Hemiplegic Shoulder Pain

An Approach to Diagnosis and Management

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KEYWORDS

• Hemiplegic shoulder pain • Poststroke shoulder pain • Stroke rehabilitation
• Shoulder subluxation

KEY POINTS

- Hemiplegic shoulder pain (HSP) occurs in most patients with hemiplegia, and has an adverse effect on functional outcomes.
- Evaluation and management is challenging, as HSP remains a clinical diagnosis, and many of the available treatments for HSP lack sufficient or robust support in the medical literature.
- The pathogenesis of HSP is multifactorial and includes neurologic and mechanical factors, often in combination, which vary among those affected.
- The systematic approach discussed in this article is intended help practitioners to accurately identify the factors contributing to each patient's pain, and to prescribe the most effective treatment based on the available evidence.

INTRODUCTION

Stroke, or cerebrovascular accident, is the third leading cause of death and the leading cause of adult long-term disability in the United States. Impairments from stroke vary widely, but one of the most common is hemiplegic shoulder pain (HSP). Pain and loss of function in the upper limb is a significant detriment to quality of life. HSP is a challenge to patients and their health care providers, as it reduces participation in rehabilitation, discourages motion, hinders recovery, and adversely affects function. The causes of HSP are multifactorial, have neurologic and mechanical causes, and can be generated peripherally in the limb or centrally within the brain.

Although HSP has been recognized and discussed in the medical community for decades, the evidence in the medical literature lacks sufficient quantity and quality, and

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is inconsistent in its conclusions. It can be confusing to manage HSP when each of its components has its own controversies in treatment. For example, even if adhesive capsulitis is identified as a contributor to HSP, debate remains regarding the best treatment practice for adhesive capsulitis itself. The purpose of this article is to assist the reader in developing a strategy for the management of HSP. No patient is exactly the same, so a one-size-fits-all treatment is unlikely to be effective. Instead, the focus should be on a consistent approach to ensure that all components of the diagnosis are addressed appropriately.

SCOPE AND SIGNIFICANCE

Every year in the United States 795,000 people suffer a new or recurrent stroke: 1 stroke every 40 seconds. More than 7 million Americans older than 20 years have had a stroke. Stroke is the third leading cause of death and the leading cause of long-term disability, costing the United States $18.8 billion annually, and with a lifetime cost of $140,000 per patient with ischemic stroke.[1] Of those who survive a stroke, approximately half have hemiplegia. Although 70% of those with hemiplegia will achieve ambulatory status, half are left with a nonfunctional arm.[2] The incidence of HSP is widely reported in previous literature, ranging from 16% to 84% but most commonly reported as near 70%.[3]

It is not only pain but associated psychological distress that limits a patient's participation in the rehabilitation process. The presence of HSP is strongly correlated with a prolonged hospital stay and lower Barthel functional score in the first 12 weeks after stroke.[4] Of patients who had a Barthel Index score of less than 15, 59% experienced shoulder pain during their hospital stay, compared with 25% of patients with a Barthel Index score greater than 15.[5] Patients with HSP are less likely to return to their home.[6] Conversely, improvement of upper limb function within the first 5 weeks after a stroke can result in improved use of the affected limb in functional tasks.[7]

PREDICTORS AND PROGNOSIS IN HEMIPLEGIC SHOULDER PAIN

HSP has a significant impact on function both during and after rehabilitation. A meta-analysis of 58 studies assessed outcomes of overall upper limb recovery according to age, sex, lesion site, initial motor impairment, motor-evoked potentials, and somatosensory-evoked potentials.[8] Only initial measures of impairment and function predicted long-term outcome. Age in itself is not clearly a risk factor on its own, but those of older age are more likely to have preexisting abnormality that affects impairment. Additional risk factors for developing shoulder pain within the first 6 months after stroke include impaired voluntary motor control, diminished proprioception, tactile extinction, abnormal sensation, spasticity of the elbow flexor muscles, restricted range of motion (ROM) for both shoulder abduction and shoulder external rotation trophic changes, and type 2 diabetes mellitus.[9] Barlak and colleagues[10] found a significant correlation between HSP and adhesive capsulitis and complex regional pain syndrome, but none between HSP and grade of subluxation, spasticity, impingement syndrome, or thalamic pain.

In addition to new impairments following a stroke, the practitioner must also consider the likelihood of pre-existing abnormality, whether symptomatic or not, which may contribute to pain in the shoulder. Shoulder pain is a common musculoskeletal complaint made to primary care physicians and a reason for referral to a musculoskeletal specialist. Rotator cuff disorders are the most common source of such pain. Partial tears of the rotator cuff are frequently seen as early as age 50 years, with the risk of severe injury increasing in the 60s and 70s age groups.[11] Degeneration

of articular surfaces may reduce ROM, and damage to soft tissue can increase joint laxity.[12] Further complicating a proper diagnosis are data that suggest a poor correlation between symptoms and findings on physical examination. Dromerick and colleagues[13] found that examination findings consistent with injury to the supraspinatus and long head of the biceps are more consistently associated with early onset of HSP, regardless of whether the patient reported pain.

SHOULDER ANATOMY

The human shoulder is a complex ball-and-socket joint that allows multidirectional reach. This agility comes at the sacrifice of stability.[14] The extensive ROM is due largely to the shallow depth of the glenoid fossa, with only 25% of the humeral head coming into contact with the glenoid. This agility is necessary to properly position the hands for a large variety of functional tasks. The only true joint directly connecting the entire upper quarter to the trunk is the sternoclavicular joint. Stability of movement, therefore, depends on both static and dynamic stabilizers (Fig. 1). Stability is provided by the surrounding muscles and ligaments. The glenohumeral ligaments serve as the primary static stabilizers and include the superior, middle, and inferior

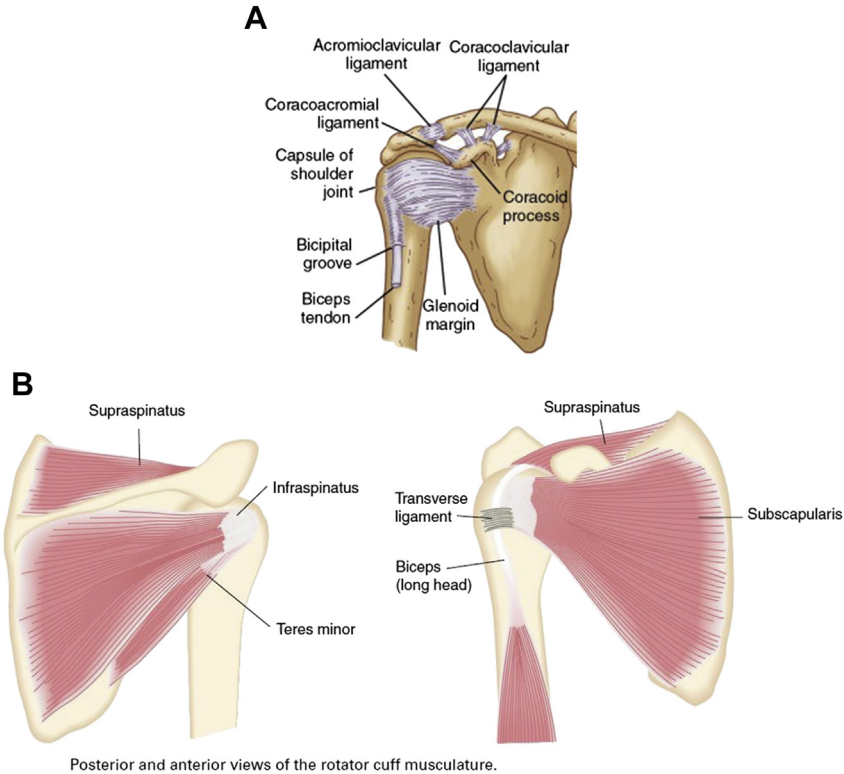

Fig. 1. Static (*A*) and dynamic (*B*) stabilizers of the shoulder. Image *B* shows the posterior (*left*) and anterior (*right*) views of the rotator cuff musculature. (*From* [*A*] O'Donoghue DH. Treatment of injuries to athletes, 4th ed. Philadelphia: Saunders, 1984; with permission; and [*B*] DeLee JC, Drez D, Miller MD. DeLee and Drez's Orthopaedic Sports Medicine, 3rd Ed. Philadelphia: Saunders, 2009, p. 989; with permission.)

glenohumeral ligaments. The primary dynamic stabilizers are the rotator cuff muscles, whose attachments form a cuff around the head of the humerus.

