Management of Hemiplegic Shoulder Pain

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Abstract Stroke remains one of the leading causes of death in the United States. However, due to advancements in medical care, more and more Americans are surviving the acute event and living with disability of varying degrees. One factor that has been shown to decrease functional recovery and quality of life in this population is hemiplegic shoulder pain (HSP). Due to the diverse nature of possible etiologies, the exact cause of HSP in a patient is often obscure, thus making effective treatment equally as elusive. Therapy remains a mainstay of treatment in HSP of any etiology, especially when coupled with functional electrical stimulation. Both corticosteroid and botulinum toxin A injections have been shown to be efficacious in certain subpopulations experiencing HSP and studies centered around new technology in the realm of implantable microstimulators show promising results.

Keywords Hemiplegic shoulder pain · Post-stroke shoulder pain · Complex regional pain syndrome · Shoulder-hand syndrome · Functional electrical stimulation

Introduction

According to the CDC, about 800,000 new strokes occur each year in the United States. This statistic has remained relatively stable over the last 20 years. However, with advancements in medical care, mortality rates have started to

decrease. There were 133,000 deaths due to stroke in 2008, nearly 20,000 less than in 1998. Regardless, stroke remains the fourth leading cause of death in the United States [1]. Over 660,000 Americans survive stroke each year, and it has been noted that nearly half of survivors over the age of 65 will be left with moderate to severe disability [2]. One factor that has not only been shown to decrease functional recovery and quality of life [3–5], but also lead to serious disability in many people with stroke is hemiplegic shoulder pain (HSP). Historically, prevalence has varied drastically between studies (from 5 to 84 %). Postulated reasons include disparities in defining shoulder pain and differences in recruitment criteria [6•]. Recent population-based studies out of New Zealand and Sweden found similar prevalences of 22–23 %. The largest study to date of 1,000 patients in a Turkish rehabilitation hospital found a 55 % prevalence [7–9]. Researchers have shown that HSP reduces participation in the rehabilitation process, leads to poorer recovery of arm function, lowers rates of discharge home and leads to increased length of hospital stay [10]. HSP is both a prevalent and important problem that remains frustrating to treat for physicians. Due to the diverse range of possible etiologies, the exact cause of HSP is often obscure, making efficacious treatment equally as elusive. The purpose of this paper is to explore the pathophysiology underlying HSP as well as the most current treatment options and in doing so, outline a systematic clinical approach to understanding and caring for a person suffering with HSP.

Pathophysiology

Because of the wide array of pathologies potentially underlying the development of HSP, the precise etiology is difficult to assess. It is impossible to treat HSP effectively

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without first understanding the mechanism of injury or disease. In 2011, Kalichman et al. [6•] created a comprehensive narrative review on the etiology and associated factors of HSP, identifying three specific types of possible pathologic processes: (1) soft-tissue lesions, (2) impaired motor control (specifically muscle tone changes), and (3) altered peripheral and central nervous system activity. Importantly, the authors noted that each could present as separate phenomenon, coexist, or evolve to trigger each other's development.

Soft tissue injuries are some of the most common etiologies of HSP. Included in this category are rotator cuff tendinopathies and tears, biceps tendinopathy, subacromial and subdeltoid bursitis, adhesive capsulitis and myofascial pain of the shoulder region [6•]. Many studies over the past two decades have demonstrated a high incidence of impingement syndrome and other rotator cuff pathologies in hemiplegic patients. One reported 61 % of patients suffering from HSP in an acute rehabilitation setting had impingement syndrome, while 33 % had evidence of a rotator cuff tear [4]. Another used MRI to show that 35 % of chronic stroke survivors with HSP had a tear of the biceps, rotator cuff or deltoid tendons, while 53 % exhibited tendinopathy [11]. An important study suggested that such injuries may be due in part to trauma sustained during post-stroke rehabilitation. Najenson et al. [12] noted that rotator cuff tears or tendinopathies could be related to passive abduction greater than 90° or forced abduction without lateral rotation, specifically referring to the use of overhead pulleys. Adhesive capsulitis has been found in as many as 43–77 % of stroke survivors [6•]. It should be noted that adhesive capsulitis can be both the cause and effect of HSP. Bender et al. [13] suggested that HSP may trigger the development of adhesive capsulitis due to immobilization of the affected limb, disuse atrophy or contracture, while Joynt observed that adhesive capsulitis during the acute stage is often quite painful [14]. A detailed history and physical examination coupled with appropriate imaging help differentiate between these soft tissue injuries.

