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The Effects of Muscle Tone on Shoulder Pain in the Post-CVA Population

Jane Ubben
Grand Valley State University

Kim Moyle
Grand Valley State University

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By

Jane Ubben
Kim Moyle

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ABSTRACT

The purposes of this study were to determine whether post-CVA hemiplegic shoulder pain is related to hypertonicity, whether muscle tone of the shoulder differed between patients with pain and those without, and to determine whether a relationship exists between hypotonicity or hypertonicity and amount of range of motion (ROM), glenohumeral subluxation, or sensory disturbances, in those who experience pain. Data was analyzed for significance using the Wilcoxon Matched-Pairs Signed Ranks Test , Mann-Whitney U-Test, Chi-Square and Fisher's Exact Tests ($\alpha = 05$). Review of 109 charts revealed a significant relationship between ROM limitations and pain. ROM limitations were significantly related to hypertonicity, but hypertonicity was not found to be related to pain when those with and without pain were compared. Comparing muscle tone prior to and following the onset of pain, hypertonicity was found to be significantly related to shoulder pain, $p < 0.01$. Shoulder pain was significantly related to sensory deficits and unilateral neglect. ROM limitations may produce pain secondary to contracture development or to abnormal biomechanics of the shoulder joint. Hypertonicity of shoulder musculature may contribute to limitations in ROM. Sensory deficits may lead to trauma resulting in pain. Need for further research regarding effects of muscle tone on pain development in the hemiplegic shoulder was identified.

DEDICATION

This research project is dedicated to those patients who suffered a stroke, unaware of its contribution to our study. May further research be done from this project to help decrease shoulder pain in the post-CVA population.

We would also like to dedicate this thesis to our families and friends for their support and understanding during our college years.

Also a special dedication to Arin, from Jane, for his patience with his mom throughout the last three years.

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CHAPTER 1

INTRODUCTION

Hemiplegia is the paralysis of one side of the body, which occurs with lesions involving the cerebral hemisphere or brainstem, and is considered the classic sign of neurovascular disease of the brain (Ryerson, Levit, 1987). Lesions are produced by thrombotic, embolic, or hemorrhagic processes, with the upper extremity more severely affected in all three classifications of stroke, except with involvement of the anterior cerebral artery (Cailliet, 1980).

Upper extremity function is an integral part of self-care activities, balance, transfer activities, wheelchair mobility and ambulation (Cailliet, 1980). Functional recovery following stroke will be affected in large part by shoulder function, because the shoulder moves the arm in space and provides stability and control for upper extremity function. Recovery following stroke is variable, but functional recovery of the upper extremity is usually less complete than that of the lower extremity (Cailliet, 1980).

One factor that complicates rehabilitation efforts in activities such as balance retraining, participation in self-care activities and ambulation training is shoulder pain. The reported incidence of shoulder pain in hemiplegia ranges from 5% (Parker, Wade, Langton Hewer, 1986), to 84% (Najenson, Yacubovich, Pikielini, 1971). Variation may be due to hemiplegic patient selection, the object of the study, and the researcher's definition of pain (Roy, 1988). Functional recovery in the patient with hemiplegic shoulder pain will be deterred because the patient will avoid use of the extremity for activities such as those listed above (Caldwell, Wilson, Braun, 1969). Pain also has a detrimental effect on the patient's motivation.

contractures, subluxation (Moskowitz, Goodman, Smith, Mellins, 1969), and sensory disturbances (DeCourval, et al., 1990), have all been postulated as causes of pain. Other suggested causes of shoulder pain in hemiplegia include trauma to joint structures (Anderson, 1985), degenerative and arthritic problems of the glenohumeral joint (Cofield, 1990), and pain referred from the cervical and visceral regions (Bennett, 1983). ROM limitations and subluxation have been associated with abnormal tone, i.e., an increase or decrease in resistance to passive movement, or sensory deficits of the affected upper extremity (Tobis, 1957, Andrews, Bohannon, 1989). Traumatization of the shoulder joint has been associated with sensory deficits of the affected upper extremity (DeCourval, et al., 1990).

Dystonia is an inherent part of the natural sequence of recovery following stroke. Described by Twitchell in 1951, this sequence includes (1) an initial loss of voluntary function with a decrease or loss of tendon reflexes, (2) hypotonicity, (3) an increase in deep tendon reflexes with possible clonus, and (4) clasp-knife phenomenon appearing in the elbow flexors.

Pain involving the hemiplegic shoulder may develop within the first few weeks following a CVA, or may develop at any time during the first year (DeCourval et al., 1990). Tobis, (1957), attributes pain to the flaccid stage of recovery, while Bobath (1972), contends that pain is not a problem until spasticity develops. Dardier and Reid, (1972), state that in their experience, the occurrence of shoulder pain in hemiplegia is more frequent with spasticity. These researchers suggest that this may be due to the fact that pain sensation is less frequently impaired when spasticity is present, than when the affected shoulder is flaccid and subluxed (Dardier, Reid, 1972).

Hemiplegic shoulder pain remains an enigma, and presents a difficult problem in rehabilitation of the stroke patient. Development of preventative treatment measures to alleviate this problem depends on a better understanding of the etiology of shoulder pain in hemiplegia. A relationship between muscle tone and pain has been theorized, but without consensus as to whether pain is associated with hypotonicity or hypertonicity. This study will look at the muscle tone of the affected upper extremity in the post-CVA patient in an attempt to determine its role in the development of shoulder pain.

The questions to be explored by this research study are: (1) Is the onset of pain in the hemiplegic post-CVA shoulder related to a change in muscle tone? (2) Is there a significant difference in muscle tone in the affected upper extremity between those patients who experience shoulder pain and those who do not? (3) Of those patients who experience shoulder pain, is there a relationship between the presence of hypotonicity or hypertonicity and the amount of ROM, glenohumeral subluxation, or sensory disturbance, including unilateral neglect?

CHAPTER 2

LITERATURE REVIEW

Each year in the United States, an estimated 500,000 new strokes occur, with approximately 300,000 survivors, the majority of whom will have some form of residual deficit (McDowell, 1990). Of these survivors, approximately 30% will be left dependent on others (Kelly-Hayes, et al., 1989). According to Thomas Anderson, (1978), ambulation is possible with minimal voluntary motor return in the lower extremity, the affected extremity primarily being used for weight bearing. However, usefulness of the upper extremity (finger dexterity and fine coordinated movements) is dependent on nearly complete return of volitional motor control (Anderson, 1978). Motor return generally begins proximally, with distal return occurring later in recovery except with lesions of the anterior cerebral artery (Anderson, 1978). The shoulder provides the musculoskeletal base for both proximal and distal control.

Normal Shoulder

The bony anatomy and muscular mechanics of the shoulder girdle are suggestive of its complexity. The shoulder girdle is comprised of four joints: (1) the sternoclavicular joint, (2) the acromioclavicular joint, (3) the scapulothoracic joint and (4) the glenohumeral joint. The scapulothoracic joint is not a true joint, as movement which occurs here is related to movement at the acromioclavicular joints. The placement of the scapula on the thoracic cage provides stability for movement of the arm (Moore, 1985), and maintains the glenoid fossa's angle with the frontal plane, which is approximately 40 degrees, the shoulder joint's physiological plane (Jensen, 1980). The glenoid fossa accommodates approximately one-third of the humeral head (Anderson, 1985). This relatively small area of bony contact provides the shoulder with a large degree of mobility.

