Shoulder Pain in Hemiplegia

(brachial plexus injury, diagnosis, occupational therapy, shoulder dislocation)

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The recovery of a patient following cerebrovascular accident (CVA) is affected by the extent of brain damage and the development of complications. Development of a painful shoulder is one serious complication that can interfere with the patient's entire rehabilitation program, including ambulation training and self-care activities (1). A painful shoulder can limit the patient's general mobility, because when a patient protects his or her arm, this restricts both active and passive movements. This limits the patient's activities, such as rolling in bed, transferring, putting on a shirt or blouse, and bending to reach his or her feet to put on shoes and socks. The incidence of painful shoulders in patients with hemiplegia can be quite high. One survey found the incidence to be as high as 70% (1). Another study found that 43% of the patients developed shoulder pain, regardless of the amount of therapy given (2).

Shoulder pain in the hemiplegic patient is a perplexing problem. Some of the suggested etiologies of shoulder pain include immobilization of the upper extremity, trauma to the joint structures, and subluxation of the gleno-humeral joint. However, shoulder pain often develops from a combination of these problems. To treat the patient effectively, the occupational therapist must understand the basic anatomy and kinesiology of the shoulder complex, the potential causes of shoulder pain, and the pros and cons of various techniques used to prevent and treat shoulder pain.

Immobilization of the Upper Extremity

Immobilization of the upper extremity can occur in the involved side of the body following CVA. This immobilization can be due to decreased active movement and/or spasticity, both of which can limit passive and active movement. This immobilization can impair circulation in the upper extremity and affect the autonomic nervous system, which may result in a reflex sympathetic dystrophy or shoulder-hand syndrome.

The shoulder-hand syndrome is characterized by a deep, burning pain, trophic changes, such as changes in skin temperature and color, limitation of movement, and edema (3). The swelling of the hand is caused by a combination of edema and thickening of the soft tissues of the hand (4), particularly around the joints (5). The symptoms may vary from mild to severe

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and may last from weeks to years (3).

Cailliet (6, p 114) suggested three stages in the shoulder-hand syndrome. In the first stage, the following occur: (a) a limited range of motion (ROM) in the shoulder, (b) swelling of the dorsum of the hand, (c) loss of normal wrinkles in the skin (skin becomes shiny in appearance), (d) a limited ROM in finger flexion, (e) hypersensitivity to touch, pressure, or variations in movement, and (f) pain on wrist extension.

In the second stage, the following occur: (a) shoulder pain subsides and ROM increases, (b) edema decreases, but fingers become stiff, (c) sensitivity decreases, and (d) osteoporosis is seen on X-rays.

In the third stage, the following occur: (a) progressive atrophy of the bone, skin, and muscles, and (b) an increase in limitation of movement in the hand, wrist, and fingers (leaving the hand painless but in a useless clawed position). Cailliet (6, p 115) estimated that 65% of patients who develop shoulder-hand syndrome initially show symptoms one to three months after stroke.

Moberg (7) describes the evolution of the shoulder-hand syndrome through the theory of the “shoulder-hand pump.” In this pump, the arterial circulation of the upper extremity occurs through cardiac pumping action and is assisted by gravity. The venous return is dependent on a valve system and is assisted by the extrinsic influence of muscle and hand function, which simulate a pumping action. The venous return is also aided by repeated elevation of the arm above cardiac level. Two of the major pumps in the upper extremity are located in the axilla and in the hand. Repeated movement of the shoulder and hand through an adequate range is needed to activate these pumps. In the hand specifically, most of the arterial supply is located in the volar aspect, whereas most of the venous drainage is located in the dorsal aspect; thus, if the pumps fail, edema is often seen over the dorsal aspect of the hand. A failure of the pumps can lead to changes in joint structures, atrophy, osteoporosis, contractures, pain, and possibly a reflex sympathetic dystrophy or shoulder-hand syndrome.

Moberg (7) also explains the cyclical effect of the syndrome. Limited movement of the shoulder will cause adhesions to develop in the shoulder joint in addition to causing the hand to become edematous, because one of the pumps is impaired. Edema in the hand will limit movement, particularly finger flexion. This will cause shortening of the collateral ligaments at the metacarpophalangeal and interphalangeal joints, further limiting movement and impairing circulation. The limitation of two of the major pumps with joint changes and the resulting pain starts the cycle over. A frozen shoulder and hand is the end result.