Impaired motor control, more specifically spasticity or flaccidity of the shoulder girdle muscles, has been shown to contribute to HSP. Flaccidity may lead to shoulder joint instability and subluxation, which in turn can cause significant soft tissue and nerve dysfunction including peripheral nerve compression, adhesive changes in associated tendons, and traction injuries to the brachial plexus [15–17]. Though it seems intuitive that these pathologic changes would cause pain, the precise relationship between shoulder subluxation and HSP remains unclear. Increased tone of the muscles responsible for scapular movement may also result in abnormal scapulohumeral rhythm and lead to impingement of the rotator cuff or other structures in the subacromial space [18]. Other studies support this finding empirically. One found that patients experienced pain reduction and

improvement in range of motion after phenol motor point blocks were performed on the subscapularis muscle, while another found reductions in reported pain scores with surgical release of muscular contractures in the shoulder girdle [19, 20]. To further illustrate this point, a Cochrane review of six randomized, controlled trials showed that one injection of botulinum toxin A into a spastic shoulder muscle effectively decreased pain and increased range of motion in patients with HSP when compared to placebo and intra-articular steroid injection [21•].

Altered peripheral and central nervous activity may also contribute to HSP. However, this association remains ambiguous. Researchers have suggested that peripheral nerve entrapment, central sensitization and central post-stroke pain all play a role in HSP, though studies remain incongruent and inconclusive [6•]. Complex regional pain syndrome (CRPS or shoulder-hand syndrome) in stroke survivors has been more formally studied with a reported incidence ranging between 12.5 and 61.4 %, with the variation likely due to inconsistent diagnostic criteria [22, 23]. Usually appearing within the first 3 months following stroke, CRPS involves dysfunction of the sympathetic nervous system. It has been postulated to originate from several different cortical processes, including tissue damage due to the primary lesion and localized neurogenic inflammation causing peripheral sensitization [24, 25]. Researchers have yet to show empiric evidence of a causal relationship between HSP and CRPS.

Therapeutic Modalities

Occupational therapy remains the first line of treatment in HSP. Proper positioning of the hemiplegic upper limb is among the major goals of treatment in early stroke management and has been long shown to prevent trauma to shoulder joint components [26]. This can be achieved with numerous devices, including pillows, custom made foam cushions and troughs that attach to wheelchair armrests. It should be noted that there have been no studies that show a causal relationship between incorrect positioning and HSP. Carr and Kenney [27] explored all relevant literature on the subject and noted a general concurrence that the affected shoulder should be protracted, the arm forward, the wrist neutral and the fingers extended. They did not account for the individual's need to change position throughout the day. Some authors have suggested wearing a sling around the clock to provide support and correct positioning of the hemiplegic arm while also preventing shoulder subluxation during movement [28]. This supposition hinges on the belief that subluxation of the shoulder causes or contributes to HSP. Unfortunately, there are many studies investigating this relationship and none have clearly defined an association

[29]. Based on these studies as well as his own research, Hurd et al. [30] concluded that HSP could not be prevented by the use of a sling. Some authors even argue that a sling holds the upper limb in a position that encourages the natural direction of muscle tightening and shortening following a stroke [31]. This can lead to misuse or disuse of the limb as well as muscle contractures, all of which are known contributors to the development of HSP.

One of the most common therapeutic techniques is centered on the idea that patients should be moved and positioned into reflex inhibiting positions, thereby directing the hemiparetic limb away from abnormal increases in muscle tone [31]. According to Bobath [32], reflex inhibiting patterns prevent the development of inefficient movement and ensure that normal tone is maintained in the affected limb. She also states that integration of the affected upper limb into all daily activities is imperative for successful recovery. Unfortunately, empirical evidence for application of this theory remains inconclusive, as it does for all motor remediation theories [31]. Call and Shepherd [33] endorsed daily, specific stretches and range of motion exercises in the prevention of HSP. By performing each daily exercise for 20 min, it was thought that changes in soft tissue length in the shoulder complex could be prevented. Later studies refuted this idea, finding no differences between levels of pain for those that performed the stretches and those that did not [34].

