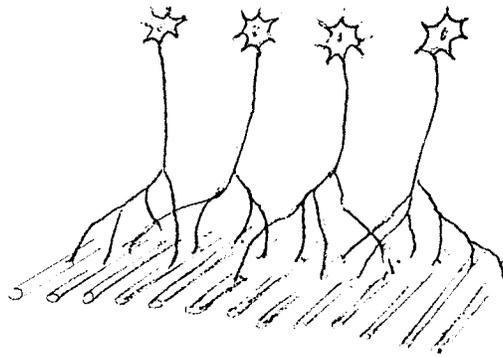
The hypothesis that the number of individual muscle fibers per reinnervated motor unit is gradually decreased over time in poliomyelitis survivors was first proposed by Wiechers (1985). Although mature reinnervated motor units were normal electrophysiologically, neuromuscular jitter was found in poliomyelitis survivors indicative of loss of or impaired innervation of single muscle fibers. He thus concluded there was an association with the amount of neuromuscular jitter and the time since acute poliomyelitis infection.

Grimby proposed "excessive" muscle fiber hypertrophy is apparent in muscles with the greatest reductions in strength secondary to the initial loss of motoneurons (Grimby, 1989). The motor units with higher fiber densities experienced greater increased neuromuscular jitter and blocking (Wiechers 1985). As a consequence, these motoneurons had a greater metabolic demand over time than normal motoneurons; productive or transmission of neural nutrients from soma to nerve terminals may have been overwhelmed (Dalakas 1991). It is speculated that the outlying sprouts will eventually degenerate and axonal arborizations will retract (Dalakas 1991). The more muscle fibers that drop off and undergo atrophy, the more apparent the muscular weakness in the poliomyelitis survivor (Dalakas 1991).

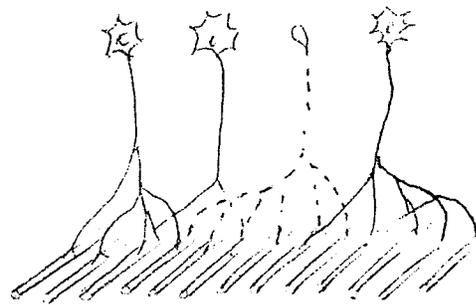
Motoneurons lose their ability to regenerate sprouts, possibly due to physiological stress or the imbalance between the metabolic supply and demand within the motoneuron (Dalakas 1991). Wiechers (1981) reported evidence that exhaustive exercise, during which the intensity is greater than usual for the muscle, may produce muscle fiber damage including fiber splitting. This fiber splitting can occur with chronic denervation and the new myofibers may further tax these enlarged motor units. Poliomyelitis survivors may experience an ongoing denervation and reinnervation process (Cashman 1987a).

Cashman cautions that one must always keep in mind that "overuse" of muscles weakened by poliomyelitis and that operate at maximal capacity with minimal or no reserve, can result in irreversible damage to muscle fibers, to terminal axon sprouts, or even to the somas of motoneurons themselves (Cashman 1987a). Unfortunately, the level and type of acceptable activity for the poliomyelitis survivor is not clear and deterioration of the axon terminals still does not represent the location of the specific pathogenic problem. Neuromuscular transmission requires protein synthesis in the cell soma for maintenance and growth of nerve terminals. Thus, the dysfunction of the axon terminal could be at the cell soma, the axon, or at the terminal boutons.

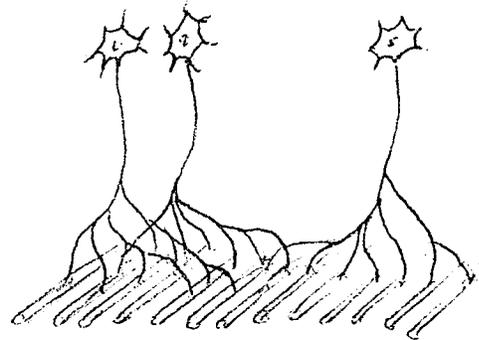
Normal



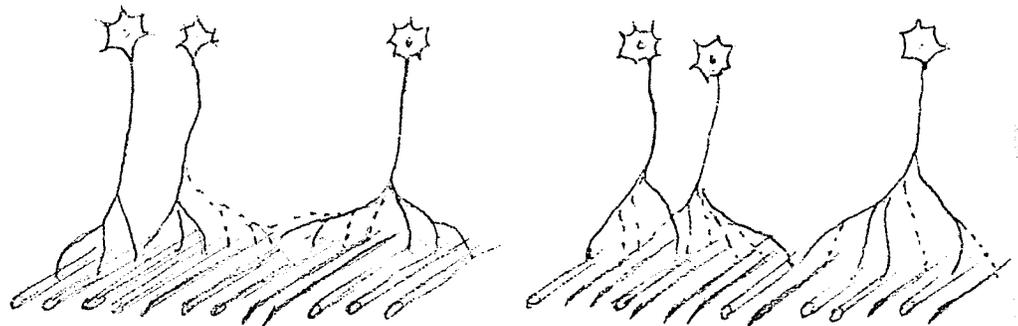
Acute Poliomyelitis



Recovery and continuous remodeling



Post-poliomyelitis pathology



**Figure 10** : Loss of individual muscle fibers in reinnervated motor unit over time (taken from Dalakas 1991).

## PSYCHOLOGICAL IMPACT OF POLIOMYELITIS

The poliomyelitis survivors were hard-driving, time-conscious overachievers as a result of their rehabilitation/recovery and it is reported that many survivors developed a Type A poliomyelitis personality (Frick 1987, Bruno 1987, Bruno 1989, Bruno 1991). The behavior patterns they developed were both physically and mentally challenging in all aspects of their personal and professional lives. They strive for perfection in pursuit of social acceptance.

"The early polio prescription was to establish goals, work hard, and push oneself beyond perceived limits. This message 'fit' well within the highly valued American work ethic. The work ethic requires the individual to take responsibility for overcoming personal adversities by working hard in the pursuit of goals." (Bruno 1991).

The physiotherapy for poliomyelitis survivors continued for years after the acute infection in an effort to achieve "complete recovery" (Meyer 1947). The rehabilitation process for the 1940's and early 1950's consisted of the application of resistive and repetitive strengthening exercises with the intention of encouraging nerve fiber sprouting (Smith 1980). In this period, "persistence in strenuous activity was the great rehabilitation virtue and the pursuit of ambulation second only to the quest for the Holy Grail" (Mailhot 1980). The poliomyelitis survivors discarded their assistive devices as soon as possible in spite of the resulting discomfort, fatigue or awkwardness. They were striving for the "appearance of complete physical normalcy" (Schechter 1961).

In this era, it is believed that "strenuous" exercise was necessary to accomplish reinnervation of orphaned muscle fibres affected by the acute viral infection. Today, any exercise program for the weakness associated with poliomyelitis requires a progressive approach to treatment. Clinicians would be ill-advised to apply an exercise program that would be regarded as "strenuous" to a poliomyelitis survivor. The difficulty in ascertaining whether or not individuals having new health problems partook in an aggressive exercise program, creates a confounding variable.

The literature revealed that the poliomyelitis survivor was potentially at risk to lose the functions that they worked so hard to regain in the rehabilitation/recovery period (Bennett 1958). The advice of the rehabilitation clinicians shifted from the "use it or lose it" to a "conserve it or lose it" philosophy. Some of poliomyelitis survivors perceived this change in philosophy as a "second disability" (Frick 1985), others thought of it as a request to "stop living" (Bruno 1991).

Psychosocial stress may exacerbate symptoms for the poliomyelitis survivors that are confronted with new health problems (Bruno 1991). The clinician should appreciate the perception of the individual that is re-evaluating their cultural expectations, beliefs, and ideals (Scheer 1991). During this transition and life reorganization, the Type A characteristics can be used constructively.

"The qualities learned and valued by polio survivors in the course of adaptation to their primary disabilities - determination, steadfast, consistency and problem solving - are essential tools for life building which can be reinforced and validated in clinical settings, and applied to their management of new secondary disabilities" (Scheer 1991).

The clinician may provide education in proper health habits that include the appropriate selection of exercise, and a balance of activities and rest periods that are dependent upon the residual strength of the individual poliomyelitis survivor. Activities, like exercise, should be performed at low intensity levels. Some behavioral modification techniques can redefine 'work' as structured daily rest periods and these individuals can be encouraged to strive for a new set of priorities in their life.

#### **TREATMENT APPROACH TO POST-POLIOMYELITIS**

The new symptoms associated with poliomyelitis are psychophysiological in nature and require a holistic approach to treatment (Bruno 1991). The poliomyelitis survivor should be approached on a psychological, physical and functional basis. A multidisciplinary approach to intervention would be optimal (Owen 1985, Agre 1989, Halstead 1991) with an emphasis on the wide variety and uniqueness of the problems associated with each poliomyelitis survivor. The treatment approach to poliomyelitis should essentially be based on the specific nature of the complaints and differential diagnosis of the new symptoms. Any other treatable causes for other signs and symptoms, such as disuse muscular atrophy, may then be ruled out.

#### **EXERCISE FOR POST-POLIOMYELITIS WEAKNESS**

DeLorme (1948) claimed that a muscle strengthening program must last a lifetime in order to maintain strength in muscles weakened by poliomyelitis. Bennett and Knowlston (1958) however, reported that "overwork" of partially denervated muscle could lead to muscle

atrophy. "Overwork" was defined as excessive continued voluntary activity of a muscle. "Overwork weakness" was defined as the long-lasting impairment of that muscle, reduced in strength and endurance, that could be reasonably related to the specific "overuse" of the muscle, and where there is a failure to regain the strength with specific exercise (Bennett 1958).

The use of exercise as a treatment intervention for the poliomyelitis survivor then came under the close scrutiny of the medical community. Alsentzer (1986) stated that "strengthening exercises add only short-lived, if any, strength and endurance in patients with post polio syndrome and they may in fact accelerate the development of weakness and loss of endurance".

Paranoia associated with the potential for increasing weakness due to the overuse of the muscles weakened by poliomyelitis, caused skepticism among clinicians about imposing exercise programs. Bradely (1985) states "an unanswered question concerns the use or dangers of physical therapy and muscle strengthening exercises in patients with the post polio syndromes". Although clinicians admit uncertainty about the effects of exercise, Bruno (1985) states "the effect of exercise on unaccustomed fatigue and impaired muscle functioning whether deleterious, therapeutic or both have not been answered empirically". Clinicians did discourage vigorous activity for the poliomyelitis population.

A muscle weakened by poliomyelitis could be susceptible to damage from exercise by:

1. contributing to the neuromuscular dysfunction of the motor units that were previously involved by acute poliomyelitis as proposed by Dalakas (1991) or
2. the exercise may strain weakened muscles and could produce traumatic injury, inflammation, edema and pain in either the muscle body, tendon or fibrous insertion (Herbison 1983).

There are reports about successfully implemented programs with the development of a carefully prescribed exercise regime (Feldman 1985, Fillyaw 1991, Owen 1985), concentrating on muscle weakness and fatigue for the poliomyelitis survivor. The limited research that is available to guide the clinician in treating poliomyelitis survivors complaining of decreasing strength is summarized in Table 6.

Muller (1970) reported that daily maximal isometric contractions (3 at 6 seconds each), performed daily, resulted in an increased strength in the muscles of children, 6-15 years after the acute onset of poliomyelitis.

Herbison (1983) suggested that an isometric (static) or progressive program performed 5-6 times per day may increase muscle strength of poliomyelitis weakened muscles.

In a case study, Twist and Ma (1986) indicate that a combination of moderate exercise with rest improved the strength of a patient with post-poliomyelitis syndrome.

Feldman (1985) introduced a "non-fatiguing" progressive exercise program for the strengthening of muscles weakened by poliomyelitis. They used an isotonic resistive exercise program based on a baseline weight for the number of repetitions that could be performed with ease. The progressive resistive program was based on 50%- 60% of this weight, and it remained constant as the number of repetitions increased. The weight was increased once 30 repetitions were attained. The program continued until the subjects reached a plateau in strength which occurred at approximately 20 - 24 weeks in duration (Feldman 1985). The results of this study also had inferences that the improved strength enhanced ADL and function.

Dean and Ross (1988) believed that an aerobic training program may reduce muscle fatigue while improving cardiorespiratory conditioning. A treadmill was used for training two poliomyelitis survivors that were complaining of new symptoms of weakness. The walking speed and time were gradually increased over the course of their training program three times a week for eight weeks. The subjects were encouraged to monitor musculoskeletal pain and fatigue. The investigators compared results with a control group of normals experiencing complaints similar to those of the subjects involved in the training program. The subjects in the training group showed a significant decrease in the volume of oxygen consumption ( $VO_2$ ), heart rate (hr) and blood pressure (bp) when compared to the untrained subjects (Dean 1988).

In another study by Dean and Ross (1991), they trained seven poliomyelitis survivors on a treadmill three times a week for 30-40 minutes over a six week period. They found no significant difference in the cardiorespiratory conditioning as measured by the  $FEV_1$  and FVC compared to the controls. However, they did report qualitative improvements in the coordination of the gait patterns during ambulation (Dean 1991).

Jones (1989) also studied the cardiorespiratory response to aerobic training by subjects with post-poliomyelitis sequelae. There were 16 subjects that exercised on a cycle ergometer at 70% of their maximal heart rate for a 16 week duration. Jones found that the exercise group was significantly higher than the controls in power output, in exercise time, maximum expired volume per unit time, and in maximal O<sub>2</sub> consumption. There were no untoward events or loss of leg strength as a result of the exercise regime as shown by isokinetic strength measurements (Jones 1989).

Gross and Schuch (1989) presented a case report of an "aggressive" exercise program for 59 year old patient with post-poliomyelitis weakness. This subject's quadriceps and hamstrings were exercised on a Cybex isokinetic dynamometer at 60' and 180'/second 3 times per week for a 6 week duration. The subject did not demonstrate a significant decrease or increase in strength. The investigators concluded there were no deleterious effects from an intensive exercise program with a relatively active post-poliomyelitis patient (Gross 1989).

Einarsson (1991) investigated the effects of a high-intensity isometric and 'isokinetic' exercise program with twelve post-poliomyelitis subjects. The training program was conducted on a Cybex II with 12 sets of 8 'isokinetic' contractions at 180' /second interspersed with 12 sets on four second isometric contractions. The total time of contractions per session was 96 seconds for 3 times per week for a six week duration. Muscle strength measurements and biopsies were conducted before and after the training sessions. They compared strength and histological findings with that of healthy controls, and found that pretraining strength was less than half of the controls' and the mean fiber areas were twice those of the controls'. The post-poliomyelitis subjects had a significant increase in isometric (mean 29%) and isokinetic (mean 24%) strength following the isokinetic/isometric regime (Einarsson 1991).

Fillyaw (1991) prescribed submaximal resistive isotonic exercises every other day on a home program basis. They trained one of the quadriceps and biceps randomly, and the contralateral limb served as a control. The subjects maintained an exercise diary and the investigators reported a compliance rate of 75%. Cybex testing was performed every six months. Results showed a strength improvement of 8.4% over a two year period for these poliomyelitis survivors that used a "non-fatiguing" progressive resistive submaximal exercise program.

reference	# PPS	control	duration	frequency	measure	exercise	results
Muller (1970)	--	no	12 weeks	daily	strain gauge	isometric	↑
Twist & Ma (1986)	1	no	8 weeks	daily and 3x /week	manual muscle test	isometric, isotonic & PNF	↑
Feldman (1987)	6	no	20 - 24 weeks	3x /week	myometer	submax resisted isotonic	↑
Dean & Ross (1988)	2	yes	8 weeks	3x /week	VO <sub>2</sub> , hr, & bp	aerobic	↑
Jones et al (1989)	16	yes	16 weeks	3x /week	VO <sub>2</sub> , hr, & bp	aerobic	↑
Gross & Schuch (1989)	1	no	6 weeks	3x /week	Cybex	isokinetic	--
Einarsson (1991)	12	yes	6 weeks	3x /week	Cybex II	isometric & isokinetic	↑
Dean & Ross (1991)	7	yes	6 weeks	3x /week	FEV <sub>1</sub> & FVC	aerobic	--
Fillyaw (1991)	17	yes	2 years	every other day	Cybex II	submax resisted isotonic	↑

**Table 6:** Literature review summary of the research conducted with exercise programs for post-poliomyelitis subjects.

## ELECTROMYOSTIMULATION

In 1977, Kots, a Russian investigator, rekindled interest in the study of the clinical effects of electromyostimulation (EMS). He claimed that the benefits included pain relief in injured areas, increased blood flow to local areas, strengthened muscle, increased muscle size and facilitated muscle contraction with an improvement in function (Johnson 1977). These findings have not been confirmed because the particular electromyostimulator is unknown.

The effects of EMS on muscular strength have been studied by many investigators. There have been reports of increased muscle girth (Cabic 1987a, Williams 1976) and increased strength (Boutelle 1985, Godfrey 1979, Johnson 1977, Laughman 1983, Selkowitz 1985, Stefanovska 1985, Wigerstad-Lossing 1986, Williams 1976) following an EMS training program. Others have shown that EMS is no more effective for increasing muscle strength (Davies 1985, Halbach 1980, Mohr 1985) than the traditional methods of exercise.

The investigators found that with the application of EMS it was possible to induce strength gains in healthy individuals (Boutelle 1985, Cabic 1987a, Cabic 1987b, Cannon 1987, Currier 1983, Currier 1984, Duchateau 1988, Laughman 1983, Romero 1982, Selkowitz 1985, Stefanovska 1985), some being all male (Cabic 1987, Duchateau 1988) and some being all female (Currier 1983, Mohr 1985). The greatest strength gains with EMS were demonstrated in patients suffering muscular atrophy secondary to various musculoskeletal knee disorders (Godfrey 1979, Johnson 1977, Wigerstad-Lossing 1986, Williams 1976). The magnitude of the strength gains with atrophic muscles could be attributable to the greater capacity of these muscles to gain force, as compared to a preconditioned muscle (Duchateau 1988, Williams 1976).

The studies with strength gains were reviewed to compare the parameters utilized. The studies were difficult to compare (like 'apples to oranges'). Some investigators used alternating current (Cabic 1987a, Cabic 1987b, Romero 1982) and others used a galvanic current (Mohr 1985); some used high frequency (Currier 1983, Laughman 1983, Selkowitz 1985, Williams 1976) and others used low frequency (Cannon 1987, Johnson 1977, Wigerstad-Lossing 1986), some used a sinusoidal wave pattern (Currier 1983, Laughman 1983, Selkowitz 1985, Williams 1976) and others used a rectangular wave pattern (Cabic 1987a, Cabic 1987b, Currier 1979, Johnson 1977, Wigerstad-Lossing 1986). It was difficult to compare the success or failure in strength gains with EMS studies since many of the details associated with the stimulation parameters and the training protocols were not provided (Boutelle 1985, Cannon 1987, Duchateau 1988, Godfrey 1979, Johnson 1977, Romero 1982).

### **Intensity level**

The current intensity, or amplitude, primarily determines the magnitude of the muscle contraction in response to the stimulation. The effectiveness of the stimulation was

reported to be related to the current tolerance; the greater the stimulus intensity, the greater the strength gains (Halbach 1980). The current levels in the EMS studies were reported to be adjusted to the subject's tolerance level (Cannon 1987, Currier 1979, Godfrey 1979, Mohr 1985, Williams 1976).

### **Pulse width**

The EMS pulse width or duration has been associated with reports of pain during the stimulated contraction (Valencic 1986). As the pulse width decreases, the amount of charge passed with each pulse then decreases. The intensity of the current required to evoke a motor response would then increase with narrower pulse width (Benton 1981).

### **Stimulus frequency**

The stimulus frequency of EMS must be sufficient to produce fused tetany; the fusing of individual mechanical responses (twitches) producing a contraction. The required frequency may vary from muscle to muscle, but would range between 30 to 35 pulses per second for the extremities (Benton 1981); the higher the stimulation rate, the faster the fatigue.

### **Duty-cycle**

A duty-cycle is an active-rest (on-off) cycle that should be included in the EMS parameters to reduce the effect of fatigue (Duchateau 1988) with the proper selection of stimulus frequency. The active 'on' time delivers a train of pulses at a specified amplitude, duration and frequency. Each pulse train will determine how long the muscle response will be maintained (Benton 1981). The inactive 'off' time is a rest period that allows the stimulated tissues to recuperate.

### **EMS motor unit recruitment**

When a muscle is stimulated by EMS, the involvement of the motor units are quite different from natural stimulation. The central nervous system activates motoneurons asynchronously, and thus the muscle fibers they innervate contract at different times,

resulting in a smooth uniform contraction. With EMS however, the stimulation of motor units is synchronous, resulting in a decrease in the ability to modulate muscle force output.

The order of motor unit activation depends on the diameter of the motor axon (Denny-Brown 1949). When a muscle is voluntarily activated and the force it exerts increases, the smaller motoneurons innervating type I fibres are activated first. The more powerful and larger type II (fast twitch) motor units are subsequently activated (Henneman 1974). With EMS, it is the type II motoneurons that are the first to be activated because they have the more excitable large diameter axons (Henneman 1974). The motoneurons that innervate the type I muscle fibres are metabolically more capable of a prolonged contraction without fatigue. They are only activated if the stimulus of the EMS is increased to reach the particular firing threshold of these smaller axons (Henneman 1974).

There are some studies that have shown that the muscles in poliomyelitis survivors adapt to the slow oxidative type fibres (Borg 1987, Borg 1988), thus the issue of muscle fatigue is not as important for these individuals.

#### **EMS discomfort**

EMS stimulation can produce an unusual sensation in the stimulated area, because the sensory axons innervating the skin are activated. It has been documented that EMS can produce an undesirable side effect that is described by some subjects as a "cramping" discomfort (Currier 1984). Few investigators commented on this subjective experience, but it was the rationalization for the withdrawal of one subject from a study (Currier 1984). The levels of pain correlated with the intensity of the tetanous contraction of the muscle developed by the electrical stimulation (Boutelle 1985). One study claimed that the subjects described the stimulation as a "pins and needles" sensation; an unusual sensation rather than a painful one (Laughman 1983).

#### **Subject motivation**

The level of motivation of the subjects can also be a factor that influences strength gains associated with the application of EMS. The higher the motivation, the higher the tolerance level to exertion, and the better the subjects tolerance of the peculiar sensations associated

with the stimulation (Williams 1976). Many of the EMS studies did not comment on procedures to control the level of motivation of the subject population (Currier 1983).

### **Hawthorne effect**

There is a potential for a Hawthorne effect with investigating the poliomyelitis survivors because they are an extreme example of a highly motivated (Type A) subject population. This characteristic enables them to tolerate the peculiar sensation associated with EMS stimulation.

Munin (1991) reported that a progressive decline of strength was not evident in their subject population of poliomyelitis survivors. This could be the result of the Hawthorne effect. The biannual measurements of isometric and 'isokinetic' strength may have been sufficient to provide the subjects with the feeling that they received exercise intervention over this three year period.

### **Voluntary exercise with EMS intervention**

For years, physiotherapists have implemented voluntary exercise programs as the conventional approach to the treatment of weakened muscles. A submaximal non-fatiguing voluntary exercise regime would be optimal to strengthen muscles with new weakness in the poliomyelitis survivor (Feldman 1985, Fillyaw 1991) because it would limit the potential for overuse that could result in further degeneration of the surviving motor units. A voluntary contraction would recruit motor units in an orderly fashion from type I to type II. The type II (fast twitch) fibers are difficult to voluntarily activate (Cabic 1987). The application of EMS would preferentially excite these larger diameter axons, dependent upon the stimulation intensity, which would allow activation of these motor units if they were not already active.

The EMS studies report strength gains in muscles that are weakened from disuse associated with various musculoskeletal knee disorders (Godfrey 1979, Johnson 1977, Wigerstad-Lossing 1986, Williams 1976) and could be applied to muscles weakened by poliomyelitis.