Moberg’s (7) theory offers one explanation for a condition that is poorly understood. Sympathetic nervous system involvement in the shoulder-hand syndrome has not been fully explained. As a division of the autonomic nervous system, part of the sympathetic nervous system is in control of vasomotor (or circulatory) responses and sudomotor (or sweat gland) responses. In the shoulder-hand syndrome, it is theorized that there are afferent pain fibers among the primarily efferent sympathetic fibers that set off a sympathetic vasomotor reaction when they are stimulated by internal or external pain (8, p 110). Some researchers suggest that there is a central neural mechanism that may facilitate the start of a shoulder-hand syndrome (5, 9). Pender (10) states that there is a psychological component in shoulder-hand syndrome that may hinder recovery.

Included in the reflex sympathetic dystrophies is the thalamic pain syndrome (8, p 109). This syndrome occurs during recovery from a thalamic infarct and is characterized by a burning pain and hypersensitivity (11, p 20; 12, p 182). Appreciation of pain in the thalamus is strongly influenced by an emotional component (13, p 26); this perhaps partially explains the psychological component of reflex sympathetic dystrophy.

A variety of approaches have been used by physicians to treat patients with shoulder-hand syndrome, including sympathectomies (14), stellate ganglion blocks, corticosteroids, and local injections (5, 15, 16). Physicians also frequently prescribe therapy, including passive and active ROM, and modalities, such as heat (4, 15) and ultrasound (17). The use of heat and paraffin baths are based on the principle of overloading of sensory systems (15). DeTakats (16) disagreed with this approach, stressing that heat is aggravating in the initial vasodilator phase when there is increased blood flow to the hand. He suggested the use of cold packs for relief for the hot, painful hand.

Occupational therapy treatment objectives for shoulder-hand syndrome are (a) prevention and (b) early recognition and treatment. To counteract the effects of immobilization and to aid in the prevention of shoulder-hand syndrome, passive ROM, self-range of motion, and active ROM activities should be done several times a day, especially activities that involve
movement at the shoulder and hand and movements that elevate the arm above cardiac level. Selection of activities that will encourage upper extremity mobilization is important to prevent further disability and dependence. If the patient demonstrates little or no active movement in the arm, bilateral activities should be used. An example of this is to have the patient hold a washcloth between clasped hands and use both upper extremities for bathing activities. If the patient demonstrates any active movement in the upper extremity, he or she should be encouraged to use this movement in functional activities to promote further mobilization of the extremity.

Spasticity is sometimes another clinical manifestation of hemiplegia that can impair passive and active movement of the extremity. If spasticity is present, efforts should focus on normalizing tone in the extremity and encouraging proper ROM. This is discussed in more detail later.

Edema is often a side effect of upper extremity immobilization. To reduce edema, the hand can be massaged (distally to proximally) to help force fluid from the hand. The patient should not allow the arm to hang dependently by his or her side for long periods of time, because this can foster development of an edematous hand. Placing the arm on a lapboard or an elevated armrest while the patient is sitting helps reduce edema. To prevent the hand from hanging dependently when the patient is standing, a sling may be considered. It is important for the occupational therapist to help the patient properly position the edematous hand during functional activities, such as wheelchair mobility, self-care activities, and tabletop activities. The therapist should keep in mind that prolonged static positioning should be avoided and that any positioning should be supplemented with ROM activities to prevent immobilization. The patient should also be encouraged to participate in functional activities to prevent further disability and dependency.

**Trauma to the Joint Structures**

Trauma to the joint structures of the shoulder has been suggested as another etiology of pain. Impingement of joint structures can easily occur in the hemiplegic patient during ROM activities, because the normal scapulo-humeral rhythm may be impaired by spastic depressors of the shoulder girdle. One study of 32 patients with hemiplegia has shown that 40% had rotator cuff injuries and that 10 of 11 patients who had severe shoulder pain also had rotator cuff injuries. This may have been because of impingement of the rotator cuff during ROM activities.