Another relatively new rehabilitation technique has not only been shown to effectively treat mild to moderate arm and hand dysfunction after stroke, but also has been noted to potentially prevent HSP [35]. Constraint induced therapy as described by Taub et al. [36] requires a patient to wear a restraining mitt on the unaffected upper limb for 90 % of waking hours. It is thought that with repetitive task training of the affected limb and restraint of the unaffected limb, the non-affected brain hemisphere will be inhibited, thus allowing reorganization of the affected side. This leads to functional recovery and prevention of learned disuse of the affected limb. Khan et al. [35] found that constraint induced therapy and conventional neurological therapy based on Bobath's theory were equivalent with respect to improvement of arm and hand dysfunction in the short and intermediate term. Interestingly, people who received constraint induced therapy were at a lower risk of developing HSP at 6 months than those that received conventional therapy [35]. It should be noted that other studies have shown low therapeutic compliance rates with constraint induced therapy and also that the benefits of this type of intensive program may not outweigh the considerable monetary and time costs needed to run it [37].

Occupational therapy remains the front line of prevention of and treatment for HSP. But, recent research studies make it clear that alone it is not sufficient to effectively treat this condition.

Medications and Injections

Providing symptomatic pain relief by prescribing nonsteroidal anti-inflammatory drugs (NSAIDs) and other oral analgesics is common practice in the management of shoulder disorders, both in the general population and in the post-stroke population [38]. Though there have been no trials that have focused on efficacy of NSAIDs specifically for the HSP population, other systematic reviews of the use of NSAIDs in the management of general shoulder pain and dysfunction have been inconclusive. One large review of 31 trials looked at a range of interventions for shoulder pain including NSAIDs [39]. After calculating effect sizes for the same reported outcome measures in the 31 trials, the authors concluded that there was little evidence to either support or refute the efficacy of NSAID use in the setting of shoulder pain [39]. Some authors support offering a trial of NSAIDs provided there are no contraindications, though there is no real evidence to support their efficacy [28]. In this case, analgesic medications should be promptly stopped if found to be ineffective.

A survey performed in The Netherlands in the year 2000 showed that most physicians believed steroid injections were effective in the management of HSP [40]. Unfortunately, local injection with corticosteroids has been shown to have disappointing results in the HSP population, unless used specifically to treat inflammatory conditions such as rotator cuff or bicipital tendonitis, subacromial bursitis or adhesive capsulitis [28]. Modest relief has been shown in some cases when corticosteroid injections are placed in the subacromial bursa. However, this is presumably in the setting of rotator cuff impingement or tendonitis [41]. As reviewed earlier, HSP has many possible etiologies, many of which would not be effectively treated with intra-articular or subacromial corticosteroid injections. In light of the fact that repeated steroid injections may have atrophic effects that could further weaken the rotator cuff, it has been suggested that they be used with caution [28]. Often physicians use a combination of steroid injection and concurrent occupational therapy to help narrow their differential diagnosis, knowing that inflammatory lesions will likely improve with this treatment program.

Suprascapular nerve blocks have long been used in the treatment of chronic shoulder pain due to rheumatoid arthritis, persistent rotator cuff lesions and resistant adhesive capsulitis [42–44]. Due to its sensory innervation of the shoulder, the suprascapular nerve is a sensible target for HSP treatments as well. However, results have been inconclusive. Lee and Khunadorn [45] reported that the efficacy of such a nerve block in HSP was poor and failed to provide complete pain relief. More recent studies have shown that it is a simple, safe and inexpensive technique that can potentially be effective in decreasing shoulder pain. Yasar et al. [46]

compared pain scores for patients receiving either intra-articular corticosteroid injections or suprascapular nerve blocks and found that both treatments were effective in decreasing HSP. One must interpret these results with caution, remembering that inflammatory lesions will respond to both interventions, while other HSP etiologies may not. Because suprascapular nerve blocks have a fairly low side-effect profile, physicians could use this intervention confidently to decrease pain related to HSP or to help understand the etiology of their patient's HSP.

CRPS comprise a specific subpopulation of those with HSP. The exact relationship between CRPS and HSP has yet to be defined; however, those identified as having this devastating disease will need early diagnosis and specific management to ensure a good prognosis. Sympathetic blockade of the stellate ganglion followed by intensive rehabilitation was noted in very early studies to be the most effective treatment [47, 48]. Refractory cases have been treated with surgical sympathectomy [47]. More recent trials have focused on non-surgical interventions, which have proven to be fairly successful. Braus et al. [49, 50] reported relief in 31/36 people treated with low-dose oral corticosteroids, a finding which was later supported by systematic review. Hamamci et al. [51] reported a significant reduction in pain and increased range of motion in 25 people treated with salmon calcitonin when compared to 16 control subjects. The use of opioids and anti-epileptic medications such as gabapentin are also considered in the treatment of CRPS.