The glenohumeral joint derives passive support from a cartilaginous labrum, glenohumeral ligaments, and joint capsule. Functional movements require coordinated movements of dynamic stabilizers. The deltoid and rotator cuff muscles (supraspinatus, infraspinatus, teres minor, and subscapularis) act on the humerus, and the position of the scapula is primarily controlled by the trapezius, serratus, and latissimus dorsi. The subscapularis rotates the humerus internally, whereas the infraspinatus and teres minor are external rotators. Abduction is primarily achieved by the deltoid and is aided by the supraspinatus. The rotator cuff muscles compress the humeral head in the glenoid fossa, thereby stabilizing the joint and providing a counterbalance to opposing forces on the humerus. Overhead activity requires simultaneous abduction by the deltoid and external rotation by the infraspinatus. Movements in a single anatomic plane, such as abduction, can only be accomplished with a predictable ratio of movement termed scapulohumeral rhythm.[15] Impairment of rotator cuff action can lead to superior subluxation of the humeral head, predisposing to impingement of the supraspinatus between the greater tubercle of the humerus and the acromion.

MECHANISMS OF INJURY

Although many mechanisms for HSP have been proposed, pinpointing the cause in individual patients can be elusive. The etiology may be multifactorial, relating to disruption of the biomechanical balance of the shoulder caused by stroke-induced weakness, spasticity, and sensory impairment. Several systems for categorizing HSP exist. A model by Ryerson and Levit identified 4 major sources of pain in patients with HSP.[16] Joint pain resulting from instability can cause sharp pain with passive or active movement. Atrophic or spastic muscle can result in a “pulling” pain with movement. Abnormal pain sensitivity can arise from inappropriate central nervous system modulation of the pain, which can vary from diffuse and achy to sharp and lancinating. Complex regional pain syndrome, though less common, is characterized by reduced ROM, dysesthesia, and trophic changes.

The difficulty in interpreting this and other descriptions of HSP is the absence of any pathognomonic relationship to any particular subtype of pain. Achy pain emanating from muscle or tendon impingement can just as likely result from an upper motor neuron disorder such as spasticity. Sharp pain, allodynia, or hyperpathia caused by a lower motor neuron disorder, such as axillary neuropathy, could present with similar symptoms associated with central causes of pain and altered sensation. To avoid such confusion, the classification of HSP is more accurately based on etiology rather than symptoms alone.

APPROACH TO DIFFERENTIAL DIAGNOSIS

To more effectively determine the factors that contribute to hemiplegic shoulder pain, the authors suggest that factors affecting HSP should be divided into 2 categories: neurologic and mechanical (**Box 1**). Neurologic factors include spasticity, brachial plexus injury, complex regional pain syndrome (CRPS), and central sensitization. Mechanical factors include shoulder subluxation, rotator cuff injury, glenohumeral joint disorders, adhesive capsulitis, and direct trauma. It is important to appreciate that the cause of pain may involve a combination of neurologic and mechanical factors.

Box 1
Components of hemiplegic shoulder pain

- Neurologic Factors
  - Upper motor neuron neurologic factors
    - Paralysis, spasticity, central poststroke pain, central sensitization
  - Lower motor neuron neurologic factors
    - Peripheral neuropathy, brachial plexus injury, complex regional pain syndrome
- Mechanical Factors
  - Shoulder subluxation, rotator cuff injury, glenohumeral joint disorders, adhesive capsulitis, myofascial pain, direct trauma

Neurologic Factors

Weakness

Weakness of the muscles supporting the shoulder joint is a commonly seen after a stroke and often persists chronically. Weakness disrupts the stabilizers of the shoulder joint and often precedes subsequent development of spasticity. It is an underlying factor common to both neurologic and mechanical factors. Weakness of the trunk muscles and the muscles stabilizing the head is also common after stroke and frequently affects posture, most commonly creating a forward flexed and stooped posture, which can further lead to anterior subluxation of the shoulder and further exacerbate rotator cuff impingement and traction on the joint capsule.

Spasticity

Muscle spasticity is commonly defined as a velocity-dependent resistance to passive stretch. It is a consequence of an upper motor neuron disorder, creating an imbalance between agonist-antagonist muscle pairs. The result in hemiplegia is typical posturing with a dominant flexor tone in the upper limbs. Overactivity of the pectoralis and subscapularis is most predominant about the shoulder, resulting in excessive humeral flexion, adduction, and internal rotation. Combined with increased activity of teres major and latissimus dorsi, spasticity inhibits active and passive abduction, extension, and external rotation at the shoulder. The consequence is inability to achieve desired ROM for activities of daily living (ADLs), and predisposition to mechanical injury (eg, rotator cuff impingement).

Of patients with HSP, approximately 85% with spastic hemiplegia experienced pain, compared with 18% in those with a flaccid hemiplegia.[17] Patients with reduced external rotation experience more pain, and use of a subscapular nerve block to a spastic subscapularis muscle has been demonstrated to reduce pain.[18] Preservation of joint mobility in patients with spasticity and prevention of contracture in those with flaccid hemiplegia are intended to reduce the incidence of HSP.

Brachial plexus and peripheral nerve injury

The brachial plexus is derived from C5-T1 roots, and arises at the lower aspect of the neck. It runs behind scalenes proximally, and behind the clavicle and pectoralis muscles distally. Injury to the plexus can be traumatic or atraumatic. In the setting of hemiplegia, the cause is most likely a traction injury caused by improper handling of the flaccid hemiplegic limb, such as pulling on the arm during transfers and repositioning.[19,20] One study based on needle electromyography (EMG) reported that 75% of supraspinatus and deltoid muscles in hemiplegic arms had neuropathic responses.[21]

The upper trunk of the plexus is most susceptible to injury. The most common isolated peripheral nerve injury in HSP is axillary neuropathy, thought to be subsequent to downward displacement of the humeral head in shoulder subluxation.[22,23] However, other studies have failed to reveal significant evidence of plexus or peripheral nerve injuries associated with HSP.[24,25] Given the conflicting evidence, it is not possible to ascertain whether plexopathy or mononeuropathy plays a substantial role in HSP. However, if a plexus or peripheral nerve injury occurs it may contribute to a cycle of pain, weakness, and progressive subluxation.

Complex regional pain syndrome

Type 1 CRPS, previously termed reflex sympathetic dystrophy or shoulder-hand syndrome, and Type 2 CRPS, previously termed causalgia, are characterized by pain that is out of proportion to the pathologic condition, peripheral and/or central autonomic abnormalities, and dystrophic changes to a limb often (but not always) following a traumatic injury. CRPS can inhibit mobility by both pain that discourages motion and the associated adhesive capsulitis that restricts it. The incidence of CRPS in patients with hemiplegia has been cited to be as high as 23%.[17] However, there is considerable variability in past reported incidence, likely attributable to various diagnostic criteria. The precise mechanism of this disorder remains unclear. There are studies demonstrating an association between shoulder-hand syndrome and spasticity, confusion, and sensory loss.[26–28] Damage to the soft tissues surrounding the hemiplegic shoulder have been implicated as a cause of shoulder-hand syndrome.[29] Abnormalities in the brain itself have also been implicated. Further study is needed before a definite causality between HSP and CRPS can be confirmed.

