11 Hemiplegic Shoulder Pain & Complex Regional Pain Syndrome

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Abstract

Shoulder pain resulting from hemiplegia is a common clinical consequence of stroke. Hemiplegic shoulder pain can occur as early as two weeks post-stroke but an onset of two to three months is more typical. Shoulder pain can negatively affect rehabilitation outcomes as good shoulder function is a prerequisite for successful transfers, maintaining balance, effective hand function, and performing activities of daily living. Similarly, complex regional pain syndrome is pervasive in hemiplegic patients and can lead to significant medical complications. This review summarizes the existing literature with regard to hemiplegic shoulder pain after stroke. Topics include prevalence, causes, impact, and management of hemiplegic shoulder pain with in-depth discussion of shoulder subluxation, spasticity, contractures, and rotator cuff disorders. Management techniques including shoulder positioning and related aids, intraarticular and intramuscular injections, electrical stimulation, active physical therapies, and other miscellaneous interventions are addressed. Finally, a discussion of the physiology, incidence, diagnosis, and treatment of complex regional pain syndrome is included.
Key Points

Hemiplegic Shoulder Pain

- There is a wide variety of potential risk factors for hemiplegic shoulder pain.
- The association between shoulder subluxation and hemiplegic shoulder pain is unclear.
- Hemiplegic shoulder pain may be associated with spastic muscle imbalance and contracted shoulder.
- Hemiplegic shoulder pain is not associated with rotator cuff disorders.
- There is high variability in the reported frequency of hemiplegic shoulder pain.
- Hemiplegic shoulder pain may be associated with poorer upper limb abilities and lower quality of life.
- Sustained positioning and static stretching of the hemiplegic shoulder may not be effective in reducing pain or improving motor function.
- Slings may be effective in reducing subluxation and pain of the hemiplegic shoulder. While a wide variety of options are available, it is unclear which is the most effective.
- Strapping/taping may be effective in reducing hemiplegic shoulder pain. While a wide variety of options are available, it is unclear which is the most effective.
- Active therapies for the hemiplegic shoulder may be effective in reducing pain, increasing range of motion, and improving motor function. While a wide variety of options are available, it is unclear which is the most effective.
- Surface neuromuscular electrical stimulation may be effective in reducing subluxation and improving range of motion in the hemiplegic shoulder, although its effectiveness may be negatively correlated with stroke onset.
- Intramuscular neuromuscular electrical stimulation may be effective in reducing hemiplegic shoulder pain, although its effectiveness may be negatively correlated with stroke onset.
- Transcutaneous electrical nerve stimulation may be effective in improving range of motion in the hemiplegic shoulder, although it may only be effective at higher intensity.
- Functional electrical stimulation may be effective in reducing subluxation and improving motor function in the hemiplegic shoulder.
- Peripheral nerve stimulation may be effective in reducing hemiplegic shoulder pain; however the evidence is limited.
- Interferential electrical stimulation may be effective in reducing hemiplegic shoulder pain; however the evidence is limited.
- High-voltage pulsed galvanic stimulation may be effective in reducing subluxation and joint displacement in the hemiplegic shoulder; however the evidence is limited.
- Extracorporeal shockwave therapy may be effective in reducing hemiplegic shoulder pain; however the evidence is limited.
- Botulinum toxin may be effective in reducing pain and improving range of motion in the hemiplegic shoulder, but only when delivered in higher doses.
• Given conflicting findings, further research is required to determine the efficacy of triamcinolone acetonide injections in treating hemiplegic shoulder pain.

• Hyaluronic acid may be effective in treating hemiplegic shoulder pain, although further research is warranted.

• Suprascapular nerve blocks may be effective in treating hemiplegic shoulder pain, although further research is warranted.

• Segmental neuromyotherapy may be effective in improving hemiplegic upper limb motor function; however, the evidence is limited.

• Surgical interventions may be effective in reducing hemiplegic shoulder pain; however, the evidence is limited.

• Acupuncture may be effective in reducing pain, increasing range of motion, and improving motor function in the hemiplegic shoulder.

• Massage therapy may reduce hemiplegic shoulder pain and promote physiological relaxation, although further research is warranted.

• Combined acupressure and aromatherapy may reduce hemiplegic shoulder pain; however, the evidence is limited.

Complex Regional Pain Syndrome

• Complex regional pain syndrome is characterized by numerous peripheral and central nervous system changes in the absence of obvious nerve injury.

• The pathophysiology of complex regional pain syndrome is not fully understood.

• There is high variability in the reported frequency of complex regional pain syndrome.

• There is no ideal diagnostic test for the identification of CRPS.

• Corticosteroids are an effective treatment for reducing the severity of complex regional pain syndrome.

• Nerve blocks may be effective in managing the symptoms of complex regional pain syndrome, although further research is required.

• Mirror imagery therapy is an effective treatment for reducing pain and improving motor function in complex regional pain syndrome.

• Combined physiotherapy and aerobic exercise may be effective in reducing pain and improving psychosocial outcomes in complex regional pain syndrome.

• Passive range of motion exercises may prevent the development of complex regional pain syndrome, although further research is required.

• Acute injections of calcitonin may prevent the development of complex regional pain syndrome, although further research is required.

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11.1 Hemiplegic Shoulder Pain

Shoulder pain resulting from hemiplegia is a common clinical consequence of stroke and can result in significant disability (Najenson et al., 1971; Poduri, 1993). The pathogenesis of hemiplegic shoulder pain (HSP) is multifactorial and includes neurologic and mechanical factors, often in combination, which vary among individuals post stroke (Table 11.1.1).

Factors most frequently associated with HSP are glenohumeral subluxation (Grossens-Sills & Schenkman, 1985; Moskowitz et al., 1969; Savage & Robertson, 1982; Shai et al., 1984), adhesive capsulitis, (Blok & Bayer, 1978; Braun et al., 1971; Fugl-Meyer et al., 1974; Grossens-Sills & Schenkman, 1985; Hakuno et al., 1984; Rizk et al., 1984) and spasticity, particularly of the subscapularis and pectoralis muscles (Caldwell et al., 1969; Moskowitz, 1969; Moskowitz et al., 1969). Suggested causes of HSP include complex regional pain syndrome (CRPS) (Chu et al., 1981; Davis et al., 1977; Perrigot et al., 1975), or injury to the rotator cuff musculotendinous unit (Najenson et al., 1971; Nepomuceno & Miller, 1974). The role of central post-stroke pain in the etiology of shoulder pain is unclear (Walsh, 2001).

Based on clinical and arthographic findings, Lo et al. (2003) catalogued the different types of shoulder dysfunction in patients with HSP within a year of stroke: 50% had adhesive capsulitis, 16% had complex regional pain syndrome, and 4% had rotator cuff tears. As well, 63% of patients had a single type of shoulder dysfunction, while 34% had two types (Lo et al., 2003). In another imaging study, researchers examined a potential association between structural changes in the hemiplegic shoulder and pain (Shah et al., 2008): 35% of patients with shoulder pain exhibited a tear of the rotator cuff, biceps, or deltoid muscle, and 53% exhibited tendinopathy of the rotator cuff, bicep, or deltoid muscle. However, these changes were not related to severity of shoulder pain (Shah et al., 2008).

Multivariable analyses by Kim et al. (2014) determined significant factors associated with an increased risk of developing HSP: age greater than 70, poor arm motor function, supraspinatus tendon tear/tendinosis, biceps tendon effusion, and adhesive capsulitis. Similarly, Karaahmet et al. (2014) found that HSP was associated with longer disease duration and poor arm motor function. Rajaratnam et al. (2007) identified three factors that predict the development of HSP in acute stroke with 98% accuracy: (1) a positive Neer test; (2) shoulder pain during the hand behind the neck manoeuvre; and (3) a difference of greater than 10° of passive external rotation at the shoulder joint.

Conclusions Regarding Factors Associated with Hemiplegic Shoulder Pain

Factors associated with hemiplegic shoulder pain include older age, longer disease duration, poor arm function, muscle imbalance, rotator cuff tear, subscapularis/pectoralis spasticity, glenohumeral subluxation, bursitis, tendonitis, adhesive capsulitis, and complex regional pain syndrome.

There is a wide variety of potential risk factors for hemiplegic shoulder pain.
11.2 Shoulder Subluxation and Hemiplegic Shoulder Pain

11.2.1 Pathophysiology
Shoulder subluxation is best defined as changes in the mechanical integrity of the glenohumeral joint that results in an incomplete dislocation, where articulating surfaces of the glenoid fossa and humeral head remain in contact. Reliable clinical measurements of the subacromial space used in clinical research include callipers (Boyd & Torrance, 1992), ultrasound, CT, and MRI (McCreesh et al., 2015). The glenohumeral joint is multiaxial and has a range of motion exceeding that of other joints in the body. In order to achieve this mobility, the glenohumeral joint must sacrifice stability. Stability is achieved through the rotator cuff, a musculotendinous sleeve that maintains the humeral head in the glenoid fossa, while at the same time allowing shoulder mobility. During the initial period following a stroke the hemiplegic arm is flaccid or hypotonic. Therefore the shoulder musculature, in particular the rotator cuff musculotendinous sleeve, cannot perform its function of maintaining the humeral head in the glenoid fossa and there is a high risk of shoulder subluxation (Figure 11.2.1).

![Figure 11.2.1A Normal Shoulder](image1)
*The humeral head is maintained in the glenoid fossa by the supraspinatus muscle.*

![Figure 11.2.1B Shoulder Subluxation](image2)
*The supraspinatus muscle is flaccid during the initial phase of hemiplegia. The weight of the unsupported arm can cause the humeral head to sublux downward in the glenoid fossa.*

Shoulder subluxation is a common problem in individuals with hemiplegia post stroke. During the initial flaccid stage of hemiplegia, the involved extremity must be adequately supported or the weight of the arm will result in shoulder subluxation. Improper positioning in bed, lack of support in the upright position, and pulling on the hemiplegic arm during transfers all contribute to glenohumeral subluxation.
Inferior subluxation commonly occurs secondary to prolonged downward pull on the arm, against which hypotonic muscles offer little resistance (Chaco & Wolf, 1971). The resulting mechanical effect is overstretching of the glenohumeral capsule, especially its superior aspect, and flaccid supraspinatus and deltoid muscles (Basmajian & Bazant, 1959; Shahani et al., 1981).

11.2.2 Scapular Rotation
Other factors appear to play a role in subluxation of the glenohumeral joint. Basmajian and Bazant (1959) proposed that, in the normal state, subluxation of the humeral head is prevented by upward angulation of the glenoid fossa and the upper part of the shoulder capsule (the coracohumeral ligament and supraspinatus muscle). They hypothesized that, after a hemiplegic stroke, the upward angulation of the scapula is lost. Cailliet (1980) added that the scapula assumes a depressed and downward rotated position in the flaccid stage, as the paretic serratus anterior and the upper part of the trapezius muscles no longer support the scapula. The combination of flaccid supportive musculature, the supraspinatus muscle in particular, and a downward rotated scapula was presumed to predispose the head of the humerus to undergo inferior subluxation relative to the glenoid fossa. In a later study, however, Ikai et al. (1998) reported that the presence of subluxation was found in only 2% of patients with downward scapular rotation.

Using 3D X-Ray technology, Prévost et al. (1987) studied the movement of the scapula and the humerus in stroke patients by comparing the affected and non-affected shoulders. Images demonstrated a significant difference between the affected and non-affected shoulders in terms of the vertical position of the humerus (i.e. degree of subluxation) and glenoid fossa in relation to the scapula. There was no significant relationship noted between the orientation of the scapula and the severity of subluxation. The authors concluded that the scapular position was not an important factor in the occurrence of inferior subluxation in hemiplegia (Prévost et al., 1987), which has been supported by the findings of subsequent studies (Culham et al., 1995; Niessen et al., 2008; Price & Pandyan, 2001).

11.2.3 Pain in Shoulder Subluxation
It has long been assumed that if shoulder subluxation is not corrected, a pattern of traction on the flaccid shoulder will result in pain, decreased range of motion, and contracture (Grossens-Sills & Schenkman, 1985; Moskowitz, 1969; Roy et al., 1994; Savage & Robertson, 1982; Shai et al., 1984). However, it remains controversial as to whether it causes HSP (Bender & McKenna, 2001; Fitzgerald-Finch & Gibson, 1975; Moskowitz et al., 1969; Shahani et al., 1981). While some observational studies have reported a significant correlation between subluxation and pain in the hemiplegic shoulder (Aras et al., 2004; Lin et al., 2014; Lo et al., 2003; Paci et al., 2007; Suethanapornkul et al., 2008), several others failed to find such a relationship (Barlak et al., 2009; Bohannon, 1988; Bohannon & Andrews, 1990; Ikai et al., 1998; Joynt, 1992; Lin et al., 2014; Mohamed et al., 2014; Van Langenbergh & Hogan, 1988; Wanklyn et al., 1996; Zorowitz et al., 1996).

