11 Painful Hemiplegic Shoulder

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Abstract

Shoulder pain resulting from hemiplegia is a common clinical consequence of a focal cerebral insult resulting from a vascular lesion (ie. hemorrhagic or ischemic stroke). Painful hemiplegic shoulder can occur as early as 2 weeks post stroke but an onset time of 2-3 months post stroke is more typical. Shoulder pain can 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. 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 causes and management of hemiplegic shoulder pain with in-depth discussion of shoulder subluxation, spasticity, contractures, and rotator cuff disorders. Management techniques including shoulder positioning, slings and aids, surgical interventions, injections, and electrical stimulation along with other miscellaneous treatments are addressed. Finally, a full discussion of the physiology, incidence, and treatment of shoulder hand syndrome is included.
Key Points

Hemiplegic Shoulder Pain

- There is a high variability in the reported incidence of shoulder pain.
- Various causes/risk factors of hemiplegic shoulder pain include muscle imbalance and poor arm function, pectoralis/subscapularis spasticity, glenohumeral subluxation, bursitis, tendonitis/tear, adhesive capsulitis, and shoulder-hand syndrome.
- It is uncertain whether shoulder subluxation causes hemiplegic shoulder pain.
- There may be an association between shoulder contractures and hemiplegic shoulder pain. Spasticity in shoulder adductors and internal rotators, especially subscapularis and pectoralis major, have been implicated in hemiplegic shoulder pain.
- Painful hemiplegic shoulder may be associated with reduced motor function and increased stroke severity.
- Evidence suggests that shoulder positioning may not improve pain or motor impairments related to shoulder subluxation.
- There is limited evidence that shoulder slings influence clinical outcomes.
- Strapping/taping the hemiplegic shoulder does not appear to improve upper limb function, but may reduce pain.
- Aggressive range of motion exercises (i.e. pullies) results in a markedly increased incidence of painful shoulder; a gentler range of motion program is preferred. Adding ultrasound treatments does not appear to improve shoulder range of motion.
- Treatment with surface neuromuscular electrical stimulation (NMES) early (< 6 months) post-stroke may reduce shoulder subluxation but not pain associated with shoulder hemiplegia. Surface NMES delivered after 6 months provides no additional benefits over conventional therapy on shoulder subluxation. Intramuscular NMES however, has been found to reduce shoulder pain up to 12 months post-therapy.
- High-voltage pulsed galvanic stimulation (HVPGS) may reduce shoulder subluxation but not pain evoked by the hemiplegic shoulder, while interferential electrical stimulation (IES) may reduce pain immediately after treatment.
- More research is needed to determine the efficacy of TENS for improving hemiplegic shoulder function and pain.
- More research is needed to determine if surgically resecting the subscapularis and the pectoralis muscle tendons improves range of motion in patients with shoulder hemiplegia.
- It is uncertain whether botulinum toxin reduces hemiplegic shoulder pain or improves range of motion.
- More studies of greater methodological quality and higher power are needed determine the efficacy of Triamcinolone acetonide injections at reducing hemiplegic shoulder pain.
- Further research is needed to determine the benefits of aromatherapy in combination with acupressure regarding its effects on reducing pain caused by shoulder hemiplegia.
• Massage therapy may reduce hemiplegic shoulder pain, anxiety, and other physiological functions (i.e. blood pressure, and heart rate) however, more research is still warranted.

• Although nerve block injections appear to be superior over saline injections or ultrasound therapy, more research is needed.

• Further research is needed to determine the effect of segmental neuromyotherapy on hemiplegic shoulder pain.

**Complex Regional Pain Syndrome**

• The pathophysiology of complex regional pain syndrome is poorly understood.

• The incidence rate of complex regional pain syndrome varies greatly among post-stroke individuals depending on the timing and the type of assessment used.

• No one diagnostic test is ideal for the identification of CRPS. Further research is required.

• Oral corticosteroids appear to improve complex regional pain syndrome; however, more research is needed.

• A mirror therapy program appears to improve shoulder-hand syndrome.

• There is not enough evidence to understand the effect of motion exercises on complex regional pain syndrome.

• Although early treatment (<4wks post-stroke) with calcitonin is found to prevent CRPS, more research is need to understand its effect in the long term and across various stroke severities.
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11.1 Incidence of Hemiplegic Shoulder Pain

Shoulder pain resulting from hemiplegia is a common clinical consequence of a focal cerebral insult resulting from a vascular lesion (i.e. hemorrhagic or ischemic stroke). The incidence of shoulder pain varies between studies, with estimates which range from 48% to 84% (Najenson et al., 1971; Poulin et al., 1990) (See Table 11.1.1).

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bohannon et al. (1986)</td>
<td>The charts of 50 patients with hemiplegia secondary to stroke admitted for inpatient rehabilitation were reviewed. Pain, assessed during passive shoulder external rotation, was reported by 36 (73%) patients</td>
</tr>
<tr>
<td>van Ouwenaller et al. (1986)</td>
<td>219 stroke patients initially hospitalized for stroke were assessed and followed up for an average of 11 months, were studied. 157 (72%) reported experiencing shoulder pain on at least one occasion during the study period.</td>
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<td>Jespersen et al. (1995)</td>
<td>Retrospective chart review of 173 admissions for inpatient stroke rehabilitation. Self-reported shoulder pain was reported by 38 (22%) patients. 27 patients developed pain during rehabilitation while 11 patients developed pain following discharge during the 6 month follow-up period.</td>
</tr>
<tr>
<td>Zorowitz et al. (1996)</td>
<td>20 patients with clinical evidence of subluxation, admitted within 6 weeks of stroke were assessed for pain using a 0-10 visual analog scale. Pain scores of 4-10 were reported by 9 (45%) patients. The remainder had no, or minimal pain.</td>
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<td>Gamble et al. (2000)</td>
<td>182 unselected, consecutive patients admitted to hospital and assessed for pain within 2 weeks of stroke. Pain was assessed using a visual analog scale. 31(25%) reported shoulder pain.</td>
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<td>Langhorne et al. (2000)</td>
<td>311 acute, consecutive stroke patients admitted to hospital were assessed for the presence of pain. Shoulder pain was reported in 9% of patients during hospitalization, 15% (from discharge-6 months), 11% (from 6-18 months) and 12% (18-30 months)</td>
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<td>Ratnasabapathy et al. (2003)</td>
<td>Self-reported shoulder pain among survivors of a population based sample of 1,761 stroke survivors from New Zealand. One week after stroke painful shoulder was reported by 256/1474 (17%), 261/1,336 (20%) at one month and 284/1,201 (23%) at six months.</td>
</tr>
<tr>
<td>Lindgren et al. (2007)</td>
<td>Population-based cohort of 416 first-ever stroke patients were assessed for shoulder pain using a 0-100 mm visual analog scale. Shoulder pain was initially reported by 71 (22%) patients. At 4 months, 71/327 (22%) of the remaining patients reported shoulder pain by 16 months, 74/305 (24%) reported pain.</td>
</tr>
<tr>
<td>Dromerick et al. (2008)</td>
<td>46 consecutive stroke rehabilitation inpatients were examined prospectively within 2 weeks of admission. Pain was self-reported in 17 (37%), 7 with pre-existing pain.</td>
</tr>
<tr>
<td>Sackley et al. (2008)</td>
<td>600 hospitalized stroke survivors were followed over a one-year period to assess the incidence of various complications, including shoulder pain. At 3 months, 44/122 (36%) reported shoulder pain. At 6 and 12 months, 37/89 (42%) and 34/73 (47%) reported shoulder pain.</td>
</tr>
<tr>
<td>Klit et al. (2011)</td>
<td>15.1% of 608 stroke survivors who responded to a postal questionnaire indicated that they experienced new onset of shoulder pain within 2 years of stroke.</td>
</tr>
<tr>
<td>Hansen et al. (2012)</td>
<td>299 acute stroke patients were interviewed at 4 days following stroke onset and at 3 and 6 months by telephone to determine the incidence of post-stroke pain. Newly developed shoulder pain was present at stroke onset (1.5%), 3 months (13.1%) and 6 months (16.1%)</td>
</tr>
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</table>

Shoulder pain, by itself, can result in significant disability (Najenson et al., 1971; Poduri, 1993). Although it can occur as early as 2 weeks post stroke, an onset time of 2-3 months post stroke is more typical (Poduri, 1993). In a prospective study, Gamble et al. (2000) reported that 52/152 (34%) developed shoulder pain following stroke, 28% by two weeks and 87% by two months. By 6 months, the pain had resolved in 80% of the patients. Lindgren et al. (2007) reported that 74 (24%) of 305 patients with first ever stroke, who remained from an original cohort of 416 patients, experienced shoulder pain by month.
Approximately half of these patients developed pain between stroke onset and 4 months. Rajaratnam et al. (2007) identified three factors that predict, with 98% accuracy, the development of hemiplegic shoulder pain following acute stroke: a positive Neer test, moderate or greater shoulder pain during the performance of the hand behind the neck manoeuvre and a difference of greater than 10 degrees of passive external rotation at the shoulder joint. Depression, reduction to light touch and reduction to temperature sensation have also been associated with shoulder pain (Gamble et al., 2000).