A submaximal voluntary contraction augmented with the application of EMS, could hypothetically involve the entire range of motor units. The advantage with the submaximal

voluntary exercise/EMS intervention is the potential strengthening of muscles that are weakened by poliomyelitis. The disadvantage is that a combined treatment regime could present a confounding variable for the interpretation of the results. It would be difficult to ascertain if any strength gains were due to the voluntary exercise, the EMS intervention or both.

## **SUMMARY OF THE LITERATURE REVIEW**

The assessment and treatment of the new problems associated with poliomyelitis presents a challenge for the clinician (Halstead 1991). There has been increasing interest in the cause of post-poliomyelitis syndrome, the nature of the clinical problem and the treatment of the survivors experiencing muscular weakness.

A dilemma that is faced clinically, is the difficulty in determining whether a poliomyelitis survivor is experiencing weakness due to post-poliomyelitis or simply due to disuse atrophy. Which ever the case may be, the post-poliomyelitis survivor may strengthen their weakened muscles by implementing an appropriate exercise program that respects their subjective feelings of pain and fatigue (Feldman 1985, Fillyaw 1991, Gross 1989, Twist 1986). There should be more research conducted in this field to reduce the paranoia associated with exercising partially denervated muscles as a residual of poliomyelitis. The exercise program should be submaximal, non-fatiguing or at a low intensity level to prevent any potential harmful effects for this subject population (Feldman 1985, Fillyaw 1991).

A controlled study was conducted, based on the existing literature, to determine the effects of submaximal voluntary exercise with electromyostimulation (EMS) intervention to the quadriceps femoris weakened by poliomyelitis.

## **OVERALL HYPOTHESIS FOR THE STUDY**

The poliomyelitis survivor that has experienced the onset of new muscular weakness will have an increase in strength of the quadriceps femoris muscle group following an exercise regime of active submaximal voluntary contractions augmented with electromyostimulation (EMS) intervention.

## **SPECIFIC HYPOTHESES FOR THE STUDY**

The specific hypotheses for the study are as follows:

1. to determine if submaximal voluntary exercise with EMS intervention has an effect on the isometric strength of the quadriceps femoris muscle weakened by poliomyelitis;
2. to determine if submaximal voluntary exercise with EMS intervention has an effect on the isovelocitv strength of the quadriceps femoris muscle weakened by poliomyelitis;
3. to determine if submaximal voluntary exercise with EMS intervention has an effect on the endurance of the quadriceps femoris muscle weakened by poliomyelitis;
4. to determine if submaximal voluntary exercise with EMS intervention has an effect on the girth measurement of the quadriceps femoris muscle weakened by poliomyelitis;
5. to determine if submaximal voluntary exercise with EMS intervention to the quadriceps femoris muscle group has an impact upon the perception of pain-related disability for the poliomyelitis survivor experiencing quadriceps femoris muscular weakness; and,
6. to determine if submaximal voluntary exercise with EMS intervention to the quadriceps femoris muscle group has an impact upon the functional capabilities (transfers, stairs and ambulation) of the poliomyelitis survivor experiencing quadriceps femoris muscular weakness.

## METHODS

### DESIGN OF THE STUDY

The study followed an A B design, with subjects serving as their own controls. In the A phase (4 weeks) of the study, there was no intervention. In the B phase (4 weeks), the subjects received an active exercise/EMS intervention for a subsequent four week duration. Measurements were conducted at the commencement of the study (week #1), at the end of the control phase A (week #4) and at the completion of the active exercise/EMS phase B (week #8) of the study as illustrated in Tables 7 and 8.

pre study	mid study	post study
<b>PHASE A</b>		<b>PHASE B</b>
<b>CONTROL</b> NO INTERVENTION  (four weeks duration)		<b>EXPERIMENTAL</b> ACTIVE EXERCISE / EMS INTERVENTION  (four weeks duration)

***Table 7 : Design of the study.***

## SEQUENCE OF EVENTS FOR THE STUDY

	pre study			mid study				post study	
week	0	1	2	3	4	5	6	7	8
Recruitment	X								
Medical examination	X								
Electrodiagnosis	X								
Orientation session	X								
Informed consent	X								
Late Effects of Poliomyelitis Questionnaire	X								
Case Profile Questionnaire	X								
Activity Profile		X	X	X	X	X	X	X	X
CK levels	X				X	X	X	X	X
isovelocity dynamometry	X				X				X
isometric dynamometry	X				X				X
thigh circumference	X				X				X
skin fold measurement	X				X				X
transfer time test	X				X				X
stair time test	X				X				X
gait speed time test	X				X				X
Barthel Index	X				X				X
Pain Disability Index	X				X				X

**Table 8:** Sequence of events for the study. The primary area of investigation was for the impact of an exercise regime with EMS intervention upon: 1. muscular strength, 2. muscular endurance, 3. muscular girth, 4. pain level/disability and 5. functional level for the poliomyelitis survivor with muscular weakness of the quadriceps femoris.

## **RECRUITMENT FOR THE STUDY**

The prospective participants for the study were recruited on a voluntary basis. A Paraphrase (Appendix B), or a brief synopsis of the study, was distributed to clients that attended the Post-Polio Clinic at the Rehabilitation Hospital in Winnipeg, Manitoba. The principal investigator had clinical contacts with the poliomyelitis community. The Post-Polio Network also provided a synopsis of the study in a newsletter (Appendix B) that was circulated throughout the province of Manitoba.

It should be acknowledged that the subject population was a self-referred group. Subjects entered the study with attributes of personal interest and various preconceived expectations.

## **MEDICAL EXAMINATION**

The poliomyelitis volunteers who expressed an interest in participating in the study underwent a physical examination provided by a physiatrist. This served as the preliminary medical screening for meeting the inclusionary and exclusionary criteria for the study.

### **Inclusionary Criteria**

The inclusionary criteria for this study were based upon the diagnostic criteria that Halstead (1991) defines for post-poliomyelitis syndrome:

1. a past medical history of acute poliomyelitis;
2. electrodiagnostic evidence of previous poliomyelitis  
(fibrillations and positive sharp waves, fasciculations, giant potentials of long duration, increased fiber density, jitter and blocking);
3. a 10 year period of neurological and functional stability;
4. onset of new muscular weakness of quadriceps femoris;
5. increasing muscular fatigue or;
6. decreasing functional abilities as determined from their medical history.

### **Exclusionary Criteria**

The exclusionary criteria for the study were as follows:

1. subjects with progressive weakness of the respiratory muscles;
2. ischemic heart disease;
3. recent fractures of the lower extremities or;
4. severe or active arthritis of the lower extremities.

## DIAGNOSTIC ELECTROMYOGRAPHY

Electrodiagnostic (EMG) investigations were performed by a neurologist on the subjects' quadriceps femoris muscle group bilaterally. Four groups were identified based on their EMG results, as illustrated in Table 9.

1. Denervation/renervation (DR) represented the survivor with remote stable poliomyelitis and partial neuromuscular recovery.
2. Active denervation (AD) represented the survivor with new denervation or an unstable poliomyelitis.
3. Normal (N) represented the poliomyelitis survivor with normal EMG results.
4. Complete denervation (CD) represents 'remote' poliomyelitis with no neuromuscular recovery.

The subject population was then coded based upon the electrodiagnostic findings for both the right and the left quadriceps.

group	insertional activity	spontaneous activity	motor unit potentials	interference pattern
DR	normal	absent	↑ duration or ↑ amplitude	reduced (neurogenic)
AD	prolonged	abnormal	↑ duration or ↑ amplitude	reduced (neurogenic)
N	normal	absent	normal	normal
CD	absent	absent	absent	absent

**Table 9:** Electrodiagnostic basis for the classification of the EMG groups.

## **ORIENTATION SESSION**

Prospective participants in the study were provided with an educational session that covered the following information:

1. acute poliomyelitis with the rehabilitation/recovery stages demonstrating:
  - a) normal motor units,
  - b) death of motoneurons during the acute stage of poliomyelitis,
  - c) reinnervation process of the "orphaned" muscle fibers,
  - d) axonal sprouting, and
  - e) the dying back of the axon sprouts during post-poliomyelitis progressive muscular atrophy;
2. symptoms of post-poliomyelitis sequelae;
3. exercise approaches for the strengthening of weakened muscles by poliomyelitis;
4. avoiding the overuse of muscles weakened by poliomyelitis by respecting the sensations of pain and fatigue;
5. the role of energy conservation;
6. the physiological effects of EMS; and,
7. the content of the Informed Consent form (Appendix A).

This session was conducted to promote the understanding of post-poliomyelitis sequelae. The principal investigator was available to answer individual questions and clarify necessary information as required.

## INFORMED CONSENT

The proposed research design was approved by the Winnipeg Municipal Hospital *Pharmacy, Therapeutics, Ethics and Research Committee* (Appendix C) and by the University of Manitoba *Faculty Committee on the Use of Human Subjects in Research* (Appendix C). These committees were responsible for ensuring the respect of the subjects' rights, with protection from any undue physical or emotional trauma that could result from their participation in the study.

The subjects were better able to make an informed decision about participating in the study, following the presentation of information provided in the Orientation Session.

The prospective subjects were informed of the research problem under investigation as the Informed Consent (Appendix A) stated:

"There is controversy regarding the benefits of high intensity exercise programs for the muscles weakened in poliomyelitis survivors".

"I understand the concept of the overuse syndrome and will respect the feelings of pain and fatigue".

"There is no documentation available to support the beneficial effect of EMS on muscles weakened by poliomyelitis, however there is documentation and a physiological rationale to support the application of EMS upon weakened quadriceps femoris muscles".

"There is controversy over the generalizability of results obtained from a single muscle group study to persons as a whole".

The subjects were to have a clear understanding that their participation in the study was for a research investigation and was not intended to serve as a treatment intervention per se, and thus the Informed Consent stated:

"The EMS intervention is NOT a phyiotherapy treatment, but is for the purpose of research in attempts to understand the impact upon strengthening the quadriceps femoris muscle weakened by poliomyelitis".

The subjects were provided with a right to privacy and freedom to decide how much information was to be revealed clinically, and thus the Informed Consent stated:

" I will submit to a medical examination by Dr. S. Vallentyne and a physiotherapy assessment by L. Robertson and colleague".

"I do expect to have my confidentiality protected during my participation in the study and in any future published results. My name will be used only for communication between Dr. S. Vallentyne and L. Robertson".

There was also a right to self-determination meaning that the subjects were informed that they have the right to withdraw from the study at anytime. They were ensured that their withdrawal would in no way affect any clinical interventions nor any future care and thus the Informed Consent stated:

"Whether or not I participate in this study will not influence any treatment that I am receiving in the hospital".

"I may decline to participate or withdraw from the study at any time without affecting my future care".

There was an explanation regarding the nature and extent of the required time and energy as a consequence of participating in the study. To enable the subjects to make an informed decision about participating, the Informed Consent stated:

"The project will require a time commitment for the initial orientation, the pre, mid and post-study evaluations, and regular appointments for EMS intervention in phase B of the study (3X per week for a 4 week period)".

"The daily log has been explained to me and I agree to complete the forms daily and submit them on a weekly basis".

The EMS parameters were selected based on maximal comfort with limited fatigability. There was a subjective interpretation for the 'unusual sensation' that accompanied the EMS stimulation and thus the Informed Consent stated:

"The muscle stimulation may produce an unusual or mildly painful sensation that is similar to a tingling feeling or a 'pins and needles' phenomenon".

The CK levels from the participants was a 'necessary evil' within this research design, because it provided valuable information regarding the potential degradation of the muscle fibers that could have been attributable to the treatment intervention. In order to determine the CK levels, a blood sample was required, that entailed a venule puncture of the radial vein. Since this procedure inflicted bodily harm, the Informed Consent stated:

"I will submit to blood sampling from the radial vein on the first, fourth, fifth, sixth, seventh and eighth week of the study which could result in some minor discomfort or possible bruising from the needle insertion".

## LATE EFFECTS OF POLIOMYELITIS QUESTIONNAIRE

The pertinent demographic information, the poliomyelitis history, the medical history and the subjective entrance complaints of the subjects enrolled in the study were captured in the The Late Effects of Poliomyelitis Questionnaire (Appendix E) adopted from Feldman (1987).

The questionnaire focused on three principal areas:

1. the extent of involvement at the acute onset of poliomyelitis;
2. the extent of functional status at their maximum recovery; and,
3. the onset of new health problems or the changes in the functional status since this maximum recovery.

Halstead (1987) claims that the two main predictors for the onset of new health problems are the extent of paralysis and age of onset of the acute poliomyelitis.

The maximum recovery was defined as the period when the subject had felt they had achieved a level of maximum strength and function lasting for several years. The time duration from the onset of acute poliomyelitis to maximal recovery was approximated. The extent of the residual functional weakness that followed the rehabilitation/recovery phase of acute poliomyelitis was documented, with an indication of the types of appliances or orthoses utilized throughout the years.

The review of the types of new health problems encountered since achieving maximal recovery, was based upon the literature (Halstead 1985, Halstead 1987, Codd 1987, Speier 1987). The onset of increasing fatigue, pain, muscle weakness in previously involved and non-involved extremities and the level of their activities of daily living (ADL) were evaluated.

## CASE PROFILE QUESTIONNAIRE

Prior to commencement of the study, the participants completed a Case Profile Questionnaire (Appendix D) that the principal investigator developed, consisting of seven qualitative questions. The questionnaire was developed to provide an appreciation for the

individuals' physical and social interactions in response to the disruption of poliomyelitis in their lives and their expectations for entering this study.

### **ACTIVITY PROFILE**

The subjects maintained a log of their various daily activities. The Activity Profile (Appendix H) was simple and concise in efforts to maintain subject compliance. The amount of time required for these activities (measured at fifteen minute intervals) was to be captured in a similar method to that of Halstead and Hartley (1975). In order to facilitate additional qualitative information, the subjects were given notebooks that served as daily diaries. This exercise provided insight for the investigator, not only to the types of activities the subjects were performing simultaneously to the study, but also to how they interpreted the amount of pain or fatigue experienced as a result of these activities. The daily diaries served as a tool for self-evaluation and energy conservation planning for the participant. The completion of the diaries was encouraged although not enforced.

### **CREATINE KINASE MEASUREMENTS**

The CK levels were monitored to detect adverse muscle effects of subjects exposed to active/EMS intervention. The CK levels were monitored by using the results from week #1 and #4 as a baseline to compare the percentage change in the weekly measures during phase B of the study. On an a priori basis, it was established that participation would be discontinued if the CK level rose above 1000 U/L, or if the level exceeded five times the baseline value.

### **DYNAMOMETRY MEASUREMENTS**

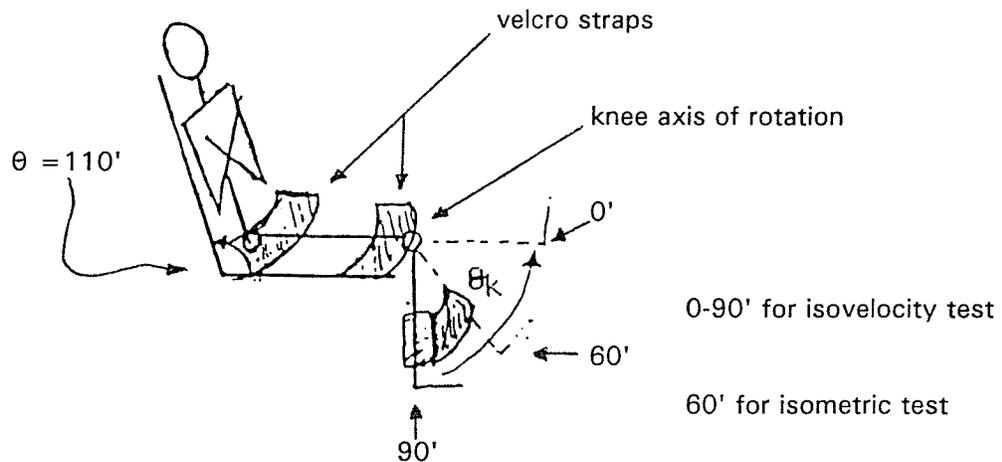
Computerized muscle dynamometry can generate data that is objective and quantifiable. This method surpasses the manual muscle test of the clinician's subjective interpretation of strength with much greater accuracy and sensitivity for strength analysis. The manual muscle test is only semi-quantifiable because it has an ordinal scale of measurement. Manual muscle testing for research should be a 'lost art' with today's technology.

In this study used a Biodex\* dynamometer to measure the isometric and isovelocitity torques generated by the quadriceps femoris muscle group about the knee joint in the saggital plane. The data points were then exported into a spreadsheet to determine:

1. peak - the maximum amplitude of torque for each repetition,
2. mean - the torque value averaged over the repetition,
3. area - the area under the torque curve per repetition.

### Dynamometry Testing Position

Stabilization and positioning of the subject are critical factors affecting the reliability and validity of the dynamometer tests of muscular performance. Thus, the subjects had their chest, pelvis and upper thighs stabilized securely with velcro straps and their hands were placed across their chest to eliminate potential muscle substitution patterns (Mohr 1985, Currier 1977). A lumbar support cushion was made available for comfort. The column height of the dynamometer was set at the estimated axis of the knee joint. The seat position was adjusted so the seat was one inch from the popliteal crease. The knee fixture was set at one inch superior to the pole of the medial malleolus. The seat angle was adjusted for 110 degrees of hip flexion (Mohr 1985; Currier 1977).



**Figure 11** : Dynamometry testing position.

\* Biodex Corporation P.O. Drawer S, Shirley, N.Y. 11967

## Isovelocity Torque

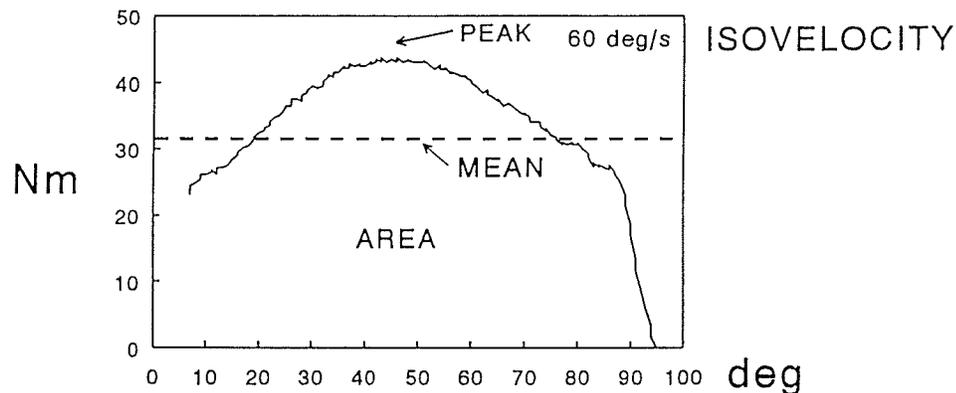
Isovelocity torque is a measurement related to the force generated by the muscle group at a specific velocity of movement (Enoka 1990). This measurement of strength is influenced by the dynamic characteristics of the muscle related to the force-velocity relationship (Hull 1938) and the moment arm/angle relationship of the muscle group of interest (personal communication with Dean Kriellaars).

The angular velocity of the Biodex dynamometer was set at 60 degrees per second for testing of the isovelocity torque of the quadriceps femoris. The principal investigator chose this slow speed concentric contraction setting because it represented the speeds observed in the functions performed by this patient population. The range of motion was set from zero to 90° of knee flexion.

The subjects were instructed to "push" at the commencement of each knee extension repetition.

The principal investigator selected the best three out of five isovelocity repetitions for torque curve analysis at the pre, mid and post study intervals.

A schematic depiction of the derivation of the isovelocity peak, mean and area are illustrated in Figure 12.



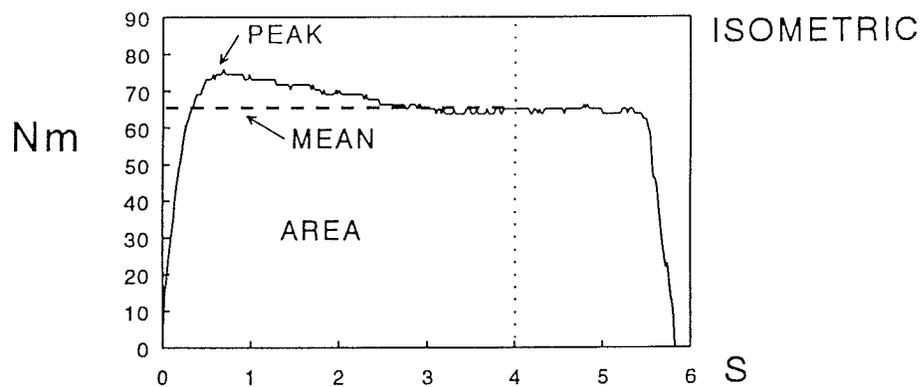
*Figure 12 : The isovelocity torque parameters; peak, mean and area of the curve.*

## Isometric Torque

Isometric torque is a measurement related to the force generated by the muscle group at a 'fixed' joint angle. There is an optimal range of lengths over which a muscle can exert a maximal force. The optimal joint angle for maximal torque product of knee flexion is approximately 60 degrees (Mohr 1985).

The Biodex dynamometer was set at zero degrees per second for testing an isometric torque of the quadriceps femoris. There were three isometric contractions performed at a knee joint angle of 60° flexion. Each contraction was held for at least a 5 second duration, with a 30 second interval between contractions. The subjects were instructed to "push-push-push" at the commencement of the contraction phase and to "now relax" at the resting phase of the test.

Due to the variation in the initiation and cessation of the extension contraction, the isometric torque curves were analyzed over a 4 second interval for consistency. A schematic depiction of the derivation of the peak, mean and area are illustrated in Figure 13.



**Figure 13** : The isometric torque parameters; peak, mean and area of the curve.

## **THIGH GIRTH MEASUREMENTS**

Skeletal muscle may undergo hypertrophy as an adaptation to an exercise strengthening program. The thigh circumference measurements, as an indirect estimate of muscle hypertrophy, were taken at 7 cm. and 20 cm. proximal to the superior border of the patella bilaterally, using a flexible cloth metric tape measure.

The thigh circumference measurement is not exclusively related to the hypertrophy of the quadriceps muscle group, but also to adipose tissue. The subject's weight and thigh skinfold measurement were recorded to account for the variations in the subcutaneous tissue status of the subject at various stages of the study. The skinfold measurement was taken medially at 20 cm. from the superior aspect of the patella bilaterally.

## **FUNCTIONAL TIME TESTS**

The subjects were timed on their functional performance in:

1. chair to bed transfer;
2. bed to chair transfer;
3. ascending a flight of 12 stairs with a 17.78 cm. rise per step;
4. descending a flight of 12 stairs with a 17.78 cm. rise per step; and,
5. ambulating five laps of a 50 meter distance on level terrain.

The subjects were instructed to use their usual walking appliances or assistive devices for the functional time tests. The subjects were requested to perform these functions at a comfortable pace and to stop the activity in the event that they experienced any increase in pain or fatigue.

A colleague physiotherapist measured the objective functional indices (transfers, stairs, and ambulation time tests) to control potential bias in the subject's performance of these experimental tasks.



## **PAIN DISABILITY INDEX**

The Pain Disability Index (Appendix F) was measured at the pre, mid and post study intervals per subject. Although there are many studies that examine variables such as pain intensity (Wolf 1978), there are relatively few scales that have dealt with the disabilities that accompany chronic pain. Pollard defines pain disability as "the extent to which chronic pain interferes with a person's ability to engage in the various life activities" (Pollard 1984).

The Pain Disability Index (Appendix F) is based on a visual analogue scale that focuses on:

1. family/home responsibilities;
2. recreation;
3. social activity;
4. occupation;
5. sexual behaviour;
6. self-care; and,
7. life-support activity.

The subjects rate their level of disability on a scale from 0 (no disability) to 10 (total disability) in the above seven areas of role activities. An overall disability score is determined by summing up the numerical ratings for a score ranging from 0 -70.

Pollard investigated whether the index could discriminate between high and low disability of two groups of patients with chronic (6 months or longer) low back pain with results statistically significant at  $p < .001$  (Pollard 1984). The reliability of the Pain Disability Index for internal consistency was reported by Tait to have a alpha coefficient of 0.871 with an item total correlation ranging from 0.56 to 0.85 with all the correlations being highly significant at  $p < 0.0001$ . (Tait 1987).

