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Treatment of Spasticity via Transcranial Electrical Stimulation in Persons with Cerebral Palsy and Severe Mental Retardation

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TREATMENT OF SPASTICITY VIA TRANSCRANIAL ELECTRICAL STIMULATION IN PERSONS WITH CEREBRAL PALSY AND SEVERE MENTAL RETARDATION

by

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TREATMENT OF SPASTICITY VIA TRANSCRANIAL ELECTRICAL STIMULATION IN PERSONS WITH CEREBRAL PALSY AND SEVERE MENTAL RETARDATION

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Although the clinical manifestations of cerebral palsy are many and varied, spasticity is a common aspect of the disorder, observed in as many as 75% of all cases (Paneth & Kiely, 1984). A double-blind and placebo-controlled and counter balanced reversal design was employed to evaluate the effectiveness of transcranial electrical stimulation (TCS) in the treatment of spastic cerebral palsy in six severely mentally impaired individuals. Although there appeared to be some improvement from pre to post measures, a comparison of the placebo and treatment conditions yielded results indicating that transcranial electrical stimulation did not increase the passive range of motion of three target joints or affect therapists' ratings of the ease of passive movement. Therapists' ratings of the pain experienced by participants during passive movement of target joints were also unaffected by the intervention. These findings appear to contradict an earlier investigation (Malden & Charash, 1985) which found transcranial electrical stimulation useful in the treatment of spasticity. Results are discussed in light of a lack of basic research supporting the hypothesized mechanism of action.

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George A. Thompson, Jr.

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INTRODUCTION

The Problem

Although the clinical manifestations of cerebral palsy are many and varied, spasticity is a common aspect of the disorder, observed in as many as 75% of all cases (Paneth & Kiely, 1984; Raney & Brashear, 1971). Cerebral palsy is a clinical syndrome, not a disease entity (Barabas & Taft, 1986). Perlstein (1950) outlines several defining characteristics of cerebral palsy including any bilateral, symmetrical, and non-progressive paralysis or associated abnormalities of posture, muscle tone and/or muscle coordination resulting from cerebral developmental defects or traumatic brain damage occurring at or soon after birth. The term cerebral palsy actually encompasses a number of clinical neurologic syndromes of quite heterogeneous etiology. These syndromes are grouped together not because they are assumed to have a common cause but because they share characteristics that are important in their clinical management (Paneth, 1986). Because of this marked heterogeneity, the plural form "cerebral palsies" might be more appropriately applied.

Although ancient descriptions of this disorder can be found (Illingworth, 1958), the modern history of cerebral palsy dates from 1843 when William J. Little, an English surgeon, first described the condition (Phelps, 1949). Winthrop Phelps (1948), an orthopedic surgeon, coined the term "cerebral palsy" and described its various

types. The non-progressive nature of this condition is one key to distinguishing it from other movement disorders. Since vascular disease or trauma can cause a spastic hemiplegia at any age, inherent in the definition of cerebral palsy is the notion of congenital or almost congenital onset (onset here referring to the timing of the insult, not the diagnosis of the illness, as the final picture does not generally appear until 1 or 2 years of life). Kurland (1957) has suggested that to be accurate the insult must have occurred between conception and one month of life. Some clinicians have extended the time of onset to the first several years of life (Barabas & Taft, 1986). Cerebral tumors, skull fractures, and cerebral hemorrhages should not be mistaken for cerebral palsy, but any consequent permanent and non-progressive movement disorder may appropriately be classified as cerebral palsy. The majority of individuals exhibiting cerebral palsy, however, do not manifest the condition as a result of late childhood factors (14% acquired) but more typically as a result of a variety of diseases and/or insults occurring during pregnancy or at the time of birth (86% congenital). It is clear from this review that prevalence-rate of cerebral palsy can vary significantly, ranging from .87 per 1,000 to 5.8 per 1,000 live births.

The National Institutes of Health (NIH, 1980) have estimated that 1-3 of every 1,000 live born develop cerebral palsy, resulting in about 9,000 new cases per year. It is important to note that the incidence of cerebral palsy may change with the advance of medical knowledge and its application (Altman, 1955). However, there is no

firm evidence that the incidence is declining; Kudrjavcev, Schoenberg, and Kurland (1984) found a decline in prevalence between the 1950s and 1960s and a leveling off since then. In the United States there has been a significant decrease in some severe forms of cerebral palsy due to the treatment and prevention of Rh incompatibility and to the availability of vastly improved obstetrical care. Unfortunately, parallel to these medical developments, prematurity as a cause of cerebral palsy seems to be increasing as a result of the improved survival rate of the premature. According to Keats (1965a) there appears to be no relationship between the incidence of cerebral palsy gender, race, or socioeconomic status. The American Academy for Cerebral Palsy (NIH, 1980) estimates the prevalence of cerebral palsy in the United States to be 750,000.

Etiology

There is considerable evidence that cerebral palsy is typically the result of a generalized cerebral insult (Perlstein, 1962). Towbin (1961) has indicated that intercranial hemorrhage plays a major role in the pathologic process of most cases. The hemorrhage may occur directly from a vessel ruptured when the dura mater is torn, or it may be widespread resulting from increased tension when the venous circulation is temporarily obstructed. Degeneration of the injured nerve cells and fibers ensues and is followed by sclerosis (Raney & Brashear, 1971; Schwartz, 1961). The affected areas may show atrophy and softening. The resulting lesions are likely to

be most severe either in the pyramidal system or in the basal ganglia. Degeneration of the corresponding tracts in the spinal cord is a consistent finding (Perlstein, 1962). In spite of the generalized nature of the cerebral insult it is important to note that there is a marked relationship between the nature and primary location of the cerebral damage and the type(s) of impairment(s) observed clinically (Keith, Norval, & Hunt, 1950). Contusion and laceration of specific cerebral tissue is likely and the resulting lesions produce different types of motor dysfunction depending on the part(s) of the brain affected.

Approximately 17.5% of all patients present with multiple etiological factors (Fishbein, 1963), most commonly: prematurity, cesarian section, dystocia (i.e., difficult labor), anoxia, respiratory distress, and breech birth. Interestingly, Nelson and Ellenberg (1981) have shown that virtually all obstetrical risk factors for cerebral palsy in normal weight infants increase the probability of cerebral palsy only when associated with birth asphyxia as indicated by a depressed 5 minute Apgar score.

It should be noted that the incidence of various etiological factors has changed significantly over the past five decades (O'Reilly, 1982). Among other factors hydrocephalus, prematurity, trauma, multiple births, and toxemia have shown a relative increase while idiopathic cases have shown a relative decrease over the same period. These changes can be traced directly or indirectly to improvements in medical technology. The relatively high incidence of

the idiopathic variable is apparently due to the lack of knowledge of many events that may have occurred during pregnancy, especially in the early stages.

Pathology

A focused hemorrhage or infarct damaging the pyramidal system results in spastic paralysis. Spasticity (hypertonus) is an exaggerated muscle contraction in response to stretch. This condition is revealed by hyperactive deep tendon reflexes and clonus. Spasticity results in difficult and inaccurate movements. In addition, because there is a marked difference in the tone of spastic muscles compared to their opposing muscles, the typical spastic quadriplegic will show flexion at the wrist and elbow, adduction and internal rotation of the arm, adduction and flexion at the hip, flexion of the knees, and pronounced ankle extension. As the individual grows the spastic muscles become relatively shorter (contracture) with resultant deformities of the limbs, pelvis, and spine. Raney and Brashear (1971) report that from 50% to 75% of all cases of cerebral palsy exhibit some degree of spasticity.

The inactivity resulting from cerebral palsy causes muscles to lose mass and strength. This condition in turn will have a negative impact on joint mobility over time. Muscle spasm is often found in the flexor muscles resulting in a marked shortening of these muscles which are typically stronger than the extensors. These spasms will tend to limit motion resulting in the formation of fibrous tissue

which in turn may produce fibrous or bony ankylosis. The long term result is the development of contracture deformity and the loss of additional function (Rushforth, 1962).

The cerebral palsied individual may have any orthopedic problem which any other child might have--club foot, congenital deformities of the spine (e.g., scoliosis/kyphosis), and so forth. These problems are not necessarily the result of cerebral palsy. There are, however, several orthopedic problems seen in the cerebral palsied population which are the result of this condition. Flexor deformities due to spasticity occur in both the upper and lower extremities. Knee-flexion deformities as seen in the bent-knee gait are common. Flexor deformities are also seen at the hip, elbow, and wrist. Another example of this type of deformity is seen in the thumb-in-palm and flexed-finger posture associated with spasticity.

Abnormal postural attitudes and deformities may result from muscular weakness. Badly pronated feet may be due to inactive tibial muscles. Weak spinal muscles may lead to loss of normal lordosis. Head drop may result from weak posterior neck muscles. The flexed position associated with loss of normal lordosis and weak abdominal muscles may lead to flaring of the rib margins.

Abnormal muscle stresses may produce changes in the architecture of certain bones. One of the most common orthopedic problem encountered in cerebral palsy is dislocation of one or both hips (Jones, 1961). In the cerebral palsied individual there is often weakness in the hip abductors and spasticity in the adductors and hamstrings

(Sharrard, 1961). This condition results in a tendency for these abnormal muscle pulls to push the head of the thigh bone out and up from the socket. Osteoporosis, a marked atrophy of the bones and generalized decrease in bone mass, is also a common finding resulting from the inactivity associated with cerebral palsy.

When compared with the expected normal intellectual curves in the population it is clear that approximately 75% of individuals with cerebral palsy exhibit some degree of mental retardation (Holman, 1953). Studies of large cerebral palsied populations indicate that serious mental retardation occurs in at least 50% of the population. Specifically, Dunsdon (1952) estimated that of 916 individuals tested 58.6% had an IQ under 69, 17.2% had an IQ between 74 and 80, and 23.75% had an IQ over 85. Cruickshank (1976) found IQ's under 85 in 75.5% of cerebral palsied individuals he examined. In addition, Kirman (1956) has indicated that 23% of the mentally retarded population exhibits some type of cerebral palsy. For many years it was believed that athetoids had intellectual ability superior to that of spastics because the athetoids' lesions were subcortical and the spastics' lesions were in the cerebral cortex. Recent comparisons, however, suggests that differences between these two groups are probably not significant. A more accurate correlation appears between intellectual development and topography of involvement. According to Cruickshank (1976), spastic paraplegics are least likely to exhibit intellectual deficits. Mental retardation is somewhat more likely to be found among hemiplegics; an even greater proportion

of spastic quadriplegics are likely to exhibit some degree of mental retardation.

For numerous reasons, effective treatment of spasticity is difficult. The relative inaccessibility of the brain to therapeutic interventions, the early onset of pathology, the apparently irreversible nature of central nervous system damage, and the poor correlation of postmortem findings to the clinical picture of patients with cerebral palsy all contribute significantly to the failure of the scientific community to find a cure for cerebral palsy. Since at present no intervention is available that will correct the brain damage that is at its root, the treatment of cerebral palsy in large part actually involves the management of the various handicapping conditions which result from the non-progressive insult to the central nervous system.

