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A Review of Bell's Palsy

Jodi Spicer

University of North Dakota

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A REVIEW OF BELL'S PALSY

by

Jodi Marie Spicer  
Bachelor of Science in Physical Therapy  
University of North Dakota, 1995

An Independent Study  
Submitted to the Graduate Faculty of the  
Department of Physical Therapy  
School of Medicine  
University of North Dakota  
in partial fulfillment of the requirements  
for the degree of  
Master of Physical Therapy

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May  
1996
This Independent Study, submitted by Jodi M. Spicer in partial fulfillment of the requirements for the Degree of Master of Physical Therapy from the University of North Dakota, has been read by the Faculty Preceptor, Advisor, and Chairperson of Physical Therapy under whom the work has been done and is hereby approved.

(Faculty Preceptor)

(Graduate School Advisor)

(Chairperson, Physical Therapy)
PERMISSION

Title A Review of Bell's Palsy

Department Physical Therapy

Degree Masters of Physical Therapy

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# TABLE OF CONTENTS

List of Figures ................................................................................................. v
List of Tables .................................................................................................. vi
Acknowledgements ........................................................................................ vii
Abstract ......................................................................................................... viii
Introduction to Bell's Palsy ........................................................................... 1
Steroid treatment in Bell's Palsy ................................................................. 6
Electrotherapy treatment in Bell's Palsy ....................................................... 10
Surgery for Bell's Palsy .................................................................................. 17
Conclusions ................................................................................................... 23
References ..................................................................................................... 26
## LIST OF FIGURES

<table>
<thead>
<tr>
<th>Figure</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Motor points of face</td>
<td>15</td>
</tr>
<tr>
<td>2. Course of facial nerve through facial canal</td>
<td>18</td>
</tr>
<tr>
<td>Table</td>
<td>Page</td>
</tr>
<tr>
<td>----------------------------------------------------------------------</td>
<td>------</td>
</tr>
<tr>
<td>1. House-Brackmann Facial Nerve Grading System</td>
<td>3</td>
</tr>
</tbody>
</table>
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ABSTRACT

Bell's Palsy is an acute unilateral weakness or paralysis of the facial muscles resulting from peripheral facial nerve dysfunction. Bell's Palsy is the most common cause of unilateral facial weakness. Since idiopathic facial paralysis was first diagnosed, several treatment options have been tried in an effort to influence early and full recovery. The role of physical therapy in the treatment of Bell's Palsy is to exercise the muscle in an attempt to keep the denervated muscle healthy while the injured axons regenerate and reinnervate the muscle. The natural course of Bell's Palsy is a spontaneous return of function in 71% of all patients that present with the condition.

The most effective treatment and intervention for Bell's Palsy remains controversial because of the spontaneous recovery of Bell's Palsy and the lack of significant scientific research on treatment parameters. The purpose of this paper is to review the literature on the effects of three treatments for Bell's Palsy. The three treatments that will be reviewed are: steroid intervention, electrical stimulation, and surgical decompression. From this review, physical therapists will be able to gain a better understanding of the role of physical therapy in the treatment of Bell's Palsy.
CHAPTER I

INTRODUCTION TO BELL'S PALSY

Bell's Palsy (BP) is a condition of unilateral facial weakness or paralysis stemming from acute peripheral facial nerve dysfunction. By definition, the peripheral facial nerve must be involved, but other cranial nerve palsies can be present, especially those of cranial nerves V, VIII, IX, and X.\(^1,2\) Although the etiology of Bell's Palsy remains unknown, several theories exist. One such theory is that it is caused by edema and entrapment of the facial nerve secondary to a viral infection.\(^3,4\) It has also been postulated that an immune dysfunction may play a role in the origin of BP.\(^3\) It has also been argued that BP is a form of polyneuropathy.\(^3\)