Muscle and joint pain have been symptoms associated with post-poliomyelitis syndrome. The Pain Disability Index takes the functional impairment incurred from post-poliomyelitis one step further by evaluating the impact of pain on the individual's perception of their social role.

## **PHASE B CLINICAL MANAGEMENT**

The electrical stimulation of the quadriceps femoris muscle group was accomplished with a two channel neuromuscular stimulator EMS Plus\*. The EMS parameters were established to avoid fatigue and reduce discomfort for the participants. The settings of the EMS parameters were held constant throughout the experiment except the current intensity level that could be adjusted to the subject's tolerance level.

### **EMS Parameters**

The EMS parameters were set as follows:

1. a biphasic AC rectangular symmetric pulse;
2. a 250 microsecond pulse width;
3. a frequency of 35 pulses per second;
4. a duty cycle (on time to off time) of 1:3;
5. simultaneous with both quadriceps stimulated;
6. ramp up at 2 seconds and ramp down and 1 second; and,
7. an amplitude of current for a tolerable muscle contraction without subjective discomfort or fatigue.

### **Electrode Application**

The skin was to be clean, and shaved if necessary, for the application of the 2.54 cm. by 15.24 cm. EMS self-adhering electrodes.

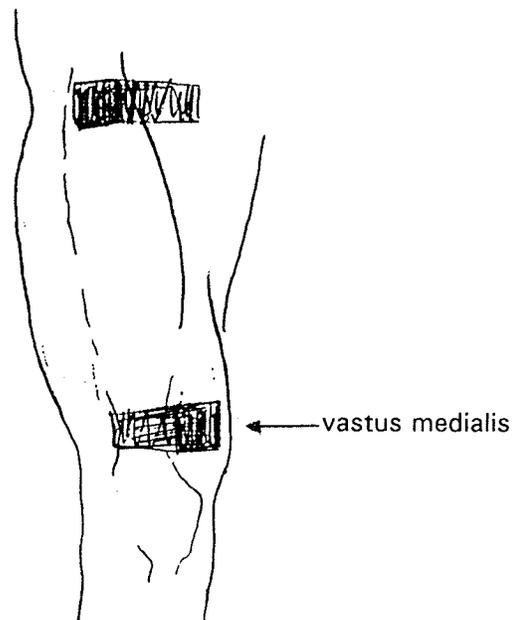
### **Electrode Placement**

The electrode sites were located:

1. inferior to the inguinal ligament over the upper margin of the rectus femoris and vastus lateralis to cover the diverging branches of the femoral nerve and
2. distally over the margin of the vastus medialis of the quadriceps femoris 7 cm. superior to the upper border of the patella.

Upon occasion, the electrodes were slightly readjusted to obtain the optimal response of the quadriceps femoris.

\* *The EMS Plus manufactured by Staodynamics, Inc., P.O. Box 1379, Longmont, CO 80502-1379*



**Figure 14 :** *Electrode placement of the EMS Plus.*

#### **Treatment Log**

A treatment log (Appendix I) was maintained per treatment visit for each of the subjects. Information included the stimulation time, the amplitude of the stimulation, the amount of weight tolerated for the voluntary contractions and a qualitative description of any subjective findings experienced during the treatment session.

#### **Training Positioning**

The subjects were in a sitting position for the training regime with their hips at approximately 110 degrees of flexion and their knees at 90 degrees of flexion with the legs in a dependent position. The subject's back was supported and a single towel could be rolled in the popliteal region for comfort.

#### **EMS Training Protocol**

Fatigue was monitored by the ability of the subject to obtain an active knee extension with the stimulus to at least 60 degrees of knee flexion from the dependent sitting position. If the subject was unable to complete the initial EMS training protocol (as described below) successively, the stage was repeated at the following session. If the subject was able to complete a stage in the EMS training protocol, they progressed to the next stage of the program until they could complete the full 60 minute treatment duration.

The EMS training protocol was as follows:

1. fifteen minutes on, fifteen minutes off, fifteen minutes on, and fifteen minutes off;
2. thirty minutes on, fifteen minutes off, and fifteen minutes on;
3. thirty minutes on, fifteen minutes off, and thirty minutes on;
4. sixty minutes on.

The subjects were requested to actively contract the quadriceps femoris muscle group simultaneously with the EMS stimulation. If the subjects were able to extend their leg 30 degrees while sitting (to knee flexion of 60 degrees), they were to hold that position for the duration of the EMS stimulation, otherwise they were to perform an isometric contraction to the best of their ability. Once the subjects were able to complete sixty minutes of the EMS protocol with an active 30 degree extension of the knee, they progressed to an application of weights to the ankles for resistance at a submaximal level.

#### **SUMMARY OF THE EXPERIMENTAL DESIGN OF THE STUDY**

Single dimension strength gains are important to investigators. However, what is more important to the post-poliomyelitis survivor is a second dimension that can be described as the overall impact of strength gains on their functional abilities.

The Barthel Index will subjectively evaluate basic self-care activities and also the primary mobility activities of the quadriceps femoris such as transfers, walking and stair climbing abilities. The mobility activities of the Barthel Index will be compared to the objective functional measures:

1. transfer time test;
2. gait speed time test; and,
3. stair time test.

A third dimension of the study relates to the impact of exercise/EMS on perceived disability due to pain or fatigue.

A multidimensional analysis of the impact of exercise/EMS is illustrated in Figure 15. Objective strength gains as well as subjective and objective functional measures are incorporated with an appreciation of the psychosocial factors that develop the unique framework of this study.

### 3. ROLE ACTIVITIES

<p>the impact of pain upon:</p> <ul style="list-style-type: none"> <li>*family/home responsibilities,</li> <li>*recreation,</li> <li>*social activity,</li> <li>*occupation,</li> <li>*sexual behaviour,</li> <li>*self-care and</li> <li>*life-support activity</li> </ul>
---

### 2. FUNCTIONAL ACTIVITIES

SUBJECTIVE FUNCTION <i>BARTHEL INDEX</i>	OBJECTIVE FUNCTION <i>TIME TESTS</i>
<ul style="list-style-type: none"> <li>*feeding</li> <li>*chair-bed-chair transfer</li> <li>*personal toilet</li> <li>*getting on &amp; off toilet</li> <li>*bathing self</li> <li>*walking on level surfaces</li> <li>*ascend &amp; descend stairs</li> <li>*dressing</li> <li>*controlling bowels</li> <li>*controlling bladder</li> </ul>	<ul style="list-style-type: none"> <li>*chair-bed-chair transfer</li> <li>*gait speed</li> <li>*ascend / descend stairs</li> </ul>

### 1. OBJECTIVE STRENGTH MEASUREMENTS

<ul style="list-style-type: none"> <li>* isometric quadriceps femoris torque</li> <li>* isovelocit y quadriceps femoris torque</li> <li>* girth measurement of thigh.</li> </ul>
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**Figure 15:** Flow chart of the interaction between the variables creating a conceptual framework for the study.

## STATISTICAL ANALYSIS

The percentage change would be analyzed per subject in the control phase A (% change from PRE to MID study intervals) and the experimental phase B (% change from the MID to the POST study intervals). The PRE study measures were taken at week #1 of the study. The MID study measures were taken at week #4 of the study. The POST study measures were taken at week #8 of the study. The percentage change in the CK levels, torque, girth and functional parameters were statistically evaluated using the paired t-test. The Bartlett test was used to examine the underlying assumption for the homogeneity of variance. The Pain Disability Index was analyzed using Friedman's two way ANOVA.

The percentage of change in the control phase A of the study was determined by the following formula:

$$\frac{\text{MID} - \text{PRE}}{\text{PRE}} \times 100$$

The percentage of change in the experimental phase B of the study was determined by the following formula:

$$\frac{\text{POST} - \text{MID}}{\text{MID}} \times 100$$

The percentage change did not require a correction for the alpha level since multiple comparisons were avoided by analyzing the relative change in phase A compared to phase B of the study. There could be a possibility of a type I error because there were a number of parameters measured within this study. This fact must be considered when interpreting the results.

Power is the calculation that considers the variance per parameter, the mean difference that one wishes to observe, the sample size and the level of significance that is established for the study. Many of the variances were not available in the parameters investigated and the mean difference to anticipate was difficult to determine. Strength gains were the key parameters and an approximate sample size of 12 would be required for a .05 level of significance.

## RESULTS

### SUBJECT POPULATION

There were 32 poliomyelitis survivors who initially volunteered to participate in the research. Eleven subjects withdrew prior to commencement of the study due to time commitments, pending vacation plans, cold weather and transportation difficulties.

Twenty-one subjects underwent initial examinations. There were three volunteers did not meet the inclusionary/exclusionary criteria for the study. The remaining 18 poliomyelitis volunteers were enrolled into the study via informed consent. No subjects dropped-out of this study.

Table 10 illustrates the selected criteria of the poliomyelitis history for the subject population under investigation within this study.

subject #	sex	present age	age of acute polio onset	year of acute polio onset	onset of new health problems	latency period
1	F	46	4	1949	5	37
2	M	55	18	1953	10	27
3	F	42	4	1952	1	37
4	M	79	1	1912	28	51
5	M	53	15	1952	2	36
6	F	43	4	1952	17	26
7	M	38	6	1958	3	29
8	M	66	29	1953	10	36
9	F	66	18	1942	10	38
10	F	49	6	1947	15	28
11	M	47	4	1947	4	39
12	F	37	.25	1953	2	35
13	F	66	5	1929	3	58
14	F	37	.06	1953	1	36
15	M	67	1.5	1924	8	58
16	F	59	21	1952	5	33
17	F	52	23	1961	6	24
18	M	37	.5	1953	2	35
	mean	52.17	8.91	1947	7.3	36.8

*Table 10: Selected characteristics of the poliomyelitis history of the subject population.*

The mean age of the eighteen subjects enrolled in the study was 52 (range of 37 to 79). There were eight men with a mean age of 55 (range of 37 to 79), and ten females with a mean age of 50 (range of 37 to 66).

The mean height of the subjects was 34.8 cm. (range of 22.1 to 30.2 cm.) and the mean weight of the subjects was 82.5 kgs. (range of 45.8 to 115.2 kgs.).

### **Onset of Acute Poliomyelitis**

The distribution for the year of onset of acute poliomyelitis of the subjects ranged from 1912 to 1961.

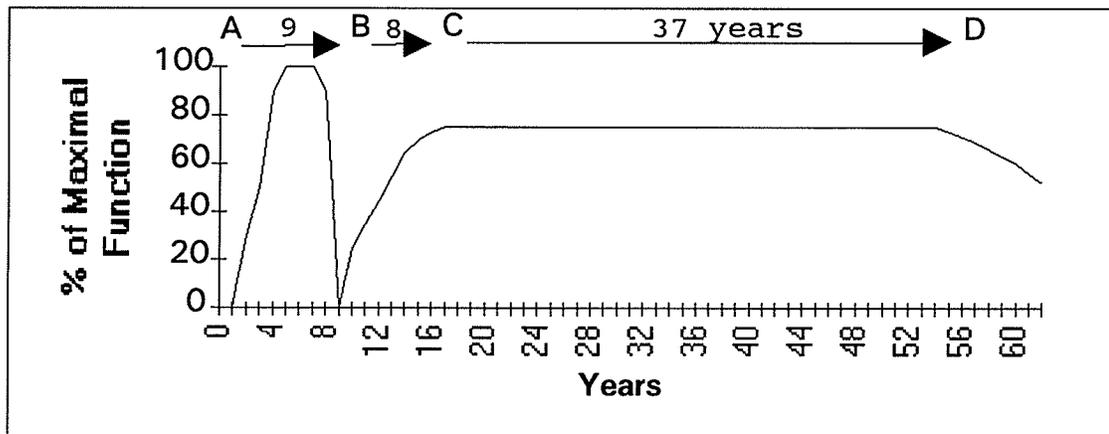
The age of onset for the acute poliomyelitis in this subject population ranged from two months to 29 years, with the mean age of onset approximately 8.91 years (point A to point B in Figure 16).

During the acute phase of poliomyelitis, all subjects reported various degrees of paralysis involving one or more of their extremities.

### **Maximum Recovery**

Maximal recovery was defined as the period when the subject felt they had achieved a level of maximum strength and function lasting for a number of years. The duration from the onset of acute poliomyelitis to maximal recovery was approximated by each subject, and ranged from 1 to 10 years. The mean time for rehabilitation/recovery in this subject population was approximately 8 years (point B to point C in Figure 16). The interval between the rehabilitation/recovery from acute poliomyelitis to the onset of new problems is often referred to as the latency period, and it ranged from 24 to 58 years for this subject population. The period of neurological and functional stability (point C to D in Figure 16) within this group lasted a mean of 37 years prior to the onset of new health problems (at point D in Figure 16).

The subject population followed a similar pattern to the functional profile as described by Halstead (1987).



**Figure 16** : The natural functional history of poliomyelitis. A = birth, B = onset of acute poliomyelitis, C = maximum recovery, D = onset of new health problems (taken from Halstead, 1987).

### Onset of New Symptoms

The one subject that had acute poliomyelitis in 1912, experienced the onset of health problems 28 years ago. Two of the subjects developed new symptoms within the past year. The existence of new health problems for this subject population had a mean interval of 7 years prior to their involvement in this study.

The new health problems and activities of daily living (ADL) for the subject population are listed in Table 11. Probably due to the nature of the inclusionary criteria for the study, fatigue and weakness in previously affected muscles were clearly the primary complaints of this subject population (100%). There were only two subjects that acknowledged a weakness evident in muscles that were not previously affected by poliomyelitis (11%). Functional activities were deteriorating for walking (67%) and climbing stairs (67%), however the ability to transfer (22%), in the subject's opinion was not seriously affected. Fourteen subjects (78%) claimed that their pain was significant to the point that it was a functionally limiting factor. One subject expressed that depression was becoming increasingly difficult to manage, and another complained that cold intolerance was a new health problem.

NEW SYMPTOMS	Number of subjects	Percentage of subjects
fatigue	18	100
pain	14	78
weakness in previously affected muscles	18	100
weakness in previously unaffected muscles	2	11
increased difficulties in walking	12	67
increased difficulties in transfers	4	22
increased difficulties in climbing stairs	12	67

*Table 11: The types of new health problems since achieving maximal recovery in the subject population.*

## CASE PROFILE QUESTIONNAIRE

### Understanding of PPS

The results of the case profile questionnaire revealed that every participant entered the study with a unique knowledge base. This knowledge was obtained through their attendance at Post-Polio conferences, the Post-Polio Network support group meetings, Post-Polio newsletters, information from associated health care professionals with expertise in treating poliomyelitis or through independently examining the poliomyelitis literature.

Thirteen of the eighteen subjects (72%) were members of the Post-Polio Network. Thirteen (72%) of the subject population were well acquainted with the onset of new health problems associated with their history of previous poliomyelitis. In spite of the wide diversity in their level of understanding about poliomyelitis, all subjects could describe post-poliomyelitis syndrome adequately.

"Some of the anterior horn cells in the spinal cord have been destroyed as a result of the acute polio. Existing horn cells still connected to the muscles have caused the muscle fiber to sprout (at the end of the muscle) to connect the muscles that have been cut off due to the destruction of the damaged horn cells. The implications are

that these muscle fibers have been overused now over the years and are deteriorating causing weakness, pain and fatigue."

"Years following the acute attack of the poliovirus, there was a gradual loss of muscle strength with increased pain and fatigue."

"When the acute polio occurred many motor nerve cells died. The remaining motor nerve cells had to send out new nerve terminals to supply signals to the muscle fibers that had lost their nerve cells. Years later these nerve terminals may be affected and losing their ability to function."

### **Understanding the symptoms associated with PPS**

It was very clear that all subjects were aware of the overuse concept and the associated symptoms for muscles weakened by poliomyelitis.

"In my case, I realized that I was experiencing overuse when the usual exercise programs were not working for me - I was unable to build endurance with increasing repetitions of a particular exercise. I became so fatigued that I could do NO more".

"All my life I have been using what muscle I have left from polio twice as much as a normal person. Look at the size of me, I would say that there would be cause for overuse - no wonder I have pain!"

"People with polio tend to overuse their weakened muscles just with ordinary activities throughout the day. They are often not aware that they are damaging their muscles at the same time. I thought that I had to put up with this pain and fatigue due to my polio weakness. Now it all makes sense, I know that I have to listen to Lorraine, and respect these feelings of pain, and take more rests - it's for my own good".

Five (28%) subjects were recently diagnosed with post-poliomyelitis syndrome (PPS) and were attempting to appreciate their progressive dysfunction and recent onset of increasing muscular weakness.

"I know that when I do too much, I become weak and exhausted, but I haven't exactly figured out what 'too much' is."

### **Allied health care interventions**

Throughout the years, the subject population underwent a wide variety of medical interventions provided from allied health professionals such as physiotherapists,

occupational therapists, chiropractors, nursing services, psychological counselling and orthotics.

Ten (56%) of the eighteen subjects were attending post-poliomyelitis hydrotherapy sessions prior to the commencement of the study and were encouraged to continue with their involvement in the program. One subject had recently commenced a physiotherapy program and was requested to postpone intervention until after the study. The subjects that were on a home exercise program had sought advice from a physician, physiotherapist or an occupational therapist. These programs were range of motion exercises with some stretching procedures specific to the needs of the participant.

Some participants expressed frustration with the inadequacies of our health care system in terms of meeting the unique needs of this patient population.

"I get a dozen different answers to the same question - doctors, nurses etc. I don't think that they know much about polio. I'm really starting to lose faith in our health care providers. First they didn't believe that I was having these problems - they told me that it was all in my head. Then I'm told that I have post polio syndrome - but they don't know what causes it. Now I am told that they are not sure what can help me.

#### **Self-administered interventions**

The subjects self-administered many other beneficial activities including walking, riding a stationary bicycle, taking hot baths, yoga, losing weight, ergonomics with proper body mechanics and energy conservation techniques.

"I currently deal with energy conservation by choosing one activity per day (eg. laundry or shopping etc.) and I have to plan my day around it so that I don't do anything else strenuous. I usually alternate mild-moderate activities with a periods of rest throughout my day."

"If you have difficulties with your polio, the secret ingredient for coping with activities is to know how to pace yourself".

#### **Subjects' expectations for the study**

When asked what their expectations were with regard to the study, the responses ranged from "nothing" to "contributing to the growing research that will ultimately help me". Many

subjects were optimistic in terms of gaining an increased knowledge base about post-poliomyelitis. Subjects that indicated a hope to strengthen their muscles, improve function or decrease their level of discomfort, were reminded that their involvement in the study was primarily for the pursuit of research and not intended for individual treatment intervention.

## **ACTIVITY PROFILE**

The activity profile revealed that one subject was pushing beyond personal limits. This individual was repeatedly reminded of the possible consequences of overuse on muscles weakened by poliomyelitis.

According to the activity profiles, that the remaining subjects had organized their day to incorporate rest periods and were making concerted efforts to plan activities alternately from minimal to moderate physical demands.

Fourteen (78%) of the eighteen subjects that returned their daily diaries. Some subjects used this media to describe their personal polio 'biographies' similar to that described by Scheer (1991). One subject wrote;

"The necessity of managing energy and activity has been an interest of mine since post polio. I have attempted to obtain the optimum level of an active lifestyle within the constraints of my physical and respiratory disabilities. The factors that I have considered have been those such as diet, state of mind, relationships, fatigue, activities, exercise and rest. Initially, and for many years, I had the desire to find the best combination of these factors, but with the overall objective of pushing my limits most of the time. With the first suggestions of a post-polio syndrome in the early eighties, it was necessary to change this life long attitude. It was now obvious that I needed more rest and resorted to the use of assistive devices in my daily life. This was a shift in my philosophy but was still within the concept of obtaining my optimum level of lifestyle. I now wonder if my main problem is that I am not getting enough exercise."

The diary portrayed subject's interpretation of post poliomyelitis syndrome and a cognitive organization of how they were adapting in context with their unique life experiences.

## ELECTRODIAGNOSTIC FINDINGS

There were 69% of the subjects that had their quadriceps femoris muscle group classified as denervation/renervation (DR), 8% as active denervation (AD), 11% as normal (N) and 11% as complete denervation (CD).

The electrodiagnostic classification of the individual subjects are summarized in Table 12.

1. denervation/renervation = DR
  - remote stable poliomyelitis with partial neuromuscular recovery
2. active denervation = AD
  - new denervation or an unstable poliomyelitis
3. normal = N
  - normal EMG findings
4. complete denervation = CD
  - remote poliomyelitis with no recovery

Subject	Left	Right
1	DR	DR
2	AD	AD
3	CD	DR
4	N	DR
5	CD	CD
6	DR	DR
7	DR	DR
8	DR	AD
9	DR	DR
10	DR	N
11	DR	N
12	DR	DR
13	DR	N
14	CD	DR
15	DR	DR
16	DR	DR
17	DR	DR
18	DR	DR

**Table 12** : *The classification of the electrodiagnostic findings of the subject population.*

Post-hoc statistical analysis of the electrodiagnostic categories were conducted on strength and functional parameters. The measures failed to reveal a relationship between the EMG results and the outcome measures.

## CK LEVELS

No subject manifested an excessive rise in CK levels (5 time increase from the phase A baseline or more than 1000 U/L) that would have necessitated withdrawal from the study.

The CK levels of the subjects are outlined in Table 13. Five subjects entered the study with clinically elevated CK levels. These levels remained elevated but stable throughout the study.

Subject	week #1	week #4	week #5	week #6	week #7	week #8
1	235	226	354	226	251	220
2	109	136	148	105	222	79
3	132	137	101	100	133	113
4	73	74	93	91	87	85
5	297	251	213	221	196	171
6	121	63	97	79	69	85
7	168	156	225	257	173	214
8	67	72	74	67	79	69
9	62	58	44	61	51	67
10	68	112	68	462	80	68
11	88	80	60	84	79	86
12	67	58	65	78	79	93
13	84	91	89	82	78	150
14	84	76	69	91	76	82
15	678	581	587	737	661	746
16	200	220	408	302	244	235
17	357	382	396	305	323	306
18	149	109	109	123	131	119

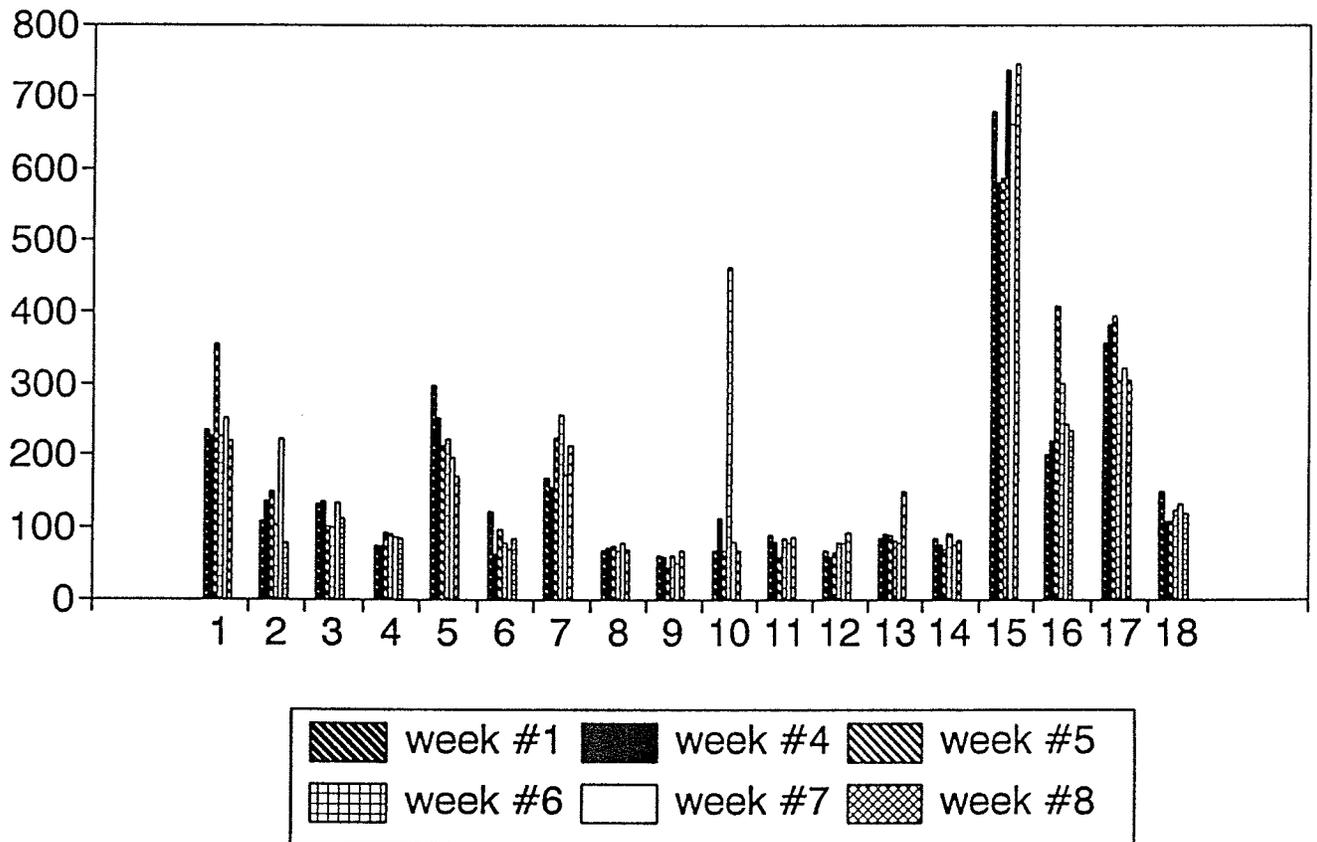
***Table 13:*** CK levels of the subject population in phase A and phase B of the study.