Shoulder pain can 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). Lo et al. (2003) catalogued the different types of shoulder dysfunction based on both clinical and arthrographic findings and reported that 16% of patients of a cohort of 32 patients with hemiplegic shoulder pain within one-year of stroke had shoulder-hand syndrome, 4% had rotator cuff tears and 50% suffered from adhesive capsulitis (frozen shoulder). Sixty-three percent of patients had a single type of shoulder dysfunction while 34% had two types (Lo et al., 2003).

**Conclusions Regarding the Incidence of Hemiplegic Shoulder Pain Following Stroke**

The reported incidence of post stroke shoulder pain varies from 9% to 73%.

There is a high variability in the reported incidence of shoulder pain.

### 11.2 Causes of Hemiplegic Shoulder Pain (HSP)

Stroke affecting the upper limbs usually results in shoulder hemiplegia which often becomes painful over time. Interestingly, hemiplegic shoulder pain (HSP) stroke-related neglect (Kaplan, 1995). Factors most frequently associated with shoulder pain are shoulder (glenohumeral) subluxation (Grossens-Sills & Schenkman, 1985; Moskowitz et al., 1969; Savage & Robertson, 1982; Shai et al., 1984), shoulder contractures or restricted shoulder range of motion (Bloch & 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) (Table 11.2.1). Suggested causes of shoulder pain 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).

In a recent study, MRI was used to examine a potential association between structural changes in hemiplegic shoulder and pain (Shah et al., 2008). In a series of 89 patients with shoulder pain, 35% of subjects exhibited a tear of at least one rotator cuff, biceps or deltoid muscle and 53% exhibited tendinopathy of at least one rotator cuff, bicep or deltoid muscle, rotator cuff tears and rotator cuff and

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**Table 11.2.1 Potential Causes of Hemiplegic Shoulder Pain**

<table>
<thead>
<tr>
<th>Anatomical Site</th>
<th>Mechanism</th>
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<tr>
<td>Muscle</td>
<td>Rotator Cuff, Muscle Imbalance, Subscapularis Spasticity, Pectoralis Spasticity</td>
</tr>
<tr>
<td>Joint</td>
<td>Glenohumeral Subluxation</td>
</tr>
<tr>
<td>Bursa</td>
<td>Bursitis</td>
</tr>
<tr>
<td>Tendon</td>
<td>Tendonitis/Tear</td>
</tr>
<tr>
<td>Joint Capsule</td>
<td>Frozen or Contracted Shoulder (Adhesive Capsulitis)</td>
</tr>
<tr>
<td>Other</td>
<td>Complex Regional Pain Syndrome (CRPS)</td>
</tr>
</tbody>
</table>
deltoid tendinopathies. These changes were not related to severity of shoulder pain (Shah et al., 2008). Results should be interpreted with caution since this study lacks comparative control analyses and therefore reliable conclusions cannot be drawn against age-matched non-stroke populations.

Multivariate analyses in an observational study performed on 94 participants revealed that age >70, poor arm motor function, and the presence of supraspinatus tendon tendinosis/tear were significant factors associated with an increased risk of developing HSP (Kim et al., 2014). Furthermore, patients with adhesive capsulitis or long head of biceps tendon effusion were found to have a higher likelihood of developing HSP compared to patients without these complications (Kim et al., 2014). In a recently study by Karaahmet et al. (2014), a greater prevalence of HSP was found among patients with supraspinatus tendinosis/tear than among those without.

**Conclusions Regarding Risk Factors of Hemiplegic Shoulder Pain**

*Causes and risk factors of hemiplegic shoulder pain include muscle imbalance and poor arm function, pectoralis/subscapularis spasticity, glenohumeral subluxation, bursitis, tendonitis/tear, adhesive capsulitis, and shoulder-hand syndrome.*

**Various causes/risk factors of hemiplegic shoulder pain include muscle imbalance and poor arm function, pectoralis/subscapularis spasticity, glenohumeral subluxation, bursitis, tendonitis/tear, adhesive capsulitis, and shoulder-hand syndrome.**

### 11.3 Shoulder Subluxation

#### 11.3.1 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. 2013). The glenohumeral joint is multiaxial and has a range of motion, which exceeds that of other joints in the body. To achieve this mobility the glenohumeral joint must sacrifice stability. Stability is achieved through the rotator cuff, a musculotendinous sleeve which 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.

Shoulder subluxation is a very common problem in hemiplegic patients. 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 while the patient is in the upright position or pulling on the hemiplegic arm when transferring the patient 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) (Figure 11.3.1).
11.3.1 A. Normal Shoulder.
The humeral head is maintained in the glenoid fossa by the supraspinatus muscle.

11.3.1 B. Shoulder Subluxation.
During the initial phase of hemiplegia, the supraspinatus muscle is flaccid. The weight of the unsupported arm can cause the humeral head to sublux downward in the glenoid fossa.

11.3.2 Scapular Rotation
There appear to be other factors playing a role in subluxation of the glenohumeral joint. Basmajian and Bazant (1959) proposed that on the normal state, subluxation of the humeral head was prevented by upward angulation of the glenoid fossa and the upper part of the shoulder capsule, the coracohumeral ligament and supraspinatus muscle. After a hemiplegic stroke they hypothesized that the upward angulation of the scapula would be lost. Cailliet (1980) added that in the flaccid stage, the scapula assumed a depressed and downward rotated position, as the paretic serratus anterior and the upper part of the trapezius muscles no longer support the scapula. The combination of flaccid supportive musculature (in particular, the supraspinatus muscle) and a downward rotated scapula was presumed to predispose the head of the humerus to undergo inferior subluxation relative to the glenoid fossa; however, the presence of subluxation was found in only one patient out of 52 who had downward scapular rotation in Ikai et al (1992).
Prévost et al. (1987), using a 3-D x-ray technique, studied the movement of the scapula and the humerus in stroke patients by comparing the affected to the non-affected shoulder. The authors demonstrated that there was a significant difference between the affected and non-affected shoulders in terms of the vertical position of the humerus (i.e. degree of subluxation) in relation to the scapula. The orientation of the glenoid fossa was also different; however, they found that with the subluxed shoulder it was actually facing less downward. There was no significant relationship noted between the orientation of the scapula and the severity of subluxation. They concluded that the scapular position was not an important factor in the occurrence of inferior subluxation in hemiplegia (Prévost et al., 1987).

Culham et al. (1995) reported that while patients with low-tone had significantly greater subluxation compared to the high-tone group, (0.52 vs. 0.21), there was no correlation between the amount of subluxation and the scapular abduction angle or the humeral abduction angle. Price et al. (Price & Pandyan, 2001) compared patients with and without stroke and reported that subluxation in stroke patients was unrelated to scapular resting position (Price & Pandyan, 2001). These authors also reported that the normal scapula tilts downward to a greater degree than found in other studies.

11.3.3 Pain in Shoulder Subluxation
Shoulder subluxation may be associated with several conditions including: shoulder pain (Grossens-Sills & Schenkman, 1985; H. Moskowitz et al., 1969; Christopher W. Roy et al., 1994; Savage & Robertson, 1982; Shai et al., 1984) and frozen shoulder or brachial plexus traction injury (Kingerly et al., 1993), although evidence for the latter is lacking (Kingerly et al., 1993). It has long been assumed that if not corrected; a pattern of traction on the flaccid shoulder will result in pain, decreased range of motion and contracture. However, not all patients with a subluxed hemiplegic shoulder experience shoulder pain and it remains controversial as to whether it causes hemiplegic shoulder pain (Bender & McKenna, 2001; Fitzgerald-Finch & Gibson, 1975; H. Moskowitz et al., 1969; Shahani et al., 1981). 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 is probably present later after stroke since “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”. Although several studies have reported an association, others have not confirmed this finding. Heterogeneity of patient characteristics and timing and method of assessment (radiological vs. clinical examination) may account for the lack of consistency among findings.