The upward slope of the glenoid fossa, along with the glenoid labrum, assist in maintaining the approximation of the glenohumeral (GH) joint (Varghese, 1981). Other structures providing stability to the GH joint include the joint capsule, the coracohumeral ligament and the rotator cuff muscles (Jensen, 1980). When the arm is relaxed and hangs dependently at the side of the body, the superior aspect of the joint capsule, including the coracohumeral ligament, is taut, preventing downward displacement of the humerus (Jensen, 1980). However, when the arm is abducted, the superior portion of the capsule and coracohumeral ligament are slack (Anderson, 1985), and proper alignment is dependent on rotator cuff and deltoid muscles (Varghese, 1981).

The bones of the shoulder joint, along with their muscular attachments, contribute to scapulo-humeral rhythm, i.e., for every two degrees of humeral abduction or forward flexion, there is one degree of scapular rotation. All four joints of the shoulder girdle must be mobile for normal scapulohumeral rhythm to take place. Normal rhythm is also dependent on force couples created by muscles acting to rotate and stabilize the scapula against the thoracic wall (Anderson, 1985). Normal scapulohumeral rhythm is necessary for functional activities involving elevation of the arm over the head. Abnormal rhythm allows impingement of soft tissue structures to occur between the acromion and the head of the humerus (Varghese, 1981). Following a lesion of the CNS, scapulohumeral rhythm is often impaired secondary to abnormal muscle tone (Ryerson, Levit, 1988).

Muscle Tone

Muscle tone is defined as "the resistance of muscle to being passively lengthened, or stretched" (Gordon, 1990). With the limb held in a gravity-eliminated position, muscle tone is assessed through slow, passive stretch of the muscle, measuring the amount of tension felt at different lengths (Harburn, Potter, 1993). According to Harburn and Potter, (1993), muscle tone is the component of neurophysiological spasticity which can be clinically assessed. Harburn and Potter, (1993), stress that all aspects of

neurophysiological spasticity cannot be clinically assessed, nor easily described, and that synonymous usage of the terms "spasticity" and hypertonicity is a common error (Harburn, Potter, 1993). Spasticity is characterized by hypertonia and hyperreflexia, with possible clonus and rigidity (Botte, Nickel, Akeson, 1988). Hypertonicity is an increase in resistance to passive movement, and is a component of spasticity (Harburn, Potter, 1993).

According to Jensen, (1980), muscle tone in the muscles surrounding a normal joint is the joint's primary source of protection. Muscle tone is comprised of (1) the intrinsic elastic stiffness of muscle, tendon and connective tissue, (2) reflexes, and (3) volitional motor control (Gordon, 1990).

Immediately following a CNS lesion, there is generally a period of paralysis or hypotonicity (i.e., decrease in resistance to passive stretch) produced by depression of neuronal activity, primarily involving the corticospinal tracts (Carpenter, 1991). There is typically a decrease in reflex activity and decrease in alpha-motor neuron excitability (Gordon, 1990). This period of paralysis or hypotonicity may last a few days, or may be present for weeks to months (Ryerson, 1990).

Generally, recovery from stroke follows characteristic stages. Following the initial state of hypotonicity, the stretch reflexes may return, along with an increase in muscle tone. Abnormal control of posture usually occurs, with the characteristic patterning of the upper extremity following upper motor neuron lesion being flexion, adduction and internal rotation (Harburn, Potter, 1993). Spasticity further enhances the abnormal posturing in flexion, and abnormal movement patterns, as the weakened antagonistic muscles cannot overcome the spastic muscles. Secondary changes in connective tissue, including shortening and development of adhesions, are also responsible for an increase in muscle tone (Harburn, Potter, 1993).

Hemiplegic Shoulder

Immobilization of an extremity leads to a decrease in blood flow, muscular atrophy, formation of adhesions and cartilaginous degeneration (Akeson, Amiel, Abel, Garfin, Woo, 1987). Immobilization of the upper extremity following stroke may occur due to hypotonicity or hypertonicity. Contractures develop as a complicaton primarily in association with spastic hemiplegia, when the shoulder joint is held in a position of adduction and internal rotation (Varghese, 1981). A retrospective study by Bohannon and associates (1986), looked at the incidence of shoulder pain in 50 hemiplegic patients, and the relationship of shoulder pain to patient age, time since onset of hemiplegia, spasticity, weakness, and range of motion of the affected shoulder. They found that over time, ROM restrictions increase, and that these limitations are associated with pain (Bohannon, Larkin, Smith, Horton, 1986).

Further study by Andrews and Bohannon, (1989), was performed to determine shoulder lateral rotation ROM in stroke patients, to compare this ROM on the affected and nonaffected sides, and to determine the relationship between these measurements and variables including age, sex, and time since onset of stroke. Intrarater and interrater reliability of the measurements were also established (Andrews, Bohannon, 1989). Measurements were performed on 25 patients who were consecutively admitted to a rehabilitation center, and who met the criterion of the study. All 25 subjects demonstrated greater passive shoulder lateral rotation on the nonaffected side than on the affected side. Intrarater and interrater measurements were both reliable. A significant correlation was found between lateral rotation ROM on the affected side and time since onset, but other variables showed no significant correlation (Andrews, Bohannon, 1989).

The amount of passive loss of shoulder ROM was related to the severity of complaint of shoulder pain in Joynt's (1992), study. Joynt examined 97 patients who were at least three months post-stroke, collecting data on shoulder abnormalities such as

subluxation, tenderness with palpation, passive ROM, amount of pain with motion, strength, sensation, tone and reflexes. History and examination led to the diagnosis of a shoulder problem in 67 patients. Demographic information revealed a number of significant findings other than that of ROM limitation and pain mentioned above. Findings included: (1) The most intense pain with movement was located laterally in the shoulder area with radiation into the arm, (2) ROM limitations were related to pain radiating down the arm, (3) lateral shoulder pain with radiation into the arm was found more often in patients with better sensation, (4) weaker patients had the highest incidence of subluxation. Twenty-eight of the 67 patients diagnosed with shoulder problems were given an injection of 1% lidocaine solution into the subacromial area. Moderate to marked pain relief was expressed by approximately 50% of those injected. Findings indicated that ROM and pain both improved similarly. Findings also indicated that patients without evident subluxation had greater improvement of ROM. The author concluded that pain may originate from the subacromial area in a significant number of cases (Joynt, 1992).

Other possible causes of hemiplegic shoulder pain may relate to the presence of hypotonic musculature. The weight of the upper extremity may produce stretching of the joint capsule, the coracohumeral ligament and rotator cuff musculature, with possible resultant pain. According to Jensen, (1980), Kioresku (1974), found the coracohumeral ligament to contain a large concentration of neuroreceptors, suggesting high susceptibility to pain. Ryerson (1990), states that the coracohumeral ligament reinforces the superior aspect of the joint capsule, and she stresses the capsule and ligament's role in joint stability. Najenson and associates (1971), contend that severe pain localized to the shoulder occurs with rotator cuff rupture, whereas pain of less severity is produced by malalignment of the GH joint.

Hakuno and associates (1984), compared the involved and noninvolved shoulder joints of 77 patients to determine whether arthrographic findings, including rotator cuff

tears, occured with hemiplegia. Findings revealed no significant difference between rotator cuff tears in paralyzed and nonparalyzed shoulders. They did, however, find that adhesive changes occurred at a significantly higher incidence in the paralyzed shoulders (23 out of 77), as compared to the nonparalyzed shoulders (2 out of 77).

Plain X-rays and arthrographies were performed on the paralyzed shoulder of 32 hemiplegic patients in a study by Najenson, Yacubovich, and Pikielni, (1971). Arthrographies were also performed on the nonparalyzed shoulder. 40% of the affected shoulders were found to have rotator cuff ruptures, while only 16% showed rotator cuff ruptures on the unaffected side. These findings are quite different from those in the study cited above (Hakuno, Sashika, Ohkawa, Itoh, 1984).