Bell's Palsy is the most common cause of unilateral facial weakness. The annual occurrence of BP is about 20 per 100,000.\(^1\) In Bell's Palsy, the right and left sides of the face are involved equally. The rate of recurring episodes of BP is 10%. The recurring episode can occur on the same side or on the opposite side as compared to the previous palsy. The ratio of affected men to women is about equal, but between the ages of ten and nineteen, BP is two times as common in women, and then, after the age of forty, men are 1.5 times more likely to acquire BP.\(^1\) This finding suggests a relationship to menarche and
Bell's Palsy is characterized by signs and symptoms of facial paralysis that include facial numbness, epiphora (abnormal overflow of tears), retroauricular pain (pain behind the ear), dysgeusia (impaired taste), hyperacusis (increased sensitivity to sound), and decreased tearing.\(^1\) Other physical findings include a unilateral shift of the palate due to the motor paralysis of branches of cranial nerve X (vagus nerve).\(^1\) A flat and expressionless face is present, and in extreme cases, a wide palpebral fissure exists. The degree and type of symptoms experienced by patients depends on the extent of nerve damage. The House-Brackmann\(^2,6\) facial nerve grading system is currently used most often by clinicians for grading the paralysis. (see Table 1) Grade I is normal function; grade VI is paralysis; grades II through V are intermediate.\(^2,6\)

The minimal evaluation for Bell's Palsy should include a thorough head and neck examination.\(^4\) A detailed history, including date of onset, duration of primary symptoms, precipitating factors, and other symptoms is required of any patient experiencing facial paralysis.\(^3\) Assessment of the cranial nerves and inspection of the skin and oral cavity mucosa, ear exam, and palpation of the parotid gland are necessary. It is critical that the eye on the involved side be evaluated. An overall assessment of the facial nerve function can be done by observing whether the patient can close the eyes (protecting the cornea), voluntarily raise the brows, smile, pucker the lips, tense the muscles of the neck, puff out the checks with pursed lips, and frown.\(^4\)
Table 1. House-Brackmann facial nerve classification system.²,⁶

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Normal</td>
<td>Normal facial function</td>
</tr>
<tr>
<td>II</td>
<td>Mild dysfunction</td>
<td>Gross: slight weakness noticeable on close inspection; may have very slight synkinesis At rest: normal symmetry and tone Motion: Forehead: moderate to good function Eye: complete closure with minimum effort Mouth: slight asymmetry</td>
</tr>
<tr>
<td>III</td>
<td>Moderate dysfunction</td>
<td>Gross: obvious but not disfiguring difference between two sides; noticeable but not severe synkinesis, contracture, and/or hemifacial spasm At rest: normal symmetry and tone Motion: Forehead: slight to moderate movement Eye: complete closure with effort Mouth: slightly weak with maximum effort</td>
</tr>
<tr>
<td>IV</td>
<td>Moderately severe dysfunction</td>
<td>Gross: obvious weakness and/or dysfiguring asymmetry At rest: normal symmetry and tone Motion: Forehead: none Eye: incomplete closure Mouth: asymmetric with maximum effort</td>
</tr>
<tr>
<td>V</td>
<td>Severe dysfunction</td>
<td>Gross: only barely perceptible motion At rest: asymmetry Motion: Forehead: none Eye: incomplete closure Mouth: slight movement</td>
</tr>
<tr>
<td>VI</td>
<td>Total paralysis</td>
<td>No movement</td>
</tr>
</tbody>
</table>
The physical examination rules out other lesions that can imitate Bell's Palsy: infection, tumor, trauma, and stroke. The sparing of forehead movement function is a cardinal sign for cerebrovascular accident. If a parotid mass is evident, a malignant neoplasm is strongly suspected. In cases that present with middle ear infections, bacteria may be the cause of the damaged nerve. If vesicles around the external auditory meatus, on the auricle, or within the external canal are present, herpes zoster oticus (Ramsay Hunt Syndrome) is suspected to be the diagnosis.

Once the diagnosis of Bell's Palsy is made, other testing is individualized on the basis of progression and severity of the condition. The single most helpful sign in determining prognosis is the appearance of palsy rather than paralysis. The severity of the palsy does not matter. As long as the palsy does not progress to paralysis, the patient can be reassured of an excellent, eventual recovery. If the face is paralyzed, the second best sign is the beginning of remission within three weeks. These patients usually can expect a favorable recovery. If the above signs are not present, commonly used tests to determine a patient's prognosis are minimal nerve excitability, maximal nerve stimulation, and electroneurography; however, electroneurography is the only test that is presently considered to be sensitive enough to determine the need for surgery. When the compound action potential (CAP) on the involved side is at least 90% reduced compared with the normal side, poor prognosis is indicated.
It was not until the publication of a study by Peitersen\(^7\) in 1982 that the natural history of BP was considered valid. Peitersen studied 1011 untreated patients over a period of 15 years. The patients were routinely checked at short intervals until remission occurred. These checks were discontinued only when normal function was returned or after a period of one year. Eighty-five percent of the patients showed the first signs of remission within three weeks after the onset. Fifteen percent of the patients did not show first remission signs until three to six months after the onset of BP. Peitersen concluded that 71\% of the patients recovered normal function of the face, 13\% had insignificant sequelae, and 16\% had permanently decreased function with contracture and associated movements. The natural course of Bell's Palsy must be considered before the treatment and the therapy program for patients with BP can be planned.