Shoulder subluxation was found to range from 29% to 57.6% in post-stroke patients (Bohannon, 1988; Lo et al., 2003; Shai et al., 1984; Suethanapornkul et al., 2008; Wanklyn et al., 1996). Shoulder pain on the other hand, was found to range to a much greater degree from 13.3% to 80%, with some findings associating the shoulder pain with shoulder subluxation (Barlak et al., 2009; Bohannon, 1988; Bohannon & Andrews, 1990; Ikai et al., 1998; Joynt, 1992; Shai et al., 1984; Suethanapornkul et al., 2008). Thus far, 31.8% to 82% of patient with pain were also found to have shoulder subluxation (Barlak et al., 2009; Shai et al., 1984). Suethanapornkul et al (2008) suggested that a significantly higher frequency of shoulder pain was found in patient with shoulder subluxation at 2-6 months post-stroke however, Joynt et al. (1992) indicates otherwise. Other studies indicate that spontaneous pain was more severe in the hemiparetic side compared to the healthy side (Lin et al., 2014) however, total asymmetry was not found to correlate with shoulder pain (Zorowitz et al., 1996). Therefore, based on the current literature, it is unclear whether shoulder subluxation is associated with shoulder pain.

Conclusions Regarding Shoulder Subluxation Post-Stroke
Shoulder subluxation may occur early on in the hemiplegic arm due to flaccid supporting shoulder musculature.

Shoulder subluxation may be a cause of shoulder pain; however, patients with shoulder subluxation do not necessarily experience pain and not all cases of hemiplegic shoulder pain suffer from subluxation.

It is uncertain whether shoulder subluxation causes hemiplegic shoulder pain.

11.4 Spasticity, Contractures and Hemiplegic Shoulder Pain (HSP)

The Pathogenesis of HSP is multifactorial and includes neurologic and mechanical factors, often in combination, which vary among those affected post-stroke.

Abnormal muscle tone, including spasticity may be directly related to HSP. 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 muscle stretch reflexes and often associated with a clasp-knife phenomena. Spasticity is one component of the upper motor neuron (UMN) syndrome and is the inevitable accompaniment of hemiplegia and an incomplete motor recovery. 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 will decrease or stop entirely. The balance of control over the muscle tips in favour of facilitation and spasticity results. Spasticity develops only if there is loss of input from both pyramidal and extrapyramidal motor systems. Spasticity presents as increased tone and reflexes on the involved side of the body.

van Ouwenaller et al. (1986) looked at various factors in 219 patients followed for one year after a stroke and identified a much higher incidence of shoulder pain in spastic (85%) than in flaccid (18%) hemiplegics. They identified spasticity as "the prime factor and the one most frequently encountered in the genesis of shoulder pain in the hemiplegic patient." However, the etiology of the subsequent shoulder pain was unclear.

Poulin de Courval et al. (1990) examined 94 hemiplegic subjects involved in a rehabilitation program after stroke and reported that subjects with shoulder pain had significantly more spasticity of the affected limb than those without pain. In contrast, Bohannon et al. (1986) conducted a statistical analysis of 50 consecutive hemiplegic patients (36 with shoulder pain) and asserted that "spasticity was unrelated to shoulder pain." Joynt (1992) also supported this finding after examining 67 patients with shoulder problems following stroke.

A study of approximately 100 stroke patients revealed that patients with severe paralysis and supraspinatus tendon pathology showed higher prevalence of HSP at 3 and 6 months (Kim et al. 2014). The peak onset and severity was found to occur at 4 months post-incident according to a recent prospective study of 301 patients with a 12 month follow up (Adey-Wakeling et al. 2015). The incidence and frequency of post-stroke shoulder pain is on the rise and expected to approach 30% (Adey-Wakeling et al. 2015). This rise is also expected to have huge implications in patients with acute stroke as quality of
life was found to be dramatically reduced as result of hemiplegic shoulder pain (Adey-Wakeling et al. 2016).

11.4.1 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. One consequence of this is the development of spastic muscle imbalance about the shoulder joint.

Clinically, the internal rotators of the shoulder predominate and is one of the last areas of shoulder function to recover. During recovery, motor units are not appropriately recruited or turned off and therefore the result is 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. Shortened 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 degrees. Muscles that contribute to spastic internal rotation/adduction of the shoulder include the subscapularis, pectoralis major, teres major and latissimus dorsi muscles. However, two muscles in particular have been implicated as most often being spastic leading to muscle imbalance. These are the subscapularis and pectoralis major muscles.

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 11.4.1.1). It is a major internal rotator of the shoulder (Hollinshead & Jenkins, 1981). The subscapularis muscle also participates in arm adduction and extension from a flexed position (Cole & Tobis, 1990). In normal individuals, nerve impulses to the subscapularis are inhibited during arm abduction; the muscle then 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 spastic hemiplegics, internal rotators, including the subscapularis muscle, are tonically active. This limits shoulder abduction, flexion and external rotation.

Bohannon et al. (1986) found limitation of external rotation of the hemiplegic shoulder was the factor which most correlated with hemiplegic shoulder pain. Zorowitz et al. (1996) also found that limitation in shoulder external rotation correlated strongly with pain. Hecht (1995) noted that "the subscapularis muscle is the primary cause of shoulder pain in spastic hemiplegia where external rotation is most limited. Although other muscles may contribute to spasticity, pain and functional contracture, the subscapularis is the keystone of the abnormal synergy pattern," (Hecht, 1995).

The subscapularis spasticity disorder is characterized by motion being most limited and pain being reproduced on external rotation. A tight band of spastic muscle is palpated in the posterior axillary fold. In support of this, Inaba and Piorkowski (1972) reported external rotation was the most painful and limited movement of the hemiplegic shoulder.

Pectoralis Spasticity Disorder
The pectoralis major muscle serves to forward flex, adduct and internally rotate the arm. Hecht (1995) has 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 appears to be problematic. This disorder is characterized by motion being most limited and pain produced on abduction. It is also noteworthy that the pectoralis major muscle is a synergist of the subscapularis muscle.

**Figure 11.4.1.1 The Subscapularis Muscle.**

The subscapularis muscle is a major internal rotator of the shoulder. As part of the typical flexor synergy pattern in spastic hemiplegics, the subscapularis is tonically active limiting not only external rotation but also shoulder abduction and flexion.

**Figure 11.4.1.2 The Pectoralis Major Muscle.**

The pectoralis major muscle serves to adduct, internally rotate and forward flex the arm at the shoulder.
The importance of other shoulder muscles; biceps, pectoralis minor muscle, and latissimus dorsi have not been studied in the stroke population. However, recent studies in the sport literature suggest that other muscles should be considered in scapular dyskinesia (Morals & Cruz 2016). A review by Kalichman & Ratmansky (2011) outlines a systematic approach to the underlying causes of HSP (see Figure 11.4.1.3), suggesting 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 hemiplegic shoulder pain. These issues may occur separately, co-exist simultaneously or may develop as a result as a trigger from a previous symptom (Kalichman & Ratmansky 2011).

Figure 11.4.1.3 Systemization of pathologies underlying hemiplegic shoulder pain. Adapted from Kalichman & Ratmansky (2011).

11.4.2 Frozen or Contracted Shoulder
A frozen or contracted shoulder is characterized clinically by limitations in range of movement, with a pattern of restriction. This condition is a frequently identified source of pain in the spastic hemiplegic shoulder (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).

The incidence of contractures in frozen shoulder was found to range between 54.6% and 76.7% (Hakuno et al., 1984; Rizk et al., 1984). Some authors reported no rotator cuff capsular tears in patients with hemiplegic shoulder pain (Rizk et al., 1984) however, Lo et al. (2003) indicated that out of the 32 participants included in the study, 54% had rotator cuff tears as diagnosed by arthrography.