Central poststroke pain and sensitization

Sensory disturbance and neglect can alter a patient's proprioception and perception of pain, predisposing the shoulder to injury. Central poststroke pain (CPSP) is another impairment deriving from stroke that can contribute to pain in the shoulder and elsewhere. Also termed thalamic pain syndrome, a lesion of the spinothalamocortical pathway may result in abnormal neural reorganization. The result is an improper generation of pain in the absence of injury, which can be reported as neuropathic, spastic, or musculoskeletal in quality. Central sensitization is a separate entity that can be observed in the presence of CRPS and CPSP, whereby abnormal responsiveness of nociceptive neurons results in dysesthesia. Sensitization often involves alterations in neurotransmitter levels, including serotonin and norepinephrine.[14]

Mechanical Factors

Shoulder subluxation

Shoulder subluxation refers to the static displacement of the humeral head in relation to the glenoid, and represents a common source of mechanical pain in HSP. Subluxation requires a disruption in the integrity of the glenohumeral joint. Clinical findings are a gap between the humeral head and the acromion. This gap can be measured with calipers, radiography, or ultrasonography, but is commonly described by finger breadths in the clinical setting. During the early stages following stroke the muscles in the hemiplegic arm are usually flaccid, thereby impairing joint stability and predisposing the shoulder to traction-type injury. The most common reason is an inability of the paralyzed shoulder girdle musculature to provide dynamic stability at the joint. Articular tissues (eg, the joint capsule) can become distended, particularly in the flaccid stage following stroke. This distension is also hypothesized to contribute to ischemia in the tendons of the supraspinatus and long head of the biceps.[30] Downward displacement of the humerus is most common during the flaccid stage, whereas

the spastic stage often leads to anterior displacement, posterior displacement, or internal rotation.[11] Anteroposterior (AP) and oblique radiographs help diagnose and characterize shoulder subluxation. Clinical diagnosis of subluxation is often achieved by measuring arm-length discrepancy or by palpating or measuring the subacromial space.[31,32]

The association between shoulder subluxation and HSP remains controversial. Paci and colleagues[30] studied 107 patients with hemiplegia in a case-control design, and measured the presence of shoulder pain in those with shoulder subluxation and those without. Patients with shoulder subluxation had significantly greater pain at admission, discharge, and at a 30- to 40-day follow-up assessment; they also had greater impairment with ADLs and required longer hospital stays. However, other studies argue that patients without subluxation are just as likely to develop pain.[33] Comparative studies of the association between shoulder subluxation and pain are limited by sample size or methodology. However, there is enough evidence to suggest that shoulder subluxation may be a contributing factor in HSP. Proper positioning, support, and correct transfer techniques by caregivers may be helpful in prevention and alleviation of pain.

Rotator cuff injury

As discussed previously, the primary purpose of the rotator cuff is to stabilize the humeral head relative to the glenoid during shoulder movements. Rotator cuff injuries are a common source of shoulder pain in the general population. Rotator cuff tears occur in 20% to 40% of the general population, with increasing incidence with age. The incidence of rotator cuff tears in hemiplegic patients ranges from 33% to 40%.[33] It is unlikely that hemiplegia is a cause of rotator cuff injury per se, but abnormal positioning, muscle imbalance caused by weakness, and spasticity can all increase the likelihood of impingement and tearing. In addition, falls can be a common occurrence during the initial onset of the stroke itself, and may be a cause of rotator cuff tear, which may go unnoticed during the initial stages of the stroke. Improper handling of the hemiplegic arm could also cause injury to the rotator cuff tendons. Treatment of rotator cuff injuries in the plegic or paretic arm is usually conservative and supportive.

Adhesive capsulitis

The term frozen shoulder is often used to describe a shoulder with decreased ROM, but the term is nonspecific and fails to determine how much of the restriction is passive (ie, a block to motion) versus active (ie, limited by pain or weakness). Adhesive capsulitis is a more specific term that refers to a condition of uncertain origin characterized by significant restriction of both active and passive shoulder motion that occurs in the absence of a known intrinsic shoulder disorder.[34] A painful shoulder may develop adhesive capsulitis because of pain inhibition of mobility, leading to subsequent disuse atrophy and contracture. The pain of adhesive capsulitis is also theorized to lead to increased immobility.[35] The decreased ROM can lead to inflammation, muscle atrophy, and contracture resulting from adhesions.[36] The prognosis of adhesive capsulitis is favorable, but requires diligence to preserve available ROM and strength. Increased immobilization from spasticity can increase the likelihood of developing adhesions.[11]

Myofascial pain

A more complete discussion of myofascial pain and trigger-point theory can be found in other articles by Dr Gerwin and by Dr Borg-Stein and Iaccarino elsewhere in this issue. As with most musculoskeletal disorders, it is important for the clinician to consider the contribution of muscle-generated pain from muscles about the shoulder

girdle, and the contribution to posture and muscle balance on the level of myofascial pain. Although there is a larger body of data regarding the impact of myofascial pain on shoulder pain in the general population, there is only one published study specifically studying myofascial pain in HSP, which demonstrated improvement of pain with dry needling of trigger points when combined with standard rehabilitation.[37]

DIAGNOSIS OF HEMIPLEGIC SHOULDER PAIN

There are no clear or widely accepted criteria for diagnosing HSP. Therefore, the authors recommend confirming the diagnosis following the same approach of dividing the workup according to suspected neurologic and mechanical factors (**Box 2**).

Neurologic Factors

History and physical examination

A proper history and physical examination are paramount, especially when symptoms can be explained by multiple causes. Important information to elicit during history taking includes preexisting shoulder pain and use of analgesics, limited functional use of the arm, prior trauma, and surgery. Regardless of the diagnosis, the key steps in the physical examination include observation (for asymmetry, deformity, and erythema), ROM, palpation, sensation, reflexes, strength, and special tests. The patient should demonstrate maximum active range of motion (AROM) before the examiner assesses full passive range of motion (PROM). Pain is most often the limiting factor in AROM, followed by weakness. If there is reduced PROM, contracture or anatomic block should be suspected. A goniometer can provide more objective monitoring of changes to ROM. Palpation is performed to assess for muscle bulk, abnormal contour or masses, or areas of tenderness. Key targets of palpation should include rotator cuff, deltoid, periscapular muscles, long head biceps tendon, other upper quarter musculature, and acromioclavicular joint. Strength testing in the C5-T1 myotomes (graded 0–5), sensory testing in the C5-T1 dermatomes (graded 0–2+), and C5-C7 reflexes (graded 0–4+) will help to localize a neurologic lesion, whether central or peripheral.

As with any neurologic injury, careful consideration to sensation and strength are useful in determining whether the lesion is central (brain and spinal cord) or peripheral, and whether the damage is focal (as in axillary neuropathy) or diffuse (as in CRPS). Because hemiplegia is an upper motor neuron disorder, it is also important to assess the presence and severity of spasticity. Muscle spasticity is determined using the Modified Ashworth Scale (**Box 3**).