As a clear relationship between spasticity and shoulder pain has been defined, it was natural that those interventions aimed at decreasing muscle tone would be used in the treatment of HSP. More specifically, botulinum toxin type A injection has been studied extensively over the last decade and has been shown to be beneficial for patients with HSP. A recent Cochrane review reported that a single injection of botulinum toxin A decreased pain and improved shoulder function in those with chronic shoulder pain due to spastic hemiplegia or arthritis [21••]. Of the six randomized trials

reviewed, five used intramuscular injection of botulinum toxin A in stroke survivors with HSP (Table 1) while one looked at intra-articular injection of 100 units of botulinum toxin A in subjects with end-stage arthritis [57]. It should be noted that laboratory observations made over the last 10 years suggest that botulinum toxin A directly inhibits peripheral nerve nociceptor activation by local neurotransmitter release, thus indirectly reducing central sensitization in spinal cord neurons [21••]. Inhibition of these pathways is very important in the treatment of chronic pain. In fact, botulinum toxin A was shown to inhibit release of substance P during in vitro studies of embryonic rat dorsal neurons [58]. Substance P acts as a neurotransmitter and is associated with inflammatory processes and pain pathways. Therefore, by inhibiting it, those pathways are also inhibited. These observations, amongst many others, suggest that there is an anti-nociceptive effect of botulinum toxin A that is likely independent of its well described paralytic action. Regardless, it has been shown to be an effective treatment for HSP.

Surface Electrical Stimulation

Electrical stimulation as a therapeutic modality was first described over 50 years ago by Liberson [59]. Application of an electrical current to the skin has been shown to stimulate lower motor nerves and muscle fibers, resulting in improved contractility and greater muscle bulk. Importantly, it has also been shown to decrease sensory cortex activation via stimulation of unmyelinated afferent neurons [60]. Clinical reports have suggested that electrical stimulation can improve muscle strength, joint malalignment, muscle tone, sensory deficits, and self-reported pain intensity [61]. There are two types of electrical stimulation: functional electrical stimulation (FES) and transcutaneous electrical stimulation (TENS). FES causes contraction of muscles in an organized fashion to facilitate the recovery of limb function and reduction of spasticity. It usually includes stimulation of the deltoid and/or supraspinatus muscles when used in the

Table 1 Muscles injected with botulinum toxin type A and the amount injected in studies looking at patients with HSP included in a recent Cochrane review

Authors	Muscles injected	Units of botulinum toxin A injected
de Boer et al. [52]	Subscapularis	100 units
Kong et al. [53]	Biceps brachii	250 units
	Pectoralis major	250 units
Lim et al. [54]	Infraspinatus	Each muscle was injected at 2 points and no more than 50 units were used in any one muscle. The maximum dose any subject received was 100 units total.
	Subscapularis	
	Pectoralis	
Marco et al. [55]	Pectoralis major	4 sites were injected with a total of 500 units
Yelnik et al. [56]	Subscapularis	500 units

treatment of HSP. TENS is a modality specifically used to modulate the pain pathway by stimulating cutaneous peripheral nerves anywhere on the body at a lower intensity and higher frequency without causing muscle contraction.

The use of FES in treatment of upper limb hemiplegia has been investigated thoroughly over the last two decades. Though studies have found that FES is associated with reduction in shoulder subluxation and improvement in motor function, very few of these studies looked at its effect on pain scores. For those that did, outcomes varied widely. A few investigators report some degree of improvement in HSP, with one specifically noting that these effects were short lived and did not last after FES was discontinued [62]. A study out of Geneva refuted the assertion that the effect was short lived. The authors noted that a group of subjects receiving FES 2 h daily, 5 days a week for 5 weeks had greater improvements in pain than their counterparts in the control group. Furthermore, the authors noted that these results improved after 1 year and remained stable for up to 24 months [63]. Alternatively, a meta-analysis conducted by Ada and Foongchomcheay found there was no evidence suggesting FES was superior to conventional therapy in the reduction of pain [64]. A recent Cochrane review looked at four studies investigating the effect of electrical stimulation on prevention and treatment of post-stroke shoulder pain. Unfortunately, they were unable to find sufficient evidence to either confirm or refute that electrical stimulation (either transcutaneous or functional) around the shoulder after stroke influences reports of pain [61]. The use of TENS in the treatment of a hemiplegic upper limb is even less understood, again with most studies focusing on functional outcomes like range of motion rather than on pain management.