Hemiplegic shoulder pain appears to be due to a combination of spastic muscle imbalance and a frozen contracted shoulder. Grossen-Sills & Schenkman (1985) found a positive relationship between shoulder pain and loss of shoulder range of motion as well as subluxation. Subluxation was however not significantly correlated with range of motion (Grossens-Sills & Schenkman, 1985).

Conclusions Regarding Frozen or Contracted Shoulder
The incidence of contractures in hemiplegic shoulder pain range from 54.6% to 76.7%.

There may be an association between shoulder contractures and hemiplegic shoulder pain. Spasticity in shoulder adductors and internal rotators, especially subscapularis and pectoralis major, have been implicated in hemiplegic shoulder pain.

11.5 Rotator Cuff Disorders

Because shoulder pain is so often associated with rotator cuff disorder it should not be surprising that it would be seen as a potentially common cause of hemiplegic shoulder pain. However, Rizk et al. (1984) failed to demonstrate any evidence of rotator cuff tears on arthrography in 30 patients with hemiplegic shoulder pain. A similar study reported a 33% incidence of rotator cuff tears in painful shoulders after strokes (Nepomuceno & Miller, 1974). Najenson et al. (1971) reported that 13 of 32 (40%) patients with severe paralysis of the upper extremity were found to have a rupture of the rotator-cuff tendon based on arthrographic findings. Partial tears of the rotator cuff musculature are common and it is always difficult determining whether they were present pre-morbidly even in previously asymptomatic patients. For instance, Yamaguchi et al. (2006) evaluated 588 patients with unilateral shoulder complaints and found that 177 patients (30.1%) had bilateral rotator cuff tears and only 199 patients (33.8%) 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). Joynt (1992) diagnosed 67 stroke patients as having hemiplegic shoulder pain. Generally, hemiplegic shoulder pain is not commonly associated with rotator cuff disorders.

11.6 Painful Hemiplegic Shoulder and Motor Impairments

A painful hemiplegic shoulder can be very limiting and has the potential to further add to the disability seen with hemiplegia. Several studies have attempted to discern the association between such variables however, the findings are conflicting.

While motor deficit has been associated with shoulder pain, a cause and effect relationship has not yet been established. Chae et al. (2007) found that pain was not a predictor of function in any of their models tested however other authors indicate a positive association between shoulder pain and motor ability. For instance, patients with pain had reduced shoulder shrug and pinch grip ability (Wanklyn et al., 1996), lower arm function and arm power (Roy et al., 1995), and generally, lower motor functional level (Aras et al., 2004). Shoulder pain was significantly associated with reflex sympathetic dystrophy, subluxation, lower Barthel scores (<15) or poorer ADLs, limited external rotation and flexion of the shoulder, reduced arm function, motor deficit, diabetes, and severity of deficit (Aras et al., 2004; Ratnasabapathy et al., 2003; Roy et al., 1995; Wanklyn et al., 1996). In patients with 6 months post-stroke, the risk of shoulder pain was found to increase with severity of upper limb motor deficit (Ratnasabapathy et al., 2003). Conversely, no significant relationship was found between shoulder pain and gender, time since onset of disease, pathogenesis of spasticity, neglect, and thalamic pain (Aras et al., 2004). Conflicting evidence also exists regarding several other associations involving hemiplegic shoulder pain. For instance, Aras et al. (2004) suggests that extension of hospital stay was not correlated with shoulder pain however, Roy et al (1995) found that shoulder pain on movement was associated with an increase length of stay at the hospital.

Conclusions Regarding Functional Impact of Hemiplegic Shoulder Pain

The development of painful hemiplegic shoulder may be associated with poorer functional outcomes, reduced motor ability, and stroke severity.

Painful hemiplegic shoulder may be associated with reduced motor function and increased stroke severity.

11.7 Management of the Painful Hemiplegic Shoulder

Management of the painful hemiplegic shoulder is difficult and response to treatment is frequently unsatisfactory (Rizk et al., 1984). The best treatment approach has not been established, in part, due to the uncertainty of the etiology of the pain. As a result, a wide variety of treatments have been used, with varying degrees of success (Snels et al., 2002). However, the management of hemiplegic shoulder pain would be greatly improved with the identification of the primary pain generator/etiology for a given patient by performing a proper, focused physical examination and selecting the appropriate intervention to address these clinical findings (Viana et al., 2012). Ideally, measures should be taken immediately following stroke to minimize the potential for the development of shoulder pain. Early passive shoulder range of motion, and supporting and protecting the involved shoulder, in the initial flaccid stage 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 associated spasticity. Careful positioning of the shoulder serves to minimize subluxation and later contractures as well as possibly promote recovery, while poor positioning may adversely affect symmetry, balance and body image.

Gilmore et al. (2004) suggests that through careful and correct positioning, the development of shoulder pain can be prevented. Bender and McKenna (2001) have noted that a primary goal of early stroke management is to prevent the development of hypertonicity (Johnstone, 1982) and to discourage inefficient patterns (Bobath, 1990). Bender and McKenna (2001) noted that the “recommended position for the upper limb is towards abduction, external rotation and flexion of the shoulder,” however, from Carr and Kenny’s (1992) review, Bender and McKenna cite that “most popular theories failed to yield consensus for exact degrees of the positioning”.

Table 11.7.1.1 Summary of Studies Investigating Positioning of the Shoulder in Stroke Patients

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>Country</th>
<th>PEDro Score</th>
<th>Methods</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ada et al. (2005b) RCT (6)</td>
<td>N=36</td>
<td>E: Sustained shoulder positioning (position 1: maximum external rotation; position 2: resting arm at 90 degrees of flexion) C: Shoulder exercises and routine upper limb care</td>
<td>• Contractures: Position 1 (+); Position 2 (-) • Pain (-)</td>
<td></td>
</tr>
<tr>
<td>Gustafsson &amp; McKenna (2006) RCT (6)</td>
<td>N=34</td>
<td>E: Static positional stretches C: Supported when seated in bed</td>
<td>• Range of Motion: Passive (-) • Pain (-) • Motor Assessment Scale (-) • Modified Barthel Index (-)</td>
<td></td>
</tr>
<tr>
<td>de Jong et al. (2006) RCT (6)</td>
<td></td>
<td>E: Positioning procedure C: Routine rehabilitation</td>
<td>• Range of Motion: Passive (-) • Modified Ashworth Scale (-)</td>
<td></td>
</tr>
</tbody>
</table>
11. Painful Hemiplegic Shoulder

---

**Table 11.7.2.1 Results from Systematic Review (Ada et al., 2005a)**

| N=19 | \( \text{E: Prolonged positioning} \) | \( \text{C: Standard rehabilitation therapy} \) |
| N=23 | \( \text{E: Standard positioning} \) | \( \text{C: Standard rehabilitation therapy} \) |

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

---

**Discussion**

Thus far, only a handful of RCTs have investigated the effect of various positioning therapies on shoulder pain and motor recovery (Table 11.7.1.1). The majority of the studies indicate that pain was not significantly improved when the patients were treated with various positioning procedures relative to other routine rehabilitation activities (De Jong et al., 2006; Gustafsson & McKenna, 2006). Range of motion or general motor was also not found to be enhanced following positioning procedures (De Jong et al., 2006; Dean et al., 2000; Gustafsson & McKenna, 2006).