The absolute values of the CK levels per subject are illustrated in Figure 17. The mean values of the weekly CK levels are illustrated in Figure 18. The average level of percentage change between week #1 and #4 is illustrated with the percentage change for week #5, #6, #7 and #8 in Figure 19, to compare the percentage change of the CK levels per subject. All but one subject remained within a +/-100% change of their baseline CK measure. One subject had a +413% change in their CK level in week #6 of the study. This was found to be within the upper limit guideline of +500% change as established for the study. This

individual denied any exceptional increase in pain or fatigue, and resumed their baseline measurement range prior to week #8 of the study.

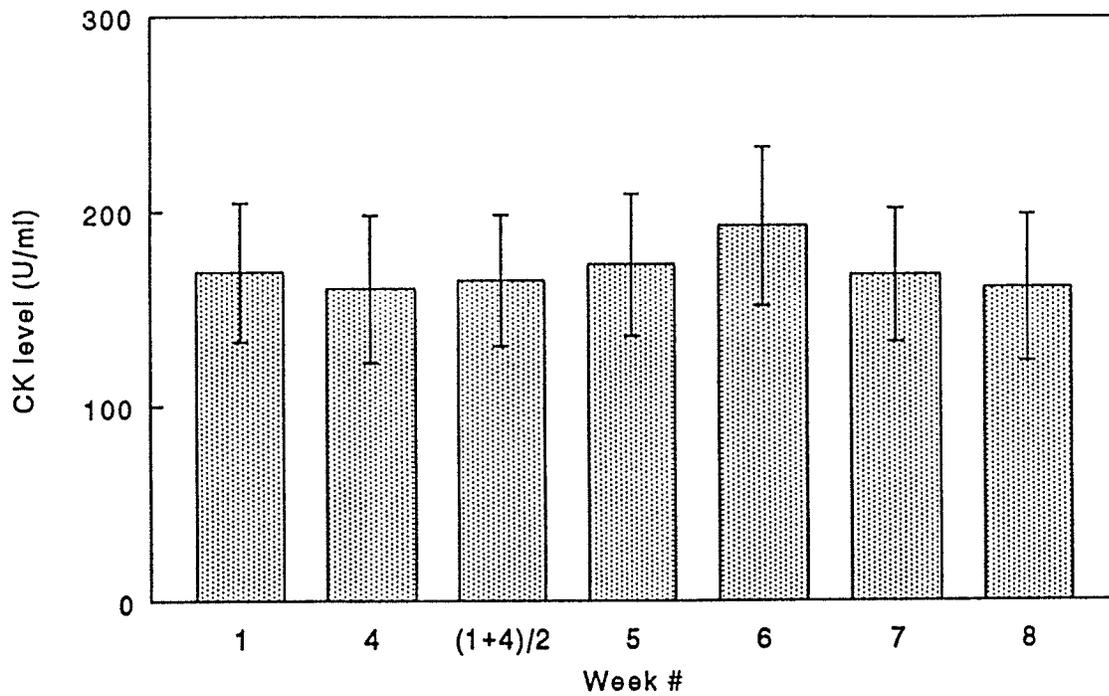
## CK LEVELS

### Range of the Absolute Values



*Figure 17: Absolute CK levels of the subjects from week #1 to #8.*

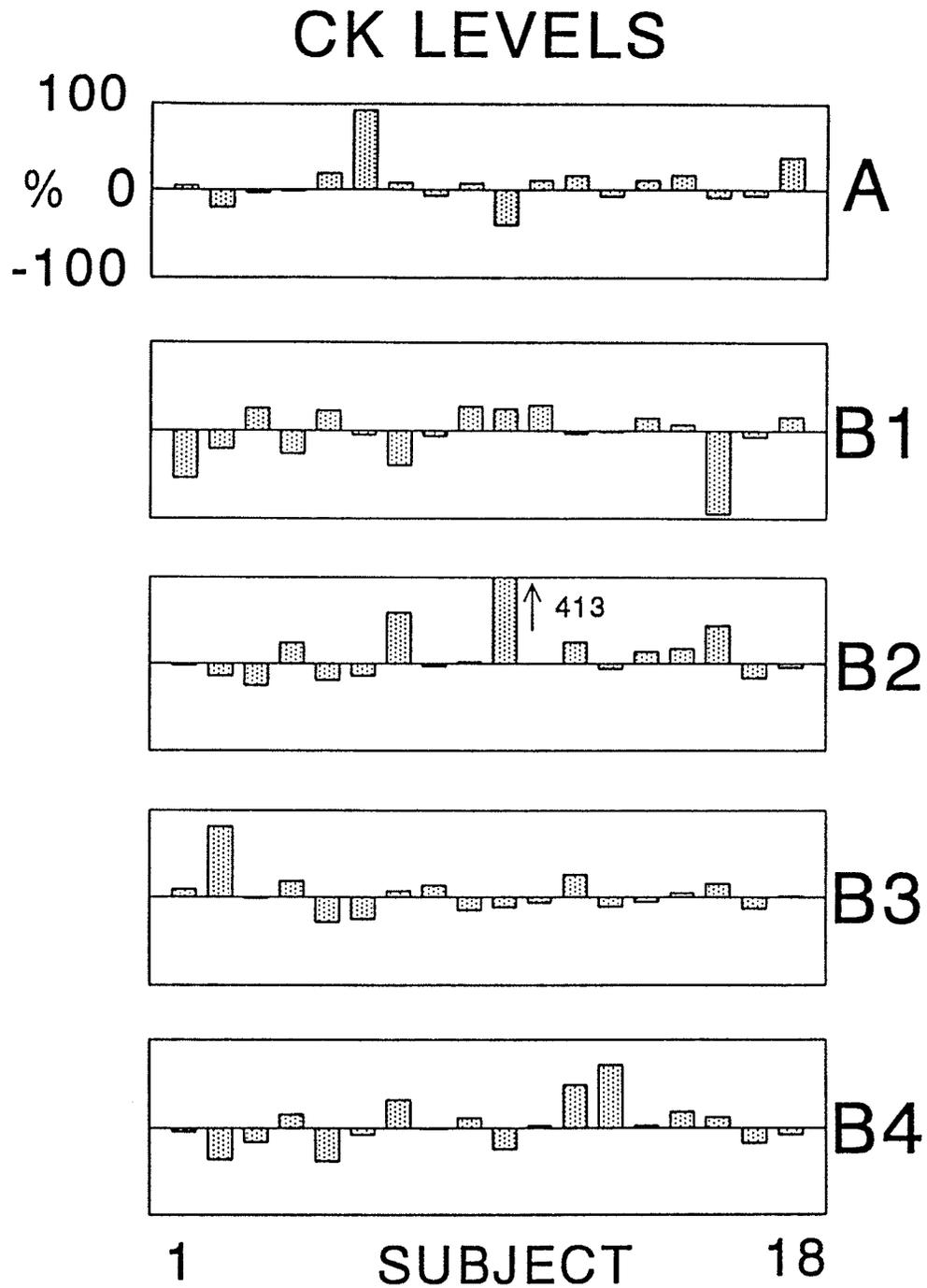
## Weekly CK Level Means



**Figure 18.** The means of the weekly CK levels.

The percentage change in the CK levels in phase A was not significant compared to the weekly measures in phase B of the study.

The CK data that was analyzed using cluster analysis uncovered a natural grouping of high, intermediate and low CK levels within the sample population. Statistical analysis revealed that strength, and functional gains were evident independent of these CK groupings.



**Figure 19** : Percentage change in the CK levels ( $\pm 100\%$  on the y axis of each graph above) per subject (#1-18 on the x axis of each graph above) in phase A (the average of the CK levels from week #1 and #4 of the study) as illustrated in the first graph and compared to the weekly percentage change in phase B at week #5 (B1), week #6 (B2), week #7 (B3) and week #8 (B4) of the study.

## **TORQUE MEASUREMENTS**

The individual subject data for the isovelocity and isometric strength parameters are found in Appendix J.

The mean values of the pre, mid and post study, left and right knee extensor isovelocity torque parameters (peak, mean and area) are illustrated in Figure 20.

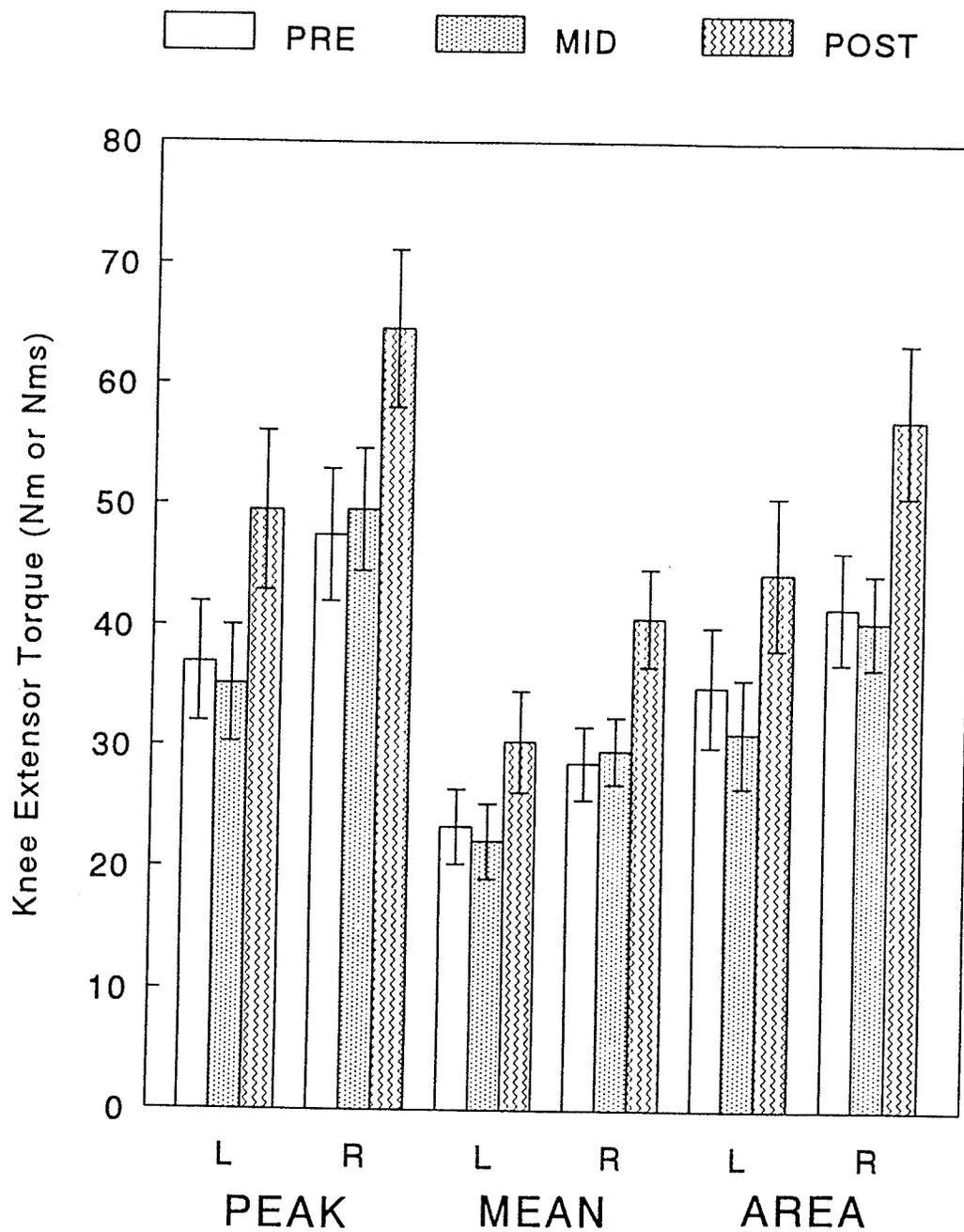
The percentage change of the strength parameters (peak, mean and area) of the left and right extensor isovelocity torque in phase A compared to phase B of the study is illustrated in Figure 21.

The mean values of the pre, mid and post study, left and right extensor isometric torque (peak, mean and area) are illustrated in Figure 22.

The percentage change of the strength parameters (peak, mean and area) of the left and right extensor isometric torque in phase A compared to phase B of the study is illustrated in Figure 23.

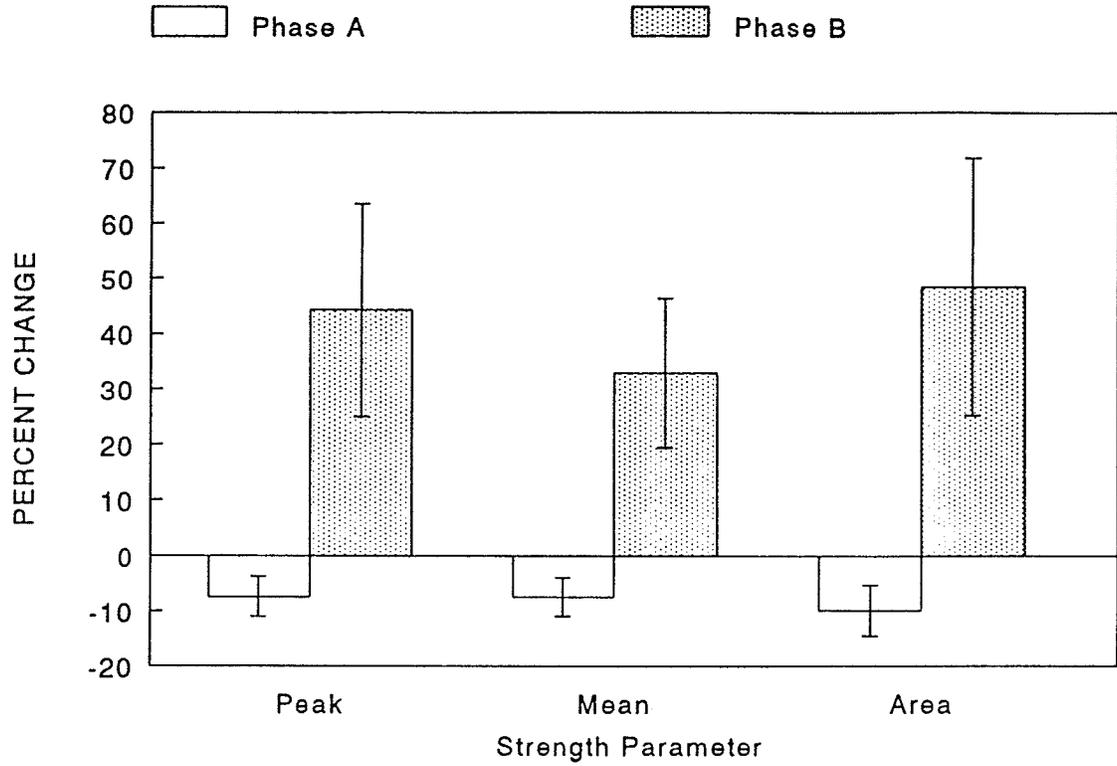
The summary of the mean percentage change of the strength parameters in phase A compared to phase B of the study is illustrated in Figure 24.

# Isovelocity Strength

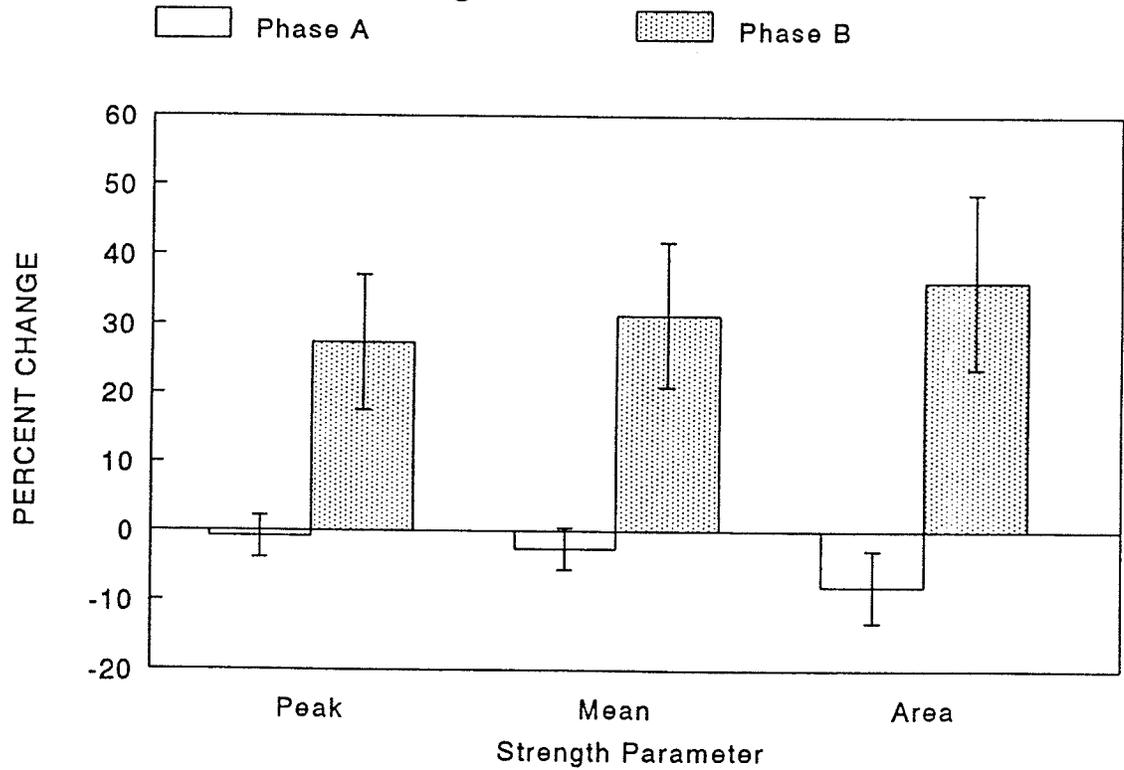


**Figure 20:** The mean values of the pre, mid and post study strength parameters of the left and right extensor isovelocity torque.

# Isovelocity Strength Left Knee Extensor

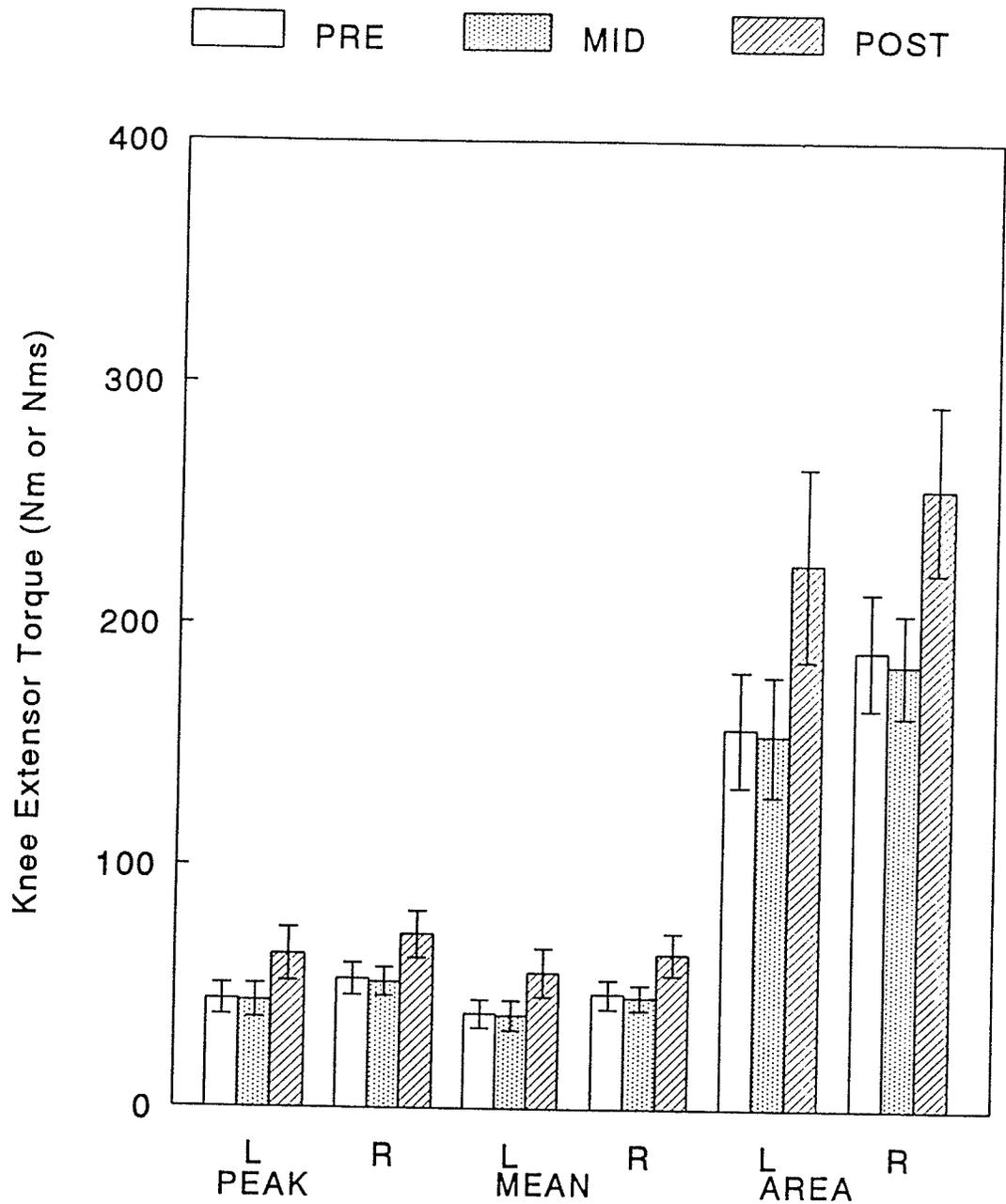


# Right Knee Extensor



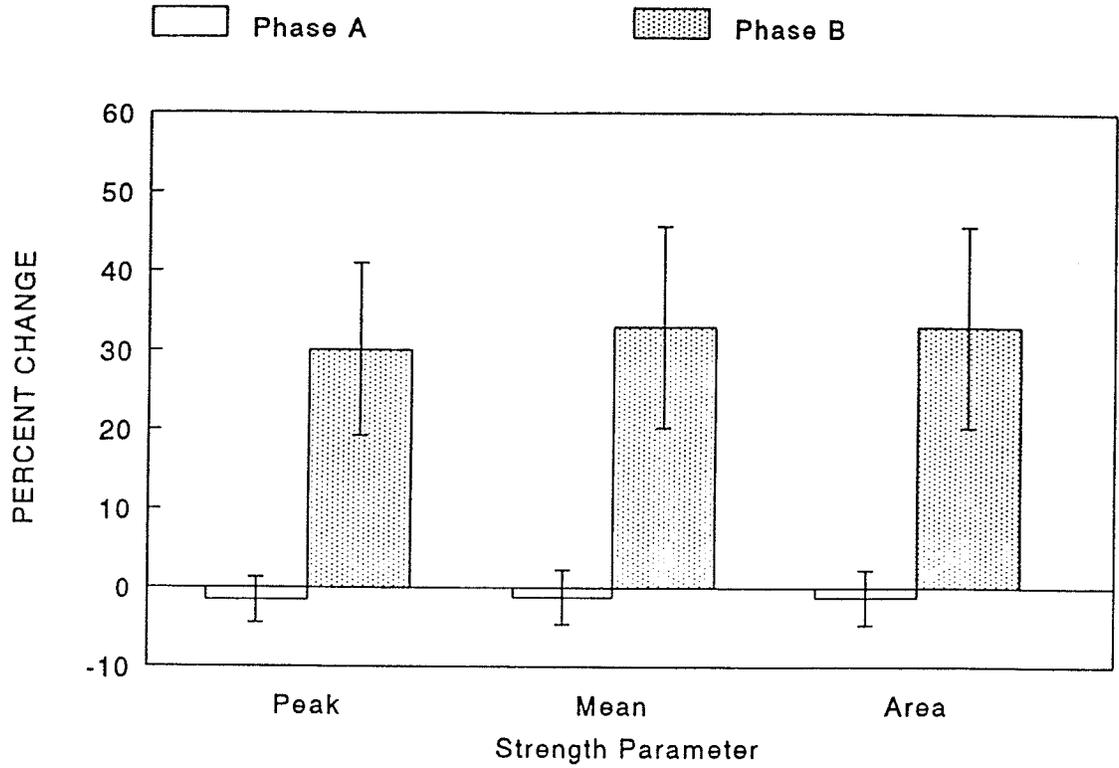
**Figure 21** : The percentage change in the strength parameters of the left and right extensor isovelocity torque in phase A compared to phase B of the study.