The most effective treatment and intervention for Bell's Palsy remains controversial. Several treatment options have been tried in an effort to influence early and full recovery. In the following chapters, three treatments will be reviewed: steroid therapy, electrotherapy, and surgical decompression. From this review, physical therapists will be able to gain a better understanding of the role of physical therapy in the treatment of Bell's Palsy.
CHAPTER II

STEROID TREATMENT IN BELL'S PALSY

When assuming the cause of paralysis in Bell's Palsy is caused by nerve edema in the facial canal, some clinicians are led to treat patients with anti-inflammatory drugs. Two small glands, one on either side of the aorta, make up what is called the adrenals. Each adrenal consists of two parts, an outer portion (cortex), and an inner portion (medulla). This organ has many functions within the body. One such function of the adrenal gland is that it acts as a storage place for hormones that are used in times of emergency, such as infection. More than 20 different hormones have been detected to originate from the adrenal cortex. Two such hormones are cortisone and hydrocortisone. Cortisone has been synthesized and used by physicians for over 40 years. In man, cortisone has an inhibitory effect on lymphoid tissue, and therefore, suppresses the inflammatory response to almost all forms of injury. This anti-inflammatory reaction may relieve pain and discomfort that is produced with inflammation. An important point to remember is that cortisone may relieve discomforting symptoms, but it does not influence the cause. Another important aspect to consider is that inflammation helps the body to mobilize its natural defenses against infection. Therefore, when patients are taking high doses of steroids,
inflammation against bacterial invaders is also inhibited. Because of these two different aspects of steroids, the effects of the use of steroids must be respected and understood.

Despite the great number of papers written that evaluate the effectiveness of steroid treatment in BP, only a few were found to be properly controlled, randomized, prospective studies. Until a definitive, statistically valid study concerning the effect of steroids in the recovery of idiopathic facial paralysis is performed, steroid treatment of BP remains controversial.

In 1972, Adour designed a double-blind randomized controlled study in which a drug or a placebo was given to the patients seen. It became clear to the physicians and to the patients that the prednisone was altering the course of the disease. The patients demanded treatment rather than continue with the clinical trial. The double-blind aspect of the study was abandoned, and from 1970 on, all patients (194) who had no contraindication to the use of steroids received prednisone. The results of the treated group were then compared to a retrospective control group (110). It was concluded in the study that prednisone did not effect partial denervation and the development of synkinesis (involuntary movement of one part of the body that occurs simultaneously with reflex or voluntary movement of another body part) and contracture, but Adour concluded that prednisone enhanced full recovery and lessened denervation.

In 1978, Wolf conducted a controlled, randomized study of 239 patients. Sixty-nine percent of the patients were seen within three days, 91% within four
days, and all patients within five days of onset. The patients in the treatment group were given 60 mg of prednisone a day for ten days. After these ten days, the dose of prednisone was tapered off in seven days. The control group received no placebo. Wolf found no difference between control, untreated, groups and groups treated with prednisone, except in autonomic synkinesis (crocodile tearing). He evaluated all patients himself and concluded that prednisone therapy is beneficial in avoiding the occurrence of autonomic synkinesis.

Stennert\textsuperscript{10} reported in an uncontrolled perspective study in 1982 that of 110 patients, 96\% had complete recovery. His protocol for the study was intravenous dexatran followed by oral cortisone and pentoxifylline (decreases platelet aggregation). The results of this study provided evidence that steroids may help in producing a complete recovery.