Subluxation of the GH joint may occur in either the flaccid or spastic stages following stroke secondary to weakness, spasticity, imbalanced muscle firing or scapular malalignment (Ryerson, 1988). When GH joint subluxation occurs during the hypotonic or flaccid stage of hemiplegia, an inferior subluxation usually results. The scapula rotates downward, the glenoid fossa no longer supports the humeral head, which then slips inferiorly and becomes internally rotated due to the weight of the arm (Ryerson, 1988). Anterior subluxation of the GH joint may occur during the spastic stage of hemiplegia. According to Ryerson, (1990), the activity of the shoulder elevators causes the scapula to rotate downward and become elevated on the rib cage. The humeral head moves anteriorly and the humerus hyperextends and becomes internally rotated, due to activity of the latissimus dorsi (Ryerson, 1990). Ryerson (1990), also describes a superior subluxation which occurs with return of deltoid and biceps firing, in which the humerus becomes internally rotated and the head of the humerus is pulled upward, in contact with the acromial process. Both the flaccid and spastic stages of hemiplegia, in association with subluxation, and without, have been considered by researchers as possible sources of pain.

A study by VanOuwenaller and associates (1986), followed 219 hemiplegic patients after CVA for one year. They distinguished between flaccid and spastic hemiplegia and looked at other factors including subluxation. Daily observation of patients revealed 72% had shoulder pain at least once. Shoulder pain occurred more often (85%) in patients with spastic hemiplegia, than with flaccid hemiplegia (18%). Subluxation occurred more often in patients with flaccid hemiplegia, but was found to be associated more often with pain in patients with spastic hemiplegia. Spasticity was diagnosed on the basis of an increased myotatic reflex, and subluxations were confirmed by roentgenologic examination.

Bohannon and Andrews, (1989) studied 24 hemiplegic patients to determine the relationship between shoulder pain and subluxation, using two different methods of clinical measurement, and to determine the interrater reliability of a clinical measurement of shoulder subluxation. The researchers did not find a significant relationship between subluxation and pain. Paretic shoulder subluxation was measured by palpation, with shoulder pain measured during passive lateral rotation. The authors clinical scale for measuring subluxation (none = 0, minimal = 1, substantial = 2), was found to be highly reliable (Bohannon, Andrews, 1990). The lack of a significant relationship between subluxation and pain found by Bohannon and Andrews, (1989), is in accordance with a previously discussed study by Joynt (1992), in which no statistical significance was found between subluxation of the affected shoulder and the amount of pain with passive movement.

Sensation

Sensory impairment has been suggested as a predisposing factor for pain in the hemiplegic shoulder (DeCourval, et al., 1990). Sensory disturbances in hemiplegia range from mild impairments such as an inability to localize touch, to a loss of recognition of the

affected half of the body (Ryerson, 1990), called unilateral neglect. The patient with neglect will not protect the arm, increasing the possibility of injury (McDowell, 1990).

DeCourval and associates (1990), looked at possible relationships between the painful shoulder in hemiplegia and unilateral neglect. Ninety-four patients were evaluated at an average of 40 days post-CVA. Shoulder pain was found in 45 subjects, and 24 subjects demonstrated unilateral neglect. No relationship was found between neglect and pain in the affected shoulder. A study by Joynt, (1992), was conducted to determine the relationship between the amount of shoulder pain and loss of ROM, subluxation, spasticity, strength and sensation. Sixty-seven hemiplegic patients were diagnosed with a shoulder problem following history and examination, which were conducted by the researcher. Joynt, (1992), concluded from his study that there was no relationship between sensory loss and amount of pain in the affected shoulder. Although this study did not look at unilateral neglect, Joynt, (1992), did find a higher incidence of shoulder pain in patients with left hemiplegia, which is more often associated with unilateral neglect (Ryerson, 1990).

Studies undertaken to determine the etiology of shoulder pain in hemiplegia have produced conflicting results. Abnormal tone often leads to secondary complications such as GH subluxation, rotator cuff tears and ROM limitations. ROM limitations have been found to relate to pain in several studies (Bohannon, Andrews, 1989; Bohannon, et al., 1986, Joynt, 1992). However, there is little consensus on whether GH joint subluxation is painful, or whether rotator cuff tears are affected by hemiplegia (Hakuno, Hironobu, Ohkawa, Itoh, 1984; Najenson, Pikielini, 1956). These studies have addressed pain as it relates to secondary complications of abnormal tone. A study on shoulder pain by VanOuwenaller, et al., (1986), made a distinction between flaccid and spastic hemiplegia, but further study addressing the changes in muscle tone over time, and muscle tone's relationship to shoulder pain in hemiplegia appears to be indicated.

Hypothesis

The hypotheses of this study were as follows:

1. The onset of shoulder pain in the post-CVA hemiplegic population is related to a change in muscle tone.

2. There is a significant difference in the muscle tone of the affected upper extremity between those patients with and those without shoulder pain. Those patients with shoulder pain will have a hypertonic upper extremity.

3. Of those patients who experience shoulder pain, ROM limitations, glenohumeral subluxation, and sensory deficits, including unilateral neglect, are associated with the presence of hypertonicity.

Definition of Terms

Hemiplegia is the paralysis of one side of the body.

Muscle tone is a normal state of balanced muscle tension. Its components include (1) elastic properties of muscle, tendon, and connective tissue, (2) reflexes, and (3) volitional motor control.

Hypotonicity is a decrease in the resistance felt with passive movement.

Hypertonicity is an increase in the resistance felt with passive movement.

Flaccid paralysis is an abnormal condition characterized by the weakening or the loss of muscle tone.

Spasticity is clinically characterized by hypertonia, hyperreflexia, possible clonus and rigidity.

Subluxation is a partial dislocation.

Unilateral neglect is the perceptual impairment in which the patients fails to recognize the affected half of their body.

CHAPTER 3

METHODOLOGY

Design

This study had a retrospective design. The population was defined as those individuals who suffered a stroke and were initially hospitalized at St.Mary's Hospital in Grand Rapids, Michigan and transferred to Mary Free Bed Rehabilitation Center for rehabilitation. The outcome observed was the presence or absence of shoulder pain and type of muscle tone in the hemiplegic upper extremity. Shoulder pain was considered any mention of pain or discomfort in the shoulder region at rest, or with motion reported more than two days within a given week. The data that was collected included the following: side affected, shoulder pain (location, characteristics, severity, and with or without motion), muscle tone of the patient's affected upper extremity (UE) (initially, during and prior to pain and at discharge), shoulder malalignment, range of motion (ROM), sensation, neglect, and pain medications. Patient charts were reviewed for this data, collected from all caregiver's notes at both hospitals.

A mini chart review at each hospital was done to pre-test and revise the data collection tool. Also, suggestions given by the Review Committee at Mary Free Bed were taken into consideration to help revise our tool. Accuracy between data collectors was checked every fifty charts. Both researchers collected data for the same three subjects with 95% accuracy between them.

Advantages of doing a retrospective study were that it was economically feasible and not as time consuming as measuring all the above factors in shoulder pain ourselves. Patient accessibility was not a problem when reviewing charts.

Study Site

St. Mary's Hospital is a 320 bed acute care hospital. Mary Free Bed is an 80 bed rehabilitation facility. These facilities are physically linked together, but are two separate institutions. A large percentage of those patients who suffered CVAs and were acutely admitted to St.Mary's were transferred to Mary Free Bed Hospital for further therapy. These are the individuals we followed on their medical course. The medical records department from each facility was contacted and approval for our study was received from both institutions' Review Boards.