Drug Treatment

The primary focus in the pharmacological treatment of cerebral palsy is the control of the deforming character of spasticity in specific muscle groups. For example, spasticity in the hip adductors can cause both a scissoring gait as well as place the hips at risk for subluxation. Similarly, gastrocnemius spasticity can cause toe walking as well as difficulty tolerating braces. Spastic hamstrings contribute significantly to the crouched, internally rotated gait seen in diplegics. Common offending muscle groups in the upper

extremities include the wrist and finger flexors as well as the elbow flexors.

Pharmaceutical agents commonly used in the treatment of cerebral palsy include agents such as dantrolene sodium, diazepam, and baclofen, and, to a lesser extent, benztropine mesylate and clonazepam (Denhoff, 1964; Denhoff & Holden, (1961). The former three agents are used primarily for treating generalized spasticity. diazepam acts primarily centrally, while dantrolene sodium's action is peripheral. Baclofen's site of action is less well defined and may include both spinal and brain centers. All three have been shown to cause quantitative relaxation in muscle tension, but most studies have shown poor effect for functional improvement in spite of relaxation achieved (Joynt & Leonard, 1980) due to the sedative effects at the necessary dose. Benztropine mesylate and clonazepam have been used more recently to provide relaxation in children who show primarily rigidity and have been found to be quite effective, although dosages need to be titrated in order to avoid over sedation (Diamond, 1986).

Chyatte and Basmajian (1973) conducted an initial investigation of the muscle relaxant (spasmolytic agent) dantrolene sodium. Their results indicated the decrease in spasticity caused by this drug brings worthwhile functional improvements. More than 90% of the subjects in this study showed some reduction of spasticity with dantrolene sodium, but gross strength also tended to diminish as the muscle relaxed. Nevertheless, motor function was usually improved

through the alleviation of spasticity more than it was impaired by concomitant weakness, particularly with regard to the function of the hands and fingers. The data indicated that net improvement depended on the degree of useful motor function unmasked by relief from spasticity balanced against drug induced weakness and/or loss of useful support of posture and movement supplied by spastic contraction. Curare-like drugs have been used to this end (Denhoff & Bradley, 1942) and while they do exhibit the desired effect they are considered too dangerous because of the narrow margin between therapeutic and toxic doses.

Denhoff, Feldman, Smith, Litchman, and Holden (1975) evaluated sodium dantrolene for efficacy and safety in 28 individuals with spastic forms of cerebral palsy in a double blind study with treatment periods of six weeks. While five of the 28 subjects showed marked benefit from the drug as compared with the placebo; in no case did all of the observers agree about improvement. Ten subjects showed marginal improvement and thirteen showed no consistent differences. Minor side effects were transient, but three individuals, in whom the drug was continued after the study period, had increased seizures. A fourth who had a normal EEG and no previous seizures developed seizures. Parents reported a small but definite improvement in activities of daily living during the period of drug administration.

Joynt and Leonard (1980) conducted a double-blind study with 20 individuals who presented with spasticity secondary to cerebral

palsy in order to compare the effects of dantrolene sodium suspension and a placebo. The drug was found to be physiologically active in reducing the force of muscle contraction but objective functional improvement, as measured by multiple performance tests, was irregular and not significant.

Since most individuals with cerebral palsy exhibit some degree of spasticity, pharmacological techniques for selective relief of spasticity may also have an appropriate role in treatment. Such techniques include motor point blocks and peripheral nerve blocks. Various agents have been used. Phenol in a 2% - 3% solution is the most commonly described in the literature.

Khalili and Betts (1967, 1970) have achieved some preliminary success in alleviating spasticity in selected muscles by means of phenol nerve blocks. In this technique carefully graded phenol solutions are used selectively to block the small gamma motoneuron axons while leaving the large alpha motoneuron axons generally unimpaired. The muscle spindles are selectively relaxed and the sensitivity of the muscle to stretch stimuli is greatly reduced. Reporting on a series of 68 patients, Khalili and Betts (1967) noted that spasticity immediately diminished in the muscle innervated by the blocked nerve. This frequently resulted in alleviation of contracture, improvement of range of motion, and less need for stretching, bracing, and splinting. In some cases, voluntary contraction improved in the muscle innervated by the blocked nerve. Relief of spasticity and improvement of voluntary stretch was

sometimes noted in muscles not innervated by the blocked nerve, in the same or the contralateral limb. Many patients showed some functional improvement in activities of daily living, though these improvements were inferred from essentially subjective and anecdotal information (Khalili & Betts, 1970). Unfortunately, benefits were not permanent since spasticity returned to pre-treatment levels over several months, necessitating a renewal of the nerve blocks.

Carpenter and Seitz (1980) found that the use of intramuscular alcohol led to reduced spasticity for varying periods of time, although periods diminished with each subsequent injection. During the period of reduced spasticity there is an opportunity for therapists and orthopedic surgeons to determine whether corrective surgery is indicated. The injections produced no adverse side effects. Diamond (1986) has also reported the use of a 50% solution of ethyl alcohol because of its ease of handling and lesser risk of systematic toxicity.

Peripheral nerve blocks (Chyatte & Basmajian, 1973) have also been used to demonstrate the effect that could be expected with a more permanent surgical procedure and are also found to be helpful by the orthopedic surgeons in facilitating the effect of stretching with serial casts. The only untoward effect that has been encountered is the temporary development of painful paresthesia (tingling sensation) in 10% to 15% of the individuals undergoing tibial nerve block. This generally lasts at most 3 to 4 weeks and can almost always be managed with mild analgesics and the soothing effects of hydrotherapy.

The effects of peripheral nerve blocks are temporary, generally lasting from four to six months. The procedure is therefore useful in "buying time", allowing therapy to be delivered more effectively. For very young children, a postponement is possible in the performance of initial invasive surgical procedures until the child has gotten past his first pre-school growth spurt. Not uncommonly, even once the block has worn off, the fact that therapy has been given more effectively for a prolonged period of time prevents the problem of spasticity from being quite as manifest as it was previously. Repeat blocks are rarely necessary.

A recent small study (Goldkamp, 1984) of the use of L-dopa in athetoid cerebral palsy indicated clinical improvement in two-thirds of the patients. However, the drug was associated with high incidence of side effects, including nausea, vomiting, and depression.

It should be noted that several pharmacological agents are commonly used for treatment of problems associated with cerebral palsy. Seizures are typically controlled pharmecutically. In addition to its muscle relaxant action, benztropine mesylate has also been applied with some success to the control of drooling (Camp, Winsberg, Green, & Abrams, 1985).

In summary, no medication evaluated to date has been found to function as more than an adjunct in the management of cerebral palsy. The use of many of these medications is limited because of significant side effects such as gastrointestinal intolerance or central nervous system depression. The efficacy of pharmacological agents in

treating cerebral palsy has not been well substantiated, particularly in regard to improving function.

Surgical Treatment

Various orthopedic and neuro-surgical procedures have been employed (Benedetti, Colombo, Alexandre, & Pellgri, 1982; Raney & Brashear, 1971; Sherk, Pasquariello, & Doherty, 1983) in the treatment of cerebral palsy. The surgical procedures now in common use in the treatment of cerebral palsy may be classified as operations upon motor nerves such as cervical rhizotomies, operations on muscles and tendons such as tendon transfers, and operations on bone such as femoral resections. Of these categories, operations upon bone appear to yield the best results (Keats, 1970; Pollack, 1962). Several operative procedures have been designed specifically to correct various problems such as nutritional insufficiency and drooling associated with cerebral palsy.

As a rule, surgical intervention for cerebral palsy management is indicated chiefly in cases of spasticity. In many such cases surgical measures serve as an adjunct to motor reeducation (Eggers & Evans, 1963). Baker (1956) has indicated that the medical community regards surgery as an adequate treatment only when it is accompanied by less intrusive measures and that it is contraindicated when the patient exhibits mental capacity insufficient for post-operative training.

A refinement of surgical technique over the past two decades has contributed to improved results of surgical intervention (Marquis, 1979). The actual procedures have not changed greatly; they continue to involve soft tissue lengthening and neurectomy to reduce deformity, establish better balance of muscle power, and decrease muscle spasticity. The gains are primarily the result of better selection of both patient and procedure improved preoperative assessment.

Keats (1970) has considered the objectives of surgical treatment in cerebral palsy to be (a) diminished level of muscle spasm, (b) equalized power for opposing muscles, (c) stabilization of poorly controlled joints, and (d) correction of deformity. A great many surgical procedures have been devised and applied; unfortunately, many have proven to be unsatisfactory upon long term follow up. Pollack (1962) in a survey of 2,500 surgically treated cases of cerebral palsy of various types noted that approximately 50% could be considered successful based on clinical impression. In this study almost all of the operations upon athetoid patients failed.

Pollack and English (1967) have described surgical procedures on muscles and tendons which include tendon transplantation, tendon lengthening, tenotomy, and myotomy. These procedures are sometimes combined advantageously with section of motor nerves to the spastic muscle group.

The success of upper extremity surgery depends not only on the mechanics of the individual joint but also on the degree of cortical

sensation and selective arm control. These factors frequently limit the goals to cosmetic correction of flexion deformities of the upper extremity without significant functional improvement. Keats (1965) has pointed out that pronation contracture of spastic origin may be relieved by suitable transplantation of the tendon of the pronator teres muscle. Complicated tendon transplantations are rarely successful; although in tenotomy, tenodesis, and tendon transplantation they may be helpful in carefully selected cases (Swanson, 1968).

Goldner (1971) has demonstrated that when the wrist is flexed and cannot be held voluntarily in the neutral position, radiocarpal arthrodesis in a position of 10 degrees of extension improves the appearance and in some cases the function of the hand. Mital (1979) has devised a procedure for treating the inability to extend the elbow due to either a fixed contracture of the flexor muscles or increased involuntary flexor muscle tone and spasticity. This operation consists of lengthening all of the primary elbow flexor muscles. The operation was judged successful in 32 elbows of 26 individuals with cerebral palsy who were followed for four years. The average gain in extension was 40 degrees and there was no loss of the patient's ability to flex the elbow or supinate the forearm. No vascular or neural complications were encountered. Independence in feeding was achieved in four patients. Six patients reported significantly improved self care skills. Seven patients acquired some ability to engage in sports and six patients who previously had

been unable to walk independently due to an inability to handle crutches could do so following this procedure.

Deformities of the lower extremities are more amenable to surgical intervention. The most common deformities are hip flexion, adduction and internal rotation, knee flexion, ankle equinus, and varus or valgus inclination of the foot. These deformities result in a crouched position commonly observed in cerebral palsy. In cases of severe adduction deformity, tenotomy of a portion of the adductor muscles, followed by immobilization of the thighs for a period of about six weeks in wide abduction, often results in substantial gait improvement (Banks & Green, 1960).

Transplantation of the anterior tibial tendon (at insertion or origin) to the lateral side of the foot may be used to improve spastic deformity (Baker & Hill, 1964). Lengthening of the Achilles tendon to correct talipes equinus is indicated only when there is unquestionable structural shortening, since spastic equinus can easily evolve into a more debilitating deformity. However, when actual shortening is present, Achilles tendon lengthening is one of the most frequently used and successful operations in the treatment of cerebral palsy (Banks & Green, 1960). After any operation for equinus, the ankle should be immobilized in 90 degrees of dorsiflexion.