In 1988, Prescott\textsuperscript{11} conducted a retrospective study of 879 patients with Bell's Palsy who had been seen over a ten year period. Prescott reported that all patients with no evidence of degeneration recovered, whether they had a partial or complete palsy. If the patients had a complete palsy, the time it took to recover was twice as long as those patients who had partial palsy. The results concluded that steroid treatment did not influence either the chance of recovery or the time required to recover. The age of the patient was the only significant factor between patients that recovered and patients that had sequelae. Consequently, the older patient may have a decreased chance of a good
outcome as compared to the younger patient.\textsuperscript{11}

A statistically definitive article that shows steroids as the treatment of choice in BP has not yet been published. However, from articles that have been written, a strong suggestion that treatment with steroids is beneficial has been noted.\textsuperscript{10} Even though steroids do not affect the cause of the disease or prevent partial denervation or contracture, steroid therapy may assist in the following aspects of BP: 1) They may prevent denervation. 2) They do prevent autonomic synkinesis. 3) They may prevent progression of incomplete paralysis to complete paralysis. 4) They may help hasten recovery.
CHAPTER III

ELECTROTHERAPY TREATMENT IN BELL'S PALSY

Therapy utilizing electrical stimulation of paralyzed muscle has been widely used for the treatment of muscle denervated by many different causes.\textsuperscript{12} There are two reasons for stimulating denervated muscle electrically. One reason is to evaluate the damage to the nerve, and therefore, establish the prognosis of the patient's condition. Another reason is to exercise the muscle in an attempt to keep the denervated muscle healthy while the injured axons regenerate and reinnervate the muscle.\textsuperscript{12} It is assumed that if the muscle is kept healthy, functional recovery is accelerated following reinnervation of the affected muscle.

It is difficult to make a decision on a patient's prognosis in the early stages of facial paralysis. Electrodiagnostic testing tries to predict the outcome of BP by determining the physiologic extent of damage to the facial nerve. Electrophysiologic tests can track the degeneration phase of BP.\textsuperscript{1} However, these tests are only useful in the first two to three weeks of the onset of the palsy. Testing is not necessary for those patients who present with only partial denervation, that is when visible voluntary movement is still present.

Marsh\textsuperscript{13} screened patients who had complete paralysis by using the
minimal nerve excitability and electroneuronography tests. Marsh found that a threshold difference on the nerve excitability test of 3.0 to 3.5 mA or greater between comparable stimulation sites on each side of the patient's face was considered to be an indication of a poor prognosis. This finding is contradicted with the findings of Prescott.14 Prescott found that a poor prognosis was indicated when a patient had no visible contraction with the use of maximal tolerated electrical stimulation, not just a 3.0 to 3.5 mA difference between the two sides of a patient's face.

Sillman and Associates15 conducted a study using electromyography (EEMG) testing to predict the prognosis of patients with Bell's Palsy. Ninety-one patients with idiopathic (62) and traumatic (29) facial paralysis underwent evoked electromyography testing within two weeks of the onset of paralysis. Nine patients that had idiopathic paralysis and 12 patients that had traumatic paralysis underwent surgical decompression of the facial nerve. The facial nerve recovery was graded using the House-Brachmann facial nerve scale. The patients were put into groups based on the maximal decline of compound muscle action potential (CAP), as determined by EEMG, and on the level of recovery the patient experienced one year after the onset of the paralysis. It was reported that among patients who did not undergo surgery, incomplete clinical recovery (grade III or higher) was significantly associated with CAP decline of greater than 90% (p>0.05) for idiopathic paralysis. But, there was no significant association between CAP decline of greater than 90% and clinical outcome in traumatic
paralysis. These findings support the use of EEMG as a prognostic tool when evaluating Bell's Palsy.

Physiological changes in denervated muscle consist of atrophy, circulatory changes, electrical activity changes, and chemical changes. Atrophy of the muscle entails a decrease in muscle size but not in the number of muscle fibers. There is a thickening of connective tissue and contractures may develop unless range of motion is maintained while the muscle is denervated. Circulatory changes that result from denervation lead to poor nutrition for the muscle, which is a factor for initial and further degeneration of the muscle fibers. Also, because of a poor nutritional state, the muscle may be more prone to injury. After two to three weeks, the denervated muscle no longer responds to alternating current (AC) stimulation, and must, as a result, be stimulated with direct current (DC). DC is required because a long duration is needed to stimulate the muscle. Although the muscle also tends to become isosensitive (loss of motor point), there still remains an area that can be stimulated to give the best muscle contraction with the least amount of current (clinical motor point). Chemical changes result in a decrease in actin and myosin which contributes to a decrease in the muscle bulk.