Sample

Our sample of patients were admitted to St.Mary's Hospital prior to January 1, 1993 with a diagnosis of an acute cerebrovascular accident and then were transferred to Mary Free Bed Hospital for further rehabilitation. (This excluded those patients who were not rehabilitation candidates, due to very good or very poor prognoses.)

One-hundred and twenty consecutive patient's charts were reviewed. Some patient charts were not available at the time of our data collection process at both hospital sites, so a total of 109 data collection forms were completely filled out.

Instruments

The tools used to collect the information are found in Appendices A, B, and C. Information used to compile these tools was taken from the Occupational Therapy and Physical Therapy evaluation forms from both St. Mary's Hospital and Mary Free Bed Rehabilitation Center.

Procedures

The Medical Records Department at Mary Free Bed Rehabilitation Center and St. Mary's Hospital were contacted, and a list was compiled of 120 post-CVA patients who were transferred from St. Mary's Hospital to Mary Free Bed Rehabilitation Center. The medical records of those patients were requested from both hospitals for the authors of this paper to review. Initial evaluation, progress and discharge notes from all care givers

were reviewed for the incidence of shoulder pain, along with all other information stated below. The primary source of information was Occupational Therapy notes. If the information was not available there, other discipline's notes were then searched.

If documentation of pain was found, its location, characteristics, and severity were recorded. Also, the presence of pain with motion or at rest was recorded. Data regarding the number of days that shoulder pain was reported within the first week following the onset of pain was also documented. The patient's ROM (of the affected and unaffected UE), sensation, reflexes, alignment, and neglect were recorded from records dated as close as possible to the occurrence of pain. (If ROM of the unaffected UE wasn't available, ROM within normal limits was assumed.) Data regarding the muscle tone of the patient's affected UE preceding and following the onset of shoulder pain, was collected as documentation was available. Documentation of the patient's position during the evaluation of muscle tone did not allow us to look at the the relationship of tone and position. Pain medications that were prescribed were also documented. The above information was collected on the form in Appendix A. Discharge information from Mary Free Bed about all variables stated above was again collected for those patients who experienced a painful shoulder and was recorded on the "Discharge" form (Appendix B).

If no pain was found to occur throughout the patient's stay at the two hospitals, the data regarding the muscle tone found at initial evaluation at St. Mary's was collected along with the following discharge data from Mary Free Bed: ROM, sensation, muscle tone, neglect, reflexes and alignment (Appendix B). Pain medications were noted for those individuals who may have had discomfort elsewhere and needed relief.

General information was also collected for each patient: age, gender, onset of CVA, UE side affected, past medical history, other diagnosis, and hand dominance. Those patients taking pain medications were grouped separately from those patients not taking pain medications for our statistical analysis. A list of pain medications commonly used in rehabilitation was used as a "quick reference" to see if a medication was for pain relief.

(See Appendix C.) Drugs not on this list were individually looked up for their affect on pain.

An identification number was given to each patient to keep their records confidential. This information was only available to the researchers and any reference to the patient was made by identification number. No patient contact was made in this study, so this deferred any potential hazards to the subjects and the need to obtain informed consent from them.

CHAPTER 4

DATA ANALYSIS/RESULTS

Characteristics of subjects

The characteristics of the subjects studied included a range of ages from 24 to 88 years with a mean of 71 years. There were 51 males and 56 females in our study. The gender of two additional subjects was not documented. Fifty-five individuals had their left upper extremity affected, and 54 had their right. Eighty-four of the subjects (73%) were premorbidly right handed, six were left handed (5%), and handedness was not documented for 19 (22%) subjects .

Twenty-six (23%) of the total sample experienced shoulder pain for a varied duration between two and forty-two days. Of those with pain, 22 (84%) had pain with passive range of motion (PROM), 8 (30%) with active ROM, and 10 (38%) had pain at rest. Documentation indicated that many subjects with shoulder pain did not have the ability to move their affected UE. Eight subjects (30%) reported previous shoulder pain and 6 (23%) had experienced post CVA trauma. Ten (38%) had record of a previous CVA. Of those with a record of a previous CVA, 5 reported previous shoulder pain, but data about the previous side affected was not collected. Documentation of the pain severity, location, and presence of radiation was not found in many of the charts and may have been difficult for practitioners to assess. Eighteen percent of those individuals with pain had some form of aphasia.

The muscle tone of individuals at discharge are located in the Figure on page 37 . Twenty-seven (25%) of the subjects had a hypotonic or flaccid upper extremity, 27 (25%) had a hypertonic upper extremity and 55 (50%) had normal muscle tone in their affected upper extremity.

Data Analysis/Results

Throughout the analysis of shoulder pain, individuals were excluded from our data who did not experience shoulder pain, but were on pain medications for other discomforts. Those individuals with shoulder pain who were on pain medications, were used just as those individuals with shoulder pain who were not on pain medications, as long as they were experiencing shoulder pain at the time.

The data found in Appendix A from those patients that experienced shoulder pain was analyzed using the Wilcoxon Matched-Pairs Signed Ranks Test ($\alpha = 0.05$) to see if the presence of pain was related to a change in muscle tone. Ordinals were given to the types of muscle tone as follows to assist with this analysis: 3 = hypertonicity, 2 = normal tone, 1 = hypotonicity, 0 = flaccidity. The muscle tone following the individual's onset of shoulder pain was compared with their muscle tone prior to shoulder pain (at initial evaluation at St. Mary's Hospital, assuming they reported no pain upon initial evaluation). Those with pain on initial evaluation were excluded from this comparison, but used later for discharge comparisons. Twenty-two individuals were used in this analysis, with nine having a change in muscle tone. See Table 1 for the data. The muscle tone increased in eight individuals (6 of whom were hypertonic). The onset of shoulder pain was found to be related to a change in muscle tone of the UE, ($p < 0.01$), with hypertonicity related to the onset of shoulder pain.

Discharge data from all patients was analyzed for a significant difference in muscle tone (hypertonicity, normal tone, hypotonicity, and flaccidity) between those with and without shoulder pain. Those individuals without pain, but on pain medications for another reason were not used for this comparison. Nineteen subjects suffered from shoulder pain and 67 did not. A Mann-Whitney U Test was used for this analysis ($\alpha = 0.05$). The categories of muscle tone were assigned ordinals as stated above. Table 2 gives the specific data which was used for analysis. No difference in muscle tone was found between those subjects with and without shoulder pain. Our hypothesis that shoulder pain

was related to hypertonicity in the affected upper extremity was disproven.

The discharge data for those patients with hypertonicity and pain was analyzed to see if there was a relationship between the presence of hypertonicity and ROM, subluxation, increased, normal, impaired or absent sensation, and neglect. The discharge data for those patients with a painful shoulder and hypotonicity (including those with a flaccid UE) were also analyzed to see if there was a relationship between the presence of hypotonicity and ROM, subluxation, sensation and neglect. For each of the five ranges of motion listed in Appendix A, the ROM of the affected shoulder was calculated as a percentage of the unaffected shoulder's ROM. These values were then categorized for statistical analysis as: 0 = normal ROM (100%), 1 = minimally decreased ROM/within functional limits (99-66%), 2 = moderately decreased ROM (66-33%), 3 = maximally decreased ROM (33-0%).

The degree of sensory impairment was generalized by taking the most frequent rating in the seven categories listed in the appendices. This nominal category was then ordinalized for statistical analysis as the following: absent sensation = 0, impaired sensation = 1, normal sensation = 2, increased sensation = 3.