Varus and valgus deformities of the foot result from an imbalance of the muscles of inversion (tibialis posterior) and eversion (peroneal). These abnormalities can be corrected by either rerouting

the posterior tibial tendon anteriorly to the medial malleolus or by calcaneal osteotomy. With the use of these rather simple procedures, recent studies have reported 80 to 90 percent improvement of foot deformities.

Operative procedures aimed at correcting equinus deformity of the ankle have a high success rate. The two most common operations for this deformity are simple Achilles tendon lengthening and gastrocnemius division which leaves the soleus muscle intact. Both procedures now yield a high rate of successful correction with recurrence rates of less than 5 percent.

A study by Schneider and Kornell (1977) points to the need to exercise caution when considering surgical intervention. These authors studied 24 individuals with spastic equinovarus resulting from cerebral palsy who were treated by anterior transfer of the posterior tibial tendon and Achilles tendon lengthening. In five patients the operation was performed bilaterally resulting in a total of 29 feet available for evaluation. After an average follow up of 5 years only 38% of the results were graded as good or satisfactory. Sixty-two percent were graded as poor because of valgus, calcaneus, or equinus deformity severe enough to require re-operation. The post-operative deformity was generally evident one or more years after surgery, often progressive, and more disabling as well as more difficult to correct than the original condition. Although the percent of acceptable results was considerably higher in hemiplegics

than for others, it was concluded that in spastic patients anterior transfers of the posterior tibial tendon should not be performed.

The basic procedure for correction of knee flexion is hamstring transfer. Eggers (1952) has described a number of different operations devised to correct incomplete knee extension on standing. Occasionally, it is sufficient to lengthen one or more of the hamstring tendons. If the patellar ligament has become elongated, it may be shortened and good results have also been obtained following transplantation of the hamstring. This is the most difficult deformity for the orthopedic surgeon to correct because intervention is almost always a failure if equinus or hip flexion remains uncorrected. The usual complication is genu recurvatum.

Ray and Ehrlich (1972) conducted a retrospective analysis of 23 patients who underwent 43 transfers of the semitendinosus muscle to the lateral intramuscular septum and of the semimembranosus muscle to the biceps. Decreased knee flexion deformity as well as improved walking were achieved in 91% of the cases studied. Unsatisfactory results were associated with complications of the procedure. Only one knee of the 43 operated on exhibited late genu recurvatum.

There have been large outcome studies of hip surgery in patients with cerebral palsy (Raney & Brashear, 1971). The most frequent procedures were adductor tenotomies and obturator neurectomies. In addition, some patients underwent release of hip flexors and derotation osteotomy of the femur. The success rate of surgery was 70 to 80 percent when the surgical indication was to simplify nursing

care for severely affected patients or to reduce deformity. Hip surgery in cerebral palsy has a lower success rate when the goal of surgery is to improve activity.

Sherk, Pasquariello, and Doherty (1983) reviewed 45 cerebral palsied patients with hip dislocation and found that some did well without treatment. For patients with hip pain the best results were obtained with extensive resection arthroplasty; soft tissue surgery alone was unsuccessful. Open reduction with femoral osteotomy achieved concentric reduction in all 15 patients who had this type of surgery. However, most retained abnormal postural reflex activity to such a degree that subsequent scoliosis, pelvic obliquity, hip extension contractures and knee flexion deformities compromised the success of the hip surgery.

Root and Spero (1981) conducted a 10 year study of cerebral palsied patients to compare the effectiveness of hip adductor transfer with tenotomy. This study involved 50 patients who had 98 adductor transfers and 52 patients who had 102 adductor tenotomies with or without neurectomy. Groups were similar with regard to the severity of their disease, age, obturator, and associated concomitant surgery. Results were evaluated in three ways: functional change, change in passive movement of the hip, and change in hip stability. Data support the position that although the adductor transfer operation takes longer and is associated with a higher incidence of post-operative drainage, the over-all improvement is greater than after adductor tenotomy with or without neurectomy. The transferred

muscle provides greater pelvic stability, decreases hip flexion contractures, and reduces instability of the hip.

Although orthopedic surgery has an important therapeutic role in the management of cerebral palsy, it does have definite limitations. Orthopedic procedures have been relatively ineffective in the treatment of upper extremity involvement and are of questionable value for the large group of patients with extrapyramidal disease. Orthopedic procedures are least successful in those patients who have the most severe deformities (Goldcamp, 1984).

In a small number of patients with severe involvement, neurosurgical procedures have been utilized. Unfortunately, neurosurgery has achieved only limited success. At this time, neurosurgical intervention in cerebral palsy must be considered experimental and not conventional therapy. Stereotaxic procedures in which either the contralateral thalamus or the ipsilateral dentate nucleus of the cerebellum is ablated have been performed on a number of patients with cerebral palsy. The results of these procedures have been unspectacular, although mild improvement has been reported in about two thirds of the patients (Goldkamp, 1984). It is not clear, however, whether the mild postoperative improvements are maintained because there have been no long term follow ups. A number of significant complications from these procedures, including seizures and respiratory problems, have been noted.

Division or partial excision of the motor nerves of spastic muscles is helpful in some cases. The goal is not to paralyze a

muscle group and thereby abolish its entire function but to produce a loss of power in the stronger muscles sufficient to result in improved muscle balance. This results in increased capability of successful muscle training. As with most surgical procedures, this type of operation has produced better results in the treatment of the lower rather than upper extremities. The operation has proved particularly suitable when applied to the branches of the obturator nerve for adductor muscle spasm and to the branches of the tibial nerve for spasm of the plantar flexors (Banks & Green, 1960). In selected cases of spastic pronation of the forearm and flexion of the wrist, section of appropriate branches of the median nerve is a helpful procedure (Samilson & Morris, 1964). Since recurrences due to nerve regeneration are somewhat common, neurectomy is less popular today than it was in the past.

Another surgical approach to control spasticity is the posterior cervical rhizotomies. Fraioli, Nucci, and Baldassarre (1978) examined the effects of bilateral cervical posterior rhizotomy on dystonia, athetosis, respirations, and other autonomic functions. Bilateral cervical posterior rhizotomies (C1-C4, C5, or C6) were performed on 16 cerebral palsied patients. The majority showed decreased muscle spasms and athetoid movements with some improvements in their posture and voluntary mobility. Five patients suffered from irregular breathing and lethargy post-operatively and four patients showed reduced diaphragmatic activity. All four of these patients

developed pneumonia which was fatal in one case. Urinary retention of up to three months was also reported.

Benedetti, Colombo, Alexandre, and Pellegri (1982) conducted a series of rhizotomies on 21 spastic patients with an average age of 9.3 years. In 18 patients in whom the clinical picture showed spastic paraparesis, a selective posterior lumbar rhizotomy was performed. Three patients with disabling spastic quadraparesis underwent posterior cervical C1-C3 rhizotomy. Follow-ups were conducted from 4 months to 3.5 years, no mortality or major complications were reported and although results were good they were related to motivation for movement, perceptual deficits, and intellectual impairment.

Surgery has also been used to correct the problems associated with cerebral palsy. McGovern (1984) has reported on the success of the Janeway gastrostomy a simple, tubeless, and safe method to provide proper nutrition to cerebral palsied unable to eat by mouth because of incoordination of the tongue and/or pharyngeal swallowing muscles. Makhani (1974) has conducted an investigation of 50 cases of surgically treated drooling. Effective correction was generally realized by converting the anterior overflow of saliva into swallowing by altering the posterior submucosal tube extension of the parotid duct. No specific evaluation of results or long term follow up were conducted.

To date, methods such as those outlined above have been less effective than desired (e.g., Davidoff, 1978; Raney & Brashear,

1971), and often exposing the individual to various unpleasant side effects.

Sensorimotor Treatment

A less restrictive approach to intervention, sensorimotor based (Gillette, 1969) physical therapy involving relaxation procedures, positioning, inhibition of pathological reflex activity, and active or passive movement of the limb is also typically employed in the treatment of spastic cerebral palsy. Physical and occupational therapies, which employ sensorimotor techniques, play a major role in the management of cerebral palsy. These disciplines have evolved in such a manner that the boundary between the two has become somewhat blurred. Many systems of sensorimotor therapy have been developed over the years and each has been given the name of its inventors (Gillette, 1969). Conclusive evidence of the advantage of one particular therapy system over another has not been presented. Most systems are now amalgamations of techniques based on basic sensorimotor theory. Denhoff (1981) has taken the position that the main objective of therapy in treating the motor impaired should be to enhance mobility while preventing complications such as joint, tendon, and/or muscle contracture.

According to Golkamp (1984) the key to success of any therapy program appears to be the consistency of application and the fact that the family is thoroughly involved from the very beginning so that carryover and supplemental interventions can be undertaken at

home. In general, priority is given to maintaining normal joint range of motion through passive range of motion (stretching or elongation exercises). Useful movement patterns can then be encouraged.

The Fay-Doman-Delacato (Fay, 1954) approach is initially one of passive movement superimposed upon the patient; only later does this method call for active participation on the part of the patient. Rood's (1954) approach uses a strong mixture of exteroceptive and proprioceptive input in developmental patterns followed by activity utilizing the stability and mobility components of the patterns. The individual's attention is directed to the activity and not to the movement patterns per se. Orthokinetics (Blashy & Fuchs, 1959) provides a continuous exteroceptive input followed by proprioceptive feedback as muscles are facilitated and inhibited. The individual is able to use the resultant muscle function in activities of daily living.

Until the 1950s physical therapists used the traditional approach of bracing and passive range of motion in an attempt to prevent contractures, maintain full range of motion, and maximize full muscle power of patients with cerebral palsy. For various physiologic reasons, such an approach alone is doomed to failure in both spastic and athetoid cerebral palsy. Furthermore, this approach does not address the problems of retained primitive reflexes and faulty postural reactions exhibited in cerebral palsy (K. Bobath, 1971).

A number of neurodevelopmental models of therapy have been devised in an effort to improve the theoretical foundation and the results of physical therapy. Currently the system most used is that originally developed by the Bobaths in the early 1960's (Bobath, 1967). Their work was begun in the 1940s with the cerebral palsied and adult acquired hemiplegics. The treatment, however, is considered appropriate to a wide variety of other dysfunctions of the central nervous system. This system views the motor disorders of cerebral palsy as the retention of tonic reflex activity and the failure to acquire normal righting reflexes and equilibrium reactions.

The concept of neurodevelopmental therapy is based on two fundamental assumptions about the nature of central nervous system dysfunction: (1) the arrest, or retardation, of normal movement is caused by the interference with normal brain maturation resulting from brain lesion, and (2) the resultant release of abnormal, or immature, postural reflex activity causes the observed abnormal patterns of posture and movement. On the basis of these concepts, treatment techniques have been developed by the Bobaths and are continually being added to and refined (Bobath, 1955).

According to Karel and Berta Bobath (1972) there are four levels of motor function: a spinal level, a brain stem level, a mid brain level, and a cortical level. These authors hypothesize that with the maturation of the central nervous system there is a gradual shift from the spinal level (involuntary level) to the cortical level

(voluntary level). This shift is viewed as being the result of the development of inhibitory function of the higher voluntary center over the reflex patterns of the lower centers. Simply stated, the damaged brain is not able to make this shift, resulting in a situation where reflexes that are adaptive in early infancy are maladaptive if they persist as the individual matures (K. Bobath, 1971; Bobath & Bobath, 1975). The individual with cerebral palsy exhibits motor behavior that is dominated by spinal and brain stem reflexes that were adaptive but now interfere with the development of midbrain and cortically controlled patterns.