To show how impingement of joint structures can occur, the basic anatomy and kinesiology of the shoulder joint must be examined. Figure 1 shows a front view of the gleno-humeral articulation. Kent describes some of the basic anatomic structures. The head of the humerus articulates with the glenoid fossa and is supported by capsular ligaments and the rotator...
cuff muscles. The coraco-humeral ligament also helps to support the joint, because it attaches from the coracoid process to the greater tubercle. The coraco-clavicular ligament binds the clavicle and scapula so that a constant relationship is maintained between the two during movement. The acromion process acts like a hood to protect the head of the humerus. The coraco-acromial ligament also forms a roof over the head of the humerus and separates the subacromial bursae from the acromio-clavicular joint. This ligament has a sharp edge and may cause impingement of the subacromial bursae or supraspinatus tendon when the arm is elevated above shoulder level.

To achieve full forward flexion or full abduction of the shoulder during passive or active ROM of the upper extremity, the humerus must be externally rotated to move the greater tubercle out of the path of the acromion as the arm elevates. Upward rotation of the scapula is also needed. This is explained using the concept of scapulo-humeral rhythm, which states that for every 15° of elevation of the arm, 5° results from movement of the scapula rotating upwards and 10° results from movement at the gleno-humeral joint (see Figure 2). It is a two-to-one ratio. In full 180° of elevation of the arm, 120% of movement is at the gleno-humeral joint, and 60% is from upward rotation of the scapula (6, p 43). If the scapula does not rotate upwards, only 120% of elevation of the arm can be achieved, at which point the humerus is blocked by the acromion, thus causing impingement of the bursae, biceps tendon, or rotator cuff. This impingement can cause inflammation of these structures and result in shoulder pain. Because of the close proximity of these structures, inflammation of one structure generally leads to inflammation of the surrounding structures (8, p 48). It is important to note that among the surrounding structures is the subdeltoid fascia, which is well supplied with blood vessels and sympathetic nerves (8, p 49). Inflammation of this structure may set off a sympathetic vasomotor reaction and result in a shoulder-hand syndrome.

Trauma to the hemiplegic shoulder can also occur from improper handling of the affected upper extremity. This can occur when too much traction is placed on the arm when transferring the patient or when the arm is twisted during an abnormal movement, such as catching the arm behind the back when transferring or rolling in bed. Trauma can also occur from improper positioning of the upper extremity.

Bobath (21) suggests treatment techniques to prevent and treat shoulder pain, including ROM activities, proper handling, and upper extremity positioning (as explained by L. LaPitz and J. Davis in a course on Neurodevelopmental Treatment for Adult Hemiplegia, Pittsburgh, PA, May 1982). When performing passive ROM, the therapist should ensure proper scapulo-
humeral rhythm to prevent impingement and possible shoulder pain. The scapula should be relaxed and mobile prior to elevation of the arm, and the therapist should passively assist upward rotation of the scapula (21, p 98).

When performing active ROM, the patient should avoid the combined motion of scapular retraction with forward flexion of the arm, because this can cause impingement from improper scapulo-humeral rhythm. Attaining proper scapulo-humeral rhythm can be assisted by treatment media positioning and activity grading. The therapist should choose activities that encourage and assist the patient with protraction and upward rotation of the scapula. In activities requiring the patient to reach for or place objects, the placement of materials in front of the patient at or below waist level encourages and assists scapular protraction and forward flexion of the shoulder. The therapist can also guide the proper movement as necessary. As the patient improves, the height of the activity is gradually raised.

Activities that encourage scapular protraction are recommended for self-range of motion. These activities tend to decrease spasticity and assist in upward rotation of the scapula. Two such activities follow. With the patient sitting in a chair, have him or her clasp his or her hands and then lean forward to reach for the floor. With the patient sitting at a table, have him or her clasp hands and push his or her arms forward on the table. It is easy to see the increase in passive shoulder movement that occurs when doing these activities. An advantage of these activities is that the patient monitors his own pain threshold and is therefore less apprehensive about mobilizing the upper extremity.

To ensure proper handling and protection of the upper extremity when helping the patient to transfer or move in bed, the person assisting should place his or her hand on the scapula of the hemiplegic arm rather than holding onto the patient's arm. This action protects the patient's arm and gives the helper better control in assisting the patient. When the patient rolls to either side in bed, the helper can assist by keeping the scapula protracted while watching that the patient does not improperly position the arm. The patient can also be taught proper protection techniques, such as hand clasping, full arm extension, and shoulder girdle protraction (to prepare for rolling from side to side in bed).