The Mann-Whitney U Test ($\alpha = 0.05$) was used to analyze if there was a relationship between an increase or decrease in muscle tone in individuals who suffer from shoulder pain and an increase or decrease in shoulder range of motion or sensation. To test for a relationship between muscle tone and subluxation or neglect, a Fisher's Exact Test was used with $\alpha = 0.05$. The specific data used for analysis are given in Table 3.

No difference was found between the two groups when sensation, shoulder ROM, presence of neglect or shoulder subluxation were compared. Due to information not documented in the charts, the number of subjects varied for the subluxation and unilateral neglect comparison. Our hypothesis that those patients with shoulder pain and hypertonicity would experience ROM limitations, glenohumeral subluxation, and sensory deficits, including unilateral neglect was disproven.

Following our data collection process, we were also interested in comparing shoulder subluxation, shoulder ROM, sensation and neglect in individuals with hypertonicity and those with hypotonicity (including flaccidity), regardless of shoulder pain. Also, we looked at shoulder subluxation, shoulder ROM, sensation and neglect in individuals with pain and no pain (regardless of muscle tone). This information was taken from the discharge data collected. The Mann-Whitney U Test for ordinal data was again used when analyzing these groups for sensation and shoulder ROM, $\alpha = 0.05$. A Chi-Square was used to analyze the presence of subluxation and neglect between these groups, $\alpha = 0.05$, $df = 1$. Throughout this analysis, varying numbers are due to information not documented or found in the charts. The data regarding muscle tone regardless of pain can be found in Table 4.

When analyzing data of those individuals with hypertonicity and hypotonicity (regardless of shoulder pain) a Chi-Square test indicated no relationship between subluxation and neglect. Sensation was also found to be independent of muscle tone in our study. There was a significant difference in shoulder ROM between those individuals with hypotonicity and those with hypertonicity ($p = 0.008$). A one tailed Mann-Whitney U test found the passive ROM of individuals with hypertonicity to be significantly decreased in the affected UE when compared with individuals with hypotonicity ($p = 0.008$).

Table 5 summarizes the data used to compare those individuals with and without shoulder pain and the presence of secondary impairments of the upper extremity. No significant difference was found between the two groups when subluxation was studied. A significant relationship was found between unilateral neglect and shoulder pain, $X^2_{(0.05, 1)} = 4.65$, $p < 0.05$. A significant difference in the shoulder ROM ($p < 0.00004$) was found. A one-tailed Mann-Whitney U analysis showed a significant decrease in shoulder ROM of those with pain versus those without pain ($p < 0.0001$). A significant difference in the sensation of the affected UE was found ($p = 0.008$). A one-tailed Mann-Whitney U

test showed a significant decrease in the sensation of those individuals with shoulder pain versus those without pain, ($p = 0.008$).

CHAPTER 5

DISCUSSION AND IMPLICATIONS

Discussion of findings

Although limitations of this study prevent specific conclusions from being drawn, our results did not support the hypotheses (1) that there is a significant difference in muscle tone in the affected upper extremity between those patients who experience shoulder pain and those who do not, and (2) that the presence of hypertonicity is related to the amount of ROM, glenohumeral subluxation or sensory disturbances, including unilateral neglect, in patients experiencing shoulder pain.

Comparisons of muscle tone before and after development of shoulder pain revealed that the onset of pain in this post-CVA sample was associated with a change in muscle tone. Hypertonicity was demonstrated in six of the nine individuals whose muscle tone changed. An increase in muscle tone in the sample studied may suggest a protective mechanism following onset of shoulder pain. Another explanation for the change in muscle tone could be the natural progression of muscle tone from flaccidity to normal tone or hypertonicity which occurs following stroke. An increase in muscle tone also occurred in those subjects who did not experience shoulder pain.

Muscle tone comparisons between patients who experienced shoulder pain and those who did not, revealed no significant differences, suggesting that muscle tone has no relationship with the development of pain in the affected shoulder. These findings, however, disagree with those of VanOuwenaller and associates (1986). This study on shoulder pain in the hemiplegic population demonstrated a significantly greater percentage of painful shoulders in patients with hypertonic musculature of the affected upper extremity. A possible explanation for these findings is the stage of recovery at which the population was studied. Our study included patients from date of admission for stroke, through discharge from Mary Free Bed Rehabilitation Center, encompassing a relatively short duration. VanOuwenaller and associates' (1986) study followed patients from onset

of CVA for an average of 11 months. Studies of long-standing effects of abnormal muscle tone may differ from these findings.

Another important factor that may have influenced our finding that shoulder pain is not related to muscle tone is that the subjects studied had a relatively good outcome following rehabilitation (50 % had normal muscle tone in their affected UE at discharge from rehab). This is not typical in the post-CVA population, and thus may explain the small percentage of subjects having shoulder pain.

Comparisons were made in the pain group, between muscle tone and amount of ROM, GH subluxation, sensory deficits and unilateral neglect, in order to determine the impact of secondary complications of abnormal muscle tone on pain. Of those patients who experienced shoulder pain, the presence of hypertonicity was not found to be significantly related to amount of ROM, glenohumeral subluxation or sensory deficits, including unilateral neglect. No significant difference was found between the number of patients with hypertonicity and GH subluxation and those with hypotonicity and GH subluxation. Sensory deficits were also similar in number between patients with pain and hypertonicity and those with pain and hypotonicity. No relationship was found between the pain groups (those with pain and hypertonicity and those with pain and hypotonicity) and ROM limitations or unilateral neglect. Secondary complications did not relate to abnormal tone in those patients with pain. These findings may be due to the small sample size of pain patients after being partitioned into subgroups.

Eighty-one percent of the population studied were age 60 and older, an age group for which arthritis is a common problem and cause of shoulder pain (Cruess, 1980). Documentation revealed complaints of previous shoulder pain in 30% of patients with pain, with five patients in the pain sample having a previous diagnosis of arthritis (not necessarily localized to the shoulder). Arthritis may have been present in a larger number of the sample, but may not have been diagnosed or been symptomatic until post-CVA. Other degenerative changes, including rotator cuff tears, may preexist in this population.

Arthritis or other degenerative changes in the sample with pain may have contributed to their pain.

One area of significance was found in comparing passive range of shoulder motion of those patients who experienced shoulder pain with those patients who did not experience shoulder pain, regardless of muscle tone. Passive range of shoulder motion was found to be greatly decreased in those patients with hemiplegic shoulder pain. One half of the patients with shoulder pain and ROM limitations demonstrated limitations in a capsular pattern, with primary limitations in external rotation. This is in agreement with an earlier study by Bohannon and Andrews (1989), who tested passive shoulder lateral (external) rotation ROM between the affected and nonaffected shoulders of hemiplegic patients. Bohannon, Larkin, Smith and Horton (1986), also found this relationship between shoulder pain and shoulder external rotation in their post-CVA population. Joynt, (1992), found that shoulder pain was significant in those patients with decreased shoulder ROM in all planes.

The relationship between shoulder pain and decreased ROM in our study may suggest the development of contractures, which, according to Akeson, (1987), start forming within two weeks of immobilization of an extremity. Akeson, (1987), however, conducted his research on animals, and cautioned against drawing conclusions concerning tissue changes in humans based on this information. Of the individuals studied, two developed pain two days post-CVA, nine developed pain seven days post-CVA, and seven developed pain 21 days post-CVA. Eight patients who complained of pain had experienced pain at some point prior to their stroke, five of whom had record of a previous CVA. Data about the side affected in the previous CVA was not collected, so no speculation can be made on the long term effects of a CVA on the affected UE.