To be sure, patients with shoulder subluxation may not have HSP and patients with HSP may not have shoulder subluxation. The failure to consistently report an association may be due in part to a failure to examine the contribution of other probable etiological factors occurring concurrently. Paci et al. (2005) suggested that pain associated with subluxation likely presents later after stroke as “fibrous changes or injury can occur in connective tissue of the ligaments and joint capsule due to incorrect alignment between the humerus and the scapula”. As well, the lack of consistency among findings may be related to the heterogeneity of patient characteristics and method/timing of assessment.
Conclusions Regarding Shoulder Subluxation and Hemiplegic Shoulder Pain

Shoulder subluxation may occur early on in the hemiplegic arm due to flaccid supporting shoulder musculature and can be exacerbated by external forces.

Shoulder subluxation may be associated with hemiplegic shoulder pain, although patients with shoulder subluxation may not experience pain and patients with pain may not have subluxed shoulder.

The association between shoulder subluxation and hemiplegic shoulder pain is unclear.

11.3 Spasticity, Contractures, and Hemiplegic Shoulder Pain

11.3.1 Pathophysiology
Spasticity is defined as a disorder of motor function characterized by a velocity-dependent increase in resistance to passive stretch of muscles accompanied by hyperactive stretch reflexes and often associated with a clasp-knife response. Under normal circumstances, a delicate balance exists between facilitating and inhibiting influences upon both alpha and gamma motor neurons, which together maintain appropriate control of skeletal muscle length and strength of contraction at the spinal cord level. After a stroke, input from one or more of the supraspinal reflex inhibitors decreases or stops entirely. The balance of control over the muscle favours facilitation, resulting in spasticity. Spasticity develops only if there is loss of input from both pyramidal and extrapyramidal motor systems.

The relationship between spasticity and HSP has been explored in several observational studies. In an early study, van Ouwenaller et al. (1986) identified spasticity as "the prime factor and the one most frequently encountered in the genesis of shoulder pain in the hemiplegic patient." In patients followed for one year after stroke, the authors identified a much higher incidence of shoulder pain in spastic (85%) than in flaccid (18%) hemiplegia. Poulin de Courval et al. (1990) similarly reported that subjects with shoulder pain had significantly more spasticity of the affected limb than those without pain. In contrast, Bohannon et al. (1986) and Joynt (1992) found that spasticity was unrelated to shoulder pain in patients with post-stroke hemiplegic shoulder.

11.3.2 Spastic Muscle Imbalance
Hemiplegia following stroke is characterized by typical posturing reflecting hypertonic muscle patterns. Flexor tone predominates in the hemiplegic upper extremity and results in scapular retraction and depression as well as internal rotation and adduction of the shoulder. This posture is the consequence of damage to higher centers and subsequent release of motor groups from pyramidal and extrapyramidal control. In stroke recovery, this "synergy pattern" of muscles is inevitable where recovery is incomplete, which can result in the development of spastic muscle imbalance around the shoulder joint.

The internal rotators of the shoulder predominate but are one of the last areas of shoulder function to recover. Motor units are not appropriately recruited during recovery, yielding the simultaneous co-contraction of agonist and antagonist muscles. A shortened agonist in the synergy pattern becomes stronger and the constant tension of the agonist can become painful; stretching of these tightened spastic muscles causes more pain. Tightened muscles inhibit movement, reduce range of motion, and prevent other movements, especially at the shoulder where external rotation of the humerus is necessary for arm abduction greater than 90°. Muscles that contribute to spastic internal
rotation/adduction of the shoulder include the subscapularis, pectoralis major, teres major, and latissimus dorsi. However, two muscles in particular have been implicated as most often being spastic leading to muscle imbalance: (1) subscapularis and (2) pectoralis major.

Subscapularis Spasticity Disorder
The subscapularis muscle originates on the undersurface of the scapula and inserts on the lesser tuberosity of the humerus as well as the capsule of the shoulder joint (Figure 11.3.2.1). It is a major internal rotator of the shoulder (Hollinshead & Jenkins, 1981) and participates in arm adduction and extension from a flexed position (Cole & Tobis, 1990). In a normal state, nerve impulses to the subscapularis are inhibited during arm abduction; the muscle relaxes and allows the humerus to externally rotate, thus preventing impingement of the greater tuberosity on the acromion (Codman, 1934). As part of the typical flexor synergy pattern in those with spastic hemiplegia, internal rotators such as the subscapularis muscle are tonically active, which limits shoulder abduction, flexion, and external rotation (Bohannon et al., 1986; Hecht, 1995; Zorowitz et al., 1996).

Figure 11.3.2.1 Subscapularis Muscle

Subscapularis spasticity disorder is characterized by motion being most limited and pain being reproduced on external rotation, as a tight band of spastic muscle is palpated in the posterior axillary fold. In fact, Inaba and Piorkowski (1972) reported external rotation was the most painful and limited movement of the hemiplegic shoulder. Subsequent studies have reported that limitation of external rotation of the hemiplegic shoulder was strongly correlated with HSP (Bohannon et al., 1986; Hecht, 1995; Zorowitz et al., 1996), suggesting that the subscapularis is “the keystone of the abnormal synergy pattern” (Hecht, 1995).

Pectoralis Spasticity Disorder
The pectoralis major muscle serves to forward flex, adduct, and internally rotate the arm, and is a synergist of the subscapularis muscle (Figure 11.3.2.2). Hecht (1995) reported on a subset of hemiplegic patients with greater limitations in abduction and flexion than on external rotation. In these patients, a spastic pectoralis major muscle appeared to be most problematic. This disorder is characterized by motion being most limited and pain produced on abduction.
Figure 11.3.2.2 The Pectoralis Major Muscle

The importance of other shoulder muscles (i.e. biceps, pectoralis minor, and latissimus dorsi) have not been studied in the stroke population. A review by Kalichman & Ratmansky (2011) outlines a systematic approach to the underlying causes of HSP (Figure 11.3.2.3). The authors suggest that shoulder spasticity can lead to soft tissue lesions and/or altered peripheral and central nervous system activity, which can play a substantial role in evoking HSP. These issues may occur separately, co-exist simultaneously, or develop as a result of a trigger from a previous symptom (Kalichman & Ratmansky, 2011).

Figure 11.3.2.3 Systemization of Pathologies Underlying HSP
Adapted from Kalichman & Ratmansky (2011)
11.3.3 Contracted/Frozen Shoulder

Adhesive capsulitis, also known as the contracted or frozen shoulder, is characterized by limitations in range of movement with a pattern of restriction. The incidence of contractures in frozen shoulder has been found to range between 54.6% and 76.7% (Hakuno et al., 1984; Rizk et al., 1984). This condition is frequently identified as a source of spastic HSP (Bohannon et al., 1986; Eto et al., 1980; Fugl-Meyer et al., 1974; Grossens-Sills & Schenkman, 1985; Hakuno et al., 1984; Rizk et al., 1984).

Conclusions Regarding Spasticity, Contractures, and Hemiplegic Shoulder Pain

Spastic muscle imbalance of the shoulder adductors and internal rotators, particularly the subscapularis and pectoralis major, appears to be associated with hemiplegic shoulder pain.

Adhesive capsulitis and its associated limited range of movement appear to be associated with hemiplegic shoulder pain.

Hemiplegic shoulder pain may be associated with spastic muscle imbalance and contracted shoulder.

11.4 Rotator Cuff Disorders and Hemiplegic Shoulder Pain

Rotator cuff disorders are commonly found with shoulder pain, and thus have been proposed as a potential cause of HSP. However, Rizk et al. (1984) failed to demonstrate any evidence of rotator cuff tears on arthrography in patients with HSP. Najenson et al. (1971) reported that only 40% of patients with severe paralysis of the upper extremity were found to have a rupture of the rotator cuff tendon. A similar study reported a 33% incidence of rotator cuff tears in painful shoulders after stroke (Nepomuceno & Miller, 1974).

While partial tears of the rotator cuff musculature may be common, it is difficult determining whether they were present pre-morbidly, even in previously asymptomatic patients. Yamaguchi et al. (2006) evaluated patients with unilateral shoulder complaints and found that 30% patients had bilateral rotator cuff tears and 34% patients had unilateral rotator cuff tears. Furthermore, evidence suggests that the prevalence of asymptomatic rotator cuff tears is 20% in patients 60 to 69 years of age and 40.7% in patients 70 years of age or older (Kim et al., 2009).

Conclusions Regarding Rotator Cuff Disorders and Hemiplegic Shoulder Pain

Rotator cuff disorders do not appear to be associated with hemiplegic shoulder pain.

Hemiplegic shoulder pain is not associated with rotator cuff disorders.

11.5 Frequency of Hemiplegic Shoulder Pain

From on a selection of large-scale observational studies, there appears to be high variability in the reported frequency of HSP, as illustrated in Table 11.5.1.

Table 11.5.1 Frequency of Hemiplegic Shoulder Pain

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>Sample Size</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>van Ouwenaller et al. (1986)</td>
<td>N=219</td>
<td>72%</td>
</tr>
<tr>
<td>Study</td>
<td>Frequency</td>
<td>Timing</td>
</tr>
<tr>
<td>-----------------------------------------</td>
<td>-----------</td>
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</tr>
<tr>
<td>Jespersen et al. (1995)</td>
<td>22%</td>
<td></td>
</tr>
<tr>
<td>Gamble et al. (2000)</td>
<td></td>
<td>0-2wk: 25%</td>
</tr>
<tr>
<td>Langhorne et al. (2000)</td>
<td></td>
<td>0-1mo: 9%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1-6mo: 15%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>6-18mo: 11%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>18-30mo: 12%</td>
</tr>
<tr>
<td>Ratnasabapathy et al. (2003)</td>
<td></td>
<td>1wk: 17%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1mo: 20%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>6mo: 23%</td>
</tr>
<tr>
<td>McLean (2004)</td>
<td>24%</td>
<td></td>
</tr>
<tr>
<td>Lindgren et al. (2007)</td>
<td></td>
<td>4mo: 22%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>16mo: 24%</td>
</tr>
<tr>
<td>Sackley et al. (2008)</td>
<td></td>
<td>3mo: 36%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>6mo: 42%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>12mo: 47%</td>
</tr>
<tr>
<td>Kitisomprayoonkul et al. (2010)</td>
<td></td>
<td>2mo: 15.3%</td>
</tr>
<tr>
<td>Klit et al. (2011)</td>
<td>24mo: 15.1%</td>
<td></td>
</tr>
<tr>
<td>Hansen et al. (2012)</td>
<td></td>
<td>1wk: 1.5%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3mo: 13%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>6mo: 16%</td>
</tr>
<tr>
<td>Adey-Wakeling et al. (2015)</td>
<td></td>
<td>0wk: 10%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4mo: 21%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>12mo: 29%</td>
</tr>
</tbody>
</table>

HSP can occur as early as 1 week post stroke (Ratnasabapathy et al., 2003), although an onset of 2-3 months post stroke is more typical (Poduri, 1993). As well, incidence of HSP appears to increase over time within the first year (Hansen et al., 2012; Langhorne et al., 2000; Ratnasabapathy et al., 2003; Sackley et al., 2008), but may subside in the following months (Langhorne et al., 2000). In a recent study, Adey-Wakeling et al. (2015) reported that the frequency of HSP is on the rise.

Conclusions Regarding Frequency of Hemiplegic Shoulder Pain

The reported frequency of hemiplegic shoulder pain varies from 9% to 72%, which may be influenced by heterogeneity in the type and timing of assessment.

There is high variability in the reported frequency of hemiplegic shoulder pain.