When the helper positions the upper extremity of a patient in bed, the scapula should be fully protracted and the arm properly supported. These precautions help position the hemiplegic arm in a reflex-inhibiting position to prevent or decrease spasticity. Again, this helps maintain proper scapulo-humeral rhythm and prevent possible shoulder pain. When the patient is supine, a pillow is placed behind the scapula to protract it, the arm is slightly abducted, the elbow is extended, and the forearm is supinated. When the patient is positioned on the unaffected side, the scapula is fully protracted, the shoulder is flexed above 90%, the elbow is extended, and the arm is supported with pillows. When the patient is positioned on the affected side, the scapula is fully protracted, the shoulder is flexed above 90%, and the elbow is extended. When a patient is in this position and the scapula is winging in back, it indicates that the scapula is not fully protracted; thus, pain may develop because the patient is lying on his or her arm and compressing the humerus into the joint. When sitting, the patient should be encouraged to position his or her arm on a table with the shoulder girdle fully protracted and his or her elbow well up on the table for proper support. The therapist may recommend a lapboard to position the arm when the patient is not at a table.

For continuity, it is extremely important to educate others who will be assisting the patient, such as nurses, aides, family members, in proper positioning and handling techniques, because everyone who assists the patient has the responsibility of caring for the hemiplegic shoulder.

Brachial Plexus Injury

Another possible cause of shoulder pain in hemiplegic patients is trauma to brachial plexus and peripheral nerve lesions (22). Differential diagnosis of this lower motor neuron disorder has been based on physical examination; signs include the presence of segmental atrophy (with or without associated sensory deficits), atypical return of function in the upper extremity (or distal recovery before proximal recovery), the delayed onset of spasticity, and the presence of abnormal electromyographic (EMG) findings (22, 23).

The positive identification of brachial plexus injuries is difficult because it is often masked by the symptoms of a CVA, such as flaccidity and sensory deficits (22, 23). Investigators disagree as to the cause of abnormal EMG findings in patients who have suffered a stroke. One study concluded that abnormal EMG evidence is not found in upper motor neuron disorders unless there is a complicating peripheral nerve injury or disease (24). Other research has found that abnormal EMGs may be
caused by an upper motor neuron lesion (25, 26). Another study found that atrophy of muscles in the hemiplegic patient, sometimes associated with lower motor neuron disorders, may be the result of a central or trophic influence (27). Bhala (25) discounts traction injury to the brachial plexus following prolonged subluxation of the shoulder as a cause of abnormal EMGs. He stated that in patients he studied, abnormal EMGs were diffuse and were found in both the upper and lower extremities of the impaired side. Other researchers have found abnormal EMGs specific to the muscles innervated by the upper trunk of the brachial plexus and consistent with atrophy of the shoulder girdle musculature (23). Regardless, those who believe that brachial plexus injuries are a potential problem attribute these injuries primarily to improper upper extremity positioning and handling (23, 28). Therefore, the positioning and handling techniques just discussed should assist in the prevention and treatment of this potential problem.

Subluxation of the Gleno-Humeral Joint

Another source of shoulder pain in the hemiplegic patient may be the subluxation of the gleno-humeral joint. Some investigators believe that when the arm hangs dependently, the gleno-humeral joint is malaligned (19, 28). This puts a constant stretch on the joint capsule, impairing circulation and possibly damaging joint structures.

Basmajian and Bazant (29) offered a theory to explain the development of subluxations. To explain this theory, it is necessary to have basic knowledge about the structures that hold the humerus in joint. In the gleno-humeral joint, stability has been sacrificed for mobility. As the glenoid fossa is only about one-third the size of the head of the humerus, the stability of the joint is dependent on muscles, tendons, and the joint capsule (20). Basmajian and Bazant (29) found that the head of the humerus is held in joint by the following: (a) the slope of the glenoid fossa, (b) the tightening of the superior portion of the joint capsule and the supraspinatus muscle. When the arm is fully adducted, the superior portion of the joint capsule is taut and the inferior portion is slack. When the arm is abducted, the superior portion of the capsule becomes slack (see Figure 3). When the humerus is pulled downward, the slope of the glenoid fossa forces the head of the humerus laterally. This lateral movement is prevented by the tightening of the su-

![Figure 3](image-url)

Capsular action during gleno-humeral movement

The superior portion of the joint capsule, the coraco-humeral ligament, and the horizontally running fibers of the supraspinatus muscle. Basmajian (30) found that in many people, the downward dislocation of the adducted arm is prevented mainly by the tightening of the superior portion of the capsule and is not dependent on any muscle activity, including that of the supraspinatus or those muscles with vertically running fibers, such as the deltoid, the biceps, and the long head of the triceps. However, when the arm is even slightly abducted, the superior portion of the capsule becomes slack and the stability of the joint becomes dependent on muscle activity of the rotator cuff muscles (30). If there is no muscle activity, the humerus may sublux, because it takes up the slack in the superior portion of the capsule.