Electrodiagnosis

Electrodiagnostic testing has excellent sensitivity and specificity for nerve injury within the peripheral nervous system. Electrodiagnostic testing may have limited utility in patients with HSP. Although it may be helpful in diagnosing a peripheral neuropathy, it cannot reliably exclude shoulder pain related to centrally mediated weakness or spasticity. Nevertheless, it may be useful in situations where there is underlying or

Box 2
Diagnosing the causes of hemiplegic shoulder pain

History, physical examination, special tests/maneuvers

Imaging (radiography, magnetic resonance imaging, ultrasonography)

Electrodiagnosis

Diagnostic nerve blocks or injections (intramuscular, intra-articular)

Box 3
Modified Ashworth Scale

0: No increase in muscle tone

1: Slight increase in muscle tone, manifested by catch and release, or minimal resistance to the end range of motion (flexion or extension)

1+: Slight increase in muscle tone, manifested by catch and then minimal release through the remainder (less than half) of the range of motion

2: Moderate (marked) increase in muscle tone through most of the range of motion, but affected part is easily moved

3: Severe (considerable) increase in muscle tone through most of the range of motion, and affected part is difficult to move

4: Affected part is rigid in flexion or extension, little to no passive range of motion

concomitant possibility of brachial plexus nerve injury, peripheral mononeuropathy, or cervical radiculopathy.

Sympathetic block

A sympathetic ganglion block is a diagnostic option considered for patients with suspected CRPS. This block may assist in reducing symptoms mediated by the sympathetic nervous system, which includes alterations in skin color and temperature. These blocks will often cause a temporary Horner syndrome.

Mechanical Factors

Physical examination

The basic components of the physical examination, such as testing of strength, sensation, and reflexes, are used regardless of the cause of HSP. In addition, there are multiple specialized tests for the shoulder, but only a few most pertinent to the mechanical components of HSP (Fig. 2A–F).

Neer, Hawkins, and Jobe ("empty can") tests can assess for subacromial impingement. Apprehension and sulcus tests assess for glenohumeral joint instability. The apprehension test is performed by placing the patient in a supine position near the edge of the bed with the arm externally rotated, abducted, and in slight extension. Apprehension against further motion during the maneuver suggests anterior shoulder instability with 63% sensitivity. The sulcus test is performed in the sitting position with the affected arm at the patient's side. The examiner pulls the elbow inferiorly to measure the physiologic separation between the acromion and humeral head. Separation of 1 cm is scored as Grade 1, 1 to 2 cm is scored as Grade 2, and more than 2 cm is scored as Grade 3. Grade 3 separation indicates multidirectional glenohumeral instability, but the maneuver has only 28% sensitivity.[38]

The Neer test is performed by passive forward elevation of the arm with scapula stabilized. A modification of the test includes adding internal rotation of the humerus to approximate the acromion and greater tuberosity of the humerus. Positive pain using this maneuver suggests subacromial impingement with 88% sensitivity. The Hawkins test for impingement is positive if pain is produced with passive horizontal adduction and internal rotation. The Jobe (or empty can) test is positive for impingement if pain is produced when resistance is applied to arms elevated and internally rotated in the scapular plane (horizontal abduction to approximately 45°).

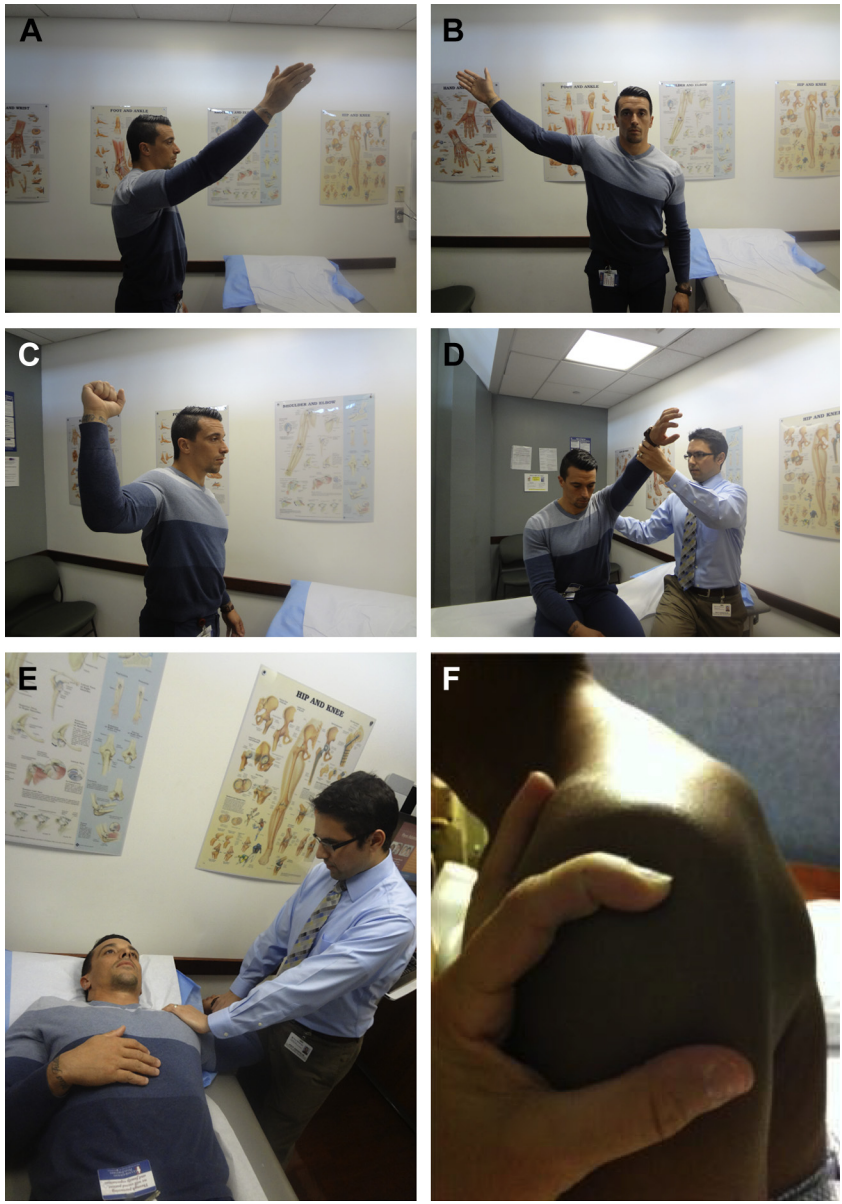

Fig. 2. Key examination maneuvers in hemiplegic shoulder pain. Examination should include active and passive flexion (*A*), abduction (*B*), and external rotation (*C*). Specialized maneuvers include Neer test modified with humeral internal rotation (*D*), apprehension test (*E*), sulcus test (*F*), hand-behind-neck (*G*), and hand-behind-back (*H*).

Fig. 2. (*continued*)

In stroke patients able to comply, a simple and efficient bedside screen for shoulder ROM includes the hand-behind-back (HBB) and hand-behind-neck (HBN) maneuvers. The HBB maneuver combines internal rotation and extension, and the HBN maneuver combines external rotation and abduction (see **Fig. 2**G, H). Differences or pain in passive or active external rotation of the shoulder can indicate the onset of HSP.

The value of the physical examination is often greatest when multiple maneuvers are positive, or when overall movement is asymmetric relative to the unaffected side. Rajaratnam and colleagues[38] concluded that HSP could be successfully diagnosed clinically by using only 3 of the aforementioned methods: Neer test, HBN, and a difference of greater than 10° passive external rotation at the shoulder joint. When combined with a report of at least moderate pain at rest, the sensitivity and positive predictive value for HSP was 96.7%. Another study of proprioception and kinematics of shoulder motion in patients with and without HSP found that those with HSP demonstrated increased lateral scapular rotation and decreased perception of passive movement.[39] Furthermore, patients who had a stroke were more likely to demonstrate abnormal scapular movement on the nonparetic side when compared with controls, arguing that rehabilitation must always take both sides of the body into account.

Diagnostic imaging

Ultimately the diagnosis of HSP is clinical, and does not necessitate diagnostic imaging. However, the use of imaging may be of benefit if the history and examination raise suspicion of underlying traumatic or structural abnormalities that may contribute to the patient's pain.