# Isometric Strength

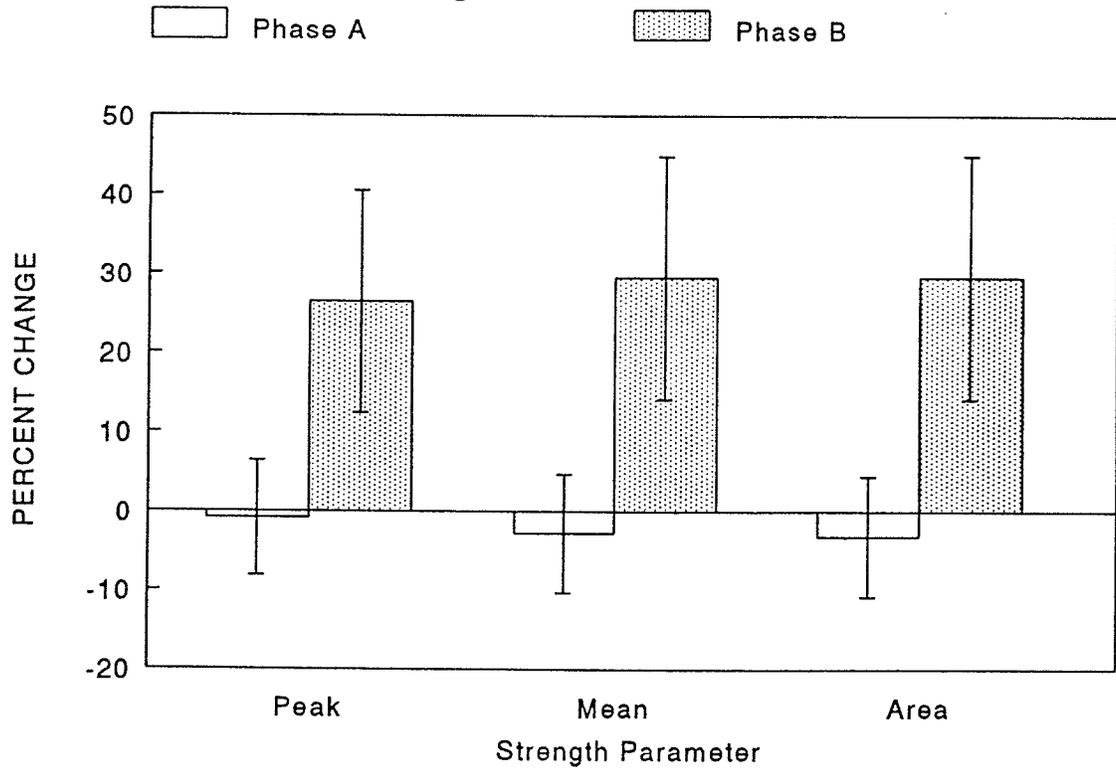


**Figure 22:** The mean values of the pre, mid and post study strength parameters of the left and right extensor isometric torque.

# Isometric Strength Left Knee Extensor

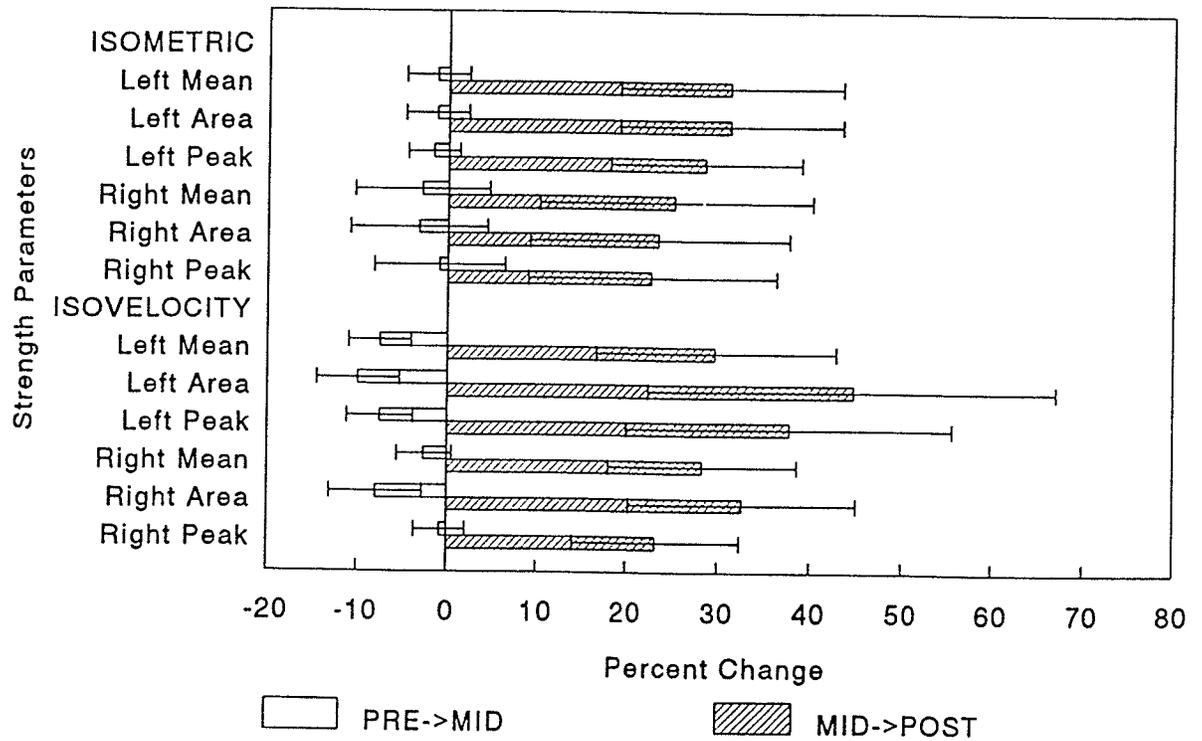


# Right Knee Extensor



**Figure 23:** The percentage change of the strength parameters of the left and right extensor isometric torque in phase A compared to phase B of the study.

## Post-Polio Study Effects of Quadriceps Training



**Figure 24:** The summary of changes of the mean percentage change of the isovelocity and isometric strength parameters in phase A compared to phase B of the study..

There was a significant increase in bilateral isovelocity torque as well as left isometric torque (peak, mean, and area values  $p < .05$ ); and there was a non-significant increase ( $p < .1$ ) in the right isometric torque.

Table 14 shows the results of the statistical analysis (paired t-test) of the percentage change in strength for phase A (pre -> mid) compared to phase B (mid -> post) of the study. The alpha level of significance and the percentage change of isometric and isovelocity torque parameters per extremity are illustrated in Table 14.

KNEE EXTENSORS		LEFT		RIGHT	
		alpha level	% change	alpha level	% change
ISOVELOCITY	peak	0.013	+51.7	0.01	+28.1
	mean	0.006	+58.3	0.004	+33.9
	area	0.008	+58.3	0.003	+44.0
ISOMETRIC	peak	0.008	+31.6	0.095	+27.3
	mean	0.014	+34.2	0.069	+32.3
	area	0.014	+34.2	0.067	+32.7

**Table 14:** The statistical analysis (paired t-test) of the isovelocity and isometric torque parameters illustrating the alpha levels and the percentage difference from phase A to phase B of the study.

## BODY WEIGHT AND GIRTH MEASUREMENTS

The pre, mid and post study thigh circumference measurements are found in Appendix K. The pre, mid and post study weights and thigh skinfold measurements are found in Appendix L.

There was no significant change in the subjects' thigh circumference, body weight or skinfold measurements throughout phase A or phase B of the study.

## FUNCTIONAL PARAMETERS

The pre, mid and post study measures of the mean transfer performance time are illustrated in Figure 25. The percentage change in transfer performance time of phase A compared to phase B is illustrated in Figure 26.

The pre, mid and post study measures of the mean stair performance time are illustrated in Figure 27. The percentage change in stair performance time of phase A compared to phase B is illustrated in Figure 28.

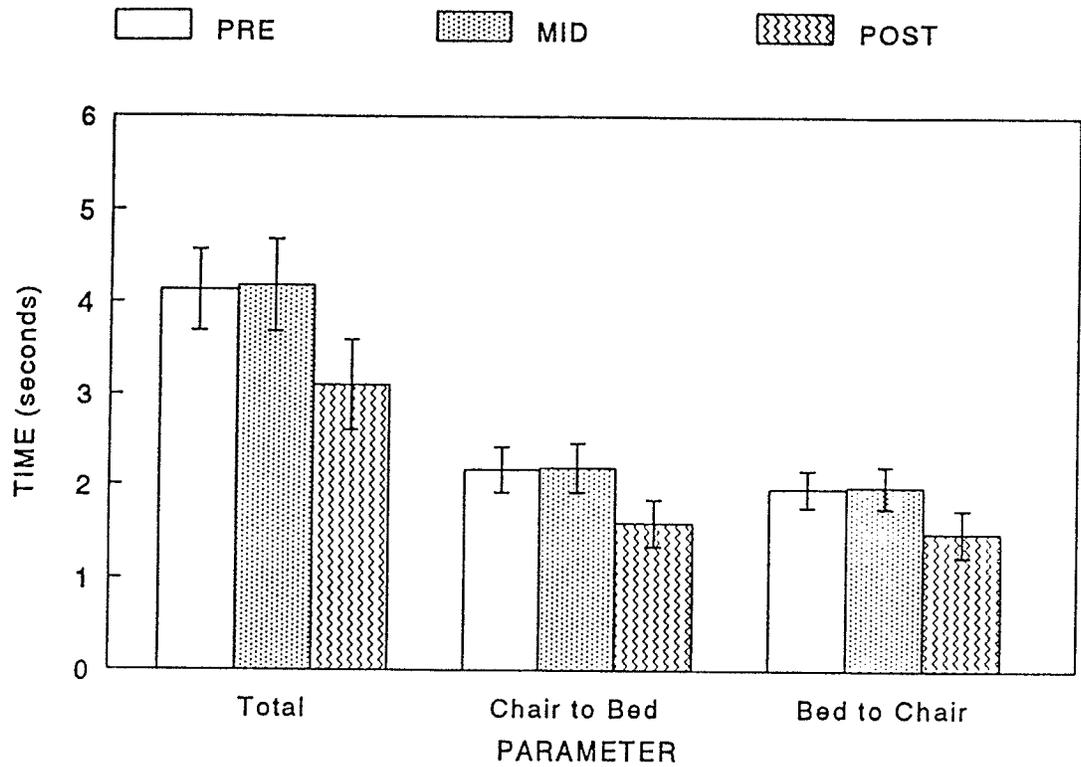
The pre, mid and post study measures of the total ambulation distance are illustrated in Figure 29. The percentage change in the total ambulation distance of phase A compared to phase B is illustrated in Figure 30.

The pre, mid, and post study measures of the mean ambulation time of lap #1, #2, #3, #4 and #5 are illustrated in Figure 31. The percentage change in the ambulation time of lap #1, #2, #3, #4 and #5 of phase A compared to phase B is illustrated in Figure 32.

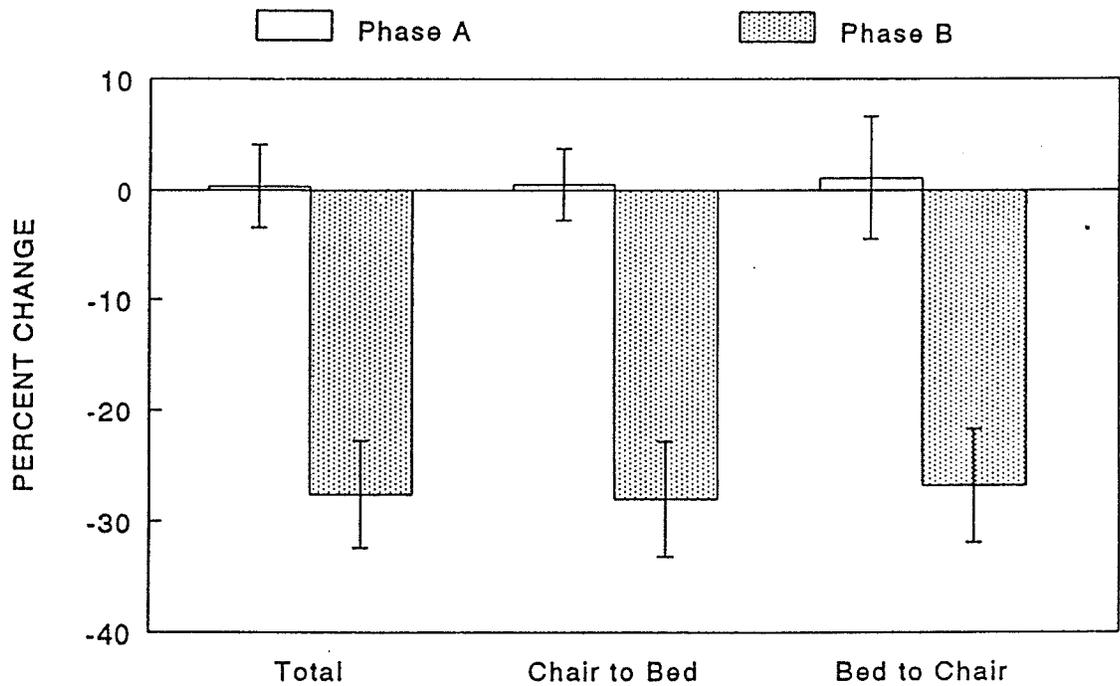
The summary of the mean percentage change in the functional parameters of phase A compared to phase B is illustrated in Figure 33.

Table 16 shows the statistical results (paired t-test) of the percentage change in the functional parameters of phase A compared to phase B. There was a significant improvement in the transfer ( $p < .01$ ), stair climbing ( $p < .01$ ), ambulation lap time tests ( $p < .01$ ) and total distance ambulated ( $p < .05$ ).

# TRANSFER PERFORMANCE

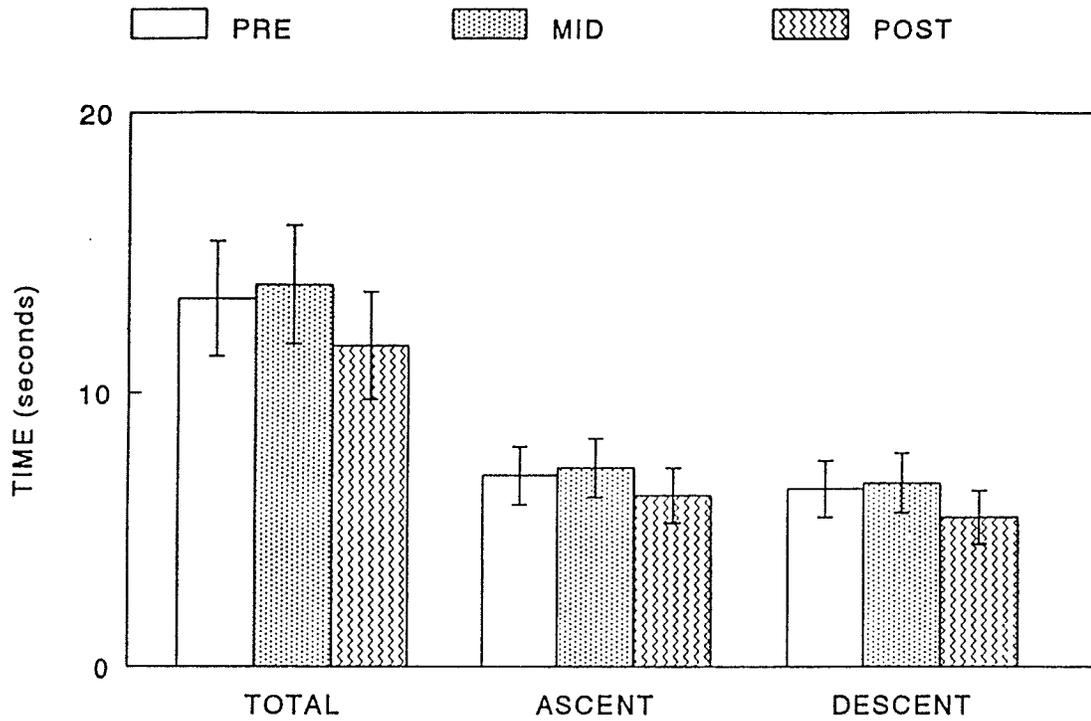


**Figure 25:** The pre, mid and post study measures of the mean transfer performance time of the subjects in phase A compared to phase B of the study.