At this point, FES remains an important part of early post-stroke therapy, more for its effects on functional outcomes such as decreased subluxation and increased range of motion than pain prevention or management. Even with this, the evidence is still not conclusive [65].

Percutaneous or Intramuscular Electrical Stimulation

One of the more exciting advancements in HSP management is currently still in the experimental phase of development. With the success of FES to improve shoulder range of motion, subluxation and to some extent pain, clinicians postulated that this type of stimulation could likely be delivered in a more efficacious manner. Percutaneous peripheral nerve stimulation was first created to deliver electric stimulation directly to the motor points of specific muscles including the supraspinatus, posterior and middle deltoid and upper trapezius via a small implanted radiofrequency microstimulator [66]. A series of pilot studies

suggested that percutaneous nerve stimulation was better tolerated by patients than FES and effective in reducing HSP [67, 68]. The first generation of intramuscular stimulators nearly eradicated HSP from patients. Researchers further refined the implantation procedure and the stimulator. Stimulations of the posterior and middle deltoid muscle locations had been shown to consistently reduce subluxation in hemiparetic shoulders and possibly help with neuromodulation of the mixed axillary nerve [69, 70]. After studying previous trials, it was postulated that a single-lead placement between the middle and posterior deltoid muscle locations would be equally efficacious at reducing pain. Wilson et al. [69] described the implantation of the single lead stimulator. The frequency, pulse duration and current are controlled by a stimulator which is connected to the implanted lead with a wire. These settings can be adjusted to optimize stimulation.

Wilson et al. [69] hypothesized that the single-lead approach would likely show similar clinical results and involve a less technically complicated placement, thus leading to wider physician adoption. They reported that a subject who received stimulation from a single lead for 6 h a day for 3 weeks had a reduction in pain score from 8/10 at baseline to 2/10 4 weeks post treatment. The person also noted substantial improvements in quality of life and at 13 months post-treatment the patient had complete resolution of his HSP. Yu et al. [70] using a different device to implant a single lead microstimulator, achieved similar results. The subject reported 8/10 baseline shoulder pain and after 12 weeks of treatment reported a pain level of 4/10. The pain further decreased to a 3/10 at 3 months follow-up. It was also noted that passive range of motion and motor function improved after 12 weeks of treatment but that quality of life and subluxation did not change [70].

Fully implantable percutaneous electrical stimulators are quite promising. The first placement of a fully internal implantable peripheral nerve stimulator was reported in 2011 [71]. In this case, the subject's baseline pain of 8/10 decreased to 1/10 by 2 weeks and reached complete resolution by 3 weeks. Improvement in quality of life and decreased pain interference with activities of daily living were also noted [71]. Loeb et al. [72] have postulated that implantable neuromuscular stimulation will successfully treat other complications of paralysis and disuse atrophy, including hand contractures, drop foot and osteoarthritis. Their devices have proved to be equally as efficacious as surface stimulators in objective measures and are better tolerated by patients [72]. Additional studies are currently being conducted to further investigate the safety and efficacy of peripheral and fully implantable systems. Though it is too early to tell, intramuscular electrical stimulators may prove to be one of the only interventions that provide long lasting relief for those people who have HSP.

Conclusion

HSP is a prevalent problem and can be devastating for those it affects. It has been shown that prevention measures prompt diagnosis and early appropriate management result in more favorable outcomes [73]. Unfortunately, studies have also noted that 20–30 % of stroke survivors with moderate to severe impairment will have HSP that is refractory to current available treatments [74, 75]. Early therapy remains the mainstay of treatment for patients. Physicians may also try certain oral medications. However, local injections, including corticosteroids and botulinum toxin A, have been shown to be more efficacious. Current research, being conducted to study percutaneous electrical stimulation in the treatment of HSP, has yielded promising results. New solutions to the problem of chronic pain and more specifically HSP may be within our reach.

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