Akeson's (1987) data may explain the development of pain in those patients with complaints 21 days post-CVA, but does not explain earlier development of pain. Another possible explanation for pain related to decreased ROM is mobilization of the affected

upper extremity. Mobilization may be active or passive, done by the patient or by a therapist, in attempts to prevent contracture development. As previously mentioned, normal scapulo-humeral rhythm is necessary for proper biomechanics of the joint. Abnormal rhythm or loss of external rotation of the humerus, which occurs normally with arm elevation may cause an impingement of soft tissue structures between the acromion and greater tuberosity of the head of the humerus. Trauma to soft tissue structures may occur with abnormal biomechanics during attempts at mobilization and this trauma could result in pain. Eighty-four percent of our subjects with shoulder pain did experience the pain with passive range of motion.

Our study also revealed a significant relationship between passive range of shoulder motion and hypertonicity, regardless of the presence of pain. When pain was disregarded, passive ROM of those patients with hypertonicity in their affected upper extremity was found to be significantly less than that of patients with hypotonicity. This finding suggests that changes in muscle with hypertonicity may contribute to decreased ROM. It is interesting to note that although limited ROM related to pain, and hypertonicity related to limited ROM, pain did not relate to hypertonicity.

No relationship was found between hypertonicity, subluxation and shoulder pain in this study. Ryerson and Levit, (1988), and Bobath, (1972), state that shoulder subluxation can be associated with increased muscle tone of the affected extremity's scapular musculature. Data concerning scapular muscle tone in isolation was not collected, as it was unavailable. Lack of data specific to the affected scapular musculature, as well as the small sample size studied may have influenced our findings.

Hypotonicity, subluxation and shoulder pain were not significantly related in our study. This is not in accordance with Tobis (1957), who described flaccid shoulder musculature and shoulder subluxation as the most common cause of shoulder pain in his experience. Findings do not agree with those of VanOuwenaller, et al., (1986), who found that anteroinferior subluxation was associated with pain A possible explanation for

this finding is the preventative treatment measures administered at Mary Free Bed Rehabilitation Center. Each documented case of shoulder subluxation was accompanied by orders for: (1) a lap board or arm trough on the patient's wheelchair, (2) the use of an arm sling during functional activities (i.e., transfers, ambulation), and (3) patient positioning in bed with a pillow under the affected upper extremity.

Another area of significance found when comparing patients who experienced shoulder pain with those who did not, regardless of muscle tone, was the association between pain and unilateral neglect. This finding is not in agreement with a previous study which found no relationship between unilateral neglect and pain (DeCourval, et al., 1990). Of note however, is that 20% of the patients in our study who exhibited unilateral neglect and experienced pain in their affected shoulder, may have experienced some sort of physical trauma following CVA (4 of 15 documented "possible CVA trauma"). Our findings may suggest that patients with unilateral neglect do not protect the affected upper extremity, leaving the shoulder highly susceptible to injury.

Another finding of significance was that impaired sensation was associated with pain when comparing patients with shoulder pain post-CVA to those without shoulder pain following CVA. This finding might be explained by possible trauma to the shoulder, as loss of proprioceptive input interferes with the patients ability to determine where their extremity is in space, and to protect the arm from injury. Thalamic modification of poorly localized incoming sensation might explain the sensation of pain reported by these patients. Carpenter (1991) states that stimuli that were previously not unpleasant may be perceived as painful, even though the threshold of excitability on the affected side is raised following a CVA.

In conclusion, ROM limitations of the affected shoulder were found to be significantly related to pain, and to hypertonicity, but not to pain with hypertonicity. Following the onset of pain, an increase in muscle tone was found to occur. A relationship was found between unilateral neglect and pain, and sensory disturbances and

pain. No significant relationship was found between pain and subluxation, either with hypertonic or hypotonic shoulder musculature.

Application to practice

Since this study found a significant relationship between shoulder pain and decreased ROM in the affected upper extremity, when taking care of individuals who have experienced a CVA, proper PROM should be performed to the affected UE to prevent contracture formation. Bohannon and Andrews (1989), stated the importance of maintaining functional ROM of the affected upper extremity in hemiplegic patients. Administering a home program of correct active assisted range of motion and positioning to the patient is essential. Educating the patient about proper biomechanics of the GHJ when administering a home program is important to decrease the chance of poor scapular mechanics that can cause impingement of the rotator cuff under the acromion process.(Bobath, 1992). Caldwell (1969) discusses a proper ordering of administering ROM to the patient in the clinic. First, ROM exercises need to be done proximally (scapula) to distally in the scapular plane (Jensen, 1980). This should be done in sitting. And finally, functional activities can be practiced.

Our findings of the relationship between shoulder pain and sensation impairment, including neglect, suggest the importance of making the individual with impaired sensation or unilateral neglect aware of his affected upper extremity. This may decrease the chance for post CVA trauma and thus shoulder pain. Instructing health care workers, as well as family members to approach the patient from the affected side will encourage attention to that side.

Inconsistent use of terminology made chart review difficult in this study. Consistent terminology, both within and between health care professions, is necessary for quality patient care. Rehabilitation facilities should develop standard terminology to be used by all health care professionals.

Limitations

The limitations of this study are as expected from a retrospective chart review. Medical documentation provides for collection of data, but in turn limits availability of certain information. Inconsistency was found between medical professionals with the use of medical terminology, and added difficulty to this chart review. Our data is only as accurate as the documentation in the charts.

Reliability of patient history is also questionable, as communication deficits are common following stroke. A number of patients suffer from aphasia following a CVA, affecting their ability to communicate whether they experience pain or not. Documentation indicated that hospital staff questioned patients as to complaints, attended to facial grimacing and outcries, and evidence of agitation. In some instances, communication boards were provided. Communication difficulties however, prevent definitive conclusions from being drawn as to whether these patients experienced pain or not, as reports often depend on subjectivity of the health care provider. Our conclusions are also limited to the two institutions which we used, and cannot be generalized to the entire hemiplegic population.

The percentage of individuals who suffer a stroke and then experience shoulder pain may actually be much greater than the twenty-three percent found in this study. The sample studied here had a very good prognosis, since they were rehabilitation candidates. The results of this study could very well be affected by such a sample, and cannot be generalized throughout the entire post-CVA population.

Fifty-percent of those individuals in this study who suffered a previous CVA also suffered from shoulder pain. Data on the previous side affected was not collected in this study. Thus, this omission in our data collecting limits us in speculating about any long term affects of CVAs.

Suggestions for further research/modifications

A prospective, longitudinal study, is recommended, with a limited number of researcher/therapists, to control interrater reliability of measurements and documentation. Studies should follow patients from date of onset of CVA, to discharge and into outpatient therapy or long-term care facilities, and should include patients from several different institutions, with a range of prognoses. This would lead to a better representation of the total post-CVA population and their outcome, and thus lead to results and conclusions that would also better represent this entire population.

Future studies should include data about treatment techniques, upper extremity function and the presence of edema in the affected upper extremity. Treatments might provide an indication as to which, if any, may prevent, produce, or eliminate pain in the hemiplegic shoulder. The presence of a previous CVA, and side affected should be taken into consideration. The individual may be suffering from previous CVA-related shoulder pain.

Patients' communication skills should be evaluated and controlled for so that accurate data could be collected. To exclude all those with aphasia may limit the researchers to a very small sample, or increase the time that the study takes to complete.

More research in needed to find the cause of shoulder pain. Shoulder pain was found to be related to a change in muscle tone (increase), possibly resulting in ROM limitations and other secondary complications that may leave the individual's arm non-functional.