11.6 Functional Impact of Hemiplegic Shoulder Pain

A painful hemiplegic shoulder has the potential to amplify the disability observed with hemiplegia. HSP can also negatively affect rehabilitation outcomes, as good shoulder function is a prerequisite for successful transfers, maintaining balance, performing activities of daily living, and for effective hand function (Rizk et al., 1984). Several studies have attempted to discern the relationship between HSP and functional outcome, but the findings have not been consistent and a cause-effect relationship has not yet been established.
Several studies have reported that HSP was associated with (Aras et al., 2004; Lindgren & Brogardh, 2014; Wanklyn et al., 1996) or predictive of (John Chae et al., 2007a; Ratnasabapathy et al., 2003; Roy et al., 1995) poorer upper limb motor function. Activities of daily living (ADLs) were found to be impacted by HSP in some studies (Roy et al., 1995; Wanklyn et al., 1996), although others did not find such an association (Caglar et al., 2016; John Chae et al., 2007a). As well, HSP has been reported to negatively impact QOL (John Chae et al., 2007a), especially in acute stroke (Adey-Wakeling et al., 2016).

**Conclusions Regarding Functional Impact of Hemiplegic Shoulder Pain**

There is Level 4 evidence that hemiplegic shoulder pain may be associated with poorer upper limb motor function and lower quality of life; the association with functional outcomes is less clear.

Hemiplegic shoulder pain may be associated with poorer upper limb abilities and lower quality of life.

11.7 Management of Hemiplegic Shoulder Pain

Management of the painful hemiplegic shoulder is difficult and response to treatment is frequently unsatisfactory (Rizk et al., 1984). The optimal treatment approach has not been established, in part due to the uncertainty of the etiology of pain. As a result, a wide variety of treatments have been used with varying degrees of success (Snels et al., 2002).

The management of HSP can be greatly improved with the identification of the primary etiology for a given patient by performing a focused physical examination and selecting the appropriate intervention to address these clinical findings. Ideally, measures should be taken immediately following stroke to minimize the potential for the development of HSP. Early passive shoulder range of motion in the initial flaccid stage, as well as supporting and protecting the shoulder, are regarded as important steps to reduce the development of shoulder pain.

11.7.1 Positioning of the Hemiplegic Shoulder

The muscles around the hemiplegic shoulder are often paralyzed, initially with flaccid tone and later with spasticity. A primary goal of early stroke management is to prevent the development of hypertonicity (Johnstone, 1982) and to discourage inefficient patterns (Bobath, 1990). Careful positioning of the shoulder serves to minimize subluxation early on and contractures later on as well as possibly promote recovery, while poor positioning may adversely affect symmetry, balance, and body image.

Gilmore et al. (2004) suggested that the development of HSP can be prevented through careful and correct shoulder positioning. Bender and McKenna (2001) noted that the “recommended position for the upper limb is towards abduction, external rotation and flexion of the shoulder”. However, the authors also noted that “most popular theories failed to yield consensus for exact degrees of the positioning”. Only a handful of RCTs have investigated the effect of various positioning therapies on HSP, which are summarized in Table 11.7.1.1.

**Table 11.7.1.1 Summary of Studies Evaluating Positioning of the Hemiplegic Shoulder**

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>Study Design (PEDro) Sample Size</th>
<th>Interventions</th>
<th>Outcomes</th>
</tr>
</thead>
</table>

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11. Hemiplegic Shoulder Pain & Complex Regional Pain Syndrome www.ebrsr.com
### Discussion

None of the included studies found significant reductions in pain or improvements in range of motion when patients were treated with various shoulder positioning or static stretching procedures relative to other routine rehabilitation activities (Ada et al., 2005b; De Jong et al., 2006; Dean et al., 2000; Gustafsson & McKenna, 2006; Turton & Britton, 2005). These procedures also showed no benefit for motor function (De Jong et al., 2006; Gustafsson & McKenna, 2006), spasticity (De Jong et al., 2006), or independence (Gustafsson & McKenna, 2006).

In a meta-analysis of these studies, Borisova and Bohannon (2009) concluded that shoulder positioning programs were not effective in preventing or reducing external rotation range of motion impairments post stroke (SMD=-0.216, 95%CI -0.573–0.141). The studies provided 20-30 minutes of positioning/stretching per session, 2-3 sessions per day, 4-7 days per week, over a total of 4-12 weeks. The authors speculated that the duration of stretching may have been insufficient, given that at least 6 hours of stretching a day was required to prevent contracture formation in the soleus muscle of individuals with cerebral palsy. The authors also suggested that the treatment may have been initiated too late following stroke to be effective.

### Conclusions Regarding Positioning of the Hemiplegic Shoulder

*There is Level 1a evidence that sustained positioning and static stretching does not reduce pain, increase range of motion, or improve motor function of the hemiplegic shoulder.*

*Sustained positioning and static stretching of the hemiplegic shoulder may not be effective in reducing pain or improving motor function.*

<table>
<thead>
<tr>
<th>Study (Ref)</th>
<th>Type</th>
<th>Intervention</th>
<th>Control</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ada et al. 2005b</td>
<td>RCT (8)</td>
<td>N=36</td>
<td>E: Sustained positioning Position 1: maximum external rotation Position 2: 90° of flexion C: Standard rehabilitation</td>
<td>• Pain (-) • Range of Motion (-) • Contracture: Position 1 (+); Position 2 (-)</td>
</tr>
<tr>
<td>Dean et al. 2000</td>
<td>RCT (7)</td>
<td>N=23</td>
<td>E: Sustained positioning C: Standard rehabilitation</td>
<td>• Pain (-) • Range of Motion (-)</td>
</tr>
<tr>
<td>De Jong et al. 2006</td>
<td>RCT (7)</td>
<td>N=19</td>
<td>E: Sustained positioning C: Standard rehabilitation</td>
<td>• Pain (-) • Range of Motion (-) • Fugl-Meyer Assessment (-) • Modified Ashworth Scale (-)</td>
</tr>
<tr>
<td>Gustafsson &amp; McKenna 2006</td>
<td>RCT (6)</td>
<td>N=34</td>
<td>E: Static positional stretches C: Standard rehabilitation</td>
<td>• Pain (-) • Range of Motion (-) • Motor Assessment Scale (-) • Modified Barthel Index (-)</td>
</tr>
<tr>
<td>Turton &amp; Britton 2005</td>
<td>UK RCT (6)</td>
<td>N=25</td>
<td>E: Static positional stretches C: Standard rehabilitation</td>
<td>• Pain (-) • Range of Motion (-) • Contracture (-)</td>
</tr>
</tbody>
</table>

*+ indicates statistically significant difference between treatment groups - indicates no statistically significant difference between treatment groups E indicates experimental group; C indicates control group*
11.7.2 Sling for the Hemiplegic Shoulder

Arm slings are often used in the initial stages following a stroke to support the affected arm. Their use is controversial due to potential disadvantages such as encouraging flexor synergies, inhibiting arm swing, contributing to contracture formation, and decrease body image causing the patient to further avoid using that arm. However, a sling remains the best method of supporting the flaccid hemiplegic arm while the patient is standing or transferring.

As tone returns to the shoulder muscles, the risk of shoulder subluxation decreases and slings can then be withdrawn. Slings tend to hold the limb in a poor position, which may accentuate the adduction and internal rotation posture and may contribute to shortening of tonically active muscles. Trials evaluating various slings for the hemiplegic shoulder are summarized in Table 11.7.2.1.

Table 11.7.2.1 Summary of Studies Evaluating Slings for the Hemiplegic Shoulder

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>Study Design (PEDro)</th>
<th>Sample Size</th>
<th>Interventions</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hartwig et al. (2012)</td>
<td>RCT (7)</td>
<td>N=41</td>
<td>E: Functional orthosis (Neuro-Lux) C: No orthosis</td>
<td>Should-Hand Syndrome score (+)</td>
</tr>
<tr>
<td>Williams et al. (1988)</td>
<td>PCT</td>
<td>N=26</td>
<td>E1: Bobath sling E2: Henderson sling C: No sling</td>
<td>Subluxation: E1, E2 vs C (+)</td>
</tr>
<tr>
<td>Hurd et al. (1974)</td>
<td>PCT</td>
<td>N=14</td>
<td>E: Sling C: No sling</td>
<td>Pain (+)*</td>
</tr>
<tr>
<td>Brooke et al. (1991)</td>
<td>PCT</td>
<td>N=10</td>
<td>E: Harris sling C: Bobath sling</td>
<td>Subluxation (+)</td>
</tr>
<tr>
<td>Moodie et al. (1986)</td>
<td>PCT</td>
<td>N=10</td>
<td>E1: Bobath sling E2: Harris sling E3: Standard sling E4: Arm trough E5: Lap tray</td>
<td>Subluxation: E3, E4, E5 vs E1, E2 (+)*</td>
</tr>
</tbody>
</table>

*+ indicates statistically significant difference between treatment groups - indicates no statistically significant difference between treatment groups * indicates no statistical inference provided E indicates experimental group; C indicates control group

Discussion
The use of a sling was found to reduce pain (Hurd et al., 1974) and subluxation (Williams et al., 1988) in the majority of the patients when compared to no sling. The effectiveness of different slings is less clear, given inconsistent findings between studies. The Bobath sling was found to be less effective than the Harris sling in one study (Brooke et al., 1991), but another study reported that both were similarly less effective than other devices (i.e. standard sling, arm trough, lap tray) (Moodie et al., 1986). When comparing different types of slings, studies failed to find significant differences between them in terms of reducing subluxation (Williams et al., 1988; Zorowitz et al., 1995).
In an RCT, Hartwig et al. (2012) examined the effect of the functional orthosis Neuro-Lux worn for 10 hours each day over a month. When compared to usual care, patients wearing the device demonstrated lower mean Shoulder-Hand Syndrome scores, and all components of the condition (i.e. pain, hand edema, limited humeral movement) were significantly lower. The majority of the participants also found the orthosis to be comfortable. However, similar to the aforementioned trials, this RCT had small sample size and did not specify the measures of pain. Future trials assessing such devices should consider using larger sample sizes and objective outcome measures.

In a Cochrane review, Ada et al. (2005a) evaluated the benefit of shoulder slings and supports. While the devices were effective in delaying the onset of pain, they were ineffective in reducing pain severity, improving arm function, or attenuating contracture. The authors concluded that there was insufficient evidence that these devices reduce or prevent shoulder subluxation post stroke. With many devices available and limited evidence of their efficacy, the best method to support the shoulder has yet to be determined.

**Conclusions Regarding Slings for the Hemiplegic Shoulder**

*There is Level 1b and Level 2 evidence that slings reduce subluxation and pain of the hemiplegic shoulder.*

*Slings may be effective in reducing subluxation and pain of the hemiplegic shoulder. While a wide variety of options are available, it is unclear which is the most effective.*

### 11.7.3 Strapping/Taping the Hemiplegic Shoulder

Strapping/taping the hemiplegic shoulder is used as a method to prevent or reduce the severity of shoulder subluxation, and may provide some sensory stimulation. There are various products and techniques that have been described previously, including those by Ancliffe (1992), Morin and Bravo (1997), and Hanger et al. (2000). More recently, researchers have explored approaches such as kinesio taping (Jaraczewska & Long, 2006), California tri-pull taping (Hayner, 2012), and neuromuscular taping (Blow, 2012). Trials evaluating the effectiveness of these strapping/taping methods are summarized in Table 11.7.3.1

<table>
<thead>
<tr>
<th>Author, Year Study Design (PEDro) Sample Size</th>
<th>Interventions</th>
<th>Outcomes</th>
</tr>
</thead>
</table>
| **Pillastrini et al.** (2016) RCT (8) N=32   | E: Neuromuscular taping C: No taping | • Pain (+)  
  • Modified Ashworth Scale (-)  
  • Range of Motion (-) |
| **Huang et al.** (2016b) RCT (7) N=49       | E: Kinesio taping C: No taping | • Pain (-)  
  • Range of Motion (-)  
  • Modified Ashworth Scale (-)  
  • Fugl-Meyer Assessment (-)  
  • Modified Barthel Index (-)  
  • Quality of Life (-) |
| **Chatterjee et al.** (2016) RCT (7) N=30    | E: California tri-pull taping C: No taping | • Pain (+)  
  • Active Shoulder Flexion (+)  
  • Acromio-Humeral Distance (-)  
  • Fugl-Meyer Assessment (-) |
**Discussion**

The specific products and techniques utilized were detailed in all but two trials (Appel et al., 2011; Heo et al., 2015). Most trials found that various forms of strapping/taping for 3-6 weeks were effective in reducing pain when compared to no intervention (Ancliffe, 1992; Chatterjee et al., 2016; Heo et al., 2015; Pandian et al., 2013; Pillastrini et al., 2016) or sham intervention (Griffin & Bernhardt, 2006). However, two trials of moderate quality reported that it was not an effective intervention for reducing pain, despite using different methods (Hanger et al., 2000; Huang et al., 2016b); the reason for these different findings is unclear. Motor impairment, functional independence, range of motion, and spasticity were not improved by strapping/taping in any trial, excluding one small, low-quality trial that found improved motor function (Appel et al., 2011).