Despite widespread enthusiasm among therapists for the neurodevelopmental model of therapy, outcome data remain sparse and unconvincing. This approach to treatment of cerebral palsy appears to be the result of an attempt by clinicians to develop some method of therapy based on logical extrapolation from the data available from the neurophysiologist at that time. Whether that logic will survive the empirical test remains to be seen.

Clinical investigations of sensorimotor methods to date have been somewhat inconclusive. There is a marked paucity of research studies that have employed rigorous scientific methodology in the evaluation of sensorimotor treatment approaches' effect on the performance of cerebral palsied individuals. Scientific documentation is needed to ascertain whether or not sensorimotor approaches can produce long term gains in motor development. Such studies are difficult to carry out because neuromuscular deficits are often highly individualized

making it difficult to obtained a matched control group. Ethical questions are involved when an infant is deprived of the possible benefits of the therapy being tested. To date the limited documentation available tends to show that in spite of the current intensive emphasis on sensorimotor based physical and occupational therapy, the only parameter to show significant improvement on test/retest has been those measures related to social-emotional and intellectual variables (Schilling, Srepp, & Patterson, 1974).

Although sensorimotor therapy retains a central role in the management of cerebral palsy, a multi-center prospective controlled study is needed to prove its efficacy and precisely delineate its salient characteristics and its role in the long term management of cerebral palsy (Marquis, 1979).

In an early study, Paine (1962) followed 177 patients retrospectively and compared 103 who had received the most intensive therapy with 74 who had not. Spastic quadriplegic and severe spastic hemiplegics appeared to receive some functional benefit from therapy, although no decrease in the need for subsequent surgery was noted. Therapy produced no demonstrable improvement in cerebral palsy patients with less severe hemiplegia or extrapyramidal lesions. Pain's work cast doubt on the universal effectiveness of physical therapy in the treatment of cerebral palsy since many of those who had no treatment attained the same level of motor competence as those given treatment.

Wright and Nicholson (1973) similarly questioned whether gains result from sensorimotor based therapy. They assigned 47 children at random to treatment and control groups and reported that gains in function occurring over one year were simply those that would be expected on a maturational basis. Rotzinger and Stoboy (1974) found that subjectively rated evaluation of gait and trampoline skills as well as motor tests led to a positive judgement of the effect of rehabilitation exercises which could not be confirmed by electromyographic investigation. Carlsen (1975) conducted a pilot study comparing the results of facilitation and functional treatment approaches on the overall development of 12 cerebral palsied individuals 1 to 5 years of age. The facilitation or "mat" approach involves activities geared toward sequential normalization of postural tone, gross coordination, and enhancing sensorimotor interaction with the environment as described by Bobath (1967), Rood (1954) and Ayers (1977). This philosophy assumes that integration on a gross sensorimotor level facilitates a maturational improvement in the finer adaptive components of development.

The functional or "table" approach is the more traditional method and is directed toward improving the child's ability in specific developmental tasks, particularly in fine motor adaptive skills and self-care skills. The "flexible" approach combines methods of both the facilitating and functional approaches. Carlsen (1975) used the Denver Developmental Screening Test and the Bayley Motor Scale to evaluate pairs of children with similar developmental ages and

randomly assigned to two treatment programs. Post-tests and subsequent statistical analyses indicated that the group of children receiving the facilitation treatment approach showed significant improvement in computed developmental age then did the group receiving the functional approach.

Scherzer, Mike, and Ilson (1976) evaluated a neurophysiological treatment procedure designed to facilitate the achievement of "motor milestones." Each program was tailored to the individual developmental needs of each subject and was composed of a combination of techniques outlined by Bobath and Bobath (1972), and Rood (1954). Twenty-four individuals below the age of 18 months participated for a minimum of six months in either experimental or control groups in this double blind study. The experimental group received active movement and positioning therapy and the control group was given passive motion exercises confined to major joint areas. Parents were trained to conduct therapy regularly at home. Medical and therapy evaluations indicated definite changes in motor, social, and management areas for those subjected to the experimental procedure and particularly among children with higher intelligence. Obtained improvement was found to be somewhat more correlated with age at entry into the study than with actual treatment time. It is important that the active patient involvement which may be a key to improvement can not typically be employed in the most severe cases.

Clark, Kreutzberg, and Chee (1977) demonstrated that the motor competence of developmentally normal infants can be improved by

controlled vestibular stimulation. In examining the therapeutic consequences of this technique Chee, Kreutzberg, and Clark (1978) used behavioral measures to compare 12 cerebral palsied children exposed to 16 sessions of horizontal and vertical stimulation of the semicircular canal over a four week period and 11 cerebral palsy children in two control groups. Blind methodology was used to assess performance. The subjects receiving vestibular stimulation exhibited greater improvement in reflexes and gross motor skills than the control group, but the study did not consider whether these gains persisted after therapy ceased. A quantitative evaluation of a series of reflexes and of gross motor skills showed a "highly significant" degree of improvement; a qualitative evaluation of each subject corroborated these findings and suggested improvements in fine motor control and social/emotional behavior.

Positive results reported by Ayres (1977) have limited generality because of the children studied, the training, and the task she employed. Learning disabled children, who exhibited involuntary motion of a choreoathetoid quality in the fingers during arm extension, were given sensory integrative therapy. Although they showed improved eye-hand coordination when compared with the control group, the difference was not statistically significant.

Sellick and Over (1980) studied 20 cerebral palsied children ranging in age from 8 to 56 months and covering most diagnostic categories. Subjects were assigned to a treatment group or a control group after having been matched in pairs. Those in the treatment

group received 16 sessions of controlled vestibular stimulation over a four week period while the control children did not. Motor function was measured one week and 18 weeks after treatment and no difference was found between the treatment and control groups. These results are discussed in relation to the Chee, Kreutzberg, and Clark (1978) study which reported that vestibular stimulation is an effective therapy for cerebral palsy children.

Sommerfeld, Frasher, Hensinger, and Beresford (1981) studied the effect of two types of physical therapy service over a five month period in 19 severely mentally impaired and cerebral palsied students aged 3 to 22 yrs. The subjects were paired and assigned to either a direct therapy treatment group or a supervised therapy management group. Ten similar students in a comparison group received no physical therapy. The study showed no significant difference in development of mature developmental reflexes, improvement of gross motor skills, or increase of passive joint motion among similar students placed in the direct, supervised, and comparison groups. The authors concluded that this pilot study should not decide the issue of whether sensorimotor physical therapy is effective for these students. But they point out that it does indicate that objective measures may be used reliably to evaluate three major areas of sensory motor development. More extensive and longer term studies are needed to determine the most beneficial form of physical therapy for this double disability population.

In two recent studies, slight improvement in function was noted in spastic quadriplegic infants who received therapy, when compared to untreated controls (Hagberg, Hagberg, & Olow, 1976; Soboloff, 1981). However, therapy had no effect on range of motion or on the persistence of primitive reflexes. Also, positive changes were better correlated with intelligence level than with therapy.

Parette and Hourcade (1983a) have pointed out that given the motor problems of infants and young children who have cerebral palsy, increasing emphasis is being placed on therapeutic strategies such as neurodevelopmental therapy. Although the underlying rationale for neurodevelopmental therapy is the inhibition of abnormal motor development, this approach may also limit the child's interaction with the environment, a prerequisite for subsequent intellectual development.

Parette and Hourcade (1983b) reviewed 18 studies involving physiotherapeutic programs for cerebral palsy and found that the crucial variable in the prognosis of therapeutic intervention was intellectual capacity in 12 of the 18. Parette, Holder, and Sears (1984) studied a group of 10 infants diagnosed as having cerebral palsy or severe motor delays and administered pre/post test assessments of both standardized and nonstandardized motor-evaluation instruments. Following six months of neurodevelopmental intervention, observed motoric gains were compared with age, IQ, infant hours in treatment, hours of parental participation and degree of involve-

ment. Correlations were found between motoric progress and age, parental participation and degree of involvement.

Hourcade and Parette (1984) reported that their evaluation of 18 studies suggested that research based inquiries into the efficacy of neurodevelopmentally based interventions are presently deficient from both a quantitative and qualitative perspective. According to these authors, the uniform provision of therapeutic intervention to all children diagnosed as having cerebral palsy may in fact be unnecessarily inhibiting the cognitive growth of the child without achieving anticipated motoric gains (Parette & Hourcade, 1983a; Parette & Hourcade, 1984).

Goldkamp (1984) examined 31 dependent cerebral palsied patients and found that neither surgery, therapy, nor time appeared to have a significant effect in reducing physical or social dependence of the patients studied. Although various factors potentially affecting outcome were investigated, only intelligence appeared to influence outcome directly.

Outcome oriented research must continue and be expanded in the area of sensorimotor therapy. However, it must also overcome the common criticisms directed toward studies to date. Specifically, researchers must avoid employing unskilled observers, designing simplistic scoring criteria that lack validity, failing to measure the integrity of independent variables, using unskilled therapists, and planning interventions with insufficient treatment duration.

Augmentative Treatment

Given the physical limitations imposed by the severe tonal abnormalities in some cerebral palsied individuals, the use of adaptive/assistive equipment to enhance mobility, hand function, and communication skills becomes a vital adjunct to therapy. Effective augmentation devices can range from the simple system for facilitating pivot transfers called the "polecat" (Brown, 1979) to complex systems employing the most up-to-date technology. The use of computer-assisted and electronically powered devices has resulted in heretofore unheard of gains in independence. The use of simple assistive devices to enhance all areas of development can be introduced at the age when most individuals would be crawling, reaching, or uttering first sounds, often as young as six or seven months (Fraser, Hensinger, & Phelps, 1986).

Fraser, Hensinger, and Phelps (1986) have provided a detailed description of their experiences in the area of fine motor activities and activities of daily living. Simple devices such as holders and cuffs strapped onto the child's hand into which utensils, crayon, etc. can be inserted can substitute for deficient grasping. Long-handled extensions allow greater range of reaching. Liberal use of velcro closures substitutes for buttons, zippers, and shoelaces. For the older child, technology has led to the development of environmental control units which can be used to operate television, telephone, page turners, feeders, etc., in a specially equipped room

so that even profoundly physically involved individuals can have some measure of functional independence.

Prostheses, braces, and splints are all terms that in some sense are synonymous with orthoses. The application of these devices to the treatment of cerebral palsy will be addressed together in this section. Long (1966) has defined an orthosis as a device added to a person's body to substitute for absent motor power, to restore function, to assist weak muscles, to position temporarily, to immobilize a body part, or to correct/prevent deformities. In the management of cerebral palsy orthotics are primarily used to provide needed support, to control involuntary movement, to prevent or correct deformities or for some combination of these objectives (Bloomberg, 1964; Hastings, 1950; Stamp, 1962). There are two basic classifications of orthoses: static and dynamic (Anderson, 1965). Dynamic devices employ moving parts while static devices do not. Movement in dynamic orthoses may be provided by another body part, battery operated motors, gas operated motors, elastics, strings, or pulleys. Although orthoses can function dynamically to increase the energy efficiency of gait, or statically to control joint alignment, the optimal brace for the individual with cerebral palsy must do both. Orthoses designed to assist weak muscles or substitute for absent motor power may enable functional activity to be performed more easily by the individual.