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TABLES

Table 1

The Change in Muscle Tone Following the Onset of Pain

Tone Prior to Pain	Tone Following Onset of Pain	Difference in Tone	Signed Rank	P-Value
3	3	0		
1	1	0		
0	0	0		
3	3	0		
2	2	0		
0	1	+ 1	+ 2	< 0.01**
0	2	+ 2	+ 4.5	
1	1	0		
1	1	0		
1	3	+ 2	+ 4.5	
1	1	0		
0	3	+ 3	+ 8	
1	3	+ 2	+ 4.5	
1	3	+ 2	+ 4.5	
0	0	0		
3	2	- 1	- 1	
0	3	+ 3	+ 8	
2	2	0		
3	3	0		
3	3	0		
0	3	+ 3	+ 8	
3	3	0		

* Refer to the text for the definition of the muscle tone represented by ordinals.
+ Tone increased, - Tone decreased
**Significant
$\alpha = 0.05$

Table 2

The Relationship Between Shoulder Pain and Muscle Tone at Discharge

	Muscle Tone				N	P-Value
	Hypertonic	Hypotonic	Flaccid	Normal		
Pain	9	6	1	3	19	0.80
No Pain	13	6	7	41	67	
					Total = 86	

$\alpha = 0.05$

Table 3

The Relationship of Shoulder Pain and Muscle Tone to Secondary Impairments

	Muscle Tone of Painful Shoulders		N	P-Value
Secondary Impairments	Hypertonic	Hypotonic/Flaccid		
Subluxation				
Yes	1	4	13	0.09
No	6	2		
Neglect				
Yes	7	5	15	0.66
No	2	1		
Shoulder ROM				
Normal	0	0		
Minimal Decrease	3	3	16	> 0.20
Moderate Decrease	6	4		
Maximum Decrease	0	0		
Sensation				
Hypersensitive	0	0		
Normal	4	3	16	> 0.20
Impaired	4	2		
Absent	1	2		

$\alpha = 0.05$

Table 4

The Relationship of Muscle Tone to Secondary Impairments

Secondary Impairments	Muscle Tone: Hypertonic	Muscle Tone: Hypotonic/Flaccid	N	P-Value
Subluxation				
Yes	6	9	46	> 0.25
No	17	14		
Neglect				
Yes	19	16	51	> 0.75
No	8	8		
Shoulder ROM				
Normal	0	4	54	0.008 **
Minimum Decrease	17	19		
Moderate Decrease	9	4		
Maximum Decrease	1	0		
Sensation				
Hypersensitive	0	0	54	0.15
Normal	15	12		
Impaired	10	10		
Absent	2	5		

** Significant

$\alpha = 0.05$, df = 1

Table 5

The Relationship of the Presence or Absence of Pain to Secondary Impairments

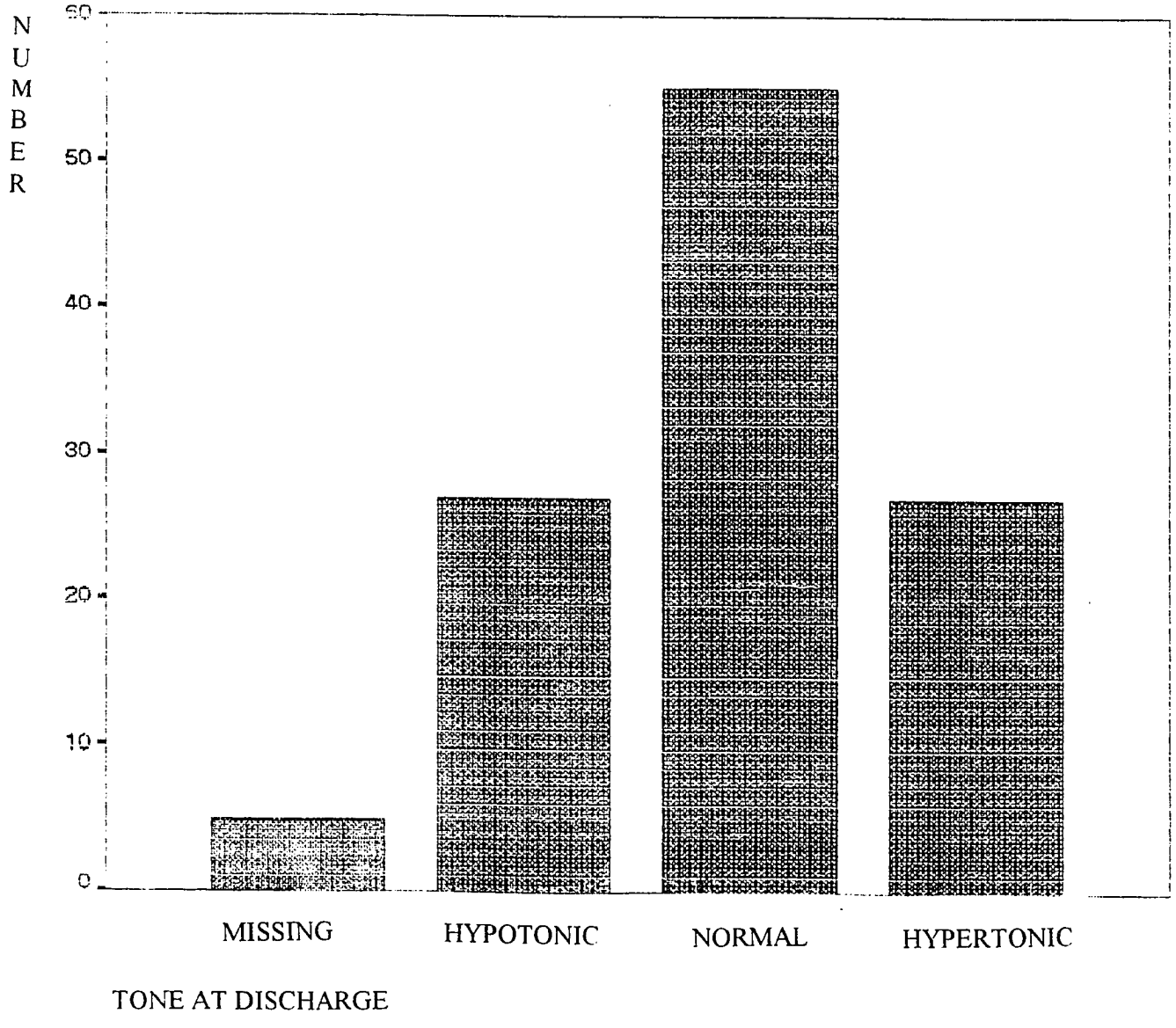
Secondary Impairments	Pain	No Pain	N	P-Value
Subluxation				
Yes	5	10	79	> 0.10
No	10	54		
Neglect				
Yes	11	24	84	< 0.05 **
No	6	43		
Shoulder ROM				
Normal	0	8	86	0.00004 **
Minimum Decrease	8	57		
Moderate Decrease	11	1		
Maximum Decrease	0	1		
Sensation				
Normal	8	46	85	0.008 **
Impaired	8	18		
Absent	3	2		
Hypersensitive	0	0		

** Significant
$\alpha = 0.05$, df = 1

FIGURES

Figure

The Discharge Muscle Tone of the Affected Upper Extremity



APPENDICES

APPENDIX A "Pain"

I.D. # _____ N.D. = Not Documented
AGE: ___
GENDER:___
DATE OF CVA:___/___/___
UE SIDE AFFECTED: R___ L___
HAND DOMINANCE: R___ L___
PMH:
Previous CVA Y___ N___ No Mention ___
Post CVA Trauma Y___ N___ No Mention ___
Previous Shoulder Pain Y___ N___ No Mention ___
OTHER DX: _____
_____

DATE OF ONSET OF PAIN: ___/___/___
LOCATION:
Shoulder: Y___ N___
Radiating: Y___ N___
Other: _____
CHARACTERISTICS: _____
SEVERITY: _____
PAIN WITH MOTION:
AROM: Y ___ N ___
PROM: Y ___ N ___
PAIN AT REST: Y ___ N ___
OF DAYS OF REPORTED PAIN THE 1ST WK. OF PAIN: _____