### Conclusions Regarding Strapping the Hemiplegic Shoulder

There is Level 1a evidence that shoulder strapping/taping reduces hemiplegic shoulder pain; however it may not improve spasticity, disability, range of motion, or motor function. Strapping/taping may be effective in reducing hemiplegic shoulder pain. While a wide variety of options are available, it is unclear which is the most effective.

### 11.7.4 Active Therapies for the Hemiplegic Shoulder

The association of spasticity, contracture, and muscle imbalance with shoulder pain suggests that a therapeutic approach designed to improve range of motion (ROM) may improve pain. Studies examining active therapies for the hemiplegic shoulder are summarized in Table 11.7.4.1.
Table 11.7.4.1 Summary of Studies Evaluating Active Therapies

<table>
<thead>
<tr>
<th>Author, Year Study Design (PEDro)</th>
<th>Sample Size</th>
<th>Interventions</th>
<th>Outcomes</th>
</tr>
</thead>
</table>
| Inaba & Piorkowski (1972) RCT (7) N=33 | E1: ROM exercises + ultrasound  
E2: ROM exercises + positioning  
C: ROM exercises + mock ultrasound | • Range of Motion (-) |
| Lynch et al. (2005) RCT (6) N=35 | E: Continuous passive ROM exercises  
C: Self-ROM exercises | • Pain (-)  
• Modified Ashworth Scale (-)  
• Fugl-Meyer Assessment (-)  
• Joint stability (-) |
| Kumar et al. (1990) RCT (5) N=28 | E1: ROM exercises  
E2: ROM exercises with skateboard  
E3: ROM exercises with overhead pulley | • Pain: E1 vs E3 (+)  
• Subluxation (-) |
| Partridge et al. (1990) RCT (5) N=65 | E: Cryotherapy  
C: Bobath therapy | • Pain: C (+) |
| Jeon et al. (2016) RCT (5) N=12 | E: ‘Monkey chair and band’ therapy  
C: Standard therapy | • Pain (+)  
• Range of Motion (+)  
• Motor Assessment Scale (+) |
| Hafsteinsdottir et al. (2007) PCT N=326 | E: Bobath therapy  
C: Standard therapy | • Pain (-) |
| You et al. (2014) PCT N=45 | E1: Stretching + joint-stabilizing exercise  
E2: Stretching exercise  
C: Standard exercise | • Motor Assessment Scale: E1 vs C (+) |

+ indicates statistically significant difference between groups  
- indicates no statistically significant difference between groups  
E indicates experimental group; C indicates control group

Discussion

The evidence regarding active therapies for the hemiplegic shoulder is highly varied in terms of interventions and findings. An early trial found that the efficacy of ROM exercises was not improved by positioning or ultrasound (Inaba & Piorkowski, 1972), while another trial found potentially detrimental effects with an overhead pulley (Kumar et al., 1990). Self-ROM exercises were found to be just as effective in improving pain, spasticity, joint stability, and motor function as continuous passive motion exercises with the OrthoLogic Danniflex600 system (Lynch et al., 2005).

Bobath therapy was more effective than cryotherapy (Partridge et al., 1990), but no more effective than standard therapy (Hafsteinsdottir et al., 2007), in reducing pain. You et al. (2014) reported that a combination of stretching and joint-stabilizing exercises were more effective than traditional exercise in improving motor function of the affected upper limb. Similarly, Jeon et al. (2016) demonstrated significantly reduced pain, increased ROM, and improved motor function with ‘monkey chair and band’ therapy when compared to standard therapy. It should be noted, however, that the former trial was non-randomized and the latter trial was small and low quality.

Conclusions Regarding Active Therapies for the Hemiplegic Shoulder

There is Level 1b evidence that continuous passive range of motion exercises are not more effective than self-range of motion exercises at improving motor function, joint stability, spasticity, or pain in the affected limb.
There is Level 1b evidence that supplementing range of motion exercises with ultrasound or positioning is not more effective than the exercises alone.

There is limited Level 2 evidence that aggressive range of motion exercises (e.g. overhead pulleys) increase hemiplegic shoulder pain when compared to the exercises alone.

There is limited Level 2 evidence that stretching and joint stabilizing exercises improve motor function of the affected limb when compared to conventional exercises.

There is limited Level 2 evidence that Bobath therapy reduces hemiplegic shoulder pain when compared to cryotherapy but not standard therapy.

There is limited Level 2 evidence that ‘monkey chair and band’ therapy improves motor function, range of motion, and pain in the affected limb when compared to standard therapy.

Active therapies for the hemiplegic shoulder may be effective in reducing pain, increasing range of motion, and improving motor function. While a wide variety of options are available, it is unclear which is the most effective.

11.7.5 Electrical Stimulation of the Hemiplegic Shoulder

Electrical stimulation (ES) is a term used to describe the application of electrical current to the skin or directly into muscle that stimulates lower motor neurons, resulting in improved contractility and greater muscle bulk (Chae et al., 2008). The treatment can be used to improve muscle strength, joint misalignment, muscle tone, sensory deficits, and self-reported pain intensity (Price & Pandyan, 2001). The supraspinatus and posterior deltoid muscles are most likely to be treated, as they are important muscles in maintaining the correct alignment of the glenohumeral joint (Paci et al., 2005). Theoretically, ES compensates or facilitates flaccid shoulder muscles, which in turn reduces the risk of shoulder subluxation.

Surface ES for the treatment of pain is typically administered by neuromuscular electrical stimulation (NMES) or transcutaneous electrical nerve stimulation (TENS). The distinction between these two forms of treatment is that TENS is only used to treat pain while NMES can be used to improve function and pain. Another form of ES, functional electrical stimulation (FES), is largely used to restore motor function by delivering short electrical pulses to muscles. ES can also be administered intramuscularly with NMES or peripheral nerve stimulation (PNS).

In a systematic review, Price and Pandyan (2001) examined all forms of ES used in the prevention and treatment of post-stroke shoulder pain. There was evidence that FES, in addition to conventional therapy, improved function but did not reduce pain when compared to conventional therapy alone. The authors concluded that there was insufficient evidence from which to draw conclusions. Ada and Foongchomcheay (2002) conducted a meta-analysis to evaluate the effect of ES on shoulder subluxation post stroke. The results suggested that early ES helped to prevent the development of hemiplegic shoulder, while later ES helped to reduce pain.

A recent meta-analysis examined 10 RCTs to determine the effect of NMES on shoulder subluxation and pain in both “early” (<6 months) and “late” (>6 months) stroke patients (Vafadar et al., 2015). Analyses revealed that conventional therapy with NMES was more effective than conventional therapy alone at preventing/reducing shoulder subluxation, although its effectiveness was not significant in the “late”
As well, analyses demonstrated no significant difference in pain between the two groups. Trials evaluating the effects of different ES are summarized in Table 11.7.5.1 (surface ES) and Table 11.7.5.2 (intramuscular ES).

### Table 11.7.5.1 Summary of Studies Evaluating Surface Electrical Stimulation

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>Study Design (PEDro)</th>
<th>Sample Size</th>
<th>Interventions</th>
<th>Outcomes</th>
</tr>
</thead>
</table>
| Church et al. (2006) | RCT (9) N=176 | E: NMES C: Sham stimulation | • Pain (-)  
• Action Research Arm Test (-)  
• Motricity Index: C (+)  
• Frenchay Arm Test: C (+) |
| De Jong et al. (2013) | RCT (8) N=48 | E: NMES + Static arm positioning C: Sham stimulation + Sham arm positioning | • Pain (-)  
• Range of Motion (+) |
| Linn et al. (1999) | RCT (6) N=40 | E: NMES C: No stimulation | • Pain: post (+); 2mo (-)  
• Subluxation: post (+); 2mo (-) |
| Kim et al. (2016) | RCT (6) N=40 | E: Extracorporeal shockwave therapy C: No stimulation | • Pain (+)  
• Constant-Murley Scale (-) |
| Suriya-Amarit et al. (2014) | RCT (6) N=30 | E: Interferential current stimulation C: Sham stimulation | • Pain (+)  
• Range of Motion (+) |
| Leandri et al. (1990) | RCT (5) N=60 | E1: High-intensity TENS E2: Low-intensity TENS C: Sham stimulation | • Range of Motion: E1 (+) |
| Kobayashi et al. (1999) | RCT (5) N=115 | E1: NMES to supraspinatus E2: NMES to middle deltoid C: No stimulation | • Subluxation: E1 vs C (+); E2 vs C (+)  
• Abduction: E1 vs C (-); E2 vs C (+) |
| Wang et al. (2000) | RCT (5) N=32 | E: FES C: No stimulation | • Subluxation, short-duration hemiplegia (+)  
• Subluxation, long-duration hemiplegia (-) |
| Fil et al. (2011) | RCT (5) N=48 | E: High-voltage pulsed galvanic stimulation + Bobath therapy C: Bobath therapy | • Subluxation (+)  
• Shoulder joint displacement (+)  
• Motor Assessment Scale (-) |
| Baker & Parker (1986) | RCT (4) N=63 | E: NMES C: No stimulation | • Subluxation (+) |
| Koyuncu et al. (2010) | RCT (4) N=50 | E: FES C: No stimulation | • Pain (-)  
• Range of Motion (-)  
• Subluxation (+) |
| Moniruzzaman et al. (2010) | RCT (4) N=45 | E: TENS C: Ultrasound therapy | • Pain (-)  
• Range of Motion (+)  
• Muscle strength (+) |
| Faghri et al. (1994) | RCT (4) N=26 | E: FES C: No stimulation | • Spasticity (+)  
• Motor function (+) |
Table 11.7.5.2 Summary of Studies Evaluating Intramuscular Electrical Stimulation

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>Study Design (PEDro)</th>
<th>Interventions</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wilson et al. (2014)</td>
<td>RCT (9)</td>
<td>E: PNS</td>
<td>Pain (+)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>C: No stimulation</td>
<td></td>
</tr>
<tr>
<td>Yu et al. (2004)</td>
<td>RCT (7)</td>
<td>E: NMES</td>
<td>Pain: 3mo (+); 6mo (+); 12mo (+)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>C: Sling</td>
<td>Pain-interference: 6mo (+); 12mo (+)</td>
</tr>
<tr>
<td>Chae et al. (2005)</td>
<td></td>
<td></td>
<td>Subluxation: 12mo (-)</td>
</tr>
<tr>
<td>Chae et al. (2007a)</td>
<td></td>
<td></td>
<td>Motor impairment: 12mo (-)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Range of Motion: 12mo (-)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Spasticity: 12mo (-)</td>
</tr>
</tbody>
</table>

+ indicates statistically significant difference between groups
- indicates no statistically significant difference between groups
E indicates experimental group; C indicates control group

Discussion

Currently, there is a large variety of types and protocols of surface ES. When applied to the supraspinatus or deltoid muscle, NMES improved abduction and reduced subluxation, but only improved abduction when delivered to the deltoid alone (Kobayashi et al., 1999). Similarly, Manigandan et al. (2014) reported that NMES to the supraspinatus, deltoid, and biceps was more effective at reducing pain and subluxation than NMES to only the supraspinatus and deltoid muscles. An early study by Baker and Parker (1986) reported that NMES to the hemiplegic shoulder effectively reduced subluxation when compared to conventional therapy alone. These findings were confirmed by a later study, although the effect was not observed at a 2-month follow-up (Linn et al., 1999). NMES was also found to be more effective than sham stimulation for improving ROM, but not for reducing pain (De Jong et al., 2006). However, Church et al. (2006) reported that patients receiving NMES had worse outcomes for arm function than those receiving sham stimulation.