Gutmann and Gutmann conducted a study on electrical stimulation of denervated muscle of rabbits. It was concluded from the study that electrical stimulation delays and diminishes muscle atrophy following denervation. It was also found that electrical stimulation accelerates the return of the muscle to its
former size following reinnervation. Gutmann and Gutmann also reported that electrically stimulated muscle reveals a decrease in fibrosis, an increase in fiber size, and an increase in excitability as compared to unstimulated muscle.

Herbison and Associates\textsuperscript{18} performed a study that analyzed stimulation (treatment) parameters. Herbison's group stimulated (interrupted, direct, square wave current of 10-mA intensity) individual denervated muscles in treatment sessions that lasted one minute (each one minute session entailed alternating five seconds of continuous stimulation and one second of rest), twice a day, five days a week, with one hour between sessions. Herbison and associates\textsuperscript{18} reported that 1) with interrupted direct current of 25-ms pulse duration and frequency of 20 Hz, the treated muscle weighed 2% more than the untreated muscle; 2) with a current of 100-ms pulse duration and frequency of 2 Hz, the muscle that was treated exceeded the untreated muscle in weight by 10%; and 3) with a current of 25-ms pulse duration and frequency of 20 Hz, the treated muscle's weight was 10% to 33% more than the untreated muscle. These results demonstrated a significant reduction of muscle atrophy using interrupted, direct current stimulation with a pulse duration of 25-ms and a frequency of 20 Hz.

Chor and associates\textsuperscript{19} studied denervated muscle of monkeys and reported that electrical stimulation did not restore muscle bulk any more than what massage and passive movement did. The protocol of this study was ten galvanic-induced contractions, once a day. Stimulation was initiated two weeks
post-nerve damage and repair. Argument has been made that the stimulation was not sufficient enough to produce an effect, and also, that stimulation was begun too late.

Schimrigk, McLaughlin, and Gruniger\textsuperscript{20} did a study that involved stimulating the crush-denervated muscle of albino rats. The protocol involved was the use of galvanic current for two minutes, three times a day. The stimulation was supramaximal (5V to 8V, 4A to 6A) and delivered at a frequency of five pulses per second (pps). It was found, histologically, that the stimulated muscle had less central nuclei and a larger number of necrotic single fibers than the untreated muscle. Schimrigk and Associates\textsuperscript{20} concluded that electrical stimulation inhibits reinnervation of denervated muscle.

Herbison, Jaweed, and Ditunno\textsuperscript{21} have suggested that the lack of success in facilitating reinnervation of denervated muscle may be due to intense stimuli (i.e., 25 mA). This intense stimulus may cause so much movement in the muscle that a local trauma to the newly formed neuromuscular junction occurs, and thereby, limits the reinnervation.

Shrode\textsuperscript{6} investigated the results of high-voltage electrical muscle stimulation and chiropractic manipulation to treat two patients with Bell's Palsy. Both patients were treated with high-voltage pulsed galvanic current at 80 peaks/sec with a seven-inch hand held probe, with intensity to the patient's tolerance for ten minutes. The eight most common motor points that were stimulated are shown in Figure 1. One case was seen for 16 treatments over a
Figure 1. Motor points of face. 

1. Frontalis m. 
2. Corrugator supercili m. 
3. Orbicularis oculi m. 
4. Nasal compressor m. 
5. Quadratus labii superioris m. 
6. Orbicularis oris m. 
7. Quadratus labii inferioris m. 
8. Temporalis m. 

Figure 1. Motor points of face. 

16
six week time span. The other case was seen for nine treatments over a three week time period. Shrode\textsuperscript{6} concluded that both patients benefited from the treatment with complete resolution of symptoms.

To summarize, evidence for electrical stimulation is that\textsuperscript{6,12,15-21} 1) appropriate electrical stimulation can cause a contraction of denervated muscle; 2) contraction of the muscle may decrease edema and venous stasis within the muscle, and therefore, delay muscle fiber degeneration and fibrosis; 3) with appropriate electrical stimulation, recovery time is shortened. Evidence against the use of electrical stimulation is that 1) contraction of denervated muscle may inhibit reinnervation by disrupting the regeneration of neuromuscular junctions; 2) the increased sensitivity of denervated muscle to trauma may increase the muscles susceptibility to damage with the use of electrical stimulation; 3) the cost and time of the prolonged treatment is not worth the rehabilitated effects.