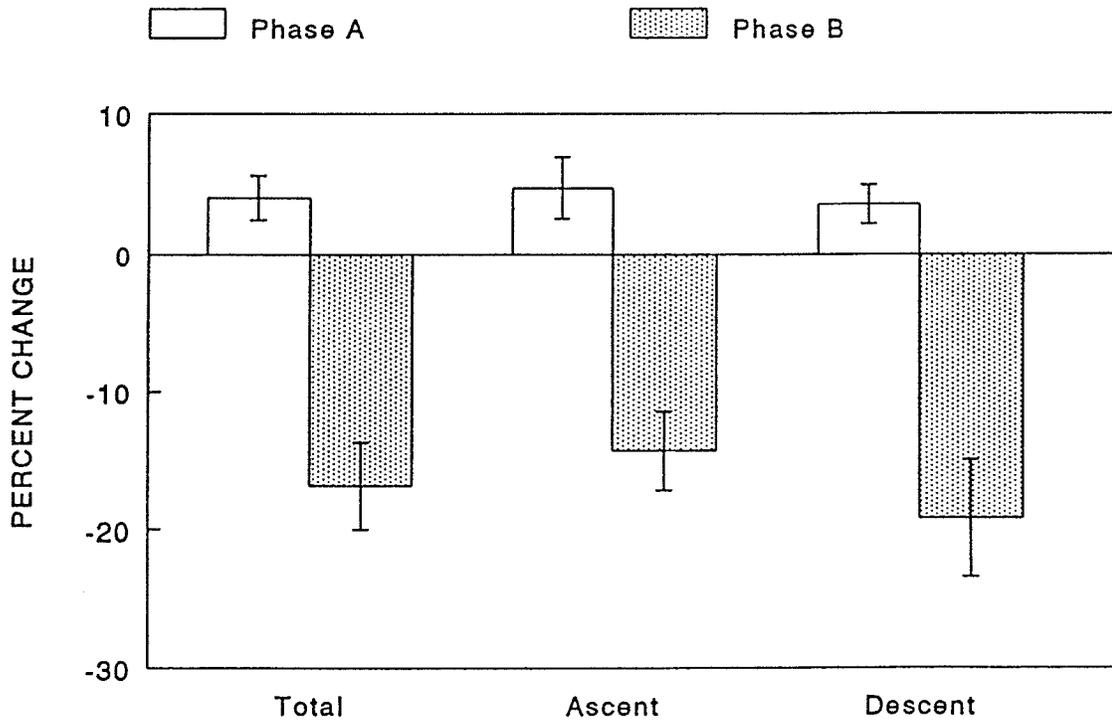


**Figure 26:** The percentage change in the mean transfer performance time of phase A compared to phase B.

# STAIRS PERFORMANCE

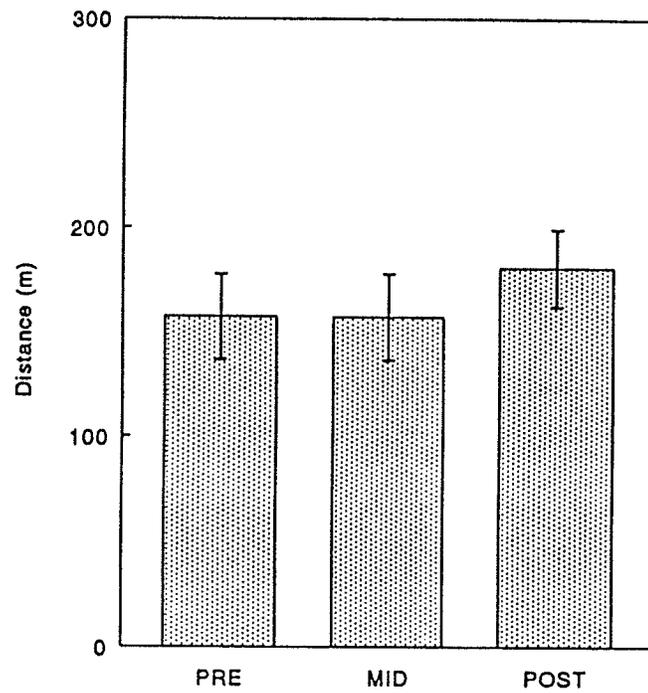


**Figure 27:** The pre, mid and post study measures of the mean stairs performance time tests.

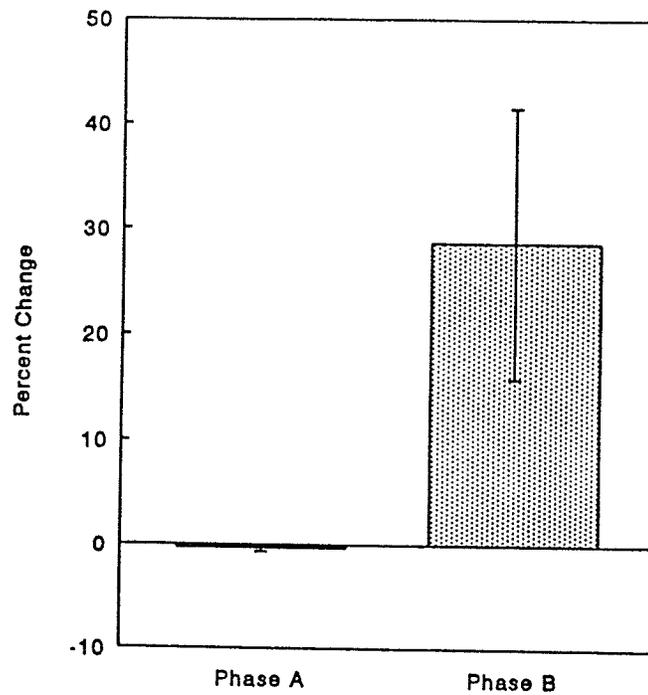


**Figure 28:** The percentage change in the stairs performance time tests in phase A compared to phase B of the study.

## Ambulation Performance

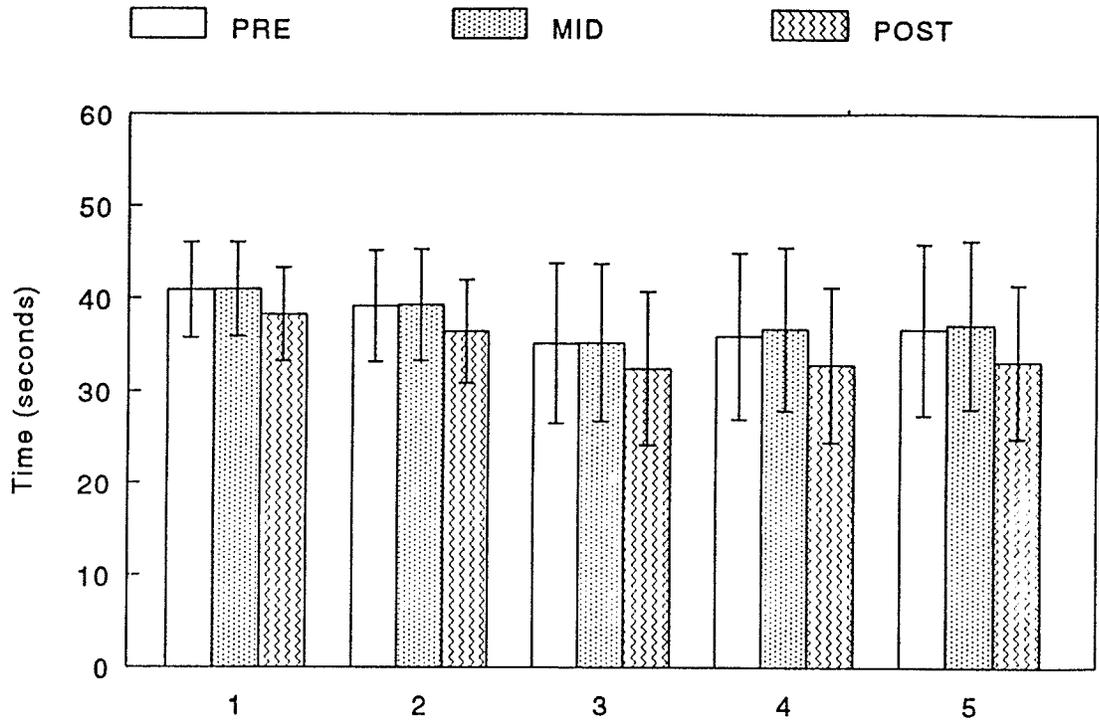


**Figure 29:** The pre, mid and post study ambulatory distance.

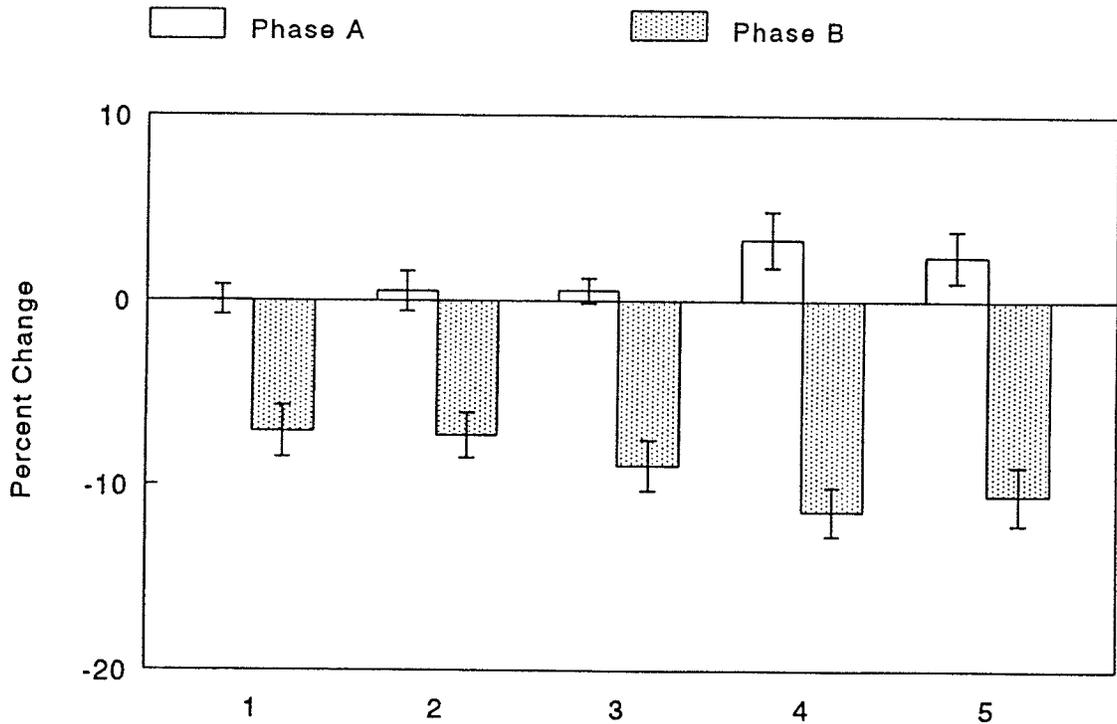


**Figure 30:** The percentage change of the ambulatory distance in phase A compared to phase B of the study.

# Ambulation Performance



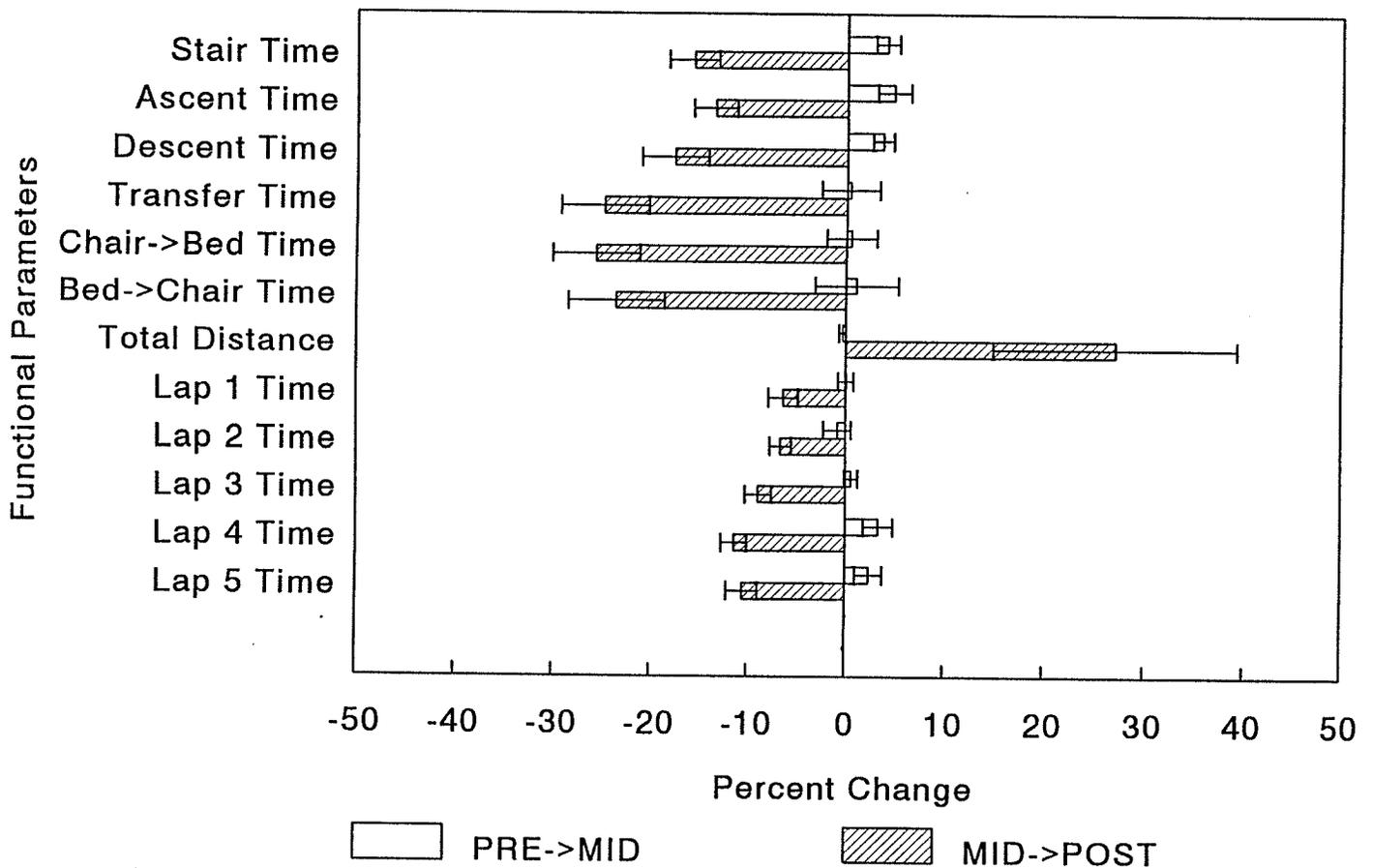
**Figure 31:** The pre, mid and post study ambulation time tests for lap #1, #2, #3, #4 and #5.



**Figure 32:** The percentage change in the ambulation time tests for lap #1, #2, #3, #4 and #5 in phase A compared to phase B of the study.

# Post-Polio Study

## Effects of Quadriceps Strengthening



**Figure 33** : The summary of the mean percentage change of the functional parameters in phase A compared to phase B of the study.

The results of the Barthel Index are illustrated in Table 15. Fourteen (78%) subjects had perfect scores on the Barthel Index.

SUBJECT #	PRE STUDY	MID STUDY	POST STUDY
1	100	100	100
2	75	75	75
3	100	100	100
4	95	95	95
5	100	100	100
6	100	100	100
7	100	100	100
8	100	100	100
9	100	100	100
10	100	100	100
11	100	100	100
12	80	80	80
13	100	100	100
14	100	100	100
15	100	100	100
16	100	100	100
17	100	100	100
18	90	90	90

*Table 15: The pre, mid and post study measures of the Barthel Index.*

The Barthel Index revealed no significant difference in the self care and mobility parameters in phase A compared to phase B, as illustrated in Table 16.

<b>FUNCTIONAL ASSESSMENT</b>	<b>measurement parameters</b>	<b>alpha level</b>	<b>% change</b>
<b>Transfers</b>	chair to bed time	0.000	-28.5
	bed to chair time	0.000	-27.9
<b>Stairs</b>	ascent time	0.000	-19.1
	descent time	0.000	-22.8
<b>Ambulation</b>	total distance	0.03	+ 29.1
	lap #1 time	0.000	-7.1
	lap #2 time	0.000	-6.0
	lap #3 time	0.000	-9.5
	lap #4 time	0.000	-14.7
	lap #5 time	0.000	-12.9
<b>Barthel Index</b>			0.0

**Table 16:** *The statistical analysis (paired t-test) of the percentage change in the functional parameters illustrating the alpha levels and the percentage difference from phase A to phase B of the study.*

## PAIN DISABILITY INDEX

The inclusionary criteria identified each subject as experiencing the onset of new muscular weakness and increasing pain or fatigue.

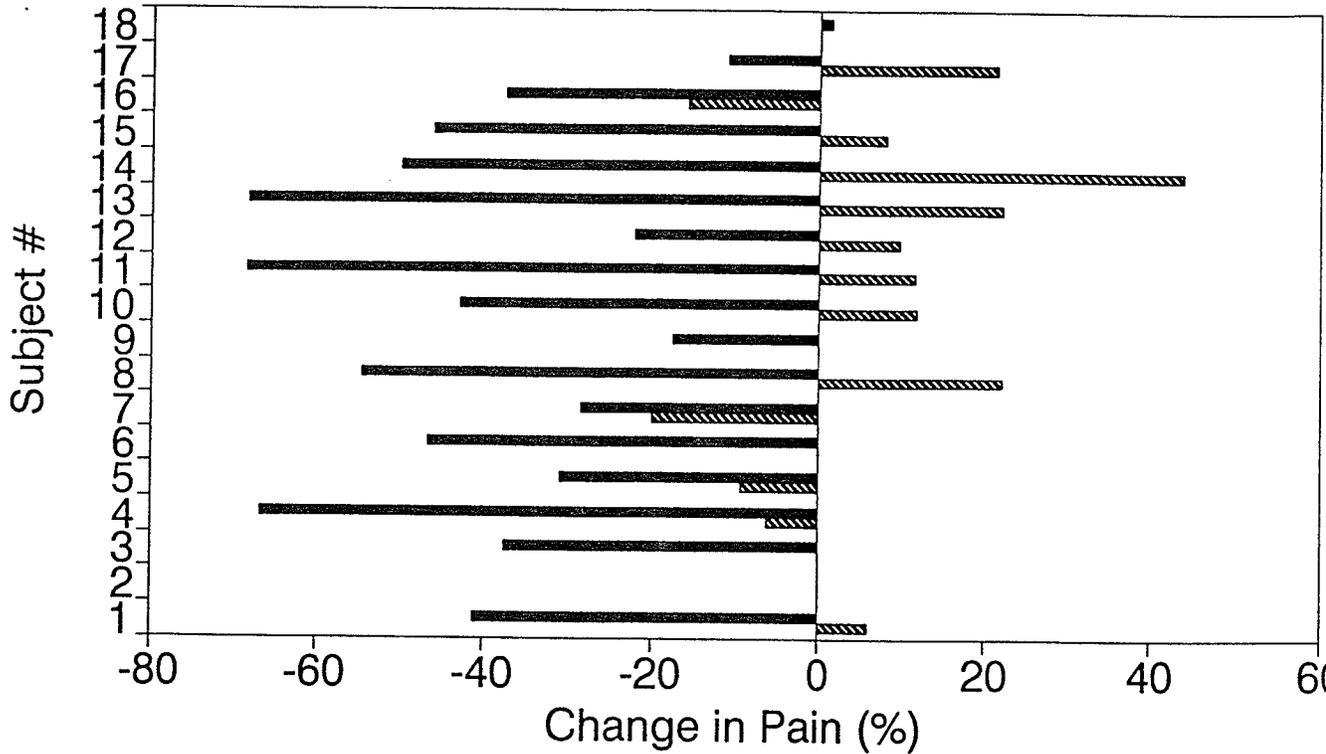
Four subjects denied pain upon entering the study. In their perception, the primary disabling symptom was fatigue associated with minor activities (as identified by \* in Table 18). These individuals completed the Pain Disability Index in relation to the fatigue that impacted on their participation in role activities.

SUBJECT #	PRE STUDY	MID STUDY	POST STUDY
1*	16	17	10
2	49	49	49
3	24	27	15
4*	16	15	5
5	32	29	20
6	15	15	8
7	35	28	20
8	27	33	20
9	40	40	33
10	25	28	16
11*	17	19	6
12	41	45	35
13	36	44	14
14	25	36	18
15*	24	26	14
16	19	16	10
17	37	45	40
18	59	59	60

*Table 17: The pre, mid and post study measurements of the Pain Disability Index.*

The pre, mid and post study results of the Pain Disability Index are illustrated in Table 17. Figure 20 illustrates the percentage change in the Pain Disability Index of the individual subjects in phase A compared to phase B of the study.

# PAIN DISABILITY INDEX



**Figure 34:** The percentage change in the Pain Disability Index per subject in phase A (solid bar) as compared to phase B (cross-hatched bar) of the study.

The statistical analysis using Friedman's two-way ANOVA for nonparametric measures and the percentage change from phase A to phase B of the study are illustrated in Table 18. There was a significant improvement ( $p < .01$ ) in the subjects' level of pain and fatigue as it impacted on the perception of their social role.

	alpha level	% change
Pain Disability Index	0.000	-43.1

**Table 18:** The statistical analysis (Friedman's two-way ANOVA) of the pain disability index illustrating the alpha levels and the percentage difference from phase A to phase B of the study.

## SUMMARY OF THE RESULTS

Submaximal voluntary exercise with EMS intervention has been found to increase isometric torque by 32% and isovelocity torque by 45% as determined by the average torque values from the peak, mean and area parameters of the quadriceps femoris strength in the muscles that are weakened by poliomyelitis. The increase in strength was not associated with any systematic change in the CK levels or with a detectable change in muscle cross sectional area as reflected in the girth and skinfold measurements. The submaximal voluntary exercise with EMS did produce improvement in the functional ability of the subjects (transfer performance improved by 28%, stair performance improved by 21%, ambulation time performance improved by 10% and the total ambulatory distance improved by 29%). The Barthel Index did not detect functional changes in the subject population. Seventeen of the eighteen subjects had a reduction in their perceived disability with respect to pain or fatigue on their level of participation in social role activities.

## DISCUSSION

Submaximal voluntary exercise with EMS resulted in a significant increase in strength, a significant reduction in pain and a significant improvement in function without an elevation in the CK levels of the post-poliomyelitis subjects with quadriceps femoris muscular weakness. The findings in this study support the implementation of a submaximal exercise/EMS program as a therapeutic measure for poliomyelitis survivors, limited by the type of poliomyelitis survivor demographics employed in this study.

There is also the possibility that submaximal voluntary exercise with EMS intervention provided a positive feedback for the subjects, with a psychological basis that could be attributable to the results of a muscle re-education effect.

The CK data that was analyzed using cluster analysis revealed a natural grouping of CK levels within the sample population. Waring (1989) described CK high level and low level groups. This study identified low, intermediate and high level groups by the distinct clustering of CK levels (under 180 U/L, between 180-400 U/L and 400-1,000 U/L). Post-hoc analysis revealed that strength gains were evident, independent of the CK groupings. It is noteworthy of mention that the high CK group showed the same outcome improvements in strength, function and pain without significant changes in their CK levels compared to the rest of the subjects under investigation.

Independent of the electrodiagnostic classification of the subjects, significant improvements were observed in the strength, function and pain parameters. One of the limitations of this study was the classification of the subject population primarily as a chronic denervation muscle status. The ability to generalize of the effects of active exercise/EMS intervention based on the few muscles classified in the active denervation group would remain questionable.

One of the proposed etiologies of the new weakness associated with post-poliomyelitis is the motoneurons that reinnervated orphaned muscle fibres in the recovery phase from the acute disease, have lost their ability to support the metabolic needs of the distal sprouts from collateral reinnervation (Dalakas 1988). If this theory were accurate, one would speculate that a symptomatic poliomyelitis population sample would have revealed a greater percentage of acute denervation in their electrodiagnostic findings, which was not the case in this study. The differences in the electromyographic results may be due to the

uniqueness of this voluntary subject sample, or that the muscle sampled was consistently applied to the vastus lateralis and not necessarily to all the muscles for which the subjects reported new weakness.

The initial level of strength in this sample population did appear to be substantially less than that reported in other studies. The data in Munin (1991) revealed that the average level was triple the average torque in the sample population within this study. The relative weakness of this sample group could also explain the high percentage changes in the isovelocity and isometric torque measures.

It should be appreciated that the sample population under investigation was symptomatic post-poliomyelitis volunteers. During the acute phase of poliomyelitis, all of these subjects reported various degrees of paresis involving one or more of their extremities, and one would conclude that this subject population experienced a fairly extensive acute viral infection.

A 29% improvement in isometric strength and 24% improvement in isovelocity strength following intervention of a resistive exercise program was found in poliomyelitis survivors as reported by Einarsson (1991). In comparison, there was a surprisingly high percentage change in the strength parameters measured within this study given the application of a submaximal non-fatiguing exercise training program. The average of the isometric strength parameters improved by 32% and the isovelocity strength improved by 45%. This could be artificially high since some of the subjects entered the study with very low levels of torque, and consequently improved by factors of 200% with only an absolute improvement of a few Newton-metres. The conclusions drawn from the percentage changes should be tempered since they are drawn from a relatively physically weak sample population.

The effect on the subjects performance resulting from knowledge of their participation in the experiment could have created a Hawthorne effect. The Hawthorne effect could be analyzed by the amount of change measured during phase A (by comparing the pre and mid study measurements), which was minimal. Alternatively, the Hawthorne effect could potentially be triggered at the time of active intervention, or phase B of this study design which would be difficult to control in the absence of a sham intervention. This difficulty imposes a limitation in the interpretation of the results of this study.

The high percentage change in the strength findings could also be attributable to a learning curve with the testing procedures. Although substitution patterns were controlled in the positioning of the subjects on the Biodex, it was still apparent that there was an increased familiarity with the instructions for the procedures in the post study measurements.

Although the right isometric strength was nonsignificant, there may have been a type II error due to the high variability of the subject population. In actuality, the alpha was .06 which approaches the .05 level of significance.

These strength changes occurred without a detectable change in thigh girth and skinfold measurements. The intervention had a limited impact on the cross sectional area of the quadriceps muscle group. However, magnetic resonance imaging or computed tomography of the individual muscles would have provided a more precise method to detect muscle cross sectional changes. As such, the thigh girth measurements must be viewed as a gross detector for the muscular changes found within this study. This does not preclude that a longer exercise regime may not result in significant changes in muscle cross sectional area.

Enoka (1991) reports that strength is not just a property of the muscle, but a property of the neuromuscular system. Thus, the nervous system involvement must have played a very important role in the development of strength for this subject population. This is termed a process of neural adaptation or increased neural efficiency with recruiting existing motor units.

The EMS applied independently would have bypassed central activation and would not necessarily have accounted for as high a degree of neural adaptation as the EMS applied with the submaximal voluntary exercise. The EMS preferentially recruited the large diameter axons. Thus, a mixed recruitment pattern would be established with the application of this combined intervention regime. This, however, creates a confounding variable that makes it virtually impossible to determine whether the EMS or the submaximal voluntary exercise was actually responsible for the strength gains achieved in this study.

There have been remarkable reports of strength gains in the geriatric population. One would conclude that disuse would be a contributing factor for the dramatic percentage change within this subject population.

It is difficult to determine clinically whether a patient is experiencing weakness due to disuse atrophy or due to the progressive post-poliomyelitis muscular atrophy. The large strength changes found in the EMS studies with atrophic muscles (Godfrey 1979, Johnson 1977, Wigerstad-Lossing 1986, Williams 1976), correlates with the magnitude of the strength changes found in this study. This would cause one to suspect that disuse atrophy may have been predominant within this particular poliomyelitis subject population.

This study provided an appreciation for the relationship between the quantitative and qualitative data. It was the significance of the quantitative strength torque parameters that provided the credibility to this study, but it was the qualitative perspective that provided the extra dimension for an appreciation of the impact of such strength gains for these poliomyelitis survivors.

Most studies did not include measures of function or pain with the investigations on strength changes (Einarsson 1991, Feldman 1985, Fillyaw 1991) in the poliomyelitis population. The design of this study was such that measures of function and pain provided a multidimensional view of the subject group under investigation. Muscular weakness as well as decreasing function and increasing pain stimulates medical consultation.

The Barthel Index is a very reliable measure, however, it was not sufficiently sensitive to detect changes in the self care and mobility parameters. Most subjects entered the study with perfect scores on this scale.

The functional time tests were good functional indicators that recognized with the improvement of strength, there is an impact upon the functional ability of the poliomyelitis subjects with weakness of the quadriceps femoris. The functional ability of the subjects was significantly improved as reflected in the time test parameters; transfer performance improved by 28%, stair performance improved by 21%, ambulation performance improved by 10% and the total ambulatory distance improved by 29%.

The improvement in function could be multifactorially dependent upon strength gains and a reduction in the level of pain. It is difficult to ascertain whether there was an efficiency in recruiting the motor units as a result of the submaximal voluntary exercise with EMS training or that there was a direct symptomatic effect that enhanced function.