When delivered intramuscularly, NMES was shown to reduce shoulder pain at 3, 6, and 12 months post stimulation (Chae et al., 2005; Yu et al., 2004). However, there were no comparable improvements in ROM, motor function, subluxation, or spasticity. Secondary analyses revealed that stroke onset was the most significant predictor of intramuscular NMES success at reducing post-stroke HSP (John Chae et al., 2007a). Factors such as age, gender, stroke type, affected side, and motor function were not found to be significant predictors of treatment success (John Chae et al., 2007b). In a more recent study, Wilson et al. (2014) reported a statistically and clinically greater pain reduction in patients treated with PNS than those receiving standard care at 6 and 10 weeks after treatment.
The results from four trials demonstrate that FES may be beneficial for improving shoulder subluxation (Chantraine et al., 1999; Koyuncu et al., 2010; Wang et al., 2000), spasticity (Faghri et al., 1994), and motor function (Chantraine et al., 1999; Faghri et al., 1994). However, the findings are conflicting as to whether FES is effective in reducing pain (Chantraine et al., 1999; Koyuncu et al., 2010). The effects of TENS were examined in two trials with different comparators. In one trial, TENS applied over the hemiplegic shoulder was effective at improving passive ROM when delivered at a high intensity, but not at a low intensity, compared to sham stimulation (Leandri et al., 1990). In the other trial, TENS was more effective than therapeutic ultrasound at improving ROM and muscle strength, but not at reducing pain (Moniruzzaman et al., 2010).

Interferential electrical stimulation (IES) is often used in physiotherapy practice to relieve pain by transmitting low-frequency electrical impulses to the muscles. In a single trial, IES was found to be effective in reducing pain at rest and during passive ROM immediately after stimulation (Suriya-amarit et al., 2014). High-voltage pulsed galvanic stimulation (HVPGS) has been used to alleviate pain by applying a high-voltage, low-amperage direct current to affected regions (Fil et al., 2011). One trial demonstrated that HVPGS was associated with reduced subluxation and joint displacement, but did not improve motor function of the affected arm. Extracorporeal shockwave therapy (ESWT) has also been used in physiotherapy to treat tendon, joint, and muscle conditions with abrupt, high-amplitude pulses of electromagnetic energy. Recently, Kim et al. (2016) demonstrated that ESWT was effective in improving HSP.

Conclusions Regarding Electrical Stimulation of the Hemiplegic Shoulder

There is Level 1a and Level 2 evidence that surface neuromuscular electrical stimulation (NMES) reduces subluxation and improves range of motion of the hemiplegic shoulder, but does not reduce pain, when compared to sham or no stimulation.

There is Level 1b evidence that intramuscular neuromuscular electrical stimulation (NMES) reduces hemiplegic shoulder pain for up to 12 months post-treatment when compared to a cuff sling, but does not improve subluxation, spasticity, or motor function.

There is Level 1b evidence that peripheral nerve stimulation (PNS) reduces hemiplegic shoulder pain when compared to no stimulation.

There is Level 1b evidence that interferential electrical stimulation (IES) reduces hemiplegic shoulder pain when compared to sham stimulation.

There is Level 1b evidence that extracorporeal shockwave therapy (ESWT) reduces hemiplegic shoulder pain when compared to no stimulation.

There is Level 2 evidence that functional electrical stimulation (FES) reduces subluxation and improves motor function of the hemiplegic shoulder when compared to no stimulation.

There is limited Level 2 evidence that transcutaneous electrical nerve stimulation (TENS) at high intensity improves passive range of motion of the hemiplegic shoulder when compared to sham stimulation.

There is limited Level 2 evidence that transcutaneous electrical nerve stimulation (TENS) improves muscle strength and range of motion when compared to ultrasound therapy.
There is limited Level 2 evidence that high-voltage pulsed galvanic stimulation (HVPGS) reduces subluxation and joint displacement in the hemiplegic shoulder when compared to no stimulation.

Surface neuromuscular electrical stimulation may be effective in reducing subluxation and improving range of motion in the hemiplegic shoulder, although its effectiveness may be negatively correlated with stroke onset.

Intramuscular neuromuscular electrical stimulation may be effective in reducing hemiplegic shoulder pain, although its effectiveness may be negatively correlated with stroke onset.

Transcutaneous electrical nerve stimulation may be effective in improving range of motion in the hemiplegic shoulder, although it may only be effective at higher intensity.

Functional electrical stimulation may be effective in reducing subluxation and improving motor function in the hemiplegic shoulder.

Peripheral nerve stimulation may be effective in reducing hemiplegic shoulder pain; however the evidence is limited.

Interferential electrical stimulation may be effective in reducing hemiplegic shoulder pain; however the evidence is limited.

High-voltage pulsed galvanic stimulation may be effective in reducing subluxation and joint displacement in the hemiplegic shoulder; however the evidence is limited.

Extracorporeal shockwave therapy may be effective in reducing hemiplegic shoulder pain; however the evidence is limited.

### 11.7.6 Botulinum Toxin Injections for the Hemiplegic Shoulder

Subscapularis spasticity is characterized by shoulder ROM being most limited by pain on external rotation, causing a spastic muscle imbalance around the shoulder in many cases. Pectoralis muscle spasticity, characterized by limitation of ROM on shoulder abduction, is seen to a lesser extent but causes a similar muscle imbalance. Intra-articular injections of botulinum toxin and other agents have been used in an effort to treat spastic muscles, reduce imbalance, and relieve HSP.

A Cochrane review by Singh and Fitzgerald (2010) examined five RCTs evaluating the efficacy of botulinum toxin for treating post-stroke shoulder pain. The authors determined that treatment was associated with reductions in pain at three and six months following injection, but not at one month. Table 11.7.6.1 summarizes trials evaluating botulinum toxin for HSP.

<table>
<thead>
<tr>
<th>Author, Year Study Design (PEDro)</th>
<th>Sample Size</th>
<th>Interventions</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marciniak et al. (2012) RCT (10)</td>
<td>N=21</td>
<td>E: 100-150U Botox</td>
<td>• Pain (-)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>C: Placebo</td>
<td>• Range of Motion (-)</td>
</tr>
<tr>
<td>Kong et al. (2007)</td>
<td></td>
<td>E: 500U Dysport</td>
<td>• Spasticity (-)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Pain (-)</td>
</tr>
</tbody>
</table>
RCT (8)  
N=17  
C: Placebo  
• Modified Ashworth Score (-)  
• Range of Motion (-)  

Marco et al. (2007)  
RCT (8)  
N=31  
E: Electrical stimulation + 500U Dysport  
C: Electrical stimulation + placebo  
• Pain (+)  
• Modified Ashworth Score (-)  
• Range of Motion (+)  

Yelnik et al. (2007)  
RCT (7)  
N=20  
E: 500U Botox  
C: Placebo  
• Pain (+)  
• Range of Motion (+)  

de Boer et al. (2008)  
RCT (6)  
N=22  
E: 100U Botox  
C: Placebo  
• Pain (-)  
• Range of Motion (-)  

+ indicates statistically significant difference between groups  
- indicates no statistically significant difference between groups  
E indicates experimental group; C indicates control group  

Discussion  
The findings of two trials demonstrated that a low dosage of botulinum toxin (100-150U) did not improve pain or spasticity in the hemiplegic shoulder post-stroke (De Boer et al., 2008; Marciniak et al., 2012). However, two of three RCTs found that administering a higher dosage (500U) reduced pain, but not spasticity, and improved ROM (Marco et al., 2007; Yelnik et al., 2007). In addition to dosage, these studies also differed with respect to muscles targeted and dilution used; all of these factors can impact efficacy. An RCT targeting subscapularis and pectoralis major with either electromyography or ultrasound and a dose of 75-100 units per muscle at 4:1 dilution would be helpful in determining if botulinum toxin is effective for HSP.  

Conclusions Regarding Botulinum Toxin Injections for the Hemiplegic Shoulder  

There is Level 1a evidence that high doses of botulinum toxin (500U) improve pain and range of motion, but not spasticity, in the hemiplegic shoulder.  

There is Level 1a evidence that low doses of botulinum toxin (100-150U) do not improve pain, spasticity, or range of motion in the hemiplegic shoulder.  

Botulinum toxin may be effective in reducing pain and improving range of motion in the hemiplegic shoulder, but only when delivered in higher doses.

11.7.7 Steroid Injections for the Hemiplegic Shoulder  
The injection of corticosteroids into various shoulder girdle joints has also been examined as a means to relieve HSP. Trials evaluating the efficacy of triamcinolone acetonide (TA) are summarized in Table 11.7.1.  

Table 11.7.1 Summary of Studies Evaluating Triamcinolone Acetonide  

<table>
<thead>
<tr>
<th>Author, Year Study Design (PEDro) Sample Size</th>
<th>Interventions</th>
<th>Outcomes</th>
</tr>
</thead>
</table>
| Rah et al. (2012)  
RCT (9)  
N=58 | E: Triamcinolone acetonide (40mg)  
C: Lidocaine | • Pain (+)  
• Flexion (+)  
• Range of Motion (+)  
• Shoulder Disability Questionnaire (+) |
<table>
<thead>
<tr>
<th>Study</th>
<th>Design</th>
<th>N</th>
<th>Intervention</th>
<th>Outcome Measures</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lim et al. (2008)</td>
<td>RCT</td>
<td>29</td>
<td>E1: Triamcinolone acetonide (40mg)</td>
<td>Pain (-), Range of Motion (-), Fugl-Meyer Assessment (-), Modified Ashworth Scale (-)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>E2: Botulinum toxin (100U)</td>
<td></td>
</tr>
<tr>
<td>Snels et al. (2000)</td>
<td>RCT</td>
<td>35</td>
<td>E: Triamcinolone acetonide (40mg)</td>
<td>Pain (-), Action Research Arm Test (-), Fugl-Meyer Assessment (-)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C: Saline</td>
<td></td>
</tr>
<tr>
<td>Yasar et al. (2011)</td>
<td>RCT</td>
<td>26</td>
<td>E: Triamcinolone acetonide (40mg)</td>
<td>Pain (-)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C: Suprascapular nerve block</td>
<td></td>
</tr>
<tr>
<td>Lakse et al. (2009)</td>
<td>RCT</td>
<td>38</td>
<td>E: Triamcinolone acetonide (40mg)</td>
<td>Pain (+), Range of Motion (+), Modified Ashworth Scale (-), Barthel Index (-), Brunnstrom Recovery Stages (-)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C: No injection</td>
<td></td>
</tr>
<tr>
<td>Baykal et al. (2013)</td>
<td>RCT</td>
<td>30</td>
<td>E: Triamcinolone acetonide (40mg)</td>
<td>Pain (-), Range of Motion (-), Modified Ashworth Scale (-), Functional Independence Measure (-), Brunnstrom Recovery Stages (-)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C: No injection</td>
<td></td>
</tr>
<tr>
<td>Jeon et al. (2014)</td>
<td>PCT</td>
<td>33</td>
<td>E1: Triamcinolone acetonide (40mg)</td>
<td>Pain (-), Range of Motion (-)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>E2: Suprascapular nerve block</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>E3: E1 + E2</td>
<td></td>
</tr>
</tbody>
</table>

+ indicates statistically significant difference between groups
- indicates no statistically significant difference between groups
E indicates experimental group; C indicates control group

Discussion

In non-controlled studies, TA demonstrated significant reductions in pain via intraarticular (Dekker et al., 1997) or subacromial injection (Chae & Jedlicka, 2009; J. Chae et al., 2007); the findings of RCTs were less consistent regarding its efficacy. In two low-quality trials, TA was found to be effective (Lakse et al., 2009) and ineffective (Baykal & Senel, 2013) in reducing pain and improving ROM when compared to no injection. Neither trial demonstrated the effectiveness of TA in improving spasticity, independence, or motor recovery. In a higher quality trial, subacromial injections of TA showed significant reductions in pain and improvements in ROM over lidocaine (Rah et al., 2012). However, intraarticular injections of TA were not found to be an effective treatment for pain, ROM, or motor function when compared to a variety of other interventions, including suprascapular nerve block (Jeon et al., 2014; Yasar et al., 2011), botulinum toxin (Lim et al., 2008), or saline placebo (Snels et al., 2000). Further research is required to determine the efficacy of corticosteroids in treating the hemiplegic shoulder when compared alternative treatments, given the limited and conflicting evidence.

Conclusions Regarding Steroid Injections for the Hemiplegic Shoulder

There is conflicting Level 1a and Level 2 evidence regarding the effectiveness of triamcinolone acetonide injections in reducing hemiplegic shoulder pain.

Given conflicting findings, further research is required to determine the efficacy of triamcinolone acetonide injections in treating hemiplegic shoulder pain.
11.7.8 Hyaluronic Acid Injections for the Hemiplegic Shoulder
Hyaluronic acid (HA) is a non-sulfated glycosaminoglycan that has been used to treat various inflammatory conditions. Trials evaluating its efficacy in treating HSP are summarized in Table 11.7.8.1.