Radiography

Radiographic imaging is a useful starting point for evaluating suspected mechanical components of HSP. An AP view will rule out fracture and help to assess for subluxation. Adding AP views with the humerus in external rotation will bring the greater tuberosity and associated soft tissue into better view, and may help reveal calcific rotator cuff tendinopathy. Rotator cuff impingement by the acromion is best evaluated with a scapular Y view. If there is concern for shoulder instability, an axillary view will evaluate the relationship of the humerus to glenoid, and an AP view with humerus in internal rotation may reveal a Hill-Sachs lesion seen in traumatic dislocation.

Conventional and Magnetic Resonance Arthrography

Conventional x-ray arthrography is rarely used in clinical practice as an isolated method of diagnosis, but can help diagnose both adhesive capsulitis and rotator

cuff tears. A normal joint will have a volume exceeding 10 mL, smooth glenohumeral capsular margin contours, and the presence of an axillary recess (a pouch of the capsule bordered by the inferior rim of the glenoid cavity and inferior portion of the humeral head). Patients with the presence of adhesive capsulitis demonstrate less than 10 mL of volume, irregular capsule margins, and a diminished or absent axillary recess. A rotator cuff injury will be demonstrated by contrast leakage from the glenohumeral joint to the subdeltoid bursa. The high sensitivity of arthrography (as high as 99%) makes this procedure the gold standard for detecting such tears.[40] However, soft tissues cannot be visualized using this method.

A 1-year study of 32 patients with HSP by Lo and colleagues[40] attempted to correlate arthrographic and clinical findings of HSP. Clinical measurements included Brunnstrom stage (**Box 4**), spasticity distribution, presence or absence of shoulder subluxation, or CRPS Type 1. Arthrographic measurements included shoulder joint volume and capsular morphology. Fifty percent of the patients had evidence of adhesive capsulitis, 44% had shoulder subluxation, 22% had rotator cuff tears, and 16% had CRPS Type 1. Disorders were often present in combination. The study determined that arthrography was useful in identifying adhesive capsulitis, and that most cases developed within 2 months of developing HSP. Most significantly, outcomes worsened the longer adhesive capsulitis remained untreated. Even when diagnostic imaging is not used, the findings emphasize the importance of initiating appropriate treatment whenever adhesive capsulitis is clinically suspected.

Magnetic resonance arthrography (MRA) has the advantage of better visualization to identify abnormality of the soft-tissue structures of the shoulder, and with little loss of sensitivity and specificity. Multiple criteria exist to diagnose adhesive capsulitis by MRA, including thickness of capsule and synovium greater than 3 mm on T2-weighted coronal sequence without fat suppression, an axillary recess diameter greater than 9.0 mm, or rotator cuff interval thickness exceeding 8.4 mm.[41,42]

A study of magnetic resonance imaging findings between stoke survivors with and without shoulder pain in the chronic stage found synovial capsule thickening, synovial capsule enhancement, and enhancement in the rotator cuff interval to be more prominent in those with shoulder pain.[43] There was no significant difference in rotator cuff tendinopathy, joint effusion, subacromial bursal fluid, acromioclavicular capsular hypertrophy, and muscle atrophy. Shoulder subluxation was not observed, and was postulated to have resolved in the more acute to subacute stages of recovery. The findings suggest that chronic mechanical limitations in HSP most closely resemble those of adhesive capsulitis.

Box 4
Brunnstrom stages of stroke recovery

1. Flaccidity (immediate after onset), no voluntary movements can be initiated
2. Spasticity appears, basic synergy patterns appear, minimal voluntary movements present
3. Increased spasticity, patient gains more voluntary control over synergies
4. Decreased spasticity, patient masters control of synergistic movement patterns
5. Further decreased spasticity, synergies lose dominance over motor acts
6. Spasticity disappears, joint movements improve, and coordination approaches normal
7. Normal function is restored

Ultrasonography

Although ultrasonography is not a new modality, there has been a recent surge in its popularity as a method of diagnosing a wide variety of musculoskeletal disorders of the shoulder. The primary advantages of ultrasonography include excellent visualization of superficial soft tissue, dynamic assessment, and lack of ionizing radiation. However, there is limited utility for some deep structures and those blocked behind bone. The pain from HSP that limits ROM may interfere with optimal positioning during ultrasonography.[44] Pathologic features easily identified by ultrasonography include rotator cuff tendinopathy, dynamic rotator cuff impingement, acromioclavicular arthrosis, and long head biceps tenosynovitis. In adhesive capsulitis, ultrasonography may reveal hypoechoic echotexture and increased vascularity within the rotator interval (triangular space bounded by superior border of the subscapularis anteriorly, anterior border of the supraspinatus tendon posteriorly, and coracoid process as base).[45]

A unique advantage to the use of ultrasonography is the ability to provide serial assessments of the shoulder throughout the course of rehabilitation. The risk of injury appears to be greatest in the early stages of recovery after stroke. Pong and colleagues[46] used ultrasonography to evaluate for soft-tissue injuries at admission and 2 weeks after completion of rehabilitation. Patients admitted at Brunnstrom stages I to III were more likely to demonstrate new or worsening injuries to the shoulder after rehabilitation in comparison with those admitted at Brunnstrom stages IV to VI. These results are consistent with those in similarly designed studies.[47,48]

MANAGEMENT OF HEMIPLEGIC SHOULDER PAIN

There are many available modalities, both pharmacologic and nonpharmacologic, for the treatment of HSP, which are outlined in **Box 5**.

Regardless of Cause

Prevention through positioning

The key to prevention of HSP is proper handling and positioning, especially in the first days after stroke. The patient depends on multiple members of the health care team to assist with positioning and transfers throughout each day. In the flaccid stage, the shoulder capsule has significant laxity and is particularly vulnerable to injury from static stabilizers. Patients who require assistance with transfers are more likely to develop shoulder pain.[5] There is no clear guideline regarding which method best reduces strain on the shoulder. However, simply raising patient and caretaker awareness of potential injuries caused by poor handling can reduce injury as a result of increased vigilance.[26]

A commonly suggested position for the shoulder is abduction, external rotation, and flexion.[33] However, there is no consensus on which position is ideal, and no one position has been proved to be significantly better in studies of the subject.[6] The aim is to achieve symmetry between the affected and unaffected shoulders, and caretakers should strive for symmetric positioning of both scapulae. Carr and Kenney[49] recommend that the shoulder be protracted, with the arm forward, wrist neutral or slightly supinated, and fingers extended. Bobath suggested a technique of positioning in a reflex-inhibiting pattern to prevent inefficient movement and maintain muscle tone.[50] The affected limb is positioned away from the direction of muscle spasticity. The precise direction varies, and depends on the muscle tone patterns of each individual. Small sample sizes in studies of positioning limit the significance of any one suggested pattern.

Box 5
Approach to treatment of hemiplegic shoulder pain

Regardless of Cause

- Prevention through positioning
- Bracing, slings, taping
- Physical therapy to optimize range of motion and strength

Neurologic Factors

- Transcutaneous electrical nerve stimulation (TENS)
- Functional electrical stimulation (FES)
- Relaxation/electromyography biofeedback
- Botulinum toxin injection
- Sympathetic blocks
- Pharmacotherapy (eg, antispasticity, neuropathic pain)

Mechanical Factors

- Pharmacotherapy (eg, anti-inflammatory)
- Corticosteroid injection
- Suprascapular nerve block
- Trigger-point injections and dry needling

Complementary and Alternative Medicine

- Acupuncture
- Aromatherapy
- Surgical treatment

Strapping and slings

Strapping is used to maintain the shoulder joint in an appropriate anatomic position to prevent or reduce subluxation. Strapping from the onset of stroke until restoration of muscle tone may prevent the incidence, or at least delay the onset, of HSP.[51,52] Taping perpendicular to a muscle inhibits activity, and taping parallel to a muscle promotes activity.[33] The exact mechanism of pain relief remains uncertain. This technique requires a trained care provider to apply the dressing, and repeat applications to prevent skin irritation. A small study comparing taping versus sham taping in patients with shoulder pain revealed decreased pain-free shoulder abduction, but no significant change in overall pain or ROM.[53] Furthermore, the study was performed without regard to specific diagnosis, and was not applied to patients with HSP. There are no high-quality studies demonstrating the benefit of taping specifically for HSP.