Unfortunately, neither splinting nor passive stretching appear to be of much value in the management of spastic cerebral palsy. While

both techniques can prevent acute contractures in flail limbs, they are not helpful in preventing contracture due to muscle imbalance. Hoffer, Garrett, Koffman, Guilford, Noble, and Rodon (1974) have concluded that braces are rarely effective in resisting impending deformity, but may buy time for preoperative evaluation and post-operative positioning. Furthermore, the weight of a splint is an additional load to be managed. The primary purpose of bracing is to provide stability and improve function. Braces are used only after careful evaluation of the patient's muscle status, posture, and deformities. It is most important that they be constructed to restrict unwanted motion without impairing desired motion. More rigid bracing of conventional metal uprights and leather pads is still used primarily for passive positioning allowing the severely involved individual to assume the upright stance and gain the physiologic benefits of verticality, i.e., a lessening of the tendency for osteoporosis and the beneficial gravitational effects on respiratory and bowel and bladder function.

Miscellaneous Treatment

Rolfing is a technique which involves the use of manual pressure on the areas of the body in which muscle tendons adhere to each other rather than sliding over one another in the normal way. Specifically, Rolfing involves a 10-hour cycle of deep manual manipulation of the myofascial tissues of the body, concentrating on areas in which asymmetries in vertical alignment have been identified. The overall

goal of Rolfing is to balance the body optimally in the gravitational field.

Perry, Jones, and Thomas (1981) conducted a study in which a series of 10 patients with mild, moderate, or severe cerebral palsy underwent Rolfing Treatment. Mildly impaired patients made gains in velocity, stride length, and cadence. The moderately impaired group made only minor gains in velocity and the severely impaired did not improve by any of the criteria used. Muscle strength and EMG measures were not altered appreciably in any of the patients. While the effects of treatment on range of motion were highly variable, increased muscle tightness in the hip and knee flexors, hip internal rotators, hip adductors, and plantar flexors was noted.

These results indicate that Rolfing can lead to improved performance in mildly affected patients because they posses the neurological capacity to make use of increased tissue mobility and thus avoid contractures. However, the increased muscle tightness which can occur probably outweighs any benefit which moderately or severely impaired patients may derive from treatment.

Sanner and Sundequist (1981) postulated that local inhibition of the pain in the contracted muscles by using acupuncture might break the reflex arc and improve the painful condition. Acupuncture has a documented but unexplained effect in relieving pain. These authors have used a method of subdermal insertion of needles in areas of abnormal muscle tenderness. The needles were kept in place for 20 minutes without any manipulations. Results of four cases indicate

that acupuncture is effective in treating painful muscle spasms. Obtained results were well maintained.

Electromyographic Treatment

Recently, another avenue of treatment involving the use of peripherally applied electrodes, has been applied to the problem of spasticity. Electromyographically (EMG) based biofeedback appears to have been at least partially successful in giving the cerebral palsied individual increased motoric self control and holds promise for future innovation (Olton & Noonberg, 1980). The term biofeedback refers to the use of techniques that enable some bodily function to be detected and transduced in order to return an amplified signal from an external source, which thus reveals the contemporary state of that function; as, for example, one might monitor one's own heartbeat, which is displayed on an oscilloscope in the form of an EKG in a series of spikes.

Much popular interest has been engendered by the application of this technology insofar as it may increase the individual's ability to control various bodily processes and therein permit self-regulation of physiological responses not previously thought to be susceptible to such control. Such control allows the individual a certain amount of "independence" from direct ongoing therapists' intervention. One type of biofeedback with potential for the treatment of cerebral palsy is electromyography (EMG). Through the use of EMG the individual can gain greater control over movement since smaller units

of movement and more subtle topographies can be detected and shaped/responded to than would be the case without EMG. Among those who first showed that even single motor units may be brought under voluntary control in this way were Basmajian (1963), Harrison and Mortenson (1962), and Hefferline (1958).

The general approach of EMG-based treatment is to measure the baseline of activity in all relevant muscles and then begin a sequence of treatment based on the presentation of contingent sensory feedback (DeBacher, 1979). First, the individual is taught to relax excessively tight muscles, often beginning with an overall relaxation procedure. Then the individual is trained to contract weak or flaccid muscles, with special attention being paid to maintaining relaxation in any previously overactive muscles. This stage may require feedback from two sets of muscles and a conjoint criterion of increasing activity in the weak muscles while decreasing activity in the strong muscle. Finally, the individual is taught to produce coordinated movements, a process that may require recording from several muscles and organizing changes in activity over time. Eventually, visual and proprioceptive feedback from the joint movement itself may come to control the movement.

Biofeedback has made at least two major contributions to the treatment of cerebral palsy. When used in conjunction with other types of therapies such as physical therapy, drugs, and/or surgery, it has provided significant improvements that were not obtained with the other treatments alone sometimes after years of the application

of the other approaches alone (Olton & Noonberg, 1980). In conjunction with other therapies, EMG has produced greater and more rapid control over muscles than other treatments alone. EMG can also be relatively inexpensive in professional time since the individual can practice without constant supervision (Johnson & Garton, 1973).

Of all the approaches to the treatment of cerebral palsy, EMG biofeedback is probably the one that has been evaluated using the most systematic clinical research methods (Cataldo, Bird, & Cunningham, 1978). Consequently, more can be reported about this area of treatment than can be said of most others. This area promises to have a major impact on the treatment of cerebral palsy in the near future, especially to the extent that can be integrated with computer technology (Finley, Etherton, Dickman, Karimian, & Simpson, 1981). However, if biofeedback is to become a viable part of effective treatment, problems such as those related to lack of maintained and transferred treatment gains will have to be addressed.

Electrical Stimulation Treatment

Transcutaneous electrical stimulation, TENS, (Wall & Sweet, 1967) has also received a significant amount of attention in the control of various types of pain (Barr, Nielsen, & Soderberg, 1986). TENS has been related to and compared with acupuncture (Fox & Melzack, 1976). The underlying assumption here is that analgesia is related to the production of beta-endorphins and/or enkephalins depending on the type of stimulation employed however, scientific validation of this

hypothesis has been difficult to achieve (Hughes, Lichstein, Whitlock, & Harker, 1984; O'Brien, Rutan, Sanborn, & Omer, 1984).

First introduced by Cooper (1978) chronic cerebellar stimulation is a procedure that involves placing silicon-mesh plates bearing platinum electrodes on the anterior and posterior lobes of the cerebellum. It has been hypothesized that chronic stimulation of the cerebellar cortex augments the inhibitory function of the cerebellum in spasticity. This hypothesis is based on the observations that trauma to the cerebellum often results in hypotonic musculature and that stimulation of the paleocerebellum can inhibit decerebrate rigidity. To say the least, this type of intervention remains controversial with reports of both success (Marquis, 1979) and failure (Bensman & Szegho, 1978).

Larson, Sances, Hemmy, and Millar (1977) conducted a study of cerebellar stimulation with 58 humans and 2 chimps. Results indicated significant if not dramatic clinical improvement. Cooper and Upton (1978) reviewed clinical results from 200 patients and neurophysiological results from 42 patients and concluded that cerebellar stimulation can improve cerebral palsy and reduce the frequency of intractable seizures. Robertson, Meek, and Smith (1980) examined the effects of focal cerebellar stimulation on oral motor control, duration of phonation, articulation, and vocal characteristics in 10 cerebral palsied patients. A pre/post evaluation indicated seven patients increased their duration of vowel phonation, four showed articulation improvement after two months of continuous stimulation,

and two patients exhibited improved oral motor control. A recent report described the results of cerebellar electrode implantation in 50 cerebral palsy patients. The investigators concluded that 34 patients had less spasticity postoperatively (Marquis, 1979). In this study, improved function in a number of the most severely handicapped patients was reported. A variety of complications were associated with the procedure, including induction of seizures, cerebrospinal fluid leaks in two patients and death in one patient. Also, mechanical problems necessitated replacement of electrode wires in 12 patients.

Several studies have questioned both the efficacy and safety of this approach to treatment. Bensman and Szegho (1978) raise the possibility of long term cerebellar damage and cite the marked lack of conclusive evidence about efficacy. These authors recommend following Federal Drug Administration standards on the use of neurological devices emphasizing the necessity of conducting animal research prior to human trials. A letter to the editor of the Journal of the American Medical Association ("Cerebellar Stimulation," 1979) cites a double blind study conducted by Russman. In this study 8 patients received systems that could be set at various levels. Results were equivocal at best and no useful effect was observed in any of the eight individuals. Finally, Wolfe, Ratusnik, and Penn (1981) conducted a long-term (2-year) evaluation of cerebellar stimulation on speech. Severity of dysarthria was not significantly altered as assessed by a panel of listeners. No speech

deterioration was noted and only 2 of 10 patients showed any positive changes in articulation.

Diamond (1986) has described a procedure for establishing peripheral nerve blocks in which controlled intensity electrical stimulation allows isolation of the peripheral nerves, following the injection of carefully graded concentrations of saline solution until the desired effect is achieved. This technique has been very successful for adductors, blocking the obturator nerve at the knee. Recently, this procedure has also been applied to the hamstrings, blocking the sciatic nerve at the level of ischial tuberosity. Overall it appears that the mode of action of these electrical approaches to treatment is not well understood, and that the interrelationship between the sympathetic nervous system and the clinical picture of spastic cerebral palsy is imprecisely defined (Wong & Jette, 1984).

Treatment Efficacy

Unfortunately, regardless of the treatment approach, when spastic cerebral palsy is accompanied by severe mental retardation, the long term effectiveness of interventions appears to be limited by cognitive deficits and poor motivation which limits the active participation of the patient (Parette, Holder, & Sears, 1984; Parette & Hourcade, 1983b)

Quite recently, Malden and Charash (1985) have reported that transcranial electrical stimulation (TCS), similar to the type of

procedure commonly employed in the treatment of headache and other kinds of pain (Gersh & Wolf, 1985), enhanced the effectiveness of physical therapy through the reduction of pain and spasticity and the suppression of abnormal reflexes in children who ranged in age from 2.5 months to 15.4 years and who suffered from spastic cerebral palsy.

In their discussion Malden and Charash (1985) urged that further research involving different designs and patients with other problems should be undertaken. These authors pointed out that any pre-trials with the equipment which may have had an effect on the results of their study should be eliminated. The purpose of the present study was to explore further the utility of TCS as an adjunct to physical therapy in the treatment of spastic individuals with severe mental retardation.

CHAPTER II

METHODS

Subjects

Five males and one female, ranging in age from four to 24 years, participated in the current investigation. Each participant was determined to be severely mentally impaired (SMI) and physically or otherwise health impaired (POHI) according to the definitions of these impairments specified by the Michigan Revised Administrative Rules for Special Education (1987). All subjects were diagnosed by local physicians as suffering from severe spastic cerebral palsy. They were all non-ambulatory and nonverbal. Each subject received regular passive range of motion therapy as part of their individual educational program.

Subject one was a 4-year-old male. He was 43 inches long and weighed 38 pounds. He had exhibited multiple impairments since birth including profound spastic quadriplegia and profound mental impairment. He was also visually impaired, hearing impaired, and exhibited a significant seizure disorder that was only partially controlled by daily administration of phenytoin. In order to maintain the subject nutritionally a gastrostomy tube had been surgically implanted 18 months prior to the present study.