The findings of this review are paralleled by those obtained from a meta-analysis, which included the results from 5 RCT, representing 126 subjects (Borisova & Bohannon, 2009). The results found that shoulder positioning programs were not effective in preventing or reducing the loss of shoulder external rotation range of motion. Interventions assessed in the 5 trials included both positioning and stretching programs (Borisova & Bohannon, 2009). In order to prevent contraction, the authors speculated that the duration of stretching may have been insufficient. While 2 hours of stretch was provided in the 2 trials that assessed this form of treatment, the authors speculated that at least 6 hours of stretching a day was required to prevent contracture formation in the soleus muscle of children with cerebral palsy (Borisova & Bohannon, 2009). The authors also suggest 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 shoulder positioning may not reduce pain, motor function, range of motion, or spasticity.*

*Evidence suggests that shoulder positioning may not improve pain or motor impairments related to shoulder subluxation.*

---

**11.7.2 Slings and Other Aids**

Arm slings are often used in the initial stages following a stroke to support the affected arm. However, their use is controversial and they can have disadvantages in that they encourage flexor synergies, inhibit arm swing, contribute 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. Ada et al. (2005a) conducted a systematic Cochrane review evaluating the benefit of shoulder slings and supports, and concluded that there is insufficient evidence that these devices reduce or prevent shoulder subluxation following a stroke. The review included only four RCTs (Ancliffe, 1992; Hanger et al., 2000; Hurd et al., 1974), one presenting unpublished data (Griffin & Bernhardt, 2006). The results are presented in Table 11.7.2.1.
Outcome | Studies Included & Intervention | Peto Odds ratio (95% CI) or Weighted Mean Difference (WMD) (95% CI)
---|---|---
Proportion of patients with pain at follow up | Hurd et al., 1974 – hemisling | OR 8.7 (1.1, 67.1) (Favours no slings)
Number of days pain was delayed with treatment | Ancliffe et al., 1992 -strapping Griffin et al., 2006 | WMD 14 days (9.7, 17.8) (Favours slings)
Pain scores on VAS (10 cm scale) | Hanger et al., 2000 – strapping | WMD 0.83 (-1.46, 3.12) (No difference)
Motor Assessment sub scores (0-18) | Hanger et al., 2000. -strapping | WMD 0.8 (-1.5, 3.1) (No difference)
Proportion of patients with contractures | Hurd et al., 1974- sling | OR 1.00 (0.1, 9.3) (No difference)
Range of shoulder external rotation at end of follow up | Hanger et al., 2000 - strapping | WMD -1.4 degrees (-10.9, 8.10) (No difference)

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. The best method to support the shoulder has yet to be determined. In the absence of empirical evidence of their efficacy, many devices are available and in common use, including a variety of slings and lapboards.

**Table 11.7.2.2 Summary of Strapping the Hemiplegic Shoulder**

<table>
<thead>
<tr>
<th>Author, Year Country PEDro Score</th>
<th>Methods</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hartwig et al., (2012) RCT (7) N=41</td>
<td>E: Conventional care + functional orthosis (Neuro-Lux) C: Conventional care</td>
<td>• Shoulder-hand syndrome score (+): limited humeral abduction (+); limited external rotation (+) • Pain (+)</td>
</tr>
<tr>
<td>Hurd et al., (1974) PCT N=14</td>
<td>E: Treated with sling C: Treated without a sling</td>
<td>• Shoulder pain (+)</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

**Discussion**

The majority of the trials assessed in this review (Table 11.7.2.2) used a crossover design whereby patients served as their own control as they tried more than one type of sling for a period of time. The use of a conventional sling was found to improve pain and reduce subluxation in the majority of the patients tested (Hurd et al., 1974; Moodie et al., 1986). On the other hand, the shoulder roll did not improve subluxation as much as the other comparable treatment options and was not found to differ from the Henderson shoulder sling (Moodie et al., 1986; Williams et al., 1988). The Hook-Hemi-Harness was found to under-correct subluxation in one study (Moodie et al., 1986) however, Brooke et al. (1991) showed that the sling improved correction of subluxation and that this treatment was favoured over the Bobath sling (Brooke et al., 1991). One study investigated the effectiveness of the arm trough and the plexiglas lap tray, and found reduced pain and subluxation in the study sample tested as a result of both treatments (Moodie et
al., 1986). The cavalier support produced significant lateral displacement of the humeral head of the affected shoulder compared to the unaffected shoulder, while the Roylan humeral cuff sling significantly reduced subluxation asymmetry (Zorowitz et al., 1995).

In a single RCT, the effect of a functional orthosis labelled Neuro-Lux was tested (Hartwig et al., 2012). Participants who wore the device for 10 hours each day demonstrated lower mean shoulder-hand syndrome scores (by 3.1 points) compared to those part of the usual care treatment (Hartwig et al., 2012). By day 28 of wearing the device, all components of shoulder and syndrome (i.e. pain, hand edema, limited movement of humeral abduction and humeral external rotation) were significantly lower compared to the score obtained from patients in the control group. The majority of the participants also found the orthosis to be comfortable (Hartwig et al., 2012). Despite positive results, the sample size was small, and the assessors were not blinded, thus limiting the interpretation of these findings. The study also does not specify the scales/outcomes used to measure pain or whether the measures were subjective or objective. Future trials assessing this device are therefore encouraged to consider larger sample sizes, and to use objective outcomes to assess shoulder-hand syndrome and its components.

Conclusions Regarding Slings in Hemiplegic Shoulder

There are a wide variety of shoulder slings/treatment options available; however, there is no consensus regarding which is the most efficacious at reducing subluxation.

There is limited evidence that shoulder slings influence clinical outcomes.

11.7.3 Strapping the Hemiplegic Shoulder
Strapping 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 three different forms of strapping the hemiplegic shoulder, which have been described previously:

Ancliffe (1992): 5-cm wide lightweight adhesive tape (Fixomull Stretch), “the first length of tape was applied to the shoulder half way along the length of the clavicle, continued across the deltoid muscle in a diagonal direction. the tape was terminated approximately one-quarter of the way of the along the spine of the scapula. A second length of tape was applied in the same direction as the first but 2 cm below. A small length of tape was applied over the shoulder to secure the ends”.

Morin & Bravo (1997): “A 10 cm-wide Elastoplast adhesive bandage was applied under tension from the forearm under the olecranon laterally to the top of the shoulder. Two other 7.5 cm-wide bandages were applied from the olecranon under the forearm to the top of the shoulder, with one passing anteriorly over the clavicle and the other posteriorly covering the spine of the scapula. No free space was left between the bandages”.

Hanger et al. (2000): Three lengths of nonstretch Elastoplast Sports tape were used. “The two main supporting tapes were applied first. Both were applied using a lifting action, starting 5 cm above the elbow, and moving up the arm front and back, crossing at the top of the shoulder. The posterior arm tape was then anchored down past the clavicle whereas the tape from the anterior aspect of the arm came across the shoulder and down past the spine of the scapula. They were both supported at the lower end by a short tape to prevent them peeling off”.

Table 11.7.3.1 Summary of Studies Regarding Strapping the Hemiplegic Shoulder

11. Painful Hemiplegic Shoulder
Author, Year Country PEDro Score | Methods | Outcomes
---|---|---
Pandian et al. (2013) RCT (8) N=162 | E: Shoulder taping  
C: Sham taping | • Pain (+)  
• Disability Index (-)
Hanger et al. (2000) RCT (7) N=98 | E: Shoulder strapping  
C: No strapping | • Shoulder-hand syndrome score (+): limited humeral abduction (+), limited external rotation (+)  
• Pain (+)
Griffin & Bernhardt (2006) RCT (7) N=33 | E: Shoulder strapping  
C: Sham shoulder strapping | • Pain (-)  
• Range of Motion (-)  
• Functional Measures (-)
Appel et al. (2011) RCT (6) N=14 | E: Shoulder strapping  
C: Rehabilitation only | • Pain-free days (+)  
• Range of Motion (-)  
• Motor Assessment Scale (-)  
• Modified Ashworth Scale (-)
Ancliffe et al. (1992) PCT N=8 | E: Shoulder strapping  
C: No strapping | • Motor Assessment Scale (+)*  
• Fugl-Meyer (+)*  
• Nine Hole Peg Test (+)*

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

Discussion

Thus far, five studies have investigated the effect of shoulder strapping of the hemiplegic shoulder on pain and motor function (Table 11.7.3.1). Of the four studies assessing pain, one RCT and one PCT reported significantly longer pain free duration compared to when the participants received sham strapping or no strapping (Ancliffe, 1992; Griffin & Bernhardt, 2006). Pandian et al (2013) found a significant improvement in pain following shoulder taping however, Hanger et al. (2000) found no benefit of shoulder strapping. Overall, the findings appear to favour the sling method for reducing pain however more studies are still warranted.

Upper limb motor function was not found to improve following shoulder strapping as illustrated by Griffin & Bernhardt (2006) and Hanger et al. (2000). Conversely, Appel et al (2011) showed that the shoulder strapping method may have improved upper limb motor function, however the study does not provide statistical inferences. Furthermore, strapping was not superior compared to sham strapping/taping at reducing disability or spasticity (Griffin & Bernhardt, 2006; Pandian et al., 2013).