This study did reveal a significant reduction in the level of discomfort of this subject population as a result of the submaximal voluntary exercise with EMS intervention. There was a 43% reduction in their perceived disability as a result of pain or fatigue upon their level of participation in social role activities.

The physiological basis could be that EMS has a 'gate control' effect. The EMS modality stimulated the surface of the skin on the anterior thigh during the training period. This stimulation could have resulted in a dorsal column blockade of pain on a temporary basis by activation of large diameter sensory axons. EMS may provide a symptomatic relief of pain in the dermatomal region of the lower extremities. The subject may learn that participation in activities does not necessarily have to be painful. The psychological basis could also reflect their delight in the improvement of their functional abilities. Nonetheless, post-poliomyelitis syndrome has a psychophysiologic basis, and therefore a holistic approach to treatment is required.

The literature that evoked "fear" into clinical application of exercise for this patient population may have a "bark that is worse than its bite". The results of this study imply that any exercise program that is based on non-fatiguing training principles will not result in a CK increase. However, it would not be possible to conclude the same effects of a controlled CK with high intensity level of exercise.

The results of this study provide an unqualified assurance that exercise programs of this nature are important to implement for the poliomyelitis population. Findings reveal that an EMS/exercise program has a significant impact on the psychosocial aspect of this subject population with respect to their level of perceived disability. It is difficult to determine if the strength gains, functional improvements and/or the reduction in pain levels, contributed to the outcome of their enhanced participation in social activities.

It is difficult to determine clinically whether a patient is experiencing weakness due to disuse atrophy or due to the progressive post-poliomyelitis muscular atrophy that is identified in the literature (Cashman 1987, Dalakas 1988). As clinicians, we have to be aware of the potential for reduction in strength if the poliomyelitis survivor continues to overwork weakened muscles. However, this should be weighed against the consequence of muscle weakness associated with disuse in the absence of therapeutic intervention.

The poliomyelitis survivor may benefit from a training program that could be implemented on a preventive basis rather than solely from a therapeutic perspective. These individuals should exercise their weakened muscles at low intensity levels (Feldman 1985, Fillyaw 1991) as identified within this study. The poliomyelitis survivor should actively participate in the planning of their exercise program by selecting a function that is important to them (Halstead 1991). The clinician would provide the expertise necessary to develop an appropriate program based on their individual needs and to analyze quantitatively strength changes as a result of these exercises. One of the available options for the poliomyelitis survivor's treatment of new weakness in the quadriceps femoris could be a submaximal voluntary exercise program with EMS. The poliomyelitis survivor should be responsible for monitoring their progress based on their level of pain and fatigue.

## CONCLUSION

The prevalence of post-poliomyelitis syndrome is increasing. There are 1.63 million poliomyelitis survivors in the United States and approximately 50% are reporting the onset of new symptoms (Parsons 1989). The latency period for confronting new health problems in these poliomyelitis survivors is approximately 40 years of age. The poliomyelitis epidemic of the 1950's is currently upon us with respect to this projected latency period. Thus there is a great need for appropriate treatment intervention for this patient population experiencing muscular weakness.

One could extrapolate that the submaximal voluntary exercise with EMS intervention is similar to any other low level exercise program that could be offered to this patient population (Feldman 1985, Dean 1991). The bonus to the combination of exercise and EMS intervention for a training program is the possibility for a 'gate control' effect to reduce the levels of discomfort. EMS may contribute to a more efficient motor unit recruitment pattern and 'muscle re-education'. These individuals may benefit from a training program that could be implemented on a preventive basis rather than solely from a therapeutic perspective.

It has been postulated that only 1-5% of the poliomyelitis population is aware that they contracted the poliovirus prior to the introduction of the Salk and Sabin vaccines (Plum 1982). Maybe the remaining 95-99% will soon be 'coming out of the woodwork' and the magnitude of their new health problems could bombard the medical profession. The time to prepare is NOW.

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INFORMED CONSENT

TITLE: Electromyostimulation (EMS) in post-poliomyelitis muscular weakness

I understand the following statements:

1. There is controversy regarding the benefits of exercise for muscles weakened for poliomyelitis survivors.
2. I understand the concept of the overuse syndrome and will respect the feelings of pain and fatigue.
3. There is no documentation available to support the beneficial effect of EMS on muscles weakened by post-poliomyelitis muscular atrophy, however there is documentation and a physiological rationale to support the application of EMS upon weakened quadriceps femoris muscles.
4. There is controversy over the generalizability of results obtained from a single muscle group study to persons as a whole.
5. I will submit to a medical examination by Dr. S. Vallentyne and a physiotherapy assessment by L. Robertson.
6. I will submit to blood sampling from the radial vein on the first, fourth, fifth, sixth, seventh and eighth week of the study which could result in some minor discomfort or possibly bruising from the needle insertion.
7. The EMS may produce an unusual or mildly painful sensation that is similar to a "pins and needles" phenomenon.
8. This EMS intervention is NOT a physiotherapy treatment, but is for the purpose of research in attempts to understand the impact upon strengthening the quadriceps femoris weakened by post poliomyelitis.
9. The project will require a time commitment for the initial orientation, the pre, mid and post-study evaluations and weekly appointments for EMS intervention in phase B of the study (3X per week for a 4 week period).
10. The risks and side effects have been explained to me and I agree to inform the investigator if a problem arises.
11. The daily activity log has been explained to me and I agree to complete the forms daily and submit them on a weekly basis.
12. The results obtained as a consequence of my participation in the EMS study will be available for Dr. S. Vallentyne and L. Robertson, whether these results are individual or in group form.

13. If I should have any questions in regard to my participation in this study, I will have them answered to my satisfaction.

14. I do not expect financial remuneration as being a participant in this study.

15. I do expect to have my confidentiality protected during my participation of the study and in any future published results. My name will be used only for communication between Dr. S. Vallentyne and L. Robertson.

16. Whether or not I participate in this study will not influence any treatment that I am receiving in the hospital.

17. I may decline to participate or withdraw from the study at any time without affecting my future care.

18. I will retain a copy of this consent documentation.

I do hereby certify that I am willing to participate in the research study on the effects of electromuscularstimulation (EMS) on the quadriceps femoris (the muscle on the front of the thigh).

Date:

Signature of participant:

Signature of investigator:

Signature of witness:



**APPENDIX C**

UNIVERSITY OF MANITOBA  
FACULTY COMMITTEE ON THE USE OF HUMAN SUBJECTS IN RESEARCH

NAME: Ms. Lorraine Robertson

OUR REFERENCE: E90:248

DATE: December 12, 1990

**YOUR PROJECT ENTITLED:**

Electromyostimulation in Post-Poliomyelitis Muscular Atrophy.

**HAS BEEN APPROVED BY THE COMMITTEE AT THEIR MEETING OF:**

Approved by Dr. J.P. Maclean on December 12, 1990.

**COMMITTEE PROVISOS OR LIMITATIONS:**

Approved as per your letter dated December 6, 1990 enclosing the amended consent form.

You will be asked at intervals for a status report. Any significant changes of the protocol should be reported to the Chairman for the Committee's consideration, in advance of implementation of such changes.

\*\* This is for the ethics of human use only. For the logistics of performing the study, approval should be sought from the relevant institution, if required.

Sincerely yours,

J. P. Maclean, M.D.,  
Chairman,  
Faculty Committee on the Use of Human  
Subjects in Research

JPM/11

TELEPHONE ENQUIRIES:

Lorraine Lester

Research - Post Polio



# THE CITY OF WINNIPEG

MUNICIPAL HOSPITAL

MORLEY AVENUE • WINNIPEG • MANITOBA • R3L 2P4

IN REPLY PLEASE REFER TO

FAX: (204) 956-4502

June 29, 1990.

## APPENDIX C

Dr. S. Vallentyne,  
Head, Rehabilitation Medicine,  
Winnipeg Municipal Hospital,  
1 Morley Avenue,  
Winnipeg, MB  
R3L 2P4

Dear Dr. Vallentyne:

RE: STUDY: "INTERMITTENT LONG TERM ELECTROLYMO-STIMULATION  
POST-POLIOMYELITIS PROGRESSIVE MUSCULAR ATROPHY":

The Pharmacy, Therapeutics, Ethics and Research Committee, at a special meeting held on Monday, June 25, 1990, approved this study to be conducted at the Winnipeg Municipal Hospital.

Yours sincerely,

D. Boonov, Secretary,  
P.T.E. & R. Committee.

DB/ap



WINNIPEG  
..where the New West begins.



**LATE EFFECTS OF POLIOMYELITIS****QUESTIONNAIRE**

This questionnaire is designed to collect information about persons who have had poliomyelitis, and to get a clearer idea about what is being called the "Late Effects of Poliomyelitis." Please answer all questions:

**I. GENERAL INFORMATION:**

Family Physician: \_\_\_\_\_  
Name . Telephone

LINKAGE ITEMS

1. Date of Birth \_\_\_\_\_  
D M Y
2. Surname \_\_\_\_\_
3. Surname at Birth \_\_\_\_\_
4. Full Names \_\_\_\_\_  
(First) (Middle)
5. Names of: Father \_\_\_\_\_  
(Last) (First) (M.I.)  
Mother \_\_\_\_\_  
(Last) (First) (M.I.)
6. Country/Province/City of Birth \_\_\_\_\_  
Country Province City
7. Full Current Address \_\_\_\_\_  
Telephone: Home \_\_\_\_\_ Work \_\_\_\_\_ Postal Code \_\_\_\_\_
8. Sex: M \_\_\_\_\_ F \_\_\_\_\_
9. Age \_\_\_\_\_
10. Date of Onset of Polio \_\_\_\_\_
11. Your Age at Time of Onset \_\_\_\_\_
12. Are You Presently Employed? Yes \_\_\_\_\_ No \_\_\_\_\_
13. If Yes, a) Number of Years Employed \_\_\_\_\_  
b) Full-time \_\_\_\_\_ Part-time \_\_\_\_\_
14. Marital Status: Single \_\_\_\_\_ Married \_\_\_\_\_ Separated \_\_\_\_\_ Divorced \_\_\_\_\_ Widow(er) \_\_\_\_\_
15. Do You Smoke? Yes \_\_\_\_\_ No \_\_\_\_\_
16. Have You Noted Any Recent Weight Gain? \_\_\_\_\_ or Weight Loss \_\_\_\_\_?

Late Effects of Poliomyelitis, Questionnaire (Continued).

II. MUSCLE WEAKNESS: Three time periods relative to your bout with polio are asked for - please place an 'X' in the appropriate boxes that describe your experience.

	At Time of Onset	After Maximal Recovery from Polio	Now
1. Respiratory	_____	_____	_____
2. Shoulder	Right ___ Left ___	Right ___ Left ___	Right ___ Left ___
3. Arm	Right ___ Left ___	Right ___ Left ___	Right ___ Left ___
4. Hand & Fingers	Right ___ Left ___	Right ___ Left ___	Right ___ Left ___
5. Hip	Right ___ Left ___	Right ___ Left ___	Right ___ Left ___
6. Leg	Right ___ Left ___	Right ___ Left ___	Right ___ Left ___
7. Foot	Right ___ Left ___	Right ___ Left ___	Right ___ Left ___
8. Abdominal Muscles	_____	_____	_____

9. Did you require the use of an "iron lung"? Yes \_\_\_ No \_\_\_

III. WALKING WEAKNESS

A. After Recovery from polio:

1. Were you able to walk after recovery? Yes \_\_\_ No \_\_\_ What Distance? \_\_\_\_\_

a) If yes, did you require the use of one cane? 2 Canes \_\_\_ Crutches? \_\_\_ Walker? \_\_\_ or did you walk without aids? \_\_\_

b) If no, were you able to use a wheelchair? Yes \_\_\_ No \_\_\_

2. Did you require the use of braces? Yes \_\_\_ No \_\_\_

a) If yes, were they: Right Left

Long Leg    \_\_\_            \_\_\_  
Short Leg    \_\_\_            \_\_\_

B. At the Present Time:

1. Are you able to walk? Yes \_\_\_ No \_\_\_ What Distance? \_\_\_\_\_

a) If yes, do require the use of one cane? \_\_\_ 2 Canes? \_\_\_ Crutches? \_\_\_ Walker? \_\_\_ or do you walk without aids? \_\_\_

b) If no, are you able to use a wheelchair? Yes \_\_\_ No \_\_\_

Late Effects of Poliomyelitis, Questionnaire (Continued)

- 2. Do you require the use of braces? Yes \_\_\_ No \_\_\_
  - a) If yes, are they:
 

	<u>Right</u>	<u>Left</u>
Long Leg	___	___
Short Leg	___	___
  - b) When were your braces last reviewed? \_\_\_\_\_  
repaired? \_\_\_\_\_
- 3. Do you have any trouble climbing stairs? Yes \_\_\_ No \_\_\_

IV. REVIEW OF PRESENT STATUS

A. Muscle Weakness:

- 1. Do you have any muscle weakness? Yes \_\_\_ No \_\_\_
  - If yes, a) date of onset of weakness \_\_\_\_\_
  - Is onset recent? (last 3 years) Yes \_\_\_ No \_\_\_
  - a) Your age at recent onset \_\_\_\_\_
  - b) Length of time between first onset of polio and recent weakness \_\_\_\_\_ (years)
- 2. Is there any relationship between recent onset of weakness and recent possible major events in your life? Yes \_\_\_ No \_\_\_
  - If yes, a) Marriage? \_\_\_ b) Parenthood? \_\_\_
  - c) Graduation? \_\_\_ d) Emotional Episode? \_\_\_
- 3. Is there a relationship between recent onset of weakness and any other episodes (i.e., major or minor events in your life)?
  - a) Illness? \_\_\_ please specify \_\_\_\_\_
  - b) Injury? \_\_\_ c) Fall? \_\_\_ d) Auto accident? \_\_\_
  - e) Surgery? \_\_\_ f) Other? \_\_\_ please specify \_\_\_\_\_

Late Effects of Poliomyelitis, Questionnaire (Continued)

**B. Pain**

1. Do you experience any pain on a regular basis? Yes \_\_\_ No \_\_\_

If yes, a) Is this pain muscular? Yes \_\_\_ No \_\_\_

If yes, please specify location \_\_\_\_\_

b) Is this pain in your joints? Yes \_\_\_ No \_\_\_

If yes, please specify which joints \_\_\_\_\_

c) Is this pain - continuous? \_\_\_\_\_

- occasional? \_\_\_\_\_

- related to fatigue? \_\_\_\_\_

- related to movement? \_\_\_\_\_

d) What is pain relieved by? \_\_\_\_\_

e) How long has pain been present? \_\_\_\_\_

**C. Fatigue**

1. Do you experience any fatigue? Yes \_\_\_ No \_\_\_

If yes, a) Is fatigue associated with muscle weakness? Yes \_\_\_ No \_\_\_

b) Is fatigue related to exercise? Yes \_\_\_ No \_\_\_

c) Is fatigue related to increased workload? Yes \_\_\_ No \_\_\_

2. Distance that you were/are able to walk:

	without fatigue PRIOR to recent decline	NOW able to walk without fatigue
a) - less than one city block		
b) - one city block		
c) - two city blocks		
d) - one mile		
e) - greater than one mile		

Late Effects of Poliomyelitis, Questionnaire (Continued)

D. Breathing Difficulty

1. Do you experience any breathing difficulty? Yes \_\_\_ No \_\_\_

If yes, a) How long have you noted this difficulty? \_\_\_\_\_ (years)

b) Has this problem become worse recently? Yes \_\_\_ No \_\_\_

If yes, Date of recent increase in difficulty \_\_\_\_\_

c) Is it associated with exercise? Yes \_\_\_ No \_\_\_

d) Is it worse at night? \_\_\_\_\_ during the day? \_\_\_\_\_

e) Have not noted a reduction in breathing capacity? Yes \_\_\_ No \_\_\_

f) Do you awaken from your sleep with shortness of breath? Yes \_\_\_ No \_\_\_

E. Other Problems

1. Do you experience any headaches regularly? Yes \_\_\_ No \_\_\_ Time of Day \_\_\_\_\_

2. Do you note any recent sleep disturbance? Yes \_\_\_ No \_\_\_

3. Have you noted that you have a recent need for more sleep? Yes \_\_\_ No \_\_\_

4. Have you noted any recent personality changes? Yes \_\_\_ No \_\_\_

5. Have you noted any recent voice changes? Yes \_\_\_ No \_\_\_

6. Do you have frequent falls? Yes \_\_\_ No \_\_\_

If yes, when did you notice an increase in falling? \_\_\_\_\_ (years ago)

F. Activities of Daily Living (ADL)

1. Do you have any problems with:

a)	Eating/swallowing.....	Yes ___	Year of onset _____	No ___
	Dressing.....	Yes ___	Year of onset _____	No ___
	Bathing.....	Yes ___	Year of onset _____	No ___
	Bladder function.....	Yes ___	Year of onset _____	No ___
	Bowel function.....	Yes ___	Year of onset _____	No ___
	Sexual function.....	Yes ___	Year of onset _____	No ___

Other (Please specify) \_\_\_\_\_

b) Do you experience difficulty in:

Going from standing to sitting position. Yes \_\_\_ Year of onset \_\_\_\_\_ No \_\_\_

Going from sitting to standup position. Yes \_\_\_ Year of onset \_\_\_\_\_ No \_\_\_

Getting in and out of a	- chair	Yes ___	Year of onset _____	No ___
	- bed	Yes ___	Year of onset _____	No ___
	- bathtub	Yes ___	Year of onset _____	No ___

G. Other Health Problems

1. Have you had any of the following?

- a) Other neurological disease? Yes \_\_\_ No \_\_\_ Duration \_\_\_\_\_ (years)
- b) Any heart disease? Yes \_\_\_ No \_\_\_ Duration \_\_\_\_\_ (years)
- c) High blood pressure? Yes \_\_\_ No \_\_\_ Duration \_\_\_\_\_ (years)
- d) Arthritis? Yes \_\_\_ No \_\_\_ Duration \_\_\_\_\_ (years)
- e) Diabetes? Yes \_\_\_ No \_\_\_ Duration \_\_\_\_\_ (years)
- f) Cancer? Yes \_\_\_ No \_\_\_ Duration \_\_\_\_\_ (years)

g) Other? (Please specify) \_\_\_\_\_

h) Are you adversely affected by:

Cold - Yes \_\_\_ No \_\_\_

Heat - Yes \_\_\_ No \_\_\_

2. Are you taking any medication on a regular basis? Yes \_\_\_ No \_\_\_

If yes, please list:

\_\_\_\_\_

\_\_\_\_\_

H. Additional Comments:

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

I hereby authorize you to share any of the above information that I have provided with my family physician noted earlier, in the interests of my optimal health care.

\_\_\_\_\_  
(Signature)

\_\_\_\_\_  
(Date)



**Appendix G**

**BARTHEL INDEX**

	with help	independent
1. Feeding (if food needs to be cut = help)	5	10
2. Moving from wheelchair to bed and return (includes sitting up in bed)	5-10	15
3. Personal toilet (wash face, comb hair, shave, clean teeth)	0	5
4. Getting on and off toilet (handling clothing, wipe, flush)	5	10
5. Bathing self	0	5
6. Walking on level surface (or if unable to walk, propel wheelchair) * score only if unable to walk	10	15
7. Ascend and descend stairs	5	10
8. Dressing (includes tying shoes, fastening fasteners)	5	10
9. Controlling bowels	5	10
10. Controlling bladder	5	10

## RATING GUIDELINES FOR BARTHEL INDEX\*

## 1. Feeding

10 = Independent. The patient can feed himself a meal when someone puts the food within his reach. He must put on an assistive device if this is needed, cut up the food alone. He must accomplish this in a reasonable time.

5 = Some help is necessary (with cutting up food, etc., as listed above).

## 2. Moving from wheelchair to bed and return

15 = Independent in all phases of this activity. Patient can safely approach the bed in his wheelchair, lock brakes, lift footrests, move safely to bed, lie down, come to a sitting position on the wheelchair, if necessary, to transfer back into it safely, and return to the wheelchair.

10 = Either some minimal help is needed in some step of this activity or the patient needs to be reminded or supervised for safety of one or more parts of this activity.

5 = Patient can come to a sitting position without the help of a second person but needs to be lifted out of bed, or if he transfers with a great deal of help.

## 3. Doing personal toilet

5 = Patient can wash hands and face, comb hair, clean teeth and shave. He may use any kind of razor but must put in blade or plug in razor without help as well as get it from drawer or cabinet. Female patients must put on own makeup.

## 4. Getting on and off toilet

10 = Patient is able to get on and off toilet, fasten and unfasten clothes, prevent soiling of clothes and use toilet paper without help. If it is necessary to use a bedpan instead of a toilet, he must be able to place it on a chair, empty it, and clean it.

5 = Patient needs help because of imbalance or in handling clothes or in using toilet paper.

## 5. Bathing self

5 = Patient may use a bathtub, a shower, or take a complete sponge bath. He must be able to do all the steps involved in whichever method is employed without another person being present.

## 6. Walking on a level surface

15 = Patient can walk at least 50 yards without help or supervision. He may wear braces or prostheses and use crutches, canes, or a walkerette but not a rolling walker. He must be able to lock and unlock braces, if used, assume the standing position and sit down,

get the necessary mechanical aids into position for use, and dispose of them when he sits. (Putting on and taking off braces is scored under dressing.)

10 = Patient needs help or supervision in any of the above but can walk at least 50 yards with a little help.

## 6a. Propelling a wheelchair

5 = If a patient cannot ambulate but can propel a wheelchair independently. He must be able to go around corners, turn around, maneuver the chair to a table, bed, toilet, etc. He must be able to push a chair at least 50 yards. Do not score this item if the patient gets score for walking.

## 7. Ascending and descending stairs

10 = Patient is able to go up and down a flight of stairs safely without help or supervision. He may and should use handrails, canes, or crutches when needed. He must be able to carry canes or crutches as he ascends or descends stairs.

5 = Patient needs help with or supervision of any one of the above items.

## 8. Dressing and undressing

10 = Patient is able to put on and remove and fasten all clothing, and tie shoe laces (unless it is necessary to use adaptations for this). The activity includes putting on and removing and fastening corset or braces when these are prescribed.

5 = Patient needs help in putting on and removing or fastening any clothing. He must do at least half the work himself. He must accomplish this in a reasonable time. Women need not be scored on use of a brassiere or girdle unless these are prescribed garments.

## 9. Continence of bowels

10 = Patient is able to control his bowels and have no accidents. He can use a suppository or take an enema when necessary.

5 = Patient needs help in using a suppository or taking an enema or has occasional accidents.

## 10. Controlling bladder

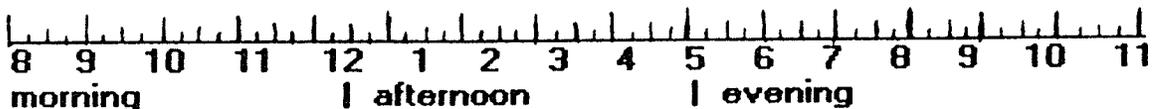
10 = Patient is able to control his bladder day and night. Patients who wear an external device and leg bag must put them on independently, clean and empty bag, and stay dry day and night.

5 = Patient has occasional accidents or cannot wait for the bedpan or get to the toilet in time or needs help with an external device.