Subject two was a 19-year-old male 51 inches long who weighed 42 pounds. He had exhibited profound multiple impairments since

birth including profound spastic quadriplegia and moderate mental impairment. Although some clinical indications of a decerebrate condition were observed, recent neurological examination concluded otherwise and supported the inclusion of this individual in the present investigation. He exhibited no functional motoric skills and relied on a rudimentary eye blink system to indicate a moderately reliable yes/no. At the time of this investigation subject two was taking no medication.

Subject three was a 24-year-old male 66 inches long who weighed 92 pounds. He had exhibited severe multiple impairments since birth including severe mental retardation and profound spastic quadriplegia. He exhibited no functional motoric skills and was able to vocally respond to yes/no questions reliably. Subject three had a history of diazepam to control spasticity but had been drug free for 24 months at the time of this investigation.

Subject four was a 21-year-old male 51 inches long who weighed 44 pounds. He had exhibited severe multiple impairments since birth including profound mental retardation and profound spastic quadriplegia. He exhibited no functional motoric or communication skills. Baclofen for the control of spasticity had been used in the past but subject four had been drug free for 18 months immediately preceding this study.

Subject five was a 17-year-old female 61 inches long who weighed 55 pounds. She had exhibited severe multiple impairments since suffering encephalitis during the first year of life including

profound mental retardation and profound spastic quadriplegia. She exhibited no functional motoric or communication skills. At the time of this investigation she exhibited a significant seizure disorder controlled by daily administration of phenytoin.

Subject six was a 13-year-old male 52 inches long who weighed 57 pounds. He had exhibited severe multiple impairments including hydrocephaly, profound mental retardation, and profound spastic quadriplegia since birth. He exhibited limited functional motoric skills but exhibited no functional communication. Subject six was receiving no medication at the time of the current investigation.

All methods and procedures were reviewed and approved by a University Institutional Review Board. Informed consent to participate in the study was obtained from the appropriate parent or guardian, and the procedures and equipment employed in the study were examined and deemed safe by a pediatrician and a pediatric neurologist prior to this investigation.

Setting and Materials

Participants attended a day training facility administered by Kalamazoo Valley Intermediate School District that serves 160 severely impaired individuals. The study was conducted at the facility in a small room located adjacent to the students' classroom. This room contained three large mats on which the passive range of movement therapy and measurements were conducted.

This experiment evaluated the effectiveness of a compact (8.6 by 8.9 by 14.6 cm, 20 oz), battery-operated (9-V) electrical stimulation device used to deliver Transcranial Stimulation. The device, originally designed for use in Transcutaneous Nerve Stimulation (TENS) for alleviating chronic peripheral nerve mediated pain, has been recommended by the manufacturer, Pain Suppression Labs, Inc. (Elmwood Park, NJ) for use in Transcranial Stimulation (TCS) as a means of reducing the spasticity associated with cerebral palsy. They also describe the device, as a neurotransmitter modulator [sic]. According to the manufacturer, this type of electrotherapy involves the application of a mild electrical current to stimulate the peripheral nervous system in an attempt to reduce or eliminate pain. They allege that such stimulation applied transcranially can have beneficial effects in the palsied population.

An independent electrical analysis revealed a square waveform (:::::::::___:::::::::___:::::::::___:::::::::___ ;K=250ms) output signal of 12 KHz repeated at 16 Hz. Output values (milliamps) for each metered reading are presented in Table 1.

Table 1

Electrical Output (Milliamps) for Transcranial Stimulator Across Setting Values

Meter Setting				
0	1	2	3	4
0.00	0.30	0.78	1.20	1.70

Voltage changes automatically (5 to 25 volts) to supply the required current as skin resistance varies.

Based on the manufacturer's recommendation, and for the purpose of this investigation, stimulation was delivered to the temples above the ears by means of sponge covered electrodes moistened with warm tap water to ensure proper contact. Electrodes were secured to the head by a soft strap with Velcro fasteners. Other materials used in the study included recording forms, tape measures, digital timers, and goniometers (J.A. Preston Corp., Clifton, NJ).

Design

A double-blind and placebo-controlled reversal design (i.e., P-T-P-T or T-P-T-P; Figure 1) was used to evaluate the efficacy of the transcranial stimulation employed (Hersen & Barlow, 1981). Treatment and placebo conditions were counterbalanced across individuals to control for effects related to the order of treatment presentation and, in an attempt to detect possible cumulative treatment effects, the number of sessions devoted to each condition was varied systematically. In order to maximize the integrity of the blind control each subject was randomly assigned to the treatment or placebo conditions of varying lengths so that the condition for any one subject was unpredictable from the physical therapists' point of view. Over the course of the study 106 placebo sessions and 115 treatment sessions were conducted.

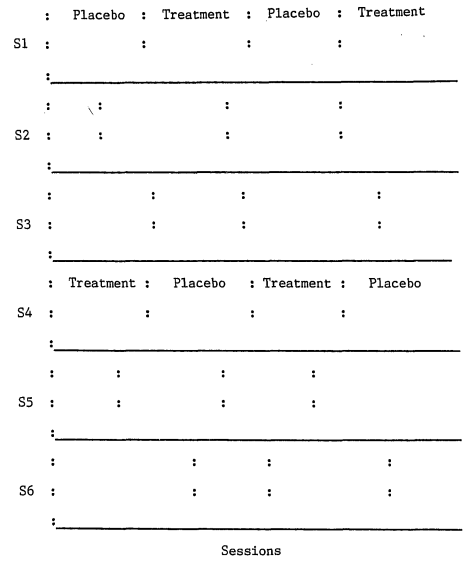

Figure 2. Staggered Counterbalanced Reversal Design

Procedures

Physical therapists familiar with the participants selected three joints for each subject (Table 2) for passive range of motion analysis. These target joints met all of the following criteria:

1. The muscles, although spastic, could be stretched.

2. Passive range was significantly limited, and impaired functional movement.

3. In spite of obvious pathology, joint flexibility was still present (i.e., the joint was not calcified).

4. All subjects exhibited difficulty "relaxing" affected limbs due to pain, anxiety, reflex activity, severe spasticity or some combination of these conditions.

5. Selection criteria were also intended to result in a representative cross section of upper and lower extremity joints typically affected by spastic cerebral palsy.

Table 2

Target Joints and Associated Degrees of Normal Range of Motion.

	Joint 1	Joint 2	Joint 3
S1	R Elbow Extension (0)	R Knee Extension (0-10)	L Shoulder Flexion (0-180)

Table 2--Continued

	Joint 1	Joint 2	Joint 3
S2	R Hip Flexion (0-125)	L Hip Extension (0-30)	L Shoulder Flexion (0-180)
S3	L Hip Flexion (0-125)	L Wrist Extension (0-70)	Between Knees Centimeters (60-70)
S4	R Knee Extension (0-10)	R Elbow Extension (0)	R Shoulder Flexion (0-180)
S5	L Hip Flexion (0-125)	R Elbow Extension (0)	Between Knees Centimeters (60-70)
S6	L Hip Adduction (0-30)	L Knee Extension (0-10)	L Shoulder Flexion (0-180)

Throughout the study, participants remained recumbent during all conditions. Sessions were conducted at the same time daily Monday through Friday, each session lasted 45 to 60 minutes. At no time were the physical therapists or participants given any information concerning the status of the equipment. Dependent measures were scored in the same manner in all phases of the investigation. The sequence of session adhered to the following protocol:

1. Measurement
2. Equipment Output Check
3. Application of equipment - 10 min
4. Equipment Output Check
5. 15 min rest for effect build up
6. Standard passive range of motion therapy without any relaxation activity.
7. Measurement
8. Placebo vs. Treatment Rating.

Prior to each session, and again immediately following completion of TCS application, measurement of passive range of motion (degrees of passive range) was completed by two licensed physical therapists familiar with the subjects. To accomplish this reliably, a certified physical therapy assistant held the participant in position as needed to measure each of 3 target joints, while one of the physical therapists employed a goniometer to assess the passive range of motion.

Two additional measures were recorded by the physical therapists. These were "ease of passive movement" and "apparent pain during movement." Ease of passive movement was defined as the amount of resistance encountered when joints were moved by the therapist. Because subjects were unable to report any pain they experienced during movement, pain was inferred from as unusual sound such as crying, movement, or facial expression such as grimacing observed during joint movement by the therapist. Therapists reported their rating of whether ease of "ease of passive movement" and "pain during movement" for each individual participant was within the usual (i.e., pre-investigation) range for the individual being assessed (in which case a "0" was recorded), improved for the individual (in which case a "+" was recorded), or worsened for that individual (in which case a "-") was recorded.

Following the completion of the pre-session measurement the session controller connected each subject to a masked TCS unit for the prescribed ten minute period at the manufacturer's recommended setting of one. After completion of the "treatment" period each subject was disconnected by the controller and allowed to rest for 15 minutes to permit build up of maximum effect. This rest period was employed based on the manufacturer's recommendations.

In all phases of the study, each subject was exposed to 7 to 15 minutes of passive range of motion therapy. Each subject's physical therapy involved passive movement of various joints, as dictated by each subject's therapeutic needs. This passive range of motion

constituted a continuation of the physical therapy procedures conducted regularly prior to the study in support of the individual's individualized education program (IEP). Specific regimes were established and followed for each subject in order to ensure consistent implementation. Upon completion of passive range of motion therapy the second measurement was taken using the procedures described above. Finally, in order to evaluate the integrity of the blind, therapists were also asked to answer the question "Was the TCS activated today?"

Placebo

During the placebo condition electrodes were moistened and attached to the subjects' head above the ears at the temples by the experimenter who served as a session controller; the electrodes were kept in place for 10 minutes during this phase, but equipment was not activated.

TCS Treatment

Active treatment was identical to the placebo phase except that the device was activated for 10 minutes. Throughout the treatment phases of this investigation, the intensity of stimulation was set at "1" (on a scale of 1 to 4). The intensity and duration of stimulation selected for evaluation were based on the manufacturer's recommendations and previous findings by Malden and Charash (1985).

Dependent Variable Reliability

For one-third of all sessions and at least once in every condition for each participant, interobserver agreement (i.e., reliability) data were collected for goniometric measurements, for both rating scale measurements, and for therapist's evaluations of whether physical therapy was improved by the day's treatment. Rating scale agreement was calculated by having a second therapist independently rate each participant's ease of passive movement and apparent pain, using the +, 0, and - system described earlier. Agreement concerning whether physical therapy was improved by each day's treatment was determined by having a second therapist, who participated in the physical therapy session, independently respond to the question listed above.

Goniometric measurements were considered in agreement if a second therapist's independent rating of maximum mobility was within three degrees of that of the primary observer. Interobserver agreement was 100% when this criterion was applied. All observers were physical therapists familiar with all subjects. Interobserver agreement was 91% (74 agreements, 7 disagreements) for ease of passive movement, and 80% (65 agreements, 16 disagreements) for pain during movement.

Dependent Variable Validity

All dependent measures were selected by two physical therapists experienced with the subjects for a minimum of five years, both agreed that they accurately reflected clinical problems exhibited by

the subjects, that improvement in these measures would be of real benefit to participants, and, that these measures should be expected to change in the desired direction if TCS was of any clinical value.