Conclusions Regarding Strapping the Hemiplegic Shoulder

*There is level 1a evidence that shoulder strapping/taping may reduce hemiplegic shoulder pain; however, it may not improve range of motion, spasticity, disability, or upper limb motor function.*

*Strapping/taping the hemiplegic shoulder does not appear to improve upper limb function, but may reduce pain.*
11.7.4 Active Therapies in the Hemiplegic Shoulder

The association of spasticity, muscle imbalance and a frozen shoulder with shoulder pain suggests that a therapeutic approach designed to improve range of motion of the hemiplegic shoulder may improve pain. See table 11.7.4.1 for studies examining active therapy for shoulder hemiplegia.

Table 11.7.4.1 Summary of Studies Regarding Active Therapy for the Hemiplegic Shoulder

<table>
<thead>
<tr>
<th>Author, Year Country PEDro Score</th>
<th>Methods</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inaba &amp; Piorkowski (1972) RCT (7) N=33</td>
<td>E1: Range of motion (ROM) exercises + positioning group E2: ROM exercises and ultrasound C: ROM exercises and mock ultrasound</td>
<td>• Range of Motion (-)</td>
</tr>
<tr>
<td>Lynch et al. (2005) RCT (6) N=35</td>
<td>E: Continuous passive motion treatments C: Self-range of motion exercises</td>
<td>• Modified Ashworth Scale (-) • Fugl-Meyer: Pain (-), Self-care (-) • Joint stability (-)</td>
</tr>
<tr>
<td>Kumar et al. (1990) RCT (5) N=28</td>
<td>E1: Range of motion exercises E2: Rehabilitation with a skate board E3: Rehabilitation with overhead pulley</td>
<td>• Pain: E3 vs. E1 (+) • Subluxation (-)</td>
</tr>
<tr>
<td>Partridge et al. (1990) RCT (5) N=65</td>
<td>E: Cryotherapy C: Bobath therapy</td>
<td>• Pain: C (+)</td>
</tr>
<tr>
<td>Tyson &amp; Chissim (2002) RCT (4) N=22</td>
<td>E: Distal hold with axilla hold (involving shoulder support) C: Distal hold without shoulder support</td>
<td>• Flexion (+)</td>
</tr>
<tr>
<td>You et al. (2014) PCT</td>
<td>E1: Stretching and joint stabilizing exercise therapy E2: Stretching exercise therapy C: Normal development therapy</td>
<td>• Range of Motion (-)</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

Discussion

Inaba & Piorkowski (1972) found no significant differences in the outcomes of patients who received: ROM exercises and positioning, ROM exercises and ultrasound or ROM exercises and mock ultrasound. Kumar et al. (1990) found that overhead pullies caused dramatically higher levels of shoulder pain than more restrained ROM exercises. Although there were no statistically significant differences in change scores between the control and the experimental group, Lynch et al. (2005) reported a trend towards improvement in the area of shoulder joint stability associated with continuous passive motion using the OrthoLogic Danniflex600 shoulder CPM system. Partridge et al. (1990) found that treatments using Bobath therapy resulted in significantly less pain than cryotherapy.

Conclusions Regarding Active Therapies in the Hemiplegic Shoulder

There is level 1b evidence that supplementing range of motion activities with ultrasound or positioning exercises may not be more effective than when performing range of motion exercises alone.

There is level 2 evidence that aggressive range of motion therapies, using overhead pullies may result in increased rates of shoulder pain.
There is level 1b evidence that continuous passive range of motion exercises are not superior over self-range of motion exercises at improving joint stability, spasticity, or pain.

There is limited level 2 evidence that stretching and joint stabilizing therapies are more effective at improving motor arm function compared to normal development therapies.

There is limited level 2 evidence that Bobath therapy for the hemiplegic shoulder may be associated with greater pain reduction than passive cryotherapy (application of local cold therapy).

**Aggressive range of motion exercises (i.e. pullies) results in a markedly increased incidence of painful shoulder; a gentler range of motion program is preferred. Adding ultrasound treatments does not appear to improve shoulder range of motion.**

### 11.7.5 Electrical Stimulation in the Hemiplegic Shoulder

Electrical neuromuscular stimulation is a term used to describe the application of electrical current to the skin, or directly into muscle that stimulates motor nerves resulting in improved contractility and greater muscle bulk. The treatment can be used to improve muscle strength, joint misalignment, muscle tone, sensory deficits and self-reported pain intensity (Price & Pandyan, 2001). Electrical stimulation for the treatment of pain is typically administered by two methods, 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. During TENS, patients usually describe a “pins-and-needles sensation.” NMES on the other hand, has been described as “electrical stimulation of lower motor neurons to cause muscle contraction”, clinically providing “both therapeutic and functional benefits” (Chae et al. 2008). Another form of electrical stimulation, functional electrical stimulation (FES), is largely used to restore motor function by delivering electrical pulses to muscles causing them to contract. Several studies have also investigated the effect of this intervention on hemiplegic shoulder pain (Faghri et al. 1994; Chantraine et al. 1999; Wang et al. 2000; Koyunchu et al. 2010). Surface FES is performed at frequencies of between 35 to 50 Hz (Paci et al. 2005).

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, NMES should help to compensate or facilitate flaccid shoulder muscles, which in turn should reduce the risk of shoulder subluxation. A case report by Chae et al. (2001) showed improvements in subluxation and pain after 6 weeks of NMES treatment and pain free after 1 year, although subluxation recurred.

Price and Pandyan (2001) conducted a systematic review of all forms of electrical stimulation (ES) used in the prevention and treatment of post stroke shoulder pain. The included studies and the results are presented in Tables 11.7.5.1 and 11.7.5.2. The authors concluded that there was insufficient evidence from which to draw conclusions. There was evidence that FES, in addition to conventional therapy, improves function but is not superior for preventing pain.

**Table 11.7.5.1 Studies included in the Systematic Review authored by Price and Pandyan (2001)**

<table>
<thead>
<tr>
<th>Study</th>
<th>Intervention</th>
<th>Length of Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Faghri et al., 1994</td>
<td>FES vs. no sham treatment</td>
<td>6 weeks</td>
</tr>
<tr>
<td>Leandri et al., 1990</td>
<td>Sham treatment vs. high intensity TENS vs. low intensity TENS</td>
<td>4 weeks</td>
</tr>
<tr>
<td>Linn et al., 1999</td>
<td>No sham treatment vs. electrical stimulation (not FES or TENS)</td>
<td>4 weeks</td>
</tr>
<tr>
<td>Sonde et al., 1998</td>
<td>No sham treatment vs. low frequency TENS</td>
<td>3 months</td>
</tr>
</tbody>
</table>
Table 11.7.5.2 Results From Studies Evaluating Any form of ES in the Treatment and Prevention of Shoulder Pain

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Significant Result (Y/N)</th>
<th>Odds Ratio* or Weighted (or Standardized) Mean Difference and 95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>New cases of shoulder pain</td>
<td>No</td>
<td>0.64 (0.19, 2.14)*</td>
</tr>
<tr>
<td>Pain intensity rating change from baseline</td>
<td>No</td>
<td>0.13 (-1.00, 1.25)</td>
</tr>
<tr>
<td>Passive humeral lateral rotation</td>
<td>Yes</td>
<td>9.17 (1.43, 16.9)</td>
</tr>
<tr>
<td>Motor score change from baseline</td>
<td>No</td>
<td>0.24 (-0.14, 0.62)</td>
</tr>
<tr>
<td>Glenohumeral subluxation compared to baseline</td>
<td>Yes</td>
<td>-1.13 (-1.66, -0.60)</td>
</tr>
<tr>
<td>Spasticity score change from baseline</td>
<td>No</td>
<td>0.05 (-0.28, 0.371)</td>
</tr>
</tbody>
</table>

Ada and Foongchomcheay (2002) also conducted a meta-analysis to examine the effect of electrical stimulation on shoulder subluxation following stroke. This review included the results from 6 RCTs (Baker & Parker, 1986; Faghri et al., 1994; Kobayashi et al., 1999; Linn et al., 1999; Wang et al., 2000). The results, presented in Table 11.7.5.3 suggest that early treatment following stroke helps to prevent the development of hemiplegic shoulder while later treatment helps to reduce pain, in addition to conventional therapy.