TONE OF AFFECTED UE:
INITIAL EVAL @ ST. MARY'S:

	Position Unknown	Seated	Supine	Standing
Flaccid	___	_____	_____	_____
Hypo	___	_____	_____	_____
Hyper	___	_____	_____	_____
Normal	___	_____	_____	_____

PRIOR TO ONSET OF PAIN AS AVAILABLE:

	Position Unknown	Seated	Supine	Standing
Flaccid	___	_____	_____	_____
Hypo	___	_____	_____	_____
Hyper	___	_____	_____	_____
Normal	___	_____	_____	_____

FOLLOWING ONSET OF PAIN AS AVAILABLE:

	Positon Unknown	Seated	Supine	Standing
Flaccid	___	_____	_____	_____
Hypo	___	_____	_____	_____
Hyper	___	_____	_____	_____
Normal	___	_____	_____	_____

REFLEXES:
Biceps DTR: Normal ___ Increased ___ Decreased ___
Triceps DTR: ___ ___ ___
SHOULDER SUBLUXATION:
N___ Y ___ 1 finger ___
2 finger ___
3 finger ___

APPENDIX A "PAIN" - PAGE 2

I. D. # _____

PROM SHOULDER:	AFFECTED		UNAFFECTED		
AFF/UAFF					
	DEGREE	pain w/PROM	DEGREE	pain w/PROM	%
ROM					
ER	___	___	ER ___	___	___
IR	___	___	IR ___	___	___
FLEX	___	___	FLEX ___	___	___
ABD	___	___	ABD ___	___	___
EXT	___	___	EXT ___	___	___

SENSATION: (CIRCLE OVERALL)

	Normal	Impaired	Absent	Increased
Light touch	___	___	___	___
Pin Prick/dull	___	___	___	___
Proprioception	___	___	___	___
Kinesthesia	___	___	___	___
Deep Pressure	___	___	___	___
Temperature:				
Hot	___	___	___	___
Cold	___	___	___	___

NEGLECT: Y___ N___ L___ R___

PAIN MEDICATIONS: **DATE OF PRESCRIPTION:**

1. _____ _____
2. _____ _____
3. _____ _____
4. _____ _____
5. _____ _____
6. _____ _____

REASON FOR PRESCRIPTION:

1. _____
2. _____
3. _____
4. _____
5. _____
6. _____

APPENDIX B "Discharge"

I.D. # _____ **N.D. = Not Documented**
AGE: ___
GENDER:___
DATE OF CVA:___/___/___
UE SIDE AFFECTED: R___ L___
HAND DOMINANCE: R___ L___
PMH:
Previous CVA Y___ N___ No Mention ___
Post CVA Trauma Y___ N___ No Mention ___
Previous Shoulder Pain Y___ N___ No Mention ___
OTHER DX: _____
_____

PAIN: Y___ N___
DATE OF ONSET OF PAIN: ___/___/___
DATE OF RESOLUTION OF PAIN: ___/___/___
DURATION OF PAIN: _____
TONE OF AFFECTED UE:
INITIAL EVAL @ ST. MARY'S:

	Position Unknown	Seated	Supine	Standing
Flaccid	___	_____	_____	_____
Hypo	___	_____	_____	_____
Hyper	___	_____	_____	_____
Normal	___	_____	_____	_____

AT DISCHARGE:

	Position Unknown	Seated	Supine	Standing
Flaccid	___	_____	_____	_____
Hypo	___	_____	_____	_____
Hyper	___	_____	_____	_____
Normal	___	_____	_____	_____

REFLEXES:
Biceps DTR: Normal ___ Increased ___ Decreased ___
Triceps DTR: ___ ___ ___
SHOULDER SUBLUXATION:
N___ Y___ 1 finger ___
2 finger ___
3 finger ___

PROM SHOULDER:	AFFECTED DEGREE	AFFECTED pain w/PROM	UNAFFECTED DEGREE	UNAFFECTED pain w/PROM	AFF/UAFF % ROM
ER	___	___	ER ___	___	___
IR	___	___	IR ___	___	___
FLEX	___	___	FLEX ___	___	___
ABD	___	___	ABD ___	___	___
EXT	___	___	EXT ___	___	___

APPENDIX B "DISCHARGE" - PAGE 2

I.D. # _____

SENSATION: (CIRCLE OVERALL)

	Normal	Impaired	Absent	Increased
Light touch	___	___	___	___
Pin Prick/dull	___	___	___	___
Proprioception	___	___	___	___
Kinesthesia	___	___	___	___
Deep Pressure	___	___	___	___
Temperature:				
Hot	___	___	___	___
Cold	___	___	___	___

NEGLECT: Y___ N___ L___ R___

AT D/C: TONE CHANGE FROM INITIAL EVAL:

Y___ N___

Same ___ Increased ___ Decreased ___

AT D/C: TONE CHANGE FROM THAT FOLLOWING THE ONSET OF PAIN:

Y___ N___

Same ___ Increased ___ Decreased ___

APPENDIX C - (PAGE 1)

A List of Drugs for Pain Management

Compiled from **PHYSICAL AND OCCUPATIONAL THERAPY: Drug Implications for Practice.** By Terry Malone. J.B. Lippincott Co.; Philadelphia 1992.

DRUG	TRADE NAME
NONNARCOTIC ANALGESICS	
Acetaminaphen	Anacin - 3, Tempra, Tylenol, Valadol, Phenaphen, Datril
Asprin (Acetylsalicylate)	Ascripin, Bufferin, Emperin, Ecotrin, Encaprin, Zopren
Choline salicylate	Arthropan
Magnesium salicylate	Magan
Salicylate combination	Trilisate
Diflunisal	Dolobid
NON - STEROIDAL ANTIINFLAMMATORY AGENTS - NSAIDS	
Aspirin	Bufferin, Empirin
Suprofin	Suprol
Fenoprofen	Nalfon
Ibuprofen	Motrin, Rufen, Nuprin
Naproxen	Naprosyn, Anapox
Sulindae	Clinoril
Indomethacin	Indocin
Tolmetin	Tolectin
Piroxicam	Feldene
Meclofenamate	Meclomen
Mecfenamic acid	Ponstel
Diflunisal	Dolobid
STERIOD - CORTICOSTEROIDS	
Cortisone	Cortef
Hydrocortisone	Cortef
Prednisolone	Delta - Cortef
Prednisone	Deltasone, Orasone
Methylprednisone	Medrol
Triamcinolone	Aristocort, Kenacort
Paramethasone	Haldrone
Dexamethasone	Decadron
Betamethasone	Celestone
LOCAL ANETHETICS	
Lidocaine (Xylocaine)	
Mepivacaine (Carbocaine)	
Prilocaine (Citanest)	
Phenol Injections	

APPENDIX C - (PAGE 2)

DRUG	TRADE NAME
NARCOTIC ANALGESICS	
Meperidine	Demerol
Hydromorphone	Dilaudid
Oxymorphone	Numorphan
Methadone	Dolophine
Codeine	
Morphine	
Butophanol	Stadol
Pentazone	Talwin
Nalbuphine	Nubain
COMPOUND ANALGESICS AGENTS	
Tylenol #1-4	Acaetaminophen + Codeine
Empirin #1-4	Aspirin + Codeine
Percodan	Aspirin + Oxycodone
Tylox	Acetaminophen + Codeine
Darvon Compound	Aspirin, Caffeine, Propoxyphene
Darvocet	Acetaminophen, Propoxyphene