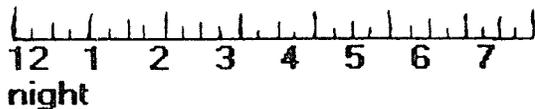
# ACTIVITY PROFILE

APPENDIX H

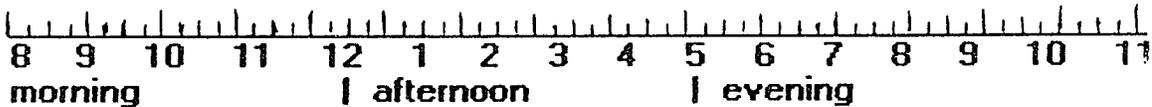
Name: \_\_\_\_\_ Date: \_\_\_\_\_



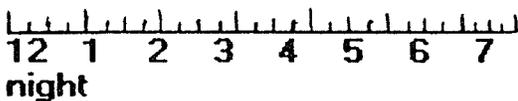
Comments:



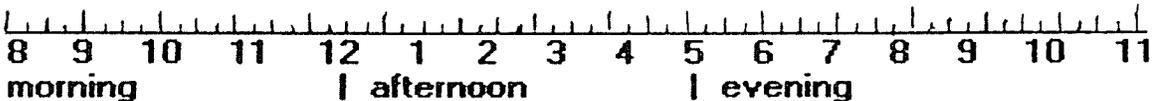
Date: \_\_\_\_\_



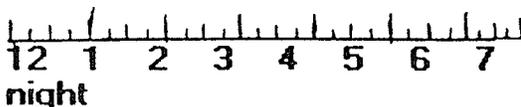
Comments:



Date: \_\_\_\_\_



Comments:



ISOVELOCITY MOMENT							APPENDIX J		
LEFT KNEE EXTENSOR MOMENT									
PEAK	PRE	PRE	PRE	MID	MID	MID	POST	POST	POST
Subject	A	B	C	A	B	C	A	B	C
#1	58.2994	73.2132	63.7226	50.1646	62.3668	47.453	101.685	81.348	93.5502
#2	63.7226	58.2994	55.5878	63.7226	61.011	56.9436	75.9248	70.5016	67.79
#3	0	0	0	0	0	0	0	0	0
#4	37.9624	36.6066	37.9624	37.9624	37.9624	35.2508	54.232	54.232	61.011
#5	0	0	0	0	0	0	0	0	0
#6	66.4342	69.1458	71.8574	59.6552	54.232	54.232	55.5878	61.011	63.7226
#7	28.4718	31.1834	29.8276	35.2508	35.2508	33.895	50.1646	48.8088	51.5204
#8	0	0	0	0	0	0	0	0	0
#9	25.7602	24.4044	25.7602	23.0486	23.0486	23.0486	36.6066	37.9624	37.9624
#10	52.8762	51.5204	52.8762	48.8088	48.8088	46.0972	82.7038	84.0596	89.4828
#11	146.426	149.138	143.715	149.138	145.071	145.071	192.524	192.524	192.524
#12	67.79	69.1458	66.4342	66.4342	66.4342	66.4342	93.5502	94.906	88.127
#13	43.3856	40.674	44.7414	42.0298	40.674	37.9624	65.0784	63.7226	62.3668
#14	0	0	0	0	0	0	0	0	0
#15	37.9624	37.9624	36.6066	37.9624	46.0972	44.7414	55.5878	51.5204	65.0784
#16	2.7116	1.3558	2.7116	0	2.7116	1.3558	4.0674	6.779	5.4232
#17	24.4044	23.0486	23.0486	20.337	17.6254	17.6254	27.116	27.116	27.116
#18	6.779	6.779	6.779	5.4232	4.0674	4.0674	0	0	0

ISOVELOCITY MOMENT							APPENDIX J		
LEFT KNEE EXTENSOR MOMENT									
MEAN	PRE	PRE	PRE	MID	MID	MID	POST	POST	POST
Subject	A	B	C	A	B	C	A	B	C
#1	31.1061	34.1729	35.0385	27.759	33.4873	26.006	47.347	47.5547	54.232
#2	42.9391	41.5804	39.9567	42.0007	41.6099	39.6311	49.8624	46.9235	44.5488
#3	0	0	0	0	0	0	0	0	0
#4	24.565	24.8839	25.5237	22.3344	20.8649	21.2042	33.4567	32.6775	34.8459
#5	0	0	0	0	0	0	0	0	0
#6	45.6637	45.3132	49.0624	42.3899	33.8418	34.6737	39.9488	41.2814	44.2494
#7	19.0013	20.008	19.224	23.8437	23.7578	22.2785	28.2706	29.2314	31.8015
#8	0	0	0	0	0	0	0	0	0
#9	16.9662	14.4915	14.7763	14.3126	14.4744	14.5189	24.8766	23.4666	24.9722
#10	31.5318	32.5021	32.0192	28.9204	29.4087	27.9857	47.7259	49.013	44.0846
#11	87.965	90.821	92.0397	93.204	91.7014	93.7664	129.4695	126.9357	127.6319
#12	44.6257	45.0012	44.725	44.8352	43.1818	43.6692	53.6275	53.0808	49.6509
#13	26.1594	26.6156	28.0312	25.6894	24.5249	23.1425	37.228	36.6995	35.289
#14	0	0	0	0	0	0	0	0	0
#15	24.6179	22.8345	23.3776	26.7614	25.5806	27.3203	39.3518	33.7255	42.1917
#16	1.4636	1.2623	1.6609	0	1.3287	1.2202	2.4211	1.7298	3.4863
#17	17.1767	16.6012	16.6012	16.6724	11.5243	10.552	11.2853	18.3374	19.4406
#18	3.9827	3.6657	3.672	3.2087	2.8439	2.1305	0	0	0

ISOVELOCITY MOMENT							APPENDIX J		
LEFT KNEE EXTENSOR MOMENT									
AREA	PRE	PRE	PRE	MID	MID	MID	POST	POST	POST
Subject	A	B	C	A	B	C	A	B	C
#1	60.0348	68.3459	69.3763	53.8524	47.8869	44.4702	85.1307	76.0875	85.1442
#2	71.7083	72.7658	68.7255	58.8214	64.4954	61.8245	82.7716	79.3007	78.4059
#3	0	0	0	0	0	0	0	0	0
#4	18.6897	20.4048	21.9504	25.0145	23.5774	23.5367	37.7565	41.5417	44.4974
#5	0	0	0	0	0	0	0	0	0
#6	67.1257	66.6105	68.1967	54.3744	51.778	51.317	51.534	51.6017	54.8692
#7	25.6517	27.2109	25.7602	31.7122	30.8851	27.8481	43.8195	41.2163	43.25
#8	0	0	0	0	0	0	0	0	0
#9	18.4931	17.6796	20.3912	15.1714	15.6324	14.9545	22.1402	28.16	29.2175
#10	43.8059	44.416	45.4057	39.3318	36.727	32.7426	75.8841	73.2674	81.3616
#11	139.8643	139.8643	137.1392	131.4177	131.133	129.3976	186.4361	189.1341	176.132
#12	73.1861	75.152	73.7962	71.288	66.0681	66.8138	84.1952	84.3986	79.938
#13	47.0869	44.7143	44.8499	34.4102	33.1086	30.0852	53.6083	53.5812	50.1104
#14	0	0	0	0	0	0	0	0	0
#15	31.2647	30.3699	31.7935	34.7898	38.6267	39.8876	47.7581	48.5648	56.5369
#16	1.288	0.3661	0.6643	0	0.6643	0.244	1.3558	0.5016	2.6845
#17	23.8756	23.0757	23.0079	24.4315	20.8929	19.5235	26.1669	25.489	23.5231
#18	1.9117	1.9795	1.7625	1.9252	1.66	0.7457	0	0	0

ISOVELOCITY MOMENT							APPENDIX J		
RIGHT KNEE EXTENSOR MOMENT									
PEAK	PRE	PRE	PRE	MID	MID	MID	POST	POST	POST
Subject	A	B	C	A	B	C	A	B	C
#1	47.453	47.453	44.7414	44.7414	40.674	40.674	77.2806	74.569	74.569
#2	52.8762	50.1646	48.8088	57.5204	47.453	46.0972	69.1458	66.4342	67.79
#3	47.453	40.674	52.8762	43.3856	48.8088	46.0972	73.2132	71.8574	69.1458
#4	40.674	35.2508	35.2508	44.7414	44.7414	42.0298	63.7226	62.3668	61.011
#5	0	0	0	0	0	0	0	0	0
#6	21.6928	20.337	18.9812	18.9812	17.6254	17.6254	6.779	5.4232	5.4232
#7	120.666	107.108	123.378	107.108	107.108	104.397	134.224	122.022	120.666
#8	missing	missing	missing	40.674	40.674	37.9624	52.8762	47.453	47.453
#9	48.8088	47.453	46.0972	44.7414	44.7414	43.3856	56.4436	51.5204	51.1646
#10	52.8762	51.5204	52.8762	50.1646	48.8088	44.7414	81.348	82.7038	82.7038
#11	165.408	159.984	162.696	158.629	159.984	155.917	195.235	192.524	199.303
#12	58.2994	61.011	59.6552	58.2994	59.6552	58.2994	81.348	79.9922	75.9248
#13	82.7038	75.9248	75.9248	82.7038	84.0596	89.4828	117.955	117.955	115.243
#14	46.0972	44.7414	47.453	52.8762	51.5204	61.011	63.7226	63.7226	66.4342
#15	48.8088	50.1646	51.5204	63.7226	59.6552	61.011	77.2806	78.6364	71.8574
#16	6.779	6.779	8.1348	6.779	6.779	5.4232	12.2022	12.2022	10.8464
#17	12.2022	13.558	12.2022	13.558	13.558	12.2022	18.9812	17.6254	18.9812
#18	18.9812	18.9812	21.6928	14.9138	14.9138	16.2696	6.779	6.779	8.1348

ISOVELOCITY MOMENT							APPENDIX J		
RIGHT KNEE EXTENSOR MOMENT									
MEAN	PRE	PRE	PRE	MID	MID	MID	POST	POST	POST
Subject	A	B	C	A	B	C	A	B	C
#1	29.3378	26.9773	27.5626	32.8603	22.7097	23.6763	49.7284	50.0791	48.5909
#2	40.1357	37.7473	38.647	38.0885	32.8371	33.6623	54.0609	50.01	50.4631
#3	33.913	31.5761	36.734	30.2238	35.9005	34.4142	44.3873	44.2456	41.9217
#4	27.9559	23.4851	22.3262	29.6219	28.4616	27.9883	42.0204	42.0203	39.8605
#5	0	0	0	0	0	0	0	0	0
#6	15.6525	13.6017	13.2084	13.0589	12.1695	11.5368	3.8298	2.7628	2.9783
#7	63.6074	53.6358	57.1848	49.1091	50.548	49.2423	72.5815	69.1614	71.1912
#8	MISSING	MISSING	MISSING	29.9016	27.7223	25.5473	35.2401	34.1018	34.3621
#9	29.5866	27.003	28.6319	29.7471	30.7359	27.626	42.8509	34.0918	35.275
#10	31.1652	32.0511	32.1316	31.9186	30.0453	29.2113	50.4926	50.6317	51.6747
#11	85.7544	96.0312	99.1039	89.714	90.3595	89.0457	123.737	122.5111	125.4463
#12	38.5851	39.9921	39.9921	40.1634	42.2755	37.341	53.3908	51.8947	52.0782
#13	44.7587	43.8347	43.2098	47.7259	43.3535	49.013	69.7015	65.4966	63.6938
#14	24.1275	23.731	24.6719	27.534	25.2622	28.0481	33.5607	34.1835	38.0411
#15	28.7174	29.2314	31.8015	36.3448	34.4355	36.5084	49.058	50.4516	45.2419
#16	4.1734	4.9569	5.1309	3.5038	3.7446	3.312	7.4569	6.8379	6.4175
#17	8.0537	9.0502	8.4806	8.8081	8.757	8.3682	12.9259	12.1685	12.5703
#18	12.9784	12.9238	13.9126	10.0919	10.2997	10.2761	3.6734	3.9792	4.9982

ISOVELOCITY MOMENT							APPENDIX J		
RIGHT KNEE EXTENSOR MOMENT									
AREA	PRE	PRE	PRE	MID	MID	MID	POST	POST	POST
Subject	A	B	C	A	B	C	A	B	C
#1	23.9195	23.7401	23.4282	25.1569	25.4348	25.5704	57.1876	55.5878	54.4218
#2	54.6998	54.7336	38.803	49.2494	43.3449	45.1075	55.859	57.0114	55.0048
#3	51.2086	45.7854	54.7336	46.5446	51.6967	48.5241	59.4789	59.2891	57.852
#4	31.6715	34.2882	30.6207	42.9517	37.8539	40.0232	60.9297	59.6688	55.8047
#5	0	0	0	0	0	0	0	0	0
#6	22.6961	21.0827	21.0013	21.2861	20.2014	18.8049	3.7285	2.9285	3.6335
#7	97.5091	89.0354	93.2112	73.1725	73.2946	72.3862	127.7435	120.3408	123.1608
#8	missing	missing	missing	32.8917	34.0984	30.9122	44.755	40.2401	40.8909
#9	39.9419	38.8843	36.3626	30.0445	31.6579	30.1123	49.0262	44.6872	42.2738
#10	46.4361	48.0767	49.1613	45.3244	41.1621	35.3457	62.6108	60.2518	63.5549
#11	133.7768	141.1659	133.7903	116.2938	113.5345	120.1781	186.8428	193.5676	195.6962
#12	66.3664	66.4342	67.5866	54.754	61.8516	62.9905	88.6286	84.5884	82.2835
#13	70.2711	70.1355	70	75.8841	73.2674	81.3616	94.794	98.2448	89.8082
#14	34.2611	35.8338	36.2676	36.6202	37.1354	40.3893	48.9986	48.1987	58.9637
#15	41.4197	43.8195	43.25	52.6999	50.9645	50.3815	66.7189	69.1187	67.4104
#16	7.4705	9.3686	11.1854	3.1183	3.145	2.3184	7.4569	6.2909	5.7757
#17	14.8189	14.2088	12.6361	12.9682	12.7852	12.6361	19.1303	19.5913	18.9812
#18	17.0017	16.0256	18.0864	11.6056	12.7716	12.9479	4.2979	4.8944	6.6977







ISOMETRIC MOMENT							APPENDIX J		
RIGHT KNEE EXTENSOR MOMENT									
PEAK	PRE	PRE	PRE	MID	MID	MID	POST	POST	POST
Subjects	A	B	C	A	B	C	A	B	C
#1	51.5204	58.2994	59.6552	46.0972	48.8088	42.0298	93.5502	89.4828	84.0596
#2	89.4828	88.127	85.4154	75.9248	75.9248	75.9248	89.4828	90.8386	86.7712
#3	47.453	52.8762	56.9436	36.6066	32.5392	35.2508	73.2132	63.7226	58.2994
#4	52.8762	51.5204	52.8762	61.011	61.011	51.5204	63.7226	66.4342	66.4342
#5	0	0	0	0	0	0	0	0	0
#6	20.337	21.6928	18.9812	20.337	18.9812	17.6254	8.1348	6.779	5.4232
#7	138.292	122.022	117.955	116.599	105.752	107.108	131.513	134.224	119.31
#8	missing	missing	missing	51.5204	58.2994	59.6552	66.4342	66.4342	62.3668
#9	52.8762	56.9436	56.9436	44.7414	46.0972	46.0972	59.6552	58.2994	58.2994
#10	46.0972	47.453	55.5878	42.0298	50.1646	43.3856	73.2132	71.8574	74.569
#11	145.071	127.445	141.003	142.359	130.157	127.445	248.111	253.535	244.044
#12	81.348	82.7038	82.7038	86.7712	90.8386	82.7038	94.906	90.8386	90.8386
#13	47.453	47.453	48.8088	70.5016	61.011	61.011	117.955	109.82	100.329
#14	27.116	24.4044	28.4718	44.7414	52.8762	46.0972	67.79	73.2132	74.569
#15	65.0784	missing	missing	78.6364	84.0596	79.9922	94.906	98.9734	101.685
#16	6.779	5.4232	0	9.4906	0	0	4.0674	5.4232	5.4232
#17	20.337	21.6928	18.9812	21.6928	21.6928	21.6928	27.116	27.116	28.4718
#18	20.337	18.9812	17.6254	12.2022	13.558	12.2022	0	0	0

ISOMETRIC MOMENT							APPENDIX J		
RIGHT KNEE EXTENSOR MOMENT									
MEANS	PRE	PRE	PRE	MID	MID	MID	POST	POST	POST
Subjects	A	B	C	A	B	C	A	B	C
#1	57.6384	54.4141	55.8239	36.2761	41.416	36.7213	83.8505	83.9584	78.7781
#2	80.4441	79.9585	74.9805	69.618	70.5758	70.306	80.6263	79.6752	79.4054
#3	42.6166	48.4041	48.1343	28.2492	28.1278	29.3622	65.4359	57.2336	51.3315
#4	46.8999	46.4412	47.0348	57.4023	57.0853	44.9707	58.6097	57.4967	59.5332
#5	0	0	0	0	0	0	0	0	0
#6	18.7654	18.7991	17.9154	18.853	17.2746	15.3995	7.1635	4.9713	3.973
#7	123.9848	112.2143	103.1015	103.5871	91.4255	96.0527	119.1688	111.0609	103.8165
#8	missing	missing	missing	47.7633	53.7801	56.3132	60.64	61.9014	57.2215
#9	44.593	45.5239	43.8038	36.4177	38.0906	38.3266	46.0972	45.1664	44.7009
#10	41.3553	42.0028	48.6739	37.3621	43.9994	38.2457	63.0143	65.4426	70.623
#11	128.6998	115.5196	125.1788	123.7016	120.6055	117.5768	216.7796	233.5484	226.3512
#12	73.2199	75.972	77.4088	64.1745	83.3041	74.8658	87.6683	85.7729	83.7628
#13	39.3182	40.674	41.0248	59.554	47.1427	47.77	104.336	95.3512	90.7509
#14	24.9036	22.0368	24.944	41.679	44.1141	42.6504	62.441	63.7563	65.9958
#15	51.1831	missing	missing	71.5539	71.709	68.6916	82.3733	85.3345	84.1274
#16	5.2805	2.4103	0	8.6724	0	0	3.865	3.8313	4.1281
#17	18.7654	18.7991	17.9154	20.2021	20.1009	19.4015	24.9036	23.9794	26.6978
#18	17.902	15.73	15.5479	9.1477	11.2456	9.7334	0	0	0

ISOMETRIC MOMENT							APPENDIX J		
RIGHT KNEE EXTENSOR MOMENT									
AREA	PRE	PRE	PRE	MID	MID	MID	POST	POST	POST
Subject	A	B	C	A	B	C	A	B	C
#1	231.1097	218.2431	223.8968	145.518	166.1397	147.2806	336.2926	336.6994	315.9421
#2	322.6262	320.6738	300.608	279.1592	282.9826	281.9251	323.2634	319.5485	318.4367
#3	170.885	194.0692	193.0117	113.2906	112.8026	117.7648	262.4015	229.4963	205.8511
#4	188.063	186.2056	188.5782	230.1742	228.8862	180.2672	234.9873	230.486	238.1327
#5	0	0	0	0	0	0	0	0	0
#6	75.2469	75.396	71.8574	75.613	69.2678	61.7567	28.743	19.9438	15.9306
#7	497.0901	449.9761	413.424	415.3357	366.5542	385.142	477.8792	445.3936	416.3253
#8	missing	missing	missing	191.5881	215.6671	224.6967	243.1898	248.247	228.3438
#9	178.8707	182.6534	175.7252	146.0603	152.7851	153.7477	184.9582	181.2298	179.3588
#10	165.8143	168.3904	195.181	149.8159	176.4303	153.4359	252.5855	262.3744	283.7418
#11	516.0581	463.1957	501.9442	495.8705	483.5324	471.3977	868.9728	936.3564	907.4912
#12	293.5714	304.6076	310.3833	257.1139	334.0013	300.1877	351.4911	343.9122	335.8181
#13	157.7202	163.157	164.5399	238.8784	189.0934	191.6152	418.3189	382.3626	363.9237
#14	99.8547	88.3846	100.0038	167.1159	176.8234	171.0206	250.3485	255.6496	264.6522
#15	205.3495	missing	missing	286.1944	287.6194	274.1156	330.3136	342.2039	335.7232
#16	2.0608	0.8677	0	10.06	0	0	15.5104	15.3748	16.5543
#17	75.2469	75.396	71.8574	81.009	80.6023	77.4162	99.8547	96.194	107.054
#18	71.8167	63.0718	62.3532	31.1021	40.4842	39.0335	0	0	0

**Appendix K**

The pre, mid and post study thigh circumference measurements in the subject population.

LEFT THIGH CIRCUMFERENCE							RIGHT THIGH CIRCUMFERENCE					
#	at 7 cm.			at 20 cm.			at 7 cm.			at 20 cm.		
	pre	mid	post	pre	mid	post	pre	mid	post	pre	mid	post
1	57.4	61.5	60.2	74.2	73.0	73.4	58.5	58.3	59.2	74.5	73.3	75.0
2	34.4	32.5	33.4	43.0	43.3	43.0	34.3	33.4	33.0	42.6	43.1	42.8
3	41.3	41.8	42.2	54.7	54.6	53.7	54.1	57.3	55.3	72.5	74.6	71.5
4	34.5	36.0	35.3	45.0	44.5	44.2	37.0	37.1	37.2	48.2	47.0	48.0
5	34.2	33.6	33.8	42.0	40.8	41.8	30.3	29.5	29.5	35.3	33.9	34.9
6	58.5	57.0	56.6	74.2	71.1	70.4	47.7	48.4	46.7	67.8	67.0	66.9
7	36.0	35.0	35.9	44.9	44.5	45.0	39.2	39.0	39.2	50.6	50.8	51.1
8	30.5	29.9	30.0	36.5	36.0	36.1	39.6	38.2	39.6	53.0	52.0	52.7
9	35.0	34.6	35.8	44.3	44.1	45.2	35.2	35.1	35.9	45.5	45.2	46.5
10	32.9	32.9	33.3	40.7	41.5	41.8	32.1	33.2	33.4	38.8	40.0	40.5
11	40.0	39.6	39.9	51.6	51.4	51.2	42.5	42.3	42.1	53.2	52.5	52.8
12	56.2	53.5	52.6	66.9	67.6	66.4	53.4	52.5	52.0	66.8	67.7	67.7
13	39.5	39.1	39.3	54.3	53.8	52.5	45.0	43.7	45.5	59.4	61.0	59.3
14	44.6	44.0	42.6	48.1	47.8	46.3	51.1	50.0	47.5	65.1	65.2	64.1
15	41.7	40.5	41.2	51.2	50.5	51.4	44.1	44.5	45.5	49.5	57.8	58.3
16	32.4	31.8	32.8	42.4	42.5	45.9	34.7	34.5	35.1	47.2	47.5	50.2
17	47.9	46.3	46.3	61.8	61.5	62.4	48.5	47.3	47.9	61.8	60.7	63.5
18	41.3	40.5	41.2	53.2	52.5	54.1	43.2	42.8	42.4	62.1	60.6	54.6
$\bar{x}$	41.0	40.5	40.7	51.6	51.2	51.4	42.8	42.6	42.6	55.8	55.5	55.6

**Appendix L**

The pre, mid and post study weights and the skinfold measures of the subject population.

#	LEFT SKINFOLD			RIGHT SKINFOLD		
	pre	mid	post	pre	mid	post
1	58.0	58.0	58.0	58.0	58.0	58.0
2	34.2	33.7	34.4	35.6	33.8	37.8
3	53.0	56.2	56.0	58.0	58.0	58.0
4	16.2	19.6	17.0	17.0	20.0	20.6
5	22.6	22.0	22.8	20.6	20.0	21.4
6	58.0	58.0	58.0	58.0	58.0	58.0
7	29.2	30.4	28.8	36.6	37.5	36.8
8	29.5	30.0	31.3	40.2	38.8	38.4
9	31.0	30.0	30.6	34.0	34.6	35.2
10	16.4	17.2	19.5	22.2	23.0	24.1
11	32.0	33.0	32.8	36.6	36.2	34.6
12	47.8	48.2	48.0	48.2	50.0	50.8
13	45.5	42.2	42.6	37.2	40.8	40.0
14	41.2	39.8	38.6	53.4	51.4	50.0
15	32.5	33.2	31.4	39.5	38.4	40.4
16	28.0	29.5	26.0	34.2	34.0	32.2
17	57.2	58.0	57.0	56.4	58.0	57.0
18	48.6	46.4	45.2	45.4	43.6	44.4
mean	<b>37.83</b>	<b>38.08</b>	<b>37.67</b>	<b>40.62</b>	<b>40.78</b>	<b>40.98</b>