In addition to strapping, shoulder slings have also been used to decrease the stress on the shoulder joint and prevent subluxation by reducing the gravitational pull on the shoulder joint. However, if the arm is incorrectly positioned, or if use of a sling is not alternated with therapeutic exercise, soft-tissue contractures may occur. Such contractures can contribute to the very pain the sling is intended to prevent. Slings are recommended primarily for a flaccid upper extremity, when the patient is upright or walking, for the purpose of protection.[54] Arm troughs and lap trays are recommended for use in a wheelchair to support the limb and prevent shoulder subluxation, as well as to prevent traumatic injury. The properly positioned tray and trough can also maintain abduction and limit excessive internal and external rotation.[55] Arm slings can support

the flaccid arm and are protective in ambulating patients, but because many slings hold the arm in flexion, adduction, and internal rotation, their use must be balanced with ROM exercises. Axillary supports such as the Bobath sling are less popular, as they have not been proved to reduce subluxation and can increase soft-tissue injury through lateral displacement of the humeral head.[11]

Like strapping, there is insufficient evidence to indicate the effectiveness, best type, or proper positioning for the use of slings. Some studies demonstrate a reduction in subluxation immediately after application of the sling, but do not prove that this reduction is maintained on resuming functional activities.[33] Regardless of the effect on pain or subluxation, another reason to use a sling is to promote efficiency during ambulation. Han and colleagues[56] performed a randomized crossover gait evaluation in hemiplegic patients with and without a sling, and found improvements in heart rate and gait speed, with decreased oxygen cost and oxygen rate in patients using the sling.

Physical therapy

Physical therapy is an essential component of poststroke rehabilitation, and plays a major role in the prevention and treatment of HSP. PROM exercises should be initiated as soon as the patient is medically stable. Care should be taken during passive abduction of the arm, as this may cause or aggravate a rotator cuff injury. If pain consistent with impingement is noted during the PROM exercises, the amplitude of movement should be decreased. Performing PROM exercises within such a pain-free range has been shown to reduce reports of shoulder pain by 43%.[57]

Physical therapy is directed at both improving upper extremity mechanics and reducing neurologic injury. Heat and cold therapy are used to decrease pain, increase mobility, and reduce inflammation. Slings and strapping may be used as an adjunct to minimize subluxation and reduce mechanical pain. Overhead exercise pulleys are strongly discouraged, as they can cause impingement and rotator cuff injury.[58] Rehabilitation methods include the Bobath and Brunnstrom approaches, and task-specific motor retraining. No particular technique has been proved to be more effective than another.[36]

Lynch and colleagues[59] studied the effectiveness of a continuous passive motion (CPM) device versus self-ROM group exercises in 32 hemiplegic patients. All patients received 3.5 hours of standard therapy daily, and the additional therapies were supervised by an occupational therapist. A blinded assessor evaluated joint strength and integrity at discharge. CPM was associated with greater shoulder stability, but there was no significant improvement in motor impairment, disability, pain, or tone.

Masiero and colleagues[60] used a single-blinded trial to evaluate the addition of early sensorimotor training using a robotic device in addition to standard therapy in 35 patients with acute stroke. Robotic devices are used to provide high-intensity, repetitive, interactive training of an impaired limb, and the controlled movement helps prevent injury and ensure maximal benefit to targeted muscle groups. Patients in the treatment group underwent a total of 20 hours (4 hours daily for 5 weeks) of programmed shoulder and elbow manipulation by the robotic device. Patients in the control group underwent robotic training of the unaffected limb. The treatment group experienced a significant reduction in impairment and gains in function, with effects maintained at an 8-month follow-up assessment.

Neurologic Factors

Transcutaneous electrical nerve stimulation

Transcutaneous electrical nerve stimulation (TENS) provides an external electrical stimulus to the affected limb, and is postulated to be effective based on the gate-

control theory of pain. At high intensity, the electrical impulse can also activate the muscles to maintain muscle bulk. TENS can be delivered with low intensity (just enough stimulation to be sensed on the skin) or high intensity (noticeable muscle contraction and near-painful skin sensation). High-intensity TENS may reduce HSP in comparison with low-intensity TENS or placebo.[61]

Functional electrical stimulation

Several studies suggest that functional electrical stimulation (FES) reduces HSP and shoulder subluxation, and improves functional strength and ability.[36] FES is most often directed at the supraspinatus and (to a lesser extent) the posterior deltoid muscles because of their role in maintaining dynamic shoulder stability (**Fig. 3**). Research suggests that FES may reduce, or even prevent, subluxation. Wang and colleagues[62] compared the effects of a 6-week FES program in 16 patients with acute hemiplegia (less than 21 days) and 16 with chronic hemiplegia (more than 1 year). Those with acute hemiplegia improved with treatment while those with chronic hemiplegia did not. In addition, the reduction of shoulder subluxation in the patients with acute hemiplegia was lost on withdrawal of FES treatment. A randomized controlled trial of 50 patients with HSP found that FES and therapy, compared with therapy alone, reduced shoulder subluxation on radiography, but without a significant difference in AROM, PROM, or pain in either group at completion of inpatient rehabilitation.[63] A review of 9 controlled trials of FES for HSP by Chae and colleagues[64] found only 2 demonstrating sustained improvement after completion of treatment. A Cochrane review concluded that FES benefits pain-free passive external rotation ROM and reduces subluxation, but does not improve shoulder pain or motor impairment.[65]

Chae and colleagues[66] evaluated the effectiveness of intramuscular electrical stimulation (IES) in a multicenter, single-blinded randomized clinical trial of 61 chronic stroke survivors. Rather than traditional FES, which uses externally applied pads to deliver an electrical stimulation through the skin, IES delivers the stimulation directly to the targeted muscles via a percutaneous electrode. Advantages include more direct stimulation, reduced pain, and the ability to use the device at home with more precise targeting of the intended muscles. Patients in the treatment group were given 6 weeks of 6-hour stimulations to the supraspinatus, posterior deltoid, middle deltoid, and upper trapezius. Patients in the control group were managed with a cuff-like sling for 6 weeks. Patients who underwent IES reported a significant reduction in pain when compared with controls, sustained at 1 year after treatment. There is a single case report of complete and sustained relief of pain 13 months after a 3-week course of IES into the deltoid muscle.[67] Another case study of a fully implanted peripheral nerve

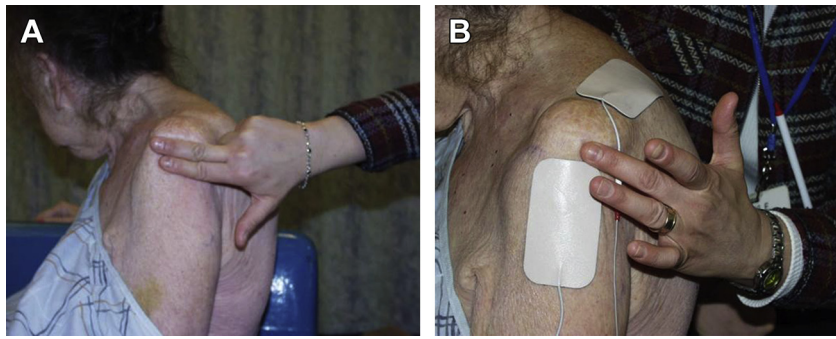

Fig. 3. Functional electrical stimulation (FES). A patient with Grade 2 shoulder subluxation (*A*) demonstrates reduced subluxation during use of FES (*B*).

stimulator targeting the axillary nerve demonstrated persistent pain relief and increased pain-free ROM, but without effects on sensation or strength.[68] IES represents a promising treatment, but is not yet widely available.