Table 11.7.5.3 Pooled Results from Ada & Foongchomcheay (2002)

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Intervention</th>
<th>Significant Result (Y/N)</th>
<th>Weighted Mean Difference and 95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shoulder subluxation (mm)</td>
<td>Early ES + CT* vs. early CT</td>
<td>Yes</td>
<td>6.5 (4.4, 8.6)</td>
</tr>
<tr>
<td>Shoulder subluxation (mm)</td>
<td>Late ES + CT vs. Late CT</td>
<td>No</td>
<td>1.9 (-2.3, 6.1)</td>
</tr>
<tr>
<td>Function (Bobath assessment chart, Motor assessment scale and Fugl-Meyer) expressed as a percentage</td>
<td>Early ES + CT vs. early CT</td>
<td>Yes</td>
<td>18.6 (0.4, 36.7)</td>
</tr>
<tr>
<td>Function (Bobath assessment chart, Motor assessment scale and Fugl-Meyer) expressed as a percentage</td>
<td>Early ES + CT vs. early CT</td>
<td>No</td>
<td>14.4 (-5.4, 34.2)</td>
</tr>
<tr>
<td>Pain (range of motion-degrees)</td>
<td>Early ES + CT vs. early CT</td>
<td>No</td>
<td>3.7 (-1.2, 8.6)</td>
</tr>
<tr>
<td>Pain (Visual analogue scale-cm)</td>
<td>Late ES + CT vs. early CT</td>
<td>Yes</td>
<td>1.6 (0.1, 3.0)</td>
</tr>
</tbody>
</table>

* CT= conventional therapy; ES= electrical stimulation

Thirteen RCTs specifically evaluated the effects of electrical stimulation on the treatment of shoulder pain (Table 11.7.5.4).

Table 11.7.5.4 Summary of RCTs Evaluating Surface Electrical Stimulation

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>PEDro Score</th>
<th>Study Sample</th>
<th>Intervention</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Church et al. (2006)</td>
<td>RCT (9)</td>
<td>N=176</td>
<td>E: sNMES</td>
<td>Action Research Arm Test (-)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C: Sham sNMES</td>
<td>Motricity Index: C (+)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Frenchay Arm Test: C (+)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Pain (-)</td>
</tr>
<tr>
<td>De Jong et al. (2013)</td>
<td>RCT (8)</td>
<td>N Sham=48</td>
<td>E: Static arm positioning + NMES</td>
<td>Passive Range of Motion: external rotation (+)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>N Int=39</td>
<td>C: Sham arm positioning + sham NMES</td>
<td>Pain (-)</td>
</tr>
</tbody>
</table>
### Table 11.7.5.5 Summary of RCTs Evaluating Intramuscular Electrical Stimulation

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>PEDro Score</th>
<th>Study Sample</th>
<th>Intervention</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Wilson et al. (2014)</strong>&lt;br&gt;RCT (9)&lt;br&gt;N&lt;sub&gt;Start&lt;/sub&gt;=25&lt;br&gt;N&lt;sub&gt;End&lt;/sub&gt;=21</td>
<td>E: PNS&lt;br&gt;C: Conventional therapy</td>
<td>Pain (+)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| **Yu et al. (2004)**<br>Chae et al. (2005) | E: Intramuscular NMES<br>C: Cuff-sling | Pain: 3mo (+); 6mo (+); 12mo (+) | Pain-interference: 6mo (+); 12mo (+) |

+ Indicates statistically significant difference between treatment groups
- Indicates no statistically significant difference between treatment groups
E indicates experimental group; C indicates control group
* No statistical inferences conducted
**RCT (7)**  
N=61

- Subluxation: 12mo (-)
- Motor impairment: 12mo (-)
- Range of Motion: 12mo (-)
- Spasticity: 12mo (-)

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

**Discussion**

Currently, there is a large variety of the types and protocols of surface electrical stimulation, each having a different clinical use. One study suggested that electrical stimulation to the hemiplegic shoulder significantly improved pain and shoulder subluxation at post-intervention however, this effect was not observed at the 2 month follow-up (Linn et al., 1999). Kobayashi et al. (1999) also found that stimulation of the supraspinatus or the middle deltoid muscle improved subluxation significantly when compared to conventional therapy however, abduction force was only improved when the stimulation was delivered to the middle deltoid muscle.

Functional electrical stimulation (FES) is often used during a motor task to evoke small muscle movements by delivering electrical impulses peripherally. Results from four RCTs demonstrate that FES may be beneficial for improving shoulder subluxation, spasticity, and selective motor function (Chantraine et al., 1999; Faghri et al., 1994; Koyuncu et al., 2010; Wang et al., 2000). Thus far, its effect on hemiplegic shoulder pain is unclear, as some studies indicates positive effects, while others indicate no benefit to FES use (Chantraine et al., 1999; Faghri et al., 1994; Koyuncu et al., 2010; Wang et al., 2000).

Neuromuscular electrical stimulation (NMES) uses electrical current to stimulate motor nerves to produce muscle contractions. Stroke literature suggests that some motor function and subluxation may be improved with surface NMES (i.e. passive range of motion), however it may not be as beneficial at reducing hemiplegic shoulder pain (Baker & Parker, 1986; Church et al., 2006; Lex D. de Jong et al., 2013). When NMES is delivered intramuscularly to stimulate motor nerves, studies revealed that shoulder pain was improved at 3, 6 and 12 months post-stimulation, however subluxation, motor function, and spasticity were not improved at 6 or 12 months post-stimulation (Chae et al., 2005; Yu et al., 2004). Furthermore, Wilson et al. (2014) reported a statistically and a clinically important difference between patients treated with intramuscular stimulation and those receiving physical therapy at 6 and 10 weeks after treatment regarding shoulder pain. Secondary analyses revealed that time from stroke onset was the most significant predictor of intramuscular NMES success at reducing post-stroke hemiplegic shoulder pain (Chae et al., 2007). When the treatment was administered before 77 weeks after stroke onset (<77 weeks), post-stroke pain was significantly reduced from 94% to 7% (Chae et al., 2007). Conversely, in patients where the treatment was delivered after 77 weeks of stroke onset, pain was not significantly altered (31% vs. 33%) (Chae et al., 2007). Factors such as age, stroke type, gender, Fugl-Meyer Score and side of hemiplegia were not found to be significant predictors of treatment success (Chae et al., 2007).

More studies are needed to understand the long and short term benefits of intramuscular electrical stimulation at improving post-stroke hemiplegic shoulder pain.

In a recent systematic review and meta-analysis, results from 10 RCTs were examined to determine the effect of NMES on shoulder subluxation and pain (Vafadar et al., 2015). The effects of the stimulation was assessed in both “early” (i.e. < 6 months) and “late” (i.e. > 6 months) stroke patients. Seven studies evaluated the effects of NMES on shoulder subluxation, six of which delivered the stimulation in the early phase post-stroke (Vafadar et al., 2015). Analyses revealed that NMES delivered in addition to conventional therapy was more effective than conventional therapy alone at reducing/preventing...
shoulder subluxation. When the stimulation was delivered late however, pooled data from three studies revealed that NMES was not significantly more effective than conventional therapy at reducing/preventing shoulder subluxation. From the 10 RCTs included in the study, nine studies evaluated the effect of NMES on shoulder pain (Vafadar et al., 2015). Pooled analyses demonstrate no significant difference in pain-free range of lateral motion or subjective pain ratings between the group treated early with NMES or conventional therapy. Only two studies evaluated the effect of late NMES on shoulder pain however, one trial did not report on statistical significance, while the second trial suggested no significant effect of NMES on pain compared to conventional therapy (Vafadar et al., 2015).

Interferential electrical stimulation (IES), which is often used in physiotherapy practice to relieve pain by transmitting electrical impulses to the muscle tissues, has only been studied in one RCT in the stroke population. Results reveal effective reduction in hemiplegic shoulder pain during passive range of motion and at rest immediately after stimulation (Suriya-amarit et al., 2014).

High voltage pulsed galvanic stimulation (HVPGS) has previously been used to alleviate pain by applying a high-voltage, low-amperage direct current to the affected regions. Thus far, only one RCT has evaluated the effects of HVPGS in a stroke population to test its efficacy at reducing hemiplegic shoulder subluxation, displacement, and motor function. The results indicate that subluxation was reported in 37.5% of the patients receiving conventional therapy, while no participant receiving HVPGS demonstrated shoulder subluxation (Fil et al., 2011). Furthermore, shoulder joint displacement values were significantly greater in the untreated group compared to the intervention group.