Table 11.7.8.1 Summary of Studies Evaluating Hyaluronic Acid

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>Study Design (PEDro)</th>
<th>Sample Size</th>
<th>Interventions</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Huang et al. (2016a)</td>
<td>RCT (8) N=26</td>
<td></td>
<td>E: Hyaluronic acid (2.5ml)</td>
<td>• Pain (+)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C: No injection</td>
<td>• Range of Motion (-)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Modified Ashworth Scale (-)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Fugl-Meyer Assessment (-)</td>
</tr>
<tr>
<td>Jang et al. (2016)</td>
<td>RCT (5) N=39</td>
<td></td>
<td>E: Hyaluronic acid (2ml)</td>
<td>• Pain (-)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C: Triamcinolone acetonide (40mg)</td>
<td>• Range of Motion (-)</td>
</tr>
</tbody>
</table>

+ indicates statistically significant difference between groups
- indicates no statistically significant difference between groups
E indicates experimental group; C indicates control group

Discussion
Two trials have evaluated HA in patients with post-stroke HSP. HA effectively reduced pain when compared to no injection, but did not improve ROM, spasticity, or motor function (Huang et al., 2016a). As well, HA was found to be as effective as TA in reducing pain and improving ROM (Jang et al., 2016).

Conclusions Regarding Hyaluronic Acid Injections for the Hemiplegic Shoulder

There is Level 1b evidence that hyaluronic acid reduces hemiplegic shoulder pain when compared to standard care.

There is Level 2 evidence that hyaluronic acid is as effective as triamcinolone acetonide in reducing hemiplegic shoulder pain.

Hyaluronic acid may be effective in treating hemiplegic shoulder pain, although further research is warranted.

11.7.9 Suprascapular Nerve Block for the Hemiplegic Shoulder
The suprascapular nerve provides sensory innervation to the shoulder muscles, and so a blockade of the nerve may provide pain relief. Trials evaluating a suprascapular nerve block (SSNB) for HSP are summarized in Table 11.7.9.1.

Table 11.7.9.1 Summary of Studies Evaluating Subscapular Nerve Block

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>Study Design (PEDro)</th>
<th>Sample Size</th>
<th>Interventions</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adey-Wakeling et al. (2013)</td>
<td>RCT (9) N=64</td>
<td></td>
<td>E: Suprascapular nerve block</td>
<td>• Pain (+)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C: Saline</td>
<td>• Modified Rankin Scale (-)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Croft Disability Index (-)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• EuroQOL (-)</td>
</tr>
<tr>
<td>Yasar et al. (2011)</td>
<td>RCT (5)</td>
<td></td>
<td>E1: Suprascapular nerve block</td>
<td>• Pain (-)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>E2: Steroid</td>
<td></td>
</tr>
</tbody>
</table>
Discussion

Three RCTs have investigated the effect of suprascapular nerve block therapy for the treatment of shoulder pain. In a high-quality trial, SSNB injections reduced pain when compared to saline placebo injection, although there was no improvement in disability or quality of life (Adey-Wakeling et al., 2013). SSNB combined with lidocaine showed a greater reduction in pain than ultrasound therapy, but had no effect on ROM (Boonsong et al., 2009). While SSNB and steroid were similarly effective in reducing shoulder pain, there was an overall reduction in pain with SNNB (Yasar et al., 2011). Further research is needed to discern the benefits of SSNB on HSP.

Conclusions Regarding Suprascapular Nerve Block for the Hemiplegic Shoulder

There is Level 1b and Level 2 evidence that suprascapular nerve block injections reduce hemiplegic shoulder pain, but do not improve range of motion, relative to saline injections or ultrasound therapy.

There is limited Level 2 evidence that suprascapular nerve block is not superior to intraarticular steroid injections in reducing hemiplegic shoulder pain.

Suprascapular nerve blocks may be effective in treating hemiplegic shoulder pain, although further research is warranted.

11.7.10 Segmental Neuromyotherapy for the Hemiplegic Shoulder

The segmental neuromyotherapy (SNMT) approach to pain aims to diagnose the precise segments involved in the sensitization process. The treatment involves a combination of modalities including therapy, local injection of anaesthetics, application of heat, and electrical stimulation. A single trial evaluating SNMT for HSP is summarized in Table 11.7.10.1.

**Table 11.7.10.1 Summary of Studies Evaluating Segmental Neuromyotherapy**

<table>
<thead>
<tr>
<th>Author, Year Study Design (PEDro) Sample Size</th>
<th>Interventions</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ratmansky et al. (2012) RCT (7) N=24</td>
<td>E: Segmental neuromyotherapy + Standard therapy C: Oral pain medication + Standard therapy</td>
<td>• Pain (-) • Neer Test (+) • Fugl-Meyer Assessment (+)</td>
</tr>
</tbody>
</table>

+ indicates statistically significant difference between groups
- indicates no statistically significant difference between groups
E indicates experimental group; C indicates control group
Discussion
Ratmansky et al. (2012) delivered SNMT, in addition to standard therapy, to identify its effects on pain, spasticity, and upper limb motor function. The results suggest that despite a significant reduction in pain with SNMT, the difference between SNMT and oral pain medication was not significant. However, treatment with SNMT showed greater improvement in motor function compared to the control.

Conclusions Regarding Segmental Neuromyotherapy for the Hemiplegic Shoulder

There is Level 1b evidence that segmental neuromyotherapy improves hemiplegic upper limb motor function, but not hemiplegic shoulder pain, when compared to oral pain medication.

Segmental neuromyotherapy may be effective in improving hemiplegic upper limb motor function; however the evidence is limited.

11.7.11 Surgery of the Hemiplegic Shoulder

Given that spastic muscle imbalance has been identified as a component of the hemiplegic shoulder, treatment designed to reverse such imbalance may relieve HSP. A pre-post study by Braun et al. (1971) evaluated surgical resection of the subscapularis and pectoralis muscle tendons. Following surgery, patients regained 90° of passive abduction and 20° of external rotation, although they eventually reported pain and discomfort after six months. In another study, Pinzur and Hopkins (1986) examined patients before and after receiving biceps tenodesis through a short deltopectoral approach. At long-term follow-up, patients showed a reduction in pain and subluxation in the hemiplegic shoulder.

Conclusions Regarding Surgery of the Hemiplegic Shoulder

There is limited Level 4 evidence that surgical resection of the subscapularis and pectoralis muscle tendons improves range of motion in the hemiplegic shoulder.

There is limited Level 4 evidence that biceps tenodesis through a deltopectoral approach reduces pain and subluxation in the hemiplegic shoulder.

Surgical interventions may be effective in reducing hemiplegic shoulder pain; however the evidence is limited.

11.7.12 Complementary & Alternative Therapies for the Hemiplegic Shoulder

Several complementary and alternative therapies exist for pain relief, including massage, acupressure, acupuncture, and aromatherapy. It has been hypothesized that such treatments enhance parasympathetic response and promote intense relaxation, altering the perception of pain (Mok & Woo, 2004; Shin & Lee, 2007). Other theories suggest that these therapies block pain signals, release various endogenous chemicals, or cause beneficial mechanical changes. Trials evaluating different complementary and alternative therapies are summarized in Table 11.7.12.1.

Table 11.7.12.1 Summary of Studies Evaluating Complementary & Alternative Therapies

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>Study Design (PEDro)</th>
<th>Interventions</th>
<th>Outcomes</th>
</tr>
</thead>
</table>
| Mendigutia-Gomez et al. (2016) | RCT (9) N=20 | E: Acupuncture  
C: Standard care | • Pain (+)  
• Range of Motion (+) |
<table>
<thead>
<tr>
<th>Study</th>
<th>Design</th>
<th>Participants</th>
<th>Interventions</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zhao et al. (2015)</td>
<td>RCT</td>
<td>N=124</td>
<td>Acupuncture</td>
<td>Pain (+), Range of Motion (+), Barthel Index (-)</td>
</tr>
<tr>
<td>Seo et al. (2013)</td>
<td>RCT</td>
<td>N=29</td>
<td>Acupuncture, herbal</td>
<td>Pain (+), Range of Motion (-), Fugl-Meyer Assessment (+)</td>
</tr>
<tr>
<td>Li et al. (2012)</td>
<td>RCT</td>
<td>N=120</td>
<td>Acupuncture + Massage</td>
<td>Pain (+), Fugl-Meyer Assessment (+), Shoulder-Hand Syndrome (+)</td>
</tr>
<tr>
<td>Shin &amp; Lee (2007)</td>
<td>RCT</td>
<td>N=30</td>
<td>Acupressure, essential oils</td>
<td>Pain (+)</td>
</tr>
<tr>
<td>Mok &amp; Woo (2004)</td>
<td>RCT</td>
<td>N=102</td>
<td>Slow-stroke back massage</td>
<td>Pain (+)</td>
</tr>
</tbody>
</table>

+ indicates statistically significant difference between groups
- indicates no statistically significant difference between groups
E indicates experimental group; C indicates control group

Discussion
Four RCTs examined the use of acupuncture. When compared to standard care, acupuncture reduced pain and spasticity, and improved range of motion, when delivered over two to three weeks (Mendigutia-Gomez et al., 2016; Zhao et al., 2015). Seo et al. (2013) found that two weeks of acupuncture reduced pain and improved motor function, although herbal point injections showed better results than the standard format. Similarly, acupressure was found to be an effective pain reliever for HSP, but was more effective with essential oils (i.e. aromatherapy) than without (Shin & Lee, 2007). Electroacupuncture, in combination with massage, yielded significantly greater reductions in pain and improvements in motor function than conventional therapy after six weeks of treatment; there was also a greater decrease in the severity of shoulder-hand syndrome with the treatment (Li et al., 2012). Massage, without adjunct therapies, showed a significant reduction in pain relative to standard care after only a week of treatment (Mok & Woo, 2004).

Conclusions Regarding Complementary & Alternative Therapies for the Hemiplegic Shoulder

There is Level 1a evidence that acupuncture reduces pain, increases range of motion, and improves motor function in the hemiplegic shoulder when compared to conventional therapy.

There is Level 1b and Level 2 evidence that massage therapy, alone or with acupuncture, reduces hemiplegic shoulder pain.

There is limited Level 1b evidence that a combination of acupressure and aromatherapy is more effective than dry acupressure in reducing hemiplegic shoulder pain.

Acupuncture may be effective in reducing pain, increasing range of motion, and improving motor function in the hemiplegic shoulder.

Massage therapy may reduce hemiplegic shoulder pain and promote physiological relaxation, although further research is warranted.
Combined acupressure and aromatherapy may reduce hemiplegic shoulder pain; however the evidence is limited.

11.8 Complex Regional Pain Syndrome (CRPS)

11.8.1 Stages and Symptoms of CRPS
CRPS can be categorized as one of two forms:

1. Type I, also referred to as shoulder-hand syndrome or reflex sympathetic dystrophy, is more common and associated with hemiplegia.
2. Type II, also referred to as causalgia, is less common and associated with traumatic injury.

CRPS is characterized by numerous peripheral and central nervous system changes in the absence of obvious nerve injury (Table 11.8.1.1). Peripheral changes include vasomotor tone with associated hand pain and swelling, exquisite hyperaesthesia, protective immobility, trophic skin changes, and vasomotor instability of the involved upper extremity (Moseley, 2004, 2006). Central changes include a disruption of sensory cortical processing, disinhibition of the motor cortex, and disrupted body schema (Moseley, 2004, 2006).

Table 11.8.1.1 Stages and Symptoms of CRPS (Iwata et al., 2002)

<table>
<thead>
<tr>
<th>Stage</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Persistent pain, described as burning or aching Extremity is edematous, warm, and hyperesthetic Pain is aggravated by movement</td>
</tr>
<tr>
<td>2</td>
<td>Early dystrophic changes in the limb Atrophy of the muscle and skin Vasospasm with hyperhidrosis</td>
</tr>
<tr>
<td>3</td>
<td>Soft-tissue dystrophy Contractures that produce frozen shoulder Pain and vasomotor changes are infrequent</td>
</tr>
</tbody>
</table>

Initially, CRPS generally presents with pain in the shoulder followed by a painful, edematous hand and wrist (Davis et al., 1977). There is frequently decreased range of motion at the shoulder and hand, while the elbow joint is spared. Passive flexion of the wrist and hand joints is painful and limited due to edema over the dorsum of the fingers. As time progresses, the extensor tendons become elevated and the collateral ligaments shorten. If untreated, it has long been thought that the condition eventually progresses to a dry, cold, bluish, and atrophied hand. However, experience would suggest that in most cases the pain and edema subside spontaneously after a few weeks.