Because of the various pros and cons found for electrical stimulation, there is much controversy over its use as a treatment for Bell's Palsy, and therefore, more research needs to be done.
CHAPTER IV

SURGERY FOR BELL'S PALSY

The rational for a surgical approach for the treatment of Bell's Palsy is based on the assumption that 1) the facial nerve is compressed within the Fallopian Canal (see Figure 2), and 2) the release of the compression enhances the return of facial movements.\textsuperscript{22} There is little argument about the need of surgery to correct functional disabilities and cosmetic deformities resulting from permanent facial paralysis, but there is controversy about whether or not surgery prevents the condition of faulty regeneration following Bell's Palsy. Given the proper evaluation and indications, the basis for a timely surgery is aimed at removing the compression in order to prevent the cosmetic and functional disabilities in those patients that have progressive degeneration of the facial nerve.

The natural course of Bell's Palsy reveals that the fate of the facial nerve is determined within the first two to three weeks after onset of the palsy.\textsuperscript{7} Those patients that present with less than 90\% maximal degeneration within three weeks of the palsy onset, generally regain satisfactory facial movement without any form of treatment.\textsuperscript{20} Those patients that present with 95\% to 100\%
1. Geniculate ganglion
2. Vestibulocochlear nerve
3. Motor nucleus of facial nerve
4. Labyrinthine segment
5. Tympanic segment
6. Mastoid segment
7. Stylo mastoid foramen
8. Stapedius nerve
9. Chora tympani nerve
10. Greater petrosal nerve

Figure 2. Course of facial nerve through facial canal.

\textsuperscript{22}
degeneration within two weeks of onset, however, have a 50% chance of permanent unsatisfactory recovery of facial function. This means that if 10% of the facial nerve fibers are still intact and remain conductive, enough endoneural tubes are also intact, and therefore, proper regeneration of the nerve is possible. Therefore, surgery for Bell's Palsy is indicated when 1) the chance of a satisfactory natural return of facial function is reduced under an admissible minimum, and 2) the majority of endoneural tubes are still intact.

The surgical procedure that is typically used is a middle cranial fossa decompression. A preauricular incision is made at the level of the tragus and is extended superiorly and anteriorly into the temporal scalp. The temporalis muscle is split and a craniotomy bone flap is removed. The dura covering the floor of the middle fossa is retracted and the facial nerve and internal auditory canal is uncovered. After decompressing the meatal foramen and labyrinthine segment to the geniculate ganglion, a muscle plug is placed over the internal auditory canal. The dura is then allowed to return to its normal anatomic position. The craniotomy bone flap is replaced and secured by suturing closed the temporalis muscle and scalp.

In 1981, Fisch conducted a study in which 14 patients with 90% or more degeneration within three weeks of palsy onset received immediate surgical treatment. These 14 patients and 13 patients who refused treatment were assessed one to three years later. Fisch concluded that 1) surgery done when the facial nerve fibers have degenerated to 90% to 94% within one to two weeks
after onset does not harm the facial nerve and may prevent further progression of degeneration; 2) surgery performed when degeneration has reached 95% to 100% within one to 14 days after onset significantly improves the recovery of facial function; and 3) surgery done when degeneration has progressed to 95% to 100% during the third week after onset does not improve the return of facial movement. Overall, it was concluded by Fisch that surgical decompression should be performed within 24 hours when degeneration has reached 90% to 94% within one to 21 days after onset of the palsy.

Giancarlo and Matucci\textsuperscript{23} treated 76 patients between 1964 and 1968 with cortisone or corticotropin. However, progression of nerve degeneration occurred in 27 of the patients. These patients were advised to have surgery to decompress the nerve. Nineteen patients accepted surgery and the eight that refused were used as the control group. Giancarlo and Matucci concluded that functional recovery of the surgical group was 73% and the recovery of the control group was 14%. This study has been criticized because the selection of the groups was not random and the assessment of the final results was expressed as a percentage of normal function based on photographs of the patient.

Mechelse,\textsuperscript{24} in 1971, conducted a study in two co-operating hospitals between 1965 and 1969. There was a total of 267 patients that were diagnosed with BP. Twenty-five of those 267 were considered to have a poor prognosis on the basis of standardized clinical and EMG criteria. The 25 patients were randomly assigned to a control group (13) and a surgical group (12). A
decompression of the vertical mastoid segment was performed (7-20 days after onset). The results between the two groups showed no significance. Only one patient out of the 25 recovered completely. Mechelse concluded that the prediction of a poor prognosis was accurate.