EMG biofeedback and relaxation exercises

A randomized crossover trial by Williams[69] of 20 patients with HSP who had no shoulder pain before stroke were given either 150 minutes of EMG biofeedback (30 minutes per day for 5 days) or 60 minutes of relaxation exercises (30 minutes per day for 2 days). Patients were assigned to the opposite group 1 week later. After the 2 weeks of intervention, both groups had increased ROM, increased muscle tone, and a 50% to 60% reduction in pain. Although the methodology of this study fails to distinguish the relative benefit of each therapy, the results suggest that such treatment provides patients with more psychological control over their pain.

Botulinum toxin

Botulinum toxin, a presynaptic acetylcholine inhibitor, has gained popularity for focal reduction of spasticity. Although targeted to reduce motor activity, the toxin also inhibits neurotransmitter release by sensory neurons. Several small studies have demonstrated improvement in ROM and reduction in pain in comparison with placebo.[17,70,71] Conversely, de Boer and colleagues[72] failed to find improvements in pain level or ROM after toxin injection into the subscapularis in 21 patients with HSP.

Lim and colleagues[73] compared botulinum toxin type A injected in the infraspinatus, pectoralis, and subscapularis with intra-articular triamcinolone acetonide injections in a randomized, double-blind, controlled study of 29 patients with HSP. There was a strong trend indicating that the botulinum toxin reduced pain and increased ROM when compared with intra-articular injection of triamcinolone. Of note, Ashworth scores were not significantly improved in the botulinum toxin group.

A smaller randomized controlled study of 17 stroke patients with HSP injected with botulinum toxin type A into the biceps and pectoralis major more than 3 months after stroke demonstrated a significant improvement in Ashworth scores for shoulder adduction and elbow flexion at week 4, but not at weeks 8 and 12. Shoulder pain and passive shoulder abduction ROM improved to a similar extent in the study and placebo groups.[71]

Another small, noncontrolled pilot study of 5 patients with HSP demonstrated decreased pain at 2 and 8 weeks after an intra-articular injection of botulinum toxin.[74] Despite the limitations of these case series, the results raise interest in the nociceptive properties of the toxin.

Sympathetic blocks for CRPS

A comprehensive explanation of CRPS management is discussed in a separate article by Dr Freedman and colleagues elsewhere in this issue. It is important to understand the essentials of recognizing and treating this condition as early as possible to prevent severe disability. There are 3 major components to management: pain management, rehabilitation, and psychological therapy.[75–80]

Pain is managed by many methods, but the basic concept is to reduce pain and altered sensitivity, prevent further injury, and increase mobility. Sympathetic blocks (often to the stellate ganglion) are used to interrupt abnormal sympathetic activity when other pharmacologic therapy fails.

Rehabilitation should use modalities for pain and edema control, and stress isometric and stress-loading exercises (repetitive and demanding motions with minimal joint motion such as scrubbing or carrying). PROM should be performed, but restricted to a

pain-free range. Psychological therapy helps reduce fear avoidance and encourage active involvement in rehabilitation.

Mechanical Factors

Pharmacotherapy

HSP originates not only via mechanical injury but also through altered sensitivity. Patients experiencing HSP will frequently require pharmacotherapy to complement other physical treatment modalities. As with most painful conditions, simple analgesics and anti-inflammatory drugs should be tried first. Acetaminophen taken before therapies can be useful and well tolerated, with less risk of adverse drug interactions and side effects than other analgesics, and is often a good medication to start with alongside a therapy program. Topical agents such as lidocaine can be helpful, and carry little risk of side effects. Although there is insufficient evidence to support or refute the effectiveness of nonsteroidal anti-inflammatory drugs (NSAIDs) in the treatment of HSP, they are considered worth a therapeutic trial if there are no contraindications. However, care must be taken in the stroke population, as many patients are already on antiplatelet treatment and often have comorbidities such as coronary artery disease, chronic kidney disease, or peptic ulcer disease (PUD).[11] NSAIDs can interfere with antiplatelet treatment, provide an unwanted anticoagulant effect, and further impair kidney function at high or chronic doses. Chronic NSAID use can also lead to PUD, which can be of greater risk in stroke patients on anticoagulants for secondary stroke prophylaxis. Topical NSAIDs carry less risk of kidney damage and PUD because of mostly local analgesic effects with limited systemic absorption.

Antiepileptic agents may be helpful with pain that seems to be of neurogenic character, as may be seen with central poststroke shoulder pain or shoulder-hand syndrome. Likewise, tricyclic antidepressants (TCAs) may have pain-relieving properties and may also aid with sleep. Other antidepressants such as selective serotonin reuptake inhibitors (SSRIs) may also be helpful with neuropathic pain. Oral pharmacologic agents that reduce spasticity help facilitate better participation in physical therapy; their use can be limited by side effects of sedation, although a bedtime dose is often well tolerated.

Small studies have demonstrated reduction in HSP with the use of oral corticosteroids.[26,81] However, the side effects associated with chronic use must be considered.

Berthier and colleagues[82] investigated the use of donepezil (without simultaneous physical therapy) in a single patient with chronic hemiplegia and observed improved sensorimotor function in the shoulder, but this finding has not been validated.

Corticosteroid injection

Corticosteroid injections targeted to the appropriate site of abnormality, most often to the glenohumeral joint or subacromial bursa, may reduce pain in patients with HSP.[33] These injections are best suited to reduce inflammatory pain from rotator cuff tears, bicipital tendonitis, subacromial bursitis, or adhesive capsulitis. If used appropriately and coupled with therapeutic exercise, the addition of steroid injections can significantly reduce pain and increase ROM for 2 to 4 weeks.[83,84] Two studies using intra-articular corticosteroid injections both demonstrated a reduction in pain but without a significant reduction in spasticity or improved function.[85,86] Pain radiating down the lateral shoulder and into the arm may reflect subacromial bursitis, for which Joynt[35] demonstrated that 50% of patients receiving 10 mL of a 1% lidocaine solution had moderate pain relief and improved ROM. Repeated injections increase the risk of weakening soft tissues and contributing to atrophic changes in the shoulder capsule.[11] Therefore, repeated or long-term use of intra-articular steroid injections

must be performed sparingly. Injections (particularly glenohumeral) are increasingly performed with fluoroscopic or ultrasound guidance, because their increased accuracy may help confirm the location of inflammatory pain, particularly if clinical suspicion remains high and there has been lack of effect after anatomic landmark-guided injection.

Suprascapular nerve block

The use of suprascapular nerve block has been studied for mechanical shoulder pain in the general population, and now with emerging studies in those with HSP. The purpose of the block is to decrease pain and allow for greater pain-free ROM, and the anesthetic medication may also be coupled with corticosteroid. A small randomized study comparing suprascapular nerve block with intra-articular injection in patients with chronic HSP found both increased ROM and decreased pain by 1 month after injection, but there was no statistical difference between the treatments.[87] Another randomized, controlled, nonblinded study comparing a block with anesthetic and corticosteroid with a placebo injection of saline in patients with HSP is currently under way.[88] A major concern with phenol motor point blocks of mixed nerves is causation of neuropathic pain. However, as reported by Chironna and Hecht,[89] the suprascapular nerve does not have a sensory component and, therefore, this risk is lower. The effect of the block varies from 3 to 9 months. Botulinum toxin can be used instead of a nerve block, although this method provides a shorter duration of action.