CRPS is not unique to patients recovering from stroke and is prevalent among patients with head injury, spinal cord injury, and even mild injury to the extremities. Typically, patients with post-stroke CRPS present with pain, hyperalgesia, joint stiffness and swelling, and autonomic abnormalities. While recovery is largely spontaneous, CRPS that persists for more than six months is often difficult to treat.

Conclusions Regarding the Stages and Symptoms of CRPS

Peripheral changes due to complex regional pain syndrome include pain, edema, dystrophy, immobility, and vasomotor instability of the affected upper limb.
Central changes due to complex regional pain syndrome include sensory cortical processing, motor cortex disinhibition, and disrupted body schema.

Complex regional pain syndrome is characterized by numerous peripheral and central nervous system changes in the absence of obvious nerve injury.

11.8.2 Pathophysiology of CRPS
The etiology of CRPS is unknown, but theoretical peripheral and central etiologies have been proposed. Peripheral etiological theories hypothesize a role for trauma to the peripheral nerves. One theory postulates ephaptic conduction between efferent sympathetic nerves and afferent somatic nerves, with the latter depolarization being perceived as pain. Central etiological theories hypothesize a disruption of autonomic nervous control from higher central nervous system centres. One theory postulates that such a disruption directly affects the internuncial pool of the spinal cord, leading to decreased inhibition of the sympathetic neurons of the lateral horn. Pain, either from contractures or subluxation, may stimulate the internuncial pool of the spinal cord and result in an abnormal sympathetic response. CRPS has often been regarded as a form of sympathetically-mediated pain involving the hemiplegic upper extremity, but a link between the abnormal sympathetic nervous system and pain has yet to be proven.

CRPS has also been proposed to be a result of paresis following stroke, mediated by disruption of the balance between intracellular and extracellular fluid (Iwata et al., 2002). Three possible mechanisms were suggested: (1) increased capillary blood pressure, caused by decreased peripheral venous return and lymph flow; (2) decreased colloidal osmotic pressure in the early stages of stroke, due to an acute phase response; and (3) enhanced permeability of capillary walls, which may result from synovial inflammation, brought about by rough management of the affected arm and hand.

In a systematic review, Geurts et al. (2000) identified five etiological studies and six therapeutic studies regarding post-stroke CRPS. The authors found that the shoulder was involved in only half of the cases, while all of the cases were characterized by painful swelling of the wrist and hand, thereby suggesting a “wrist-hand syndrome” in the other half of cases. Furthermore, they noted that CRPS hand edema was not a lymphoedema and usually coincided with increased arterial blood flow.

Conclusions Regarding the Pathophysiology of CRPS

The pathophysiology of complex regional pain syndrome is poorly understood, although several theoretical peripheral and central etiologies have been proposed.

The pathophysiology of complex regional pain syndrome is not fully understood.

11.8.3 Frequency of CRPS
There is a wide range of reported frequency of CRPS following stroke. Part of variability can be attributed to timing and form of assessment used.

Table 11.8.3.1 Frequency of CRPS

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>Sample Size</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Davis et al. (1977) N=540</td>
<td></td>
<td>12.5%</td>
</tr>
<tr>
<td>Tepperman et al. (1984)</td>
<td></td>
<td>25%</td>
</tr>
</tbody>
</table>
The frequency of CRPS appears to range between 10% and 48%. Petchkrua et al. (2000) suggested that the findings from earlier studies are overestimated, as they were obtained before patients routinely received early intensive inpatient rehabilitation. However, the results from more recent studies suggest that the actual frequency sits somewhere within the aforementioned range.

**Conclusions Regarding the Frequency of CRPS**

*The reported frequency of complex regional pain syndrome post stroke varies from 10% to 48%, which may be influenced by heterogeneity in the type and timing of assessment.*

**There is high variability in the reported frequency of complex regional pain syndrome.**

### 11.8.4 Diagnosis of CRPS

Several approaches to diagnose CRPS have been utilized, although no single test will identify all individuals with CRPS. Three sets of criteria are used routinely: (1) International Association for the Study of Pain (IASP) 1994 consensus criteria (Stanton-Hicks et al., 1995); (2) Bruehl’s (1999) criteria; and (3) Veldman’s (1993) criteria. The sensitivities and specificities of these sets of criteria range from 70% to 100% and from 36% to 94%, respectively. Common features among these criteria include: pain, allodynia, hyperalgesia, edema, changes in sweating, and limitations in range of motion. However, Tepperman et al. (1984) found that 25% of hemiplegic patients demonstrated evidence of CRPS in the involved upper extremity, but only 16.5% went on to develop the clinical syndrome.

Routine radiographs of the involved upper extremity may demonstrate a patchy, periarticular demineralization (Sudek's atrophy) as early as three to six months after the onset of clinical signs. The most sensitive diagnostic test is the technetium diphosphonate bone scan, which demonstrates increased periarticular uptake in the affected upper extremity (mostly at the shoulder and wrist); bone scan abnormalities appear earlier than the X-ray changes. Temporary resolution of symptoms with sympathetic blockade is considered diagnostic. Despite potential difficulties with the technique in terms of diagnostic validity, the accuracy of these blocks has improved with image-guided injections (e.g. ultrasound). Thermography has failed to consistently diagnose CRPS and is not considered a valid test.
Kozin et al. (1981) suggested that clinical measurements such as grip strength, tenderness, and ring size were more accurate diagnostic indicator of CRPS. Similarly, Iwata et al. (2002) suggested that a ratio of the middle finger circumference (affected vs. unaffected) greater than 1.06 at four weeks post stroke was predictive of CRPS. It has been suggested by Quisel et al. (2005) that although diagnosis through instrumentation and imaging is common, there is limited evidence that these techniques improve the diagnostic accuracy.

**Conclusions Regarding the Diagnosis of CRPS**

*Several CRPS diagnostic tests exist, although none will identify all patients with CRPS.*

**There is no ideal diagnostic test for the identification of CRPS.**

### 11.8.5 Management of CRPS

There is no definitive therapeutic intervention for CRPS, as reflected by the large number of suggested treatments. In fact, it is widely accepted that there is little evidence regarding the effectiveness of many commonly-used treatments (Pertoldi & Di Benedetto, 2005; Quisel et al., 2005). Aggressive early treatment is recommended to prevent the development of a non-functional, painful upper extremity. A one- to two-week course of high-dose corticosteroids and/or sympathetic blocks may be utilized in persistent, disabling cases. A surgical sympathectomy may be considered if nerve blocks are consistently effective but symptoms recur. Constraint-induced movement therapy and sensory discrimination training have also been proposed as possible treatments (Acerra et al., 2007). CRPS presenting for more than six months without appropriate treatment has a poor prognosis (Lieberman, 1986).

### 11.8.6 Pharmacological Interventions for CRPS

Despite a limited number of trials, a review by Geurts et al. (2000) concluded that oral corticosteroids were the most effective treatment for CRPS. Five trials evaluated the use of corticosteroids for the treatment of post-stroke CRPS and one trial evaluated the use of nerve block, all of which are summarized in Table 11.8.6.1.

**Table 11.8.6.1 Summary of Studies Evaluating Pharmacological Interventions for CRPS**

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>Study Design (PEDro)</th>
<th>Sample Size</th>
<th>Interventions</th>
<th>Outcomes</th>
</tr>
</thead>
</table>
| Kalita et al. (2006) | RCT (7) | N=60 | E: Prednisolone (40mg)  
C: Piroxicam (20mg) | CRPS Scale (+) |
| Eun Young et al. (2016) | RCT (6) | N=21 | E1: Prednisolone (80mg)  
E2: Pamidronate (180mg) | Pain: E2 (+)  
Wrist Circumference: E1 (+)  
Finger Circumference (-) |
| Rah et al. (2012) | RCT (6) | N=58 | E: Triamcinolone (40mg)  
C: Lidocaine | Pain (+)  
Shoulder Disability Questionnaire (+) |
| Yoo et al. (2012) | RCT (6) | N=42 | E: Stellate ganglion block, ultrasound guided  
C: Stellate ganglion block | Pain (+)  
CRPS Scale (-)  
Swelling (-) |
| Kalita et al. (2016) | RCT (5) |  | E: Prednisolone (10mg)  
C: Discontinuation | Pain (+)  
CRPS Scale (+) |
Discussion

Three corticosteroids were identified as potential interventions for CRPS: triamcinolone (16-40mg), methylprednisolone (8mg), and prednisolone (10-80mg). These particular glucocorticoids are commonly used to treat a variety of inflammatory conditions. An early study found that oral triamcinolone taken daily for two to three weeks effectively reduced pain in CRPS (Davis et al., 1977). Similarly, oral methylprednisolone was found to be more effective than placebo in treating such pain when taken for four weeks (Braus et al., 1994).

When delivered intravenously, triamcinolone has shown significant reductions in pain and swelling (Y. W. Kim et al., 2016), even when compared to lidocaine (Rah et al., 2012). Prednisolone injections were also found to be effective in reducing the severity of CRPS when compared to a non-steroidal anti-inflammatory drug (Kalita et al., 2006) or no treatment (Kalita et al., 2016). However, Eun Young et al. (2016) reported that prednisolone was less effective than an osteoclast-inhibiting bisphosphonate (pamidronate) in attenuating pain associated with CRPS but was more effective in reducing swelling.

Nerve blocks are another common intervention for pain management. Yoo et al. (2012) reported that a stellate ganglion block effectively reduced pain and swelling in CRPS following stroke, although not the severity of CRPS. As well, there was also a greater reduction in pain when delivering the injection via guided ultrasound than without (Davis et al., 1977; Y. W. Kim et al., 2016; Rah et al., 2012).

Conclusions Regarding Pharmacological Interventions for CRPS

There is Level 1a evidence that oral or intravenous corticosteroids reduce pain, swelling, and severity of complex regional pain syndrome.

There is Level 1b evidence that stellate ganglion nerve blocks reduce pain and swelling in complex regional pain syndrome, which may be enhanced by ultrasound guiding.

Corticosteroids are an effective treatment for reducing the severity of complex regional pain syndrome.

Nerve blocks may be effective in managing the symptoms of complex regional pain syndrome, although further research is required.

11.8.7 Mirror Imagery Therapy for CRPS

Mirror imagery is more commonly associated with the treatment of phantom limb pain, but may also be useful in the treatment of post-stroke CRPS. It is believed that mirror imagery helps create a movement illusion of the affected limb within the brain, as both real and imagined movements activate similar cortical networks. These mirror illusions may compensate for reduced or absent proprioceptive input and re-establish a pain-free relationship between sensory feedback and motor intention (Acerra et al., 2005).
During mirror imagery therapy, the patient is seated in a manner such that the unaffected limb can be viewed in the mirror and the affected limb is concealed behind the mirror, while performing a variety of movements with the upper limb. Trials evaluating the efficacy of mirror imagery therapy in treating CRPS are summarized in Table 11.8.7.1.

Table 11.8.7.1 Summary of Studies Evaluating Mirror Imagery Therapy for CRPS

<table>
<thead>
<tr>
<th>Author, Year Study Design (PEDro) Sample Size</th>
<th>Interventions</th>
<th>Outcomes</th>
</tr>
</thead>
</table>
| Pervane Vural et al. (2016) RCT (7) N=30 | E: Mirror imagery therapy C: Standard care | • Pain (+)  
• Functional Independence Measure (+)  
• Fugl-Meyer Assessment (+)  
• Brunnstrom Recovery Stages (+)  
• Modified Ashworth Scale (-) |
| Cacchio et al. (2009a) RCT (7) N=48 | E: Mirror imagery therapy C: Covered mirror therapy | • Pain (+)  
• Wolf Motor Function Test (+)  
• Motor Activity Log (+) |
| Moseley (2004) RCT (7) N=13 | E: Mirror imagery therapy C: Standard care | • Pain (+) |
| Moseley (2006) RCT (6) N=51 | E: Mirror imagery therapy C: Standard care | • Pain (+) |
| Cacchio et al. (2009b) RCT (5) N=24 | E: Mirror imagery therapy C1: Covered mirror therapy C2: Mental imagery therapy | • Pain (+)  
• Wolf Motor Function Test (+) |

+ indicates statistically significant difference between groups  
- indicates no statistically significant difference between groups  
E indicates experimental group; C indicates control group

**Discussion**

Moseley (2004, 2006) developed a Motor Imagery Program delivered in three parts: (1) limb laterality recognition; (2) imagined limb movements; and (3) mirror limb movements. Each part of the program was delivered daily for two weeks over a total of six weeks. In two separate trials, the author found that the program effectively reduced pain when compared to standard care, and such benefits were maintained at long-term follow-up. Cacchio et al. (2009a; 2009b) also conducted two trials evaluating a four-week mirror imagery therapy program. When compared to sham therapy or mental practice, active therapy was found to significantly reduce pain and improve motor function with long-lasting benefits. Most recently, these results were replicated in a similar program by Pervane Vural et al. (2016). The authors reported that mirror imagery therapy reduced pain, improved motor function, and increased functional independence better than standard care of CRPS. However, neither treatment was associated with a reduction in spasticity.