May,\textsuperscript{25} in 1979, performed a total nerve exploration on 31 patients that had Bell's Palsy. Seven of the 31 patients underwent surgery to decompress both the mastoid part of the facial nerve and the petrosal portion of the facial nerve. It was concluded that 100\% recovery was seen in these seven patients as compared to 72\% in a decompression surgery of just the mastoid part, 35\% in a supportive therapy, and 10\% in a prednisone treated protocol. May concluded that this type of surgery is not yet justified because no prospective randomized study has been done.

In 1985, May and Klein\textsuperscript{26} studied 273 patients with Bell's Palsy to assess the prognostic significance of evoked electromyography in predicting the benefit of transmastoid facial nerve surgery for decompression of the facial nerve. The results of the study showed that those patients who received surgery had no difference in recovery as compared to the natural course of Bell's Palsy.

The only study that shows statistical significance supporting surgery is the report by Fisch.\textsuperscript{22} Due to the lack of supportive studies, surgical decompression of the facial nerve as a treatment for BP remains controversial. In view of the study by Fisch,\textsuperscript{22} it is proposed that in order to gain a satisfactory (80\% to 100\%) recovery of facial function, surgical decompression is to be done within 24 hours
when degeneration reaches 90% to 94% within one to 21 days after onset of the palsy.
CHAPTER V
CONCLUSION

The medical professional who encounters a patient with the diagnosis of Bell's Palsy faces a dilemma. As stated earlier within this paper, Peitersen\textsuperscript{7} reported that the prognosis of full recovery from Bell's Palsy is quite good. Because of this natural spontaneous recovery, the choice to treat or not to treat a patient with Bell's Palsy remains controversial. In order to solve this dilemma, a physician must consider all possibilities for treatment and the effects of the treatment in order to validate the need for treatment. In this paper, three treatment programs were reviewed: steroid treatment, electrotherapy, and surgical decompression.

Published research reporting the choice treatment for Bell's Palsy is not available to date. This contributes to the controversy of steroid treatment verses no treatment. However, from current research that has been written, a strong suggestion that steroid treatment is beneficial has been noted.\textsuperscript{10} To review, steroid treatment may assist in the following: 1) They may prevent denervation. 2) They do prevent autonomic synkinesis. 3) They may prevent progression of incomplete paralysis to complete paralysis. 4) They may help hasten recovery.

The choice to use electrotherapy as the treatment for BP also is
controversial. Evidence for and against the use of electrotherapy have been presented in the paper. To review, evidence supporting the use of electrical stimulation is as follows: 1) appropriate electrical stimulation can cause a contraction of denervated muscle; 2) contraction of the muscle may decrease edema and venous stasis within the muscle, and therefore, delay muscle fiber degeneration and fibrosis; 3) with appropriate electrical stimulation, recovery time is shortened.\textsuperscript{6,18} Evidence against the use of electrical stimulation is as follows: 1) contraction of denervated muscle may inhibit reinnervation by disrupting the regeneration of neuromuscular junctions; 2) the increased sensitivity of denervated muscle to trauma may increase the muscles susceptibility to damage with the use of electrical stimulation; 3) the cost and time of the prolonged treatment is not worth the rehabilitated effects.\textsuperscript{12}

The only significant support for surgical decompression was reported in the study by Fisch.\textsuperscript{22} To review, it was proposed that in order to have any significant effect on the prognosis of Bell's Palsy, surgery should be done within 24 hours when degeneration reaches 90\% to 94\% within one to 21 days after onset of the palsy.

When pondering the question of accepting the patient for treatment and considering what treatment program to offer the patient, the physician needs to consider the consequences the disease may produce. The physician does not know if the patient will be among the 71\% who will have full recovery, even if untreated, or if the patient will be among the 29\% who will have some problems
with recovery. Not knowing the outcome or course of the disease, it will be beneficial that the physician explain to the patient the various treatment methods available and the effects of the treatments. With this patient education, the patient may also help decide on the plan of treatment and the methods of care used.

As a person who has had Bell's Palsy and has experienced full recovery, I recommend the use of a combination of steroid treatment and electrotherapy. This treatment program was successful for me. I feel that if the treatment will not hurt the condition or cause further damage, all possible methods should be used to prevent the worse case scenario.
REFERENCES