Independent Variable Integrity

To ensure that placebo and active stimulation conditions actually were arranged as intended, electrode placement and the duration and intensity of stimulation were set by a session controller and monitored by an independent observer during randomly selected sessions. Monitoring involved pre and post application equipment checks to ensure adequate electrical output, examination of electrode preparation and placement, and observation of the meter output reading on the TCS equipment. Such checks occurred during one third of all sessions and at least three times in each condition. The application of treatment and placebo conditions was judged to be correct for all sessions.

Moreover, an independent observer monitored the procedures used to manipulate joints during physical therapy and to measure passive range of motion reported at least once each experimental condition, and reported that these procedures were applied consistently (i.e., in accordance with written protocols), a point supported by the interobserver agreement data for passive range of motion reported earlier.

Integrity of Blind

During the course of the current investigation 115 treatment sessions and 106 placebo sessions were conducted. Of the 221 sessions conducted 144 (65%) were rated by the participating physical therapist as being placebo and 77 (35%) as being treatment. This evaluation of treatment/placebo was accurate 114 (52%) times, indicating that the participating physical therapists were not aware of the actual condition in effect.

CHAPTER III

RESULTS

Because of the severity of the spasticity exhibited by the participants, even the smallest positive differences between placebo and treatment conditions should be considered clinically important. Positive results might include substantially more "+" ratings in the post treatment measures for both "passive movement" and "apparent pain." Also, pre-application degrees of passive range of motion would be expected to appear improved when compared to post- application degrees of passive range of motion during treatment conditions. Such improvement in passive range of motion would result in a significant separation in graphically displayed pre and post data in the treatment conditions. Such a separation should not be apparent in the placebo conditions.

Data collected in the present study provide no indication that exposing participants to transcranial electrical stimulation improved passive range of motion (Tables 3 through 8 and Figures 2 through 7) or therapists' ratings of ease of passive movement and pain during movement experienced by the subjects (Tables 9 and 10).

Mean and range of passive range of motion data for each subject's three target joints are presented in Tables 3 through 8. These summary data indicate no difference between placebo and treatment conditions. Degrees of passive range of motion data for the three target joints from each subject are presented in Figures 2 through 7.

An examination of these data indicates all but one of the subjects tended to show some improvement in passive range of motion from pre-application to post-application samples across the length of the study. However, these gains appeared unrelated to differential exposure to treatment or placebo conditions.

Table 3

Subject 1
Range of Motion Measurements (Mean & Range)
J1=R Elbow Ext J2=R Knee Ext J3=L Shoulder Flex

	Placebo	Treatment	Placebo	Treatment
J1: Pre:	20.29	18.88	9.33	11.0
	(08-30)	(12-26)	(05-25)	(10-16)
Post:	17.14	12.88	9.4	9.88
	(05-24)	(07-19)	(05-31)	(09-10)
J2: Pre:	10.43	10.75	7.27	6.88
	(5-14)	(7-14)	(05-17)	(05-10)
Post:	10.14	7.88	6.33	5.13
	(8-12)	(5-12)	(01-13)	(03-07)
J3: Pre:	162.14	161.88	163.67	163.13
	(155-168)	(156-167)	(157-170)	(159-167)
Post:	167.88	163.50	165.73	166.88
	(157-175)	(152-170)	(155-170)	(163-170)

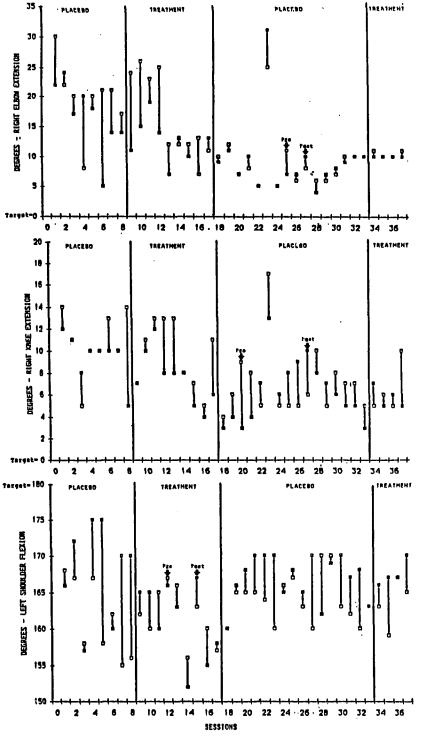

Figure 2. Range of Motion for Target Joints Across Experimental Conditions - Subject 1.

Table 4

Subject 2
Range of Motion Measurements (Mean & Range)
J1=R Hip Flex J2=L Hip Ext J3=L Shoulder Flex

		Placebo	Treatment	Placebo	Treatment
J1:	Pre:	94.80	100.00	98.23	97.47
		(85-98)	(95-115)	(88-107)	(93-104)
	Post:	98.40	97.85	97.85	98.94
		(95-101)	(90-106)	(90-106)	(97-105)
J2:	Pre:	21.80	26.83	22.00	21.71
		(19-30)	(20-30)	(16-28)	(16-31)
	Post:	23.60	27.83	21.00	21.12
		(20-30)	(20-35)	(18-25)	(16-30)
J3:	Pre:	155.80	155.17	152.23	155.06
		(154-157)	(145-164)	(130-165)	(145-162)
	Post:	157.60	152.33	154.38	155.17
		(150-165)	(147-160)	(140-162)	(151-160)

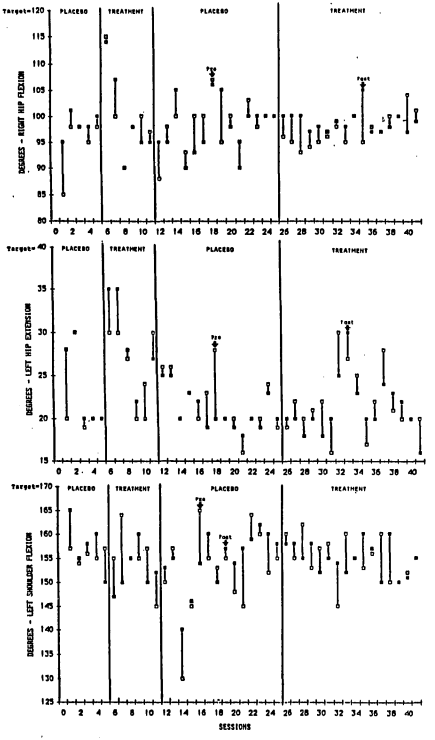

Figure 3. Range of Motion for Target Joints Across Experimental Conditions - Subject 2.

Table 5

Subject 3
Range of Motion Measurements (Mean & Range)
J1=L Hip Flex J2=L Wrist Ext J3=CM Knees

	Placebo	Treatment	Placebo	Treatment
J1: Pre:	92.00	97.31	95.80	100.29
	(85-97)	(93-100)	(90-105)	(97-105)
Post:	94.18	98.46	96.40	100.43
	(85-109)	(94-105)	(90-103)	(94-106)
J2: Pre:	11.64	11.39	9.00	12.43
	(01-20)	(08-15)	(05-11)	(10-15)
Post:	13.27	10.20	11.69	13.14
	(01-20)	(05-15)	(07-15)	(08-15)
J3: Pre:	34.91	34.23	34.80	34.43
	(33-38)	(33-36)	(33-37)	(33-37)
Post:	34.91	35.08	35.20	34.71
	(33-37)	(34-38)	(34-37)	(32-37)

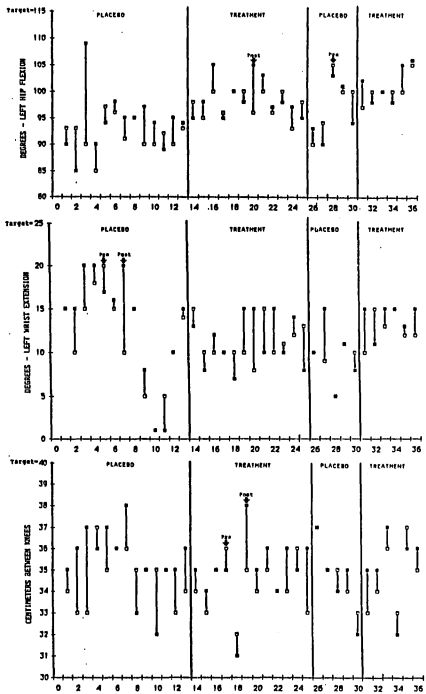

Figure 4. Range of Motion for Target Joints Across Experimental Conditions - Subject 3.

Table 6

Subject 4
Range of Motion Measurements (Mean & Range)
J1=R Knee Ext J2=R Elbow Ext J3=R Shoulder Flex

	Treatment	Placebo	Treatment	Placebo
J1: Pre:	30.50	28.78	27.89	29.33
	(27-35)	(25-33)	(25-30)	(27-33)
Post:	30.75	26.67	27.67	27.17
	(27-35)	(25-29)	(25-30)	(23-30)
J2: Pre:	24.13	24.44	23.44	24.83
	(20-28)	(20-33)	(20-30)	(18-30)
Post:	17.75	21.11	25.33	25.33
	(13-23)	(16-25)	(15-30)	(15-28)
J3: Pre:	145.75	146.00	149.33	148.66
	(140-150)	(141-150)	(143-153)	(145-152)
Post:	149.25	149.44	152.67	152.33
	(147-154)	(144-154)	(147-158)	(150-155)

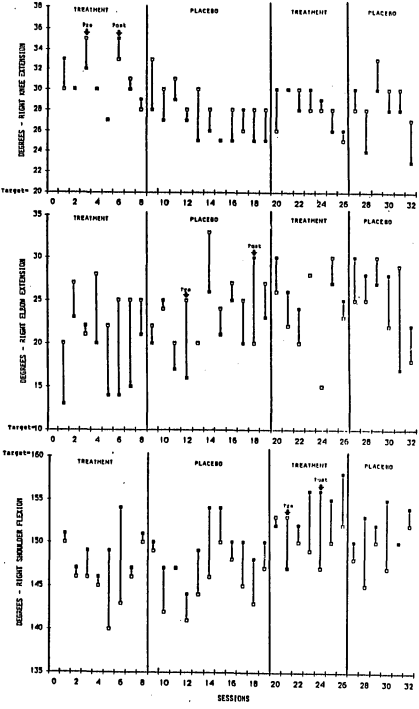

Figure 5. Range of Motion for Target Joints Across Experimental Conditions - Subject 4.