**Conclusions Regarding Mirror Imagery Therapy for CRPS**

*There is Level 1a evidence that mirror imagery therapy reduces pain and improves upper limb motor function in complex regional pain syndrome when compared to placebo or standard care.*

*Mirror imagery therapy is an effective treatment for reducing pain and improving motor function in complex regional pain syndrome.*
11.8.8 Exercise for the Prevention and Treatment of CRPS

Physiotherapy is regarded as the cornerstone of integrated treatment. However, few trials have been conducted to evaluate its effect in preventing or treating CRPS. Two relevant trials are summarized in Table 11.8.8.1.

Table 11.8.8.1 Summary of Study Evaluating Exercise for CRPS

<table>
<thead>
<tr>
<th>Author, Year Study Design (PEDro)</th>
<th>Interventions</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Topcuoglu et al. (2015)</strong></td>
<td>E: Aerobic exercise + Physical therapy&lt;br&gt;C: Physical therapy</td>
<td>• Pain (+)&lt;br&gt;• Quality of Life (+)&lt;br&gt;• Beck Depression Inventory (+)&lt;br&gt;• Functional Independence Measure (-)</td>
</tr>
<tr>
<td>RCT (6) N=40</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Kondo et al. (2001)</strong></td>
<td>E: CRPS prevention exercises&lt;br&gt;C: Standard care</td>
<td>• CRPS (+)</td>
</tr>
<tr>
<td>PCT N=152</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

+ indicates statistically significant difference between groups<br>- indicates no statistically significant difference between groups<br>E indicates experimental group; C indicates control group

A trial using historical controls examined the effectiveness of an exercise program in preventing the development of post-stroke CRPS (Kondo et al., 2001). The treatment protocol was developed in response to the observation that patients were inappropriately performing excessive passive range of motion exercises post stroke, which may have contributed to the development of CRPS. The results demonstrated that the incidence of CRPS was significantly lower (18%) in patients following the protocol compared to controls (32.4%).

In another trial, Topcuoglu (2015) developed an aerobic exercise program for the upper limbs in CRPS using an arm crank ergometer. The program, as an adjunct to physiotherapy, showed a significantly greater reduction in pain than conventional physiotherapy alone. As well, patients in the treatment group had better psychosocial outcomes than controls.

**Conclusions Regarding Exercise to Prevent and Treat CRPS**

*There is Level 1b evidence that a combination of aerobic exercise and physiotherapy reduces pain and improves psychosocial outcomes in complex regional pain syndrome when compared to conventional physiotherapy.*

*There is limited Level 2 evidence that passive range of motion exercises prevent the development of complex regional pain syndrome when compared to standard care.*

*Combined physiotherapy and aerobic exercise may be effective in reducing pain and improving psychosocial outcomes in complex regional pain syndrome.*

*Passive range of motion exercises may prevent the development of complex regional pain syndrome, although further research is required.*
11.8.9 Calcitonin for the Prevention of CRPS
Calcitonin has been used successfully to treat osteoporosis as well as a variety of pain conditions following trauma or surgery. However, its use in the treatment of CRPS following stroke has not been well studied. A single trial is summarized in 11.8.9.1.

Table 11.8.9.1 Summary of Study Evaluating Calcitonin for the Prevention of CRPS

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>Study Design (PEDro)</th>
<th>Sample Size</th>
<th>Interventions</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Matayoshi et al. (2009)</td>
<td>PCT</td>
<td>N=59</td>
<td>E: Calcitonin (20U) C: Standard care</td>
<td>• CRPS (+)</td>
</tr>
</tbody>
</table>

+ indicates statistically significant difference between groups
- indicates no statistically significant difference between groups
E indicates experimental group; C indicates control group

Matayoshi et al. (2009) evaluated the efficacy of intramuscular injections of calcitonin, in addition to regular rehabilitation, in preventing the development of post-stroke CRPS. Upon examining patients with severe stroke, the rate of CRPS was significantly lower in patients receiving calcitonin (12.5%) compared to a historical control group (57.1%). The authors suggested that early treatment with calcitonin within the first four weeks of stroke was capable of preventing CRPS, but not when the treatment was initiated after six weeks.

Conclusions Regarding Calcitonin to Prevent CRPS

There is limited Level 2 evidence that intramuscular injections of calcitonin within four weeks of stroke prevent the development of complex regional pain syndrome.

Acute injections of calcitonin may prevent the development of complex regional pain syndrome, although further research is required.
Summary

1. Factors associated with hemiplegic shoulder pain include older age, longer disease duration, poor arm function, muscle imbalance, rotator cuff tear, subscapularis/pectoralis spasticity, glenohumeral subluxation, bursitis, tendonitis, adhesive capsulitis, and complex regional pain syndrome.

2. Shoulder subluxation may occur early on in the hemiplegic arm due to flaccid supporting shoulder musculature and can be exacerbated by external forces.

3. Shoulder subluxation may be associated with hemiplegic shoulder pain, although patients with shoulder subluxation may not experience pain and patients with pain may not have subluxed shoulder.

4. Spastic muscle imbalance of the shoulder adductors and internal rotators, particularly the subscapularis and pectoralis major, appears to be associated with hemiplegic shoulder pain.

5. Adhesive capsulitis and its associated limited range of movement appear to be associated with hemiplegic shoulder pain.

6. Rotator cuff disorders do not appear to be associated with hemiplegic shoulder pain.

7. The reported frequency of hemiplegic shoulder pain varies from 9% to 72%, which may be influenced by heterogeneity in the type and timing of assessment.

8. There is Level 4 evidence that hemiplegic shoulder pain may be associated with poorer upper limb motor function and lower quality of life; the association with functional outcomes is less clear.

9. There is Level 1a evidence that sustained positioning and static stretching does not reduce pain, increase range of motion, or improve motor function of the hemiplegic shoulder.

10. There Level 1b and Level 2 evidence that slings reduce subluxation and pain of the hemiplegic shoulder.

11. There is Level 1a evidence that shoulder strapping/taping reduces hemiplegic shoulder pain; however it may not improve spasticity, disability, range of motion, or motor function.

12. There is Level 1b evidence that continuous passive range of motion exercises are not more effective than self-range of motion exercises at improving motor function, joint stability, spasticity, or pain in the affected limb.

13. There is Level 1b evidence that supplementing range of motion exercises with ultrasound or positioning is not more effective than the exercises alone.

14. There is limited Level 2 evidence that aggressive range of motion exercises (e.g. overhead pulleys) increase hemiplegic shoulder pain when compared to the exercises alone.

15. There is limited Level 2 evidence that stretching and joint stabilizing exercises improve motor function of the affected limb when compared to conventional exercises.

16. There is limited Level 2 evidence that Bobath therapy reduces hemiplegic shoulder pain when compared to cryotherapy but not standard therapy.

17. There is limited Level 2 evidence that ‘monkey chair and band’ therapy improves motor function, range of motion, and pain in the affected limb when compared to standard therapy.
18. There is Level 1a and Level 2 evidence that surface neuromuscular electrical stimulation (NMES) reduces subluxation and improves range of motion of the hemiplegic shoulder, but does not reduce pain, when compared to sham or no stimulation.

19. There is Level 1b evidence that intramuscular neuromuscular electrical stimulation (NMES) reduces hemiplegic shoulder pain for up to 12 months post treatment when compared to a cuff sling, but does not improve subluxation, spasticity, or motor function.

20. There is level 1b evidence that peripheral nerve stimulation (PNS) reduces hemiplegic shoulder pain when compared to no stimulation.

21. There is Level 1b evidence that interferential electrical stimulation (IES) reduces hemiplegic shoulder pain when compared to sham stimulation.

22. There is Level 1b evidence that extracorporeal shockwave therapy (ESWT) reduces hemiplegic shoulder pain when compared to no stimulation.

23. There is Level 2 evidence that functional electrical stimulation (FES) reduces subluxation and improves motor function of the hemiplegic shoulder when compared to no stimulation.

24. There is limited Level 2 evidence that transcutaneous electrical nerve stimulation (TENS) at high intensity improves passive range of motion of the hemiplegic shoulder when compared to sham stimulation.

25. There is limited Level 2 evidence that transcutaneous electrical nerve stimulation (TENS) improves muscle strength and range of motion when compared to ultrasound therapy.

26. There is limited Level 2 evidence that high-voltage pulsed galvanic stimulation (HVPGS) reduces subluxation and joint displacement in the hemiplegic shoulder when compared to no stimulation.

27. There is Level 1a evidence that high doses of botulinum toxin (500U) improve pain and range of motion, but not spasticity, in the hemiplegic shoulder.

28. There is Level 1a evidence that low doses of botulinum toxin (100-150U) do not improve pain, spasticity, or range of motion in the hemiplegic shoulder.

29. There is conflicting Level 1a and Level 2 evidence regarding the effectiveness of triamcinolone acetonide injections in reducing hemiplegic shoulder pain.

30. There is Level 1b evidence that hyaluronic acid reduces hemiplegic shoulder pain when compared to standard care.

31. There is Level 2 evidence that hyaluronic acid is as effective as triamcinolone acetonide in reducing hemiplegic shoulder pain.

32. There is Level 1b and Level 2 evidence that suprascapular nerve block injections reduce hemiplegic shoulder pain, but do not improve range of motion, relative to saline injections or ultrasound therapy.

33. There is limited Level 2 evidence that suprascapular nerve block is not superior to intraarticular steroid injections in reducing hemiplegic shoulder pain.

34. There is Level 1b evidence that segmental neuromyotherapy improves hemiplegic upper limb motor function, but not hemiplegic shoulder pain, when compared to oral pain medication.

35. There is limited Level 4 evidence that surgical resection of the subscapularis and pectoralis muscle tendons improves range of motion in the hemiplegic shoulder.
36. There is limited Level 4 evidence that biceps tenodesis through a deltopectoral approach reduces pain and subluxation in the hemiplegic shoulder.

37. There is Level 1a evidence that acupuncture reduces pain, increases range of motion, and improves motor function in the hemiplegic shoulder when compared to conventional therapy.

38. There is Level 1b and Level 2 evidence that massage therapy, alone or with acupuncture, reduces hemiplegic shoulder pain.

39. There is limited Level 1b evidence that a combination of acupressure and aromatherapy is more effective than dry acupressure in reducing hemiplegic shoulder pain.

40. Peripheral changes due to complex regional pain syndrome include pain, edema, dystrophy, immobility, and vasomotor instability of the affected upper limb.

41. Central changes due to complex regional pain syndrome include sensory cortical processing, motor cortex disinhibition, and disrupted body schema.

42. The pathophysiology of complex regional pain syndrome is poorly understood, although several theoretical peripheral and central etiologies have been proposed.

43. The reported frequency of complex regional pain syndrome post stroke varies from 10% to 48%, which may be influenced by heterogeneity in the type and timing of assessment.

44. Several CRPS diagnostic tests exist, although none will identify all patients with CRPS.

45. There is Level 1a evidence that oral or intravenous corticosteroids reduce pain, swelling, and severity of complex regional pain syndrome.

46. There is Level 1b evidence that stellate ganglion nerve blocks reduce pain and swelling in complex regional pain syndrome, which may be enhanced by ultrasound guiding.

47. There is Level 1a evidence that mirror imagery therapy reduces pain and improves upper limb motor function in complex regional pain syndrome when compared to placebo or standard care.

48. There is Level 1b evidence that a combination of aerobic exercise and physiotherapy reduces pain and improves psychosocial outcomes in complex regional pain syndrome when compared to conventional physiotherapy.

49. There is limited Level 2 evidence that passive range of motion exercises prevent the development of complex regional pain syndrome when compared to standard care.

50. There is limited Level 2 evidence that intramuscular injections of calcitonin within four weeks of stroke prevent the development of complex regional pain syndrome.
References


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