Complementary and alternative medicine

Acupuncture is thought to decrease myofascial pain by a neurohormonal mechanism, involving β-endorphin, dynorphin A and B, substance P, 5-hydroxytryptamine, or noradrenaline. A pilot study by Shin and Lee[90] studied the addition of acupuncture to standard rehabilitation therapy in 21 hemiplegic patients with shoulder subluxation. From the time of admission to discharge, there was significant improvement in ROM and muscle strength. A systemic review of acupuncture specifically for HSP by Lee and colleagues[91] discovered only 7 randomized controlled trials of sufficient methodology. The data suggest that acupuncture combined with therapeutic exercise is superior to either modality alone.

Aromatherapy uses plant-derived essential oils applied to the skin or inhaled through the nose to stimulate physiologic changes, including blood pressure and pulse, muscle tension, skin temperature, and blood flow. A trial comparing acupuncture with acupuncture and aromatherapy in the rehabilitation setting favored the addition of aromatherapy.[92] The limitation of this and similar studies is a failure to compare with standard rehabilitation or placebo.

Surgery

Surgical procedures are reserved only for severe shoulder pain or stiffness, most typically in the setting of adhesive capsulitis, not improved by all conservative measures. Surgery is often postponed until at least 6 months after the patient has had a stroke. Operations include release of muscle contractions, repair of rotator cuff tear, and scapular mobilization.[36] Little research has been done in this area, but a small study by Braun and colleagues[93] found that HSP was relieved in all 13 patients who had contracture release, versus no relief in patients treated without surgery. Rotator cuff repair is typically not done specifically for HSP, as it may have been present before the stroke and may offer little in the way of improvement in the plegic or paretic arm. Such repair may be more strongly considered for a traumatic rupture after stroke, but should account for the procedural risks and possibility of persistent pain from other generators of HSP.

SUGGESTED TREATMENT PROTOCOL FOR HEMIPLEGIC SHOULDER PAIN

Two things are clear regarding shoulder pain after stroke: it is a multifactorial process, and inability to prevent or treat the disorder results in poor outcomes. A review of the literature makes one thing clear: the quantity or quality of available evidence provides little guidance on how to manage this troubling complication of stroke. Until future research advances our understanding of HSP, the authors suggest a 5-step treatment approach (**Box 6**).

The first 2 steps of the protocol occur simultaneously as part of initial and subsequent evaluations of a patient with HSP. As detailed in the preceding sections, the most important part of treating a multifactorial process is to systematically consider each of the common neurologic and mechanical factors that is either present, or at risk of occurring. The treatment of a patient presenting with a flaccid upper limb after acute stroke will change as spasticity develops. Determining a history of rotator cuff tendinopathy before the cerebrovascular accident can help the clinician improve considerations for physical therapy. Once the various factors are considered, baseline measurements (eg, ROM and Modified Ashworth Scale) and appropriate diagnostic testing should be ordered to confirm diagnoses and document severity before treatment.

The third step is important at all stages of treatment, but is most pertinent during the flaccid stage after stroke and as acute rehabilitation commences. Clinicians, therapists, and family members must avoid applying excessive stress to the shoulder by reducing the effect of gravity with slings and lap trays in wheelchairs, or by reducing traction on the arm during transfers.

Only after careful examination of the shoulder and education on its proper positioning should the fourth step begin. The primary goals of rehabilitation include modalities for comfort and facilitation of movement, careful maintenance of ROM, spasticity management, and strengthening and facilitation with electrical stimulation, taping, and functional training. Only when mobility or participation in therapy is restricted by pain should the clinician introduce pharmacologic treatment for symptomatic control.

Interventional management is the fifth and final step in the treatment protocol, and is indicated when conservative measures fail. Procedures may also be considered when a patient cannot tolerate or progress through therapy because of pain, spasticity, or concern for developing CRPS.

KEY POINTS FOR TREATMENT

Positioning

- Supine: keep shoulder protracted, arm forward, wrist neutral to slightly supinated, fingers extended
- Spastic limb: keep arm abducted, externally rotated, flexed

Box 6
Approach to the treatment of hemiplegic shoulder pain

Step 1: Assess and diagnose neurologic factors contributing to HSP

Step 2: Assess and diagnose mechanical factors contributing to HSP

Step 3: Phase 1 of treatment: prevention through positioning

Step 4: Phase 2 of treatment: rehabilitation and symptomatic control

Step 5: Phase 3 of treatment: pathology-based intervention

Strapping/Taping

- Taping perpendicular to a muscle inhibits activity; taping parallel to a muscle promotes activity.

Slings and Supports

- Flaccid: use when sitting, ambulating, transferring for protection
- Spastic: avoid prolonged use to prevent contractures
- Sitting: use a lap board or arm trough positioned in slight abduction and external rotation
- Avoid axillary supports (can displace humeral head)

Physical Therapy and Modalities

- Strive for maximal amplitude of movement within a pain-free range
- Avoid overhead pulley exercises to reduce shoulder impingement
- TENS: may reduce pain, particularly when used at high intensity
- FES: apply to deltoid and supraspinatus; expect temporary reduction in shoulder subluxation
- EMG biofeedback: use to encourage active participation and psychological sense of control

Pharmacotherapy

- Neurologic:
  - Neuropathic pain: trials of TCAs or SSRIs for centrally mediated pain, gabapentinoids for peripherally mediated pain
  - Spasticity: trials of antispasmodics when ROM and positioning fail
- Mechanical pain:
  - Modalities, acetaminophen, and NSAIDs, if not contraindicated by a comorbid medical condition
  - Opioids may be considered for severe debilitating pain not responding to other measures
  - Oral corticosteroids are of little known benefit, but may be tried in short courses for debilitating pain not controlled by other measures

Injection Therapy

- Neurologic
  - Botulinum toxin intramuscular injections are beneficial for the focal reduction of spasticity
    - Muscles often targeted include subscapularis, pectoralis, infraspinatus, latissimus dorsi
    - There is emerging but insufficient evidence to suggest that intra-articular botulinum toxin may offer an antinociceptive benefit
  - CRPS: Stellate ganglion blocks are considered on when criteria for CRPS are met, and are most effective in early stages to treat autonomic symptoms
- Mechanical:
  - Corticosteroid injections are most beneficial when the pain-generating structure is correctly identified, and when there is an inflammatory component (eg, acute tendinitis rather than chronic tendinosis)
    - Structures most often targeted include the subacromial space and glenohumeral joint
  - Trigger-point injections and dry needling may benefit patients with myofascial pain; evaluate for altered posturing and kinematics (including unaffected limb)

Surgery

- Rarely indicated; usually performed after at least 6 months of failed nonsurgical treatment
- Neurologic: release of contractures if other methods for spasticity fail
- Mechanical: rotator cuff repair is usually only considered if pain clearly associated based on acute trauma, capsular release for adhesive capsulitis

Complementary/Alternative Medicine

- Acupuncture combined with therapeutic exercise may be superior to either treatment alone
- Aromatherapy may provide additional benefit (small pilot studies only)

SUMMARY

HSP is a common complication of stroke that can lead to poor functional outcomes. It is a multifactorial process that demands careful consideration of the contributing factors, both neurologic and mechanical. Efforts at prevention should be maintained throughout the course of treatment. The available evidence for nearly all treatments discussed in this article is conflicting and is limited by poor or variable methodology. The clinician is urged to consider the diagnostic and treatment approach presented in this article to ensure that all components of HSP are considered, and that treatments are provided in a logical manner. This method, along with constant vigilance by clinicians and properly educated caretakers, will provide the best opportunity to restore function and maximize quality of life.

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