If the scapula is downwardly rotated, the humerus is essentially abduced in relation to the scapula (see Figure 4) and thus is susceptible to subluxation. In the hemiplegic patient, the scapula is sometimes rotated downward either because paretic muscles are unable to maintain the neutral position of the scapula or because spastic muscles depress and rotate the scapula downward. The humerus can also be in a slightly abducted position if the patient's trunk is laterally flexed to the impaired side (see Figure 5). Cailliet (6, p 65) also suggests other causes of subluxation, such as paretic rotator cuff muscles and spastic depressors of the humerus.

The use of slings in the treatment of shoulder subluxation in the hemiplegic patient is controversial. Slings are frequently used to realign the gleno-humeral joint, thus minimizing the subluxation. The support provided by slings prevents damage to the joint structures. However, not all researchers advocate the use of slings. Friedland (31, p 247) states that it is not necessary to support a pain-free shoulder in this way because a sling will not prevent or correct a subluxation. One study (32) found no appreciable difference in shoulder ROM, subluxation, or shoulder pain in those patients who wore slings and those who did not. Bobath (21, p 106) questions whether slings properly position the arm and the gleno-humeral joint. She feels flexor spasticity is the main cause of subluxation and is reinforced when the patient wears a regular hemi-sling. Bobath used a sling with the foam roll under the axilla; however, she states this may tend to displace the head of the humerus laterally. Slings have also been identified as a contributing factor in shoulder-hand syndrome (33).

The questions of whether or not to use a sling and what type of sling to use should be given careful consideration. The therapist might ask the following questions.

- Will the sling be easy for the patient, family, or significant other to put on and take off?
- What is the patient's cognitive status? Does he or she neglect the...
Impaired side? Can he or she learn to protect the arm? If not, a sling may be indicated.

- Is the patient’s arm particularly flaccid or edematous so that he or she would need support during upright activities to prevent the arm from hanging dependently? If so, a hemi-sling may be indicated during upright activities.

- Will the sling immobilize the arm in a static position for prolonged periods and encourage contractures? If used for prolonged periods, a hemi-sling may encourage the development of spasticity and contractures, because it positions the arm in an adducted, internally rotated, elbow-flexed position.

- Will the sling impair circulation? The Bobath-type sling and the cuff on the Hook hemi-harness sling need to fit very tightly to provide the proper support. For patients with heavy arms, this arrangement may impair circulation in the arm.

- Will the sling prevent the patient from using any active movement he has in the upper extremity? The Bobath sling and the Hook hemi-harness will allow active movement; the regular hemi-sling will limit movement.

- Can proper positioning and handling techniques be substituted for using a sling? Proper positioning when the patient is in bed or sitting can prevent the arm from hanging dependently for prolonged periods. Proper handling can prevent trauma to the vulnerable joint structures.

Summary

Shoulder pain in hemiplegia remains a complicated problem. Shoulder pain may develop from a variety of etiologies, such as immobilization of the upper extremity, trauma to the joint structures (including brachial plexus injuries), and subluxation of the gleno-humeral joint. It is important for the occupational therapist to understand the basic processes that may contribute to shoulder pain so that he or she can thoroughly evaluate the patient and treatment approach for more effective management of this problem. In reviewing the literature, it appears that most shoulder pain in hemiplegic patients develops as a result of impaired circulation caused by immobilization or from trauma to the joint structures caused by improper ROM activities, positioning, or handling of the extremity. Therefore, in order to prevent and alleviate shoulder pain, efforts should be directed toward initiating activities that promote proper ROM and also proper positioning and handling of the upper extremity; this would aid circulation and prevent impingement of joint structures. The absence of pain will facilitate the patient’s participation in his or her rehabilitation program allowing the attainment of maximum functional potential.
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REFERENCES


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