Table 7

Subject 5
Range of Motion Measurements (Mean & Range)
J1=L Hip Flex J2=R Elbow Ext J3=CM Knees

	Treatment	Placebo	Treatment	Placebo
J1: Pre:	110.75	117.86	120.20	120.17
	(103-129)	(112-124)	(116-124)	(116-126)
Post:	116.17	120.57	122.80	121.50
	(105-128)	(110-125)	(117-126)	(118-125)
J2: Pre:	29.00	27.07	27.90	27.00
	(23-35)	(26-31)	(20-30)	(15-35)
Post:	27.25	29.50	25.20	25.67
	(20-30)	(26-31)	(20-28)	(20-30)
J3: Pre:	36.67	37.43	39.80	38.83
	(34-40)	(33-41)	(36-43)	(36-42)
Post:	33.00	37.43	39.80	38.83
	(35-41)	(36-42)	(40-45)	(41-42)

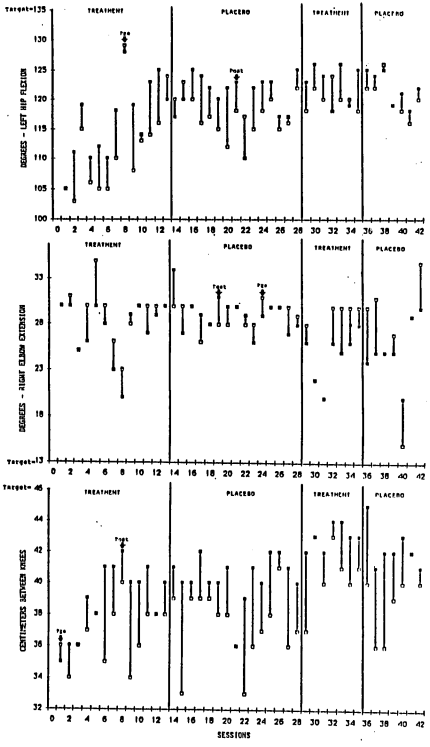

Figure 6. Range of Motion for Target Joints Across Experimental Conditions - Subject 5.

Table 8

Subject 6
Range of Motion Measurements (Mean & Range)
J1=L Hip Add J2=L Knee Ext J3=L Shoulder Flex

	Treatment	Placebo	Treatment	Placebo
J1: Pre:	21.78	21.13	18.13	21.00
	(19-26)	(18-23)	(15-23)	(20-25)
Post:	22.22	21.63	19.00	21.00
	(17-26)	(17-25)	(16-21)	(18-25)
J2: Pre:	5.00	5.38	5.88	10.43
	(02-08)	(04-07)	(04-09)	(05-20)
Post:	4.11	5.00	5.88	9.57
	(02-05)	(04-07)	(03-10)	(05-20)
J3: Pre:	155.22	163.13	160.50	158.43
	(140-165)	(155-168)	(155-165)	(146-170)
Post:	157.67	160.50	160.75	159.57
	(148-165)	(153-165)	(157-164)	(147-168)

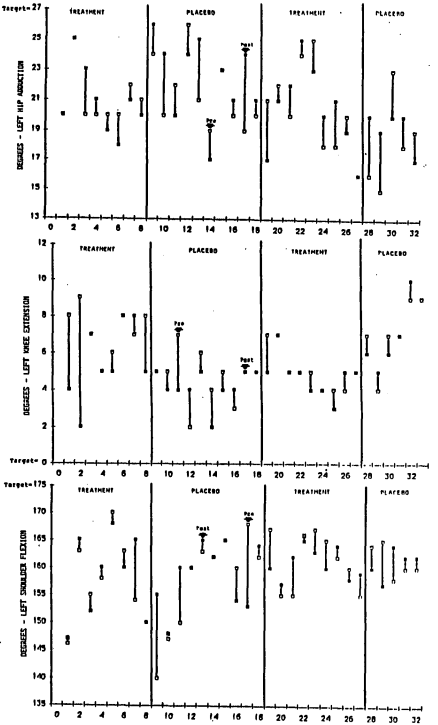

Figure 7. Range of Motion for Target Joints Across Experimental Conditions - Subject 6.

Total "ease of passive movement" and "apparent pain" ratings scored +, 0, or - across pre/post placebo and treatment conditions are presented for each subject in Tables 9 and 10. The intervention (TCS) appeared to have no impact on these measures. During pre-application, there was little or no difference between placebo (69) and treatment (64) for "ease of passive movement" or for placebo (66) and treatment (85) for "apparent pain," but a comparison of post-application measures did not show the expected increase in the number of "+" ratings, 154 to 166 for "ease of passive movement" and 169 to 166 for "apparent pain."

A comparison of pre-application and post-application data also does not support the efficacy of TCS. For "ease of passive movement," examination of "+" data reveals an improvement 64 to 166 during the treatment condition and 69 to 154 from pre-application to post-application measures in the placebo condition. The same comparison applied to "apparent pain" data yields similar results. With respect to this measure, an increase of 85 to 166 in "+" ratings was obtained during the treatment condition, however, a 66 to 169 increase was also obtained during the placebo condition.

Table 9

Ease of Passive Movement

		Placebo			Treatment		
		+	0	-	+	0	-
S1:	Pre:	35	29	2	27	21	1
	Post:	61	5	0	42	6	1
S2:	Pre:	12	39	3	13	52	4
	Post:	29	23	2	35	28	6
S3:	Pre:	2	40	7	2	57	0
	Post:	2	44	3	3	57	0
S4:	Pre:	8	36	1	6	39	6
	Post:	23	20	2	21	28	2
S5:	Pre:	6	45	9	9	45	11
	Post:	25	32	3	42	23	0
S6:	Pre:	6	33	6	7	41	3
	Post:	14	27	4	23	28	0
Total	Pre:	69	222	28	64	255	25
	Post:	154	151	14	166	170	9

Table 10

Apparent Pain Ratings

		Placebo			Treatment		
		+	0	-	+	0	-
S1:	Pre:	32	34	3	31	16	2
	Post:	60	7	2	40	8	1
S2:	Pre:	6	43	5	9	55	5
	Post:	26	22	6	31	36	2
S3:	Pre:	2	41	5	8	50	2
	Post:	8	36	4	11	47	2
S4:	Pre:	6	38	0	12	36	3
	Post:	22	21	1	20	28	3
S5:	Pre:	16	33	11	17	39	10
	Post:	38	18	4	43	19	4
S6:	Pre:	4	36	5	8	35	5
	Post:	15	26	4	21	26	1
Total	Pre:	66	225	29	85	231	40
	Post:	169	130	21	166	164	13

A final possible indicator of improved physical therapy resulting from TCS would be a decrease in the duration of each physical therapy sessions from placebo to treatment conditions. This based on the assumption that a relaxed and pain free subject would be able to be taken through the prescribed regimen quickly and without interruption. Therapy duration data were collected for each session and are presented in Table 11. A comparison of the average duration of sessions during placebo (9.40 min) with the average duration of sessions during treatment (9.32 min) indicates no effect.

Table 11

Physical Therapy Session Duration (Mean/Range)
15 Min Maximum

	Placebo	Treatment	Total
S1	8.18	9.00	8.59
	(6-11)	(7-11)	(6-11)
S2	7.83	7.39	7.61
	(7-10)	(4- 9)	(4-10)
S3	11.38	10.40	10.89
	(9-14)	(9-12)	(9-14)
S4	10.20	10.00	10.10
	(8-12)	(8-15)	(8-15)

Table 11--Continued

	Placebo	Treatment	Total
S5	11.25	11.41	11.33
	(9-14)	(8-15)	(8-15)
S6	7.53	7.71	7.62
	(5-13)	(6-10)	(5-13)
T	9.40	9.32	9.36
	(5-14)	(4-15)	(4-15)

CHAPTER IV

DISCUSSION

According to Greenwald (1975) negative results may be of interest when "an established theory clearly predicts that a difference or correlation (i.e., a treatment effect) should be found," or "when an investigator discovers a methodological weakness in a published report of positive results, correcting the weaknesses, finds that the significance vanish" (Greenwald, 1975, p. 21). A single investigation (Malden & Charash, 1985) has reported that transcranial electrical stimulation (TCS) combined with physical therapy is superior to physical therapy alone in the treatment of children with spastic cerebral palsy. Apparently on the basis of that report, the manufacturer of the device evaluated in the present study suggests that the unit may be beneficially employed with spastic individuals. Moreover, physical therapists have begun to express interest in employing such units in treating spasticity (e.g., Carris, 1986).

Although the study reported by Malden and Charash (1985) employed a reasonable design, several shortcomings are apparent. One general weakness concerns the manner in which treatment effects were assessed. Malden and Charash (1985) employed a set of four motor rating scales to assess outcomes. The reliability and validity of these scales were not demonstrated or addressed, and the professional qualifications of the individual(s) administering the scales were not reported. Also, the authors' data analysis is at best difficult to

interpret. Though they indicate that "the results of this study, depicted in Figure 7, are highly significant ($P<.001$)..." (p. 37), the specific data upon which this conclusion is based and the inferential statistic used to analyze these data are not apparent. In addition, the clinical significance of the reported changes in performance was not established.

A second general weakness involves the integrity of independent variable application. No evidence is given that stimulation actually was applied as intended (parents apparently administered stimulation in the home), and no details are provided as to the form and frequency of physical therapy across the course of the study. A third general weakness is the failure of the authors to provide a research based rationale as to why the treatment might be expected to work, i.e., to posit a mechanism of action, or to cite related research evaluating similar interventions. The latter oversight is noteworthy since electrical stimulation via implanted electrodes has been advocated by lay persons and scientists alike as beneficial in treating cerebral palsy, but appear to be of no more than limited worth (e.g., Bensman & Szegho, 1978).

Melzack, Vetere, and Finch (1983) have pointed out that there have been few double-blind studies to assess the effectiveness of these procedures. Although Malden and Charash (1985) reported that their investigation employed a double bind methodology, it is not clear that the subjects in the non-treatment group were actually "blind" since they received 70 seconds of active stimulation and

since active stimulation could easily be discriminated by the individual. In the current study the severe cognitive deficits of the patients made them blind to the treatment condition for all intents and purposes. In addition, the physical therapists who were evaluating the impact of the procedures were also unaware to the condition that was in effect at any one time.

As mentioned earlier, the mechanism through which TCS might produce beneficial effects is not apparent. Some have posited. however, that it might involve and increased production of serotonin in the brain (Malden & Charash, 1985). These authors and the manufacturer have recommended the use of urinalysis to detect and changes in the serotonin level that might result from TCS application. It should be noted that any tendency to use urinalysis to assay such chemical changes have been brought into question by a recent study conducted by Aizenstein and Scavone (1984). They reported the results of a study undertaken to determine if changes in the serotonergic central nervous system can be reflected by urinary serotonin metabolite 5-hydrooxyindoleacetic acid (5-HIAA) levels. These authors compared cerebral, spinal, and urinary levels of 5-HIAA in rats whose cerebral 5-HIAA concentration had been depleted by nucleus raphe dorsalis and medialis lesion or increased by L-tryptophan loading. No differences were found in the urinary excretion of 5-HIAA leading to the conclusion that urinary assay cannot be used to detect changes in the serotonergic CNS.

In view of its shortcomings, an attempt to replicate the experiment by Malden and Charash (1985) appeared warranted. The results of the current investigation indicate that when transcranial stimulation is applied to severely mentally impaired subjects with severe spastic cerebral palsy at the level of stimulation recommended by the manufacturer, no enhancement of the effectiveness of physical therapy was observed. These results are not supportive of the Malden and Charash (1985) results. Although any of several factors, including difference in dependent and independent measures, and participant characteristics (e.g., drug history, severity of handicap), may have been responsible for the failure to replicate the findings of Malden and Charash (1985), it is clear that transcranial electrical stimulation at the level of stimulation employed in this study is of no obvious benefit in the treatment of spastic individuals. Until the specific factors that determine whether or not such treatment is likely to be effective are clarified, it appears that statements recommending the use of transcranial stimulation in treating spasticity with severe and profound mental impairment should be made with extreme caution.

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