Sensory nerves can also be stimulated by transcutaneous electrical nerve stimulation (TENS), which delivers stimulating pulses through the overlying skin surface. Literature shows that TENS applied over the hemiplegic shoulder was only effective at improving passive range of motion when it was delivered at a high intensity, but not at a low intensity (Leandri et al., 1990). Further research is warranted to determine if TENS is useful for improving motor function or hemiplegic shoulder pain.

**Conclusions Regarding Electrical Stimulation in the Hemiplegic Shoulder**

*There is level 1a and level 2 evidence that surface neuromuscular electrical stimulation (NMES) delivered prior to 6 months post-stroke may be more effective than conventional therapy at preventing/reducing shoulder subluxation but not shoulder pain. Treatment delivered after 6 months may not be more effective than conventional therapy at reducing shoulder subluxation. The evidence for the effect of NMES on hemiplegic shoulder pain after 6 months post-stroke is currently limited.*

*There is level 1a evidence that intramuscular NMES however, may be an effective treatment of hemiplegic shoulder pain that has lasting effects (up to 12 months post-treatment).*

*There is level 1b evidence that interferential electrical stimulation (IES) is beneficial at reducing pain during range of motion and at rest in patients suffering from shoulder hemiplegia.*

*There is limited level 2 evidence that high voltage pulsed galvanic stimulation (HVPGS) is superior to conventional therapy at reducing subluxation and improving shoulder joint displacement.*

*There is limited level 2 evidence that transcutaneous electrical nerve stimulation (TENS) may only improve passive range of motion when delivered at a high intensity.*
Treatment with surface neuromuscular electrical stimulation (NMES) early (< 6 months) post-stroke may reduce shoulder subluxation but not pain associated with shoulder hemiplegia. Surface NMES delivered after 6 months provides no additional benefits over conventional therapy on shoulder subluxation. Intramuscular NMES however, has been found to reduce shoulder pain up to 12 months post-therapy.

High-voltage pulsed galvanic stimulation (HVPGS) may reduce shoulder subluxation but not pain evoked by the hemiplegic shoulder, while interferential electrical stimulation (IES) may reduce pain immediately after treatment.

More research is needed to determine the efficacy of TENS for improving hemiplegic shoulder function and pain.

11.7.6 Surgery as Treatment for Muscle Imbalance

Given that spastic muscle imbalance has been identified as a cause of hemiplegic shoulder pain, treatment designed to reverse that imbalance could potentially relieve hemiplegic shoulder-associated pain. A single pre-post study by Braun et al. (1971) showed that after surgical resection of the subscapularis and the pectoralis muscle tendons, patient regained 90° of passive abduction and 20° of external rotation. Patients eventually reported pain and discomfort after 6 months post-surgery.

Conclusions Regarding Surgery as Treatment for Hemiplegic Shoulder Pain

There is limited level 4 evidence that surgically resecting the subscapularis and pectoralis muscle tendons improves range of motion in stroke patients with a painful hemiplegic shoulder. Further research is needed to confirm these findings.

More research is needed to determine if surgically resecting the subscapularis and the pectoralis muscle tendons improves range of motion in patients with shoulder hemiplegia.

11.7.7 Botulinum Toxin Injections as Treatment for Hemiplegic Shoulder Pain

As discussed previously, subscapularis spasticity is characterized by shoulder range of motion being most limited with pain being reproduced on external rotation. This appears to correlate well with hemiplegic shoulder pain that is now thought to be a consequence of spastic muscle imbalance about the shoulder in many cases. Pectoralis muscle spasticity, characterized by limitation of range and pain on shoulder abduction, is seen to a lesser extent, causing a similar muscle imbalance. Intra-articular injections of botulinum toxin and other agents have been used in an effort to treat spastic muscles, reduce the imbalance and to relieve hemiplegic shoulder pain. See Table 11.7.7.1 for a summary of studies evaluating botulinum toxin for treating hemiplegic shoulder pain.

A Cochrane review (Singh & Fitzgerald, 2010) examined the efficacy of the use of botulinum toxin in the treatment of shoulder pain. Six RCTs were included, five of which included patients with post-stroke shoulder pain. The sixth study included patients with shoulder pain resulting from osteoarthritis and rheumatoid arthritis. Treatment with BT was associated with reductions in pain at 3 and 6 months, but not at 1 month following injection.

Table 11.7.7.1 Summary of RCTs Evaluating Botulinum Toxin
<table>
<thead>
<tr>
<th>Author, Year PEDro Score</th>
<th>Intervention</th>
<th>Main Outcome(s) Result</th>
</tr>
</thead>
</table>
| **Marciniak et al.** (2012) RCT (10) N=21 | E: 100-150 U Botox C: Placebo | • Pain (-)  
• Range of Motion (-)  
• Spasticity (-) |
| **Kong et al.** (2007) RCT (8) N=17 | E: 500 U Dysport C: Placebo | • Pain (-)  
• Modified Ashworth Score (-)  
• Range of Motion (-) |
| **Marco et al.** (2007) RCT (8) N=31 | E: TENS + 500 U Dysport C: TENS + placebo | • Pain (+)  
• Modified Ashworth Score (-)  
• Range of Motion (-) |
| **Yelnik et al.** (2007) RCT (7) N=20 | E: 500 U Botox C: Placebo | • Pain (+)  
• Range of Motion (+/-) |
| **de Boer et al.** (2008) RCT (6) N=22 | E: 100 U 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**

Studies suggest that using a low dosage of 100 to 150 Units of Botox did not improve pain, range of motion or spasticity (De Boer et al., 2008; Marciniak et al., 2012). On the other hand, two of three RCTs found that delivering a dosage of 500 units of Botox with or without TENS may reduce pain scores but not spasticity (Kong et al., 2007; Marco et al., 2007; Yelnik et al., 2007). Overall, Botox was not found to improve range of motion. Many of the studies to date differed with respect to muscles targeted, dose and dilution used and muscle targeting strategy, all of which would impact efficacy. An RCT targeting subscapularis and pectoralis major with either EMG or ultrasound using a dose of 75-100 units per muscle at 4:1 dilution would be helpful in determining if Botox would be helpful for hemiplegic shoulder pain.

**Conclusions Regarding Botulinum Toxin for Shoulder Pain**

*There is level 1a evidence that low doses of Botox (100-150U) may not improve pain, range of motion or spasticity.*

*There is level 1b evidence that 500U of Botox in combination with or without TENS may not reduce shoulder spasticity. The evidence for its use on reducing pain and improving range of motion is currently conflicting.*

*It is uncertain whether botulinum toxin reduces hemiplegic shoulder pain or improves range of motion.*

**11.7.8 Steroid Injections as Treatment for HSP**

The use of a triamcinolone acetonide injection into various shoulder girdle joints has also been examined as a means to relieve shoulder pain in patients with hemiplegia (Table 11.7.8.1).

**Table 11.7.8.1 Summary of RCTs Evaluating Triamcinolone Acetonide**
<table>
<thead>
<tr>
<th>Author, Year</th>
<th>Intervention</th>
<th>Main Outcome(s)</th>
</tr>
</thead>
</table>
| **Rah et al. (2012)**  
RCT (9)  
N=58 | E: Triamcinolone acetonide (40mg)  
C: Lidocaine | - Pain: daytime and nighttime at week 4 and 8 (+)  
- Flexion: at week 2, 4, and 8 (+)  
- External rotation: at week 2 and 4 (+)  
- Internal rotation (+)  
- Shoulder Disability Questionnaire: at week 4 and 8 (+) |
| **Lim et al. (2008)**  
RCT (9)  
N=29 | E1: Triamcinolone acetonide (40mg)  
E2: 100 Botox-A | - Pain (-)  
- Range of Motion (-)  
- Fugl-Meyer (-)  
- Modified Ashworth Scale (-) |
| **Snels et al. (2000)**  
RCT (8)  
N=35 | E: Triamcinolone acetonide (40mg)  
C: Placebo | - Pain (-)  
- Action Research Arm Test (-)  
- Fugl-Meyer (-) |
| **Yasar et al. (2011)**  
RCT (5)  
N=26 | E: Triamcinolone acetonide (40mg)  
C: Suprascapular nerve block | - Pain (-) |
| **Baykal et al. (2013)**  
RCT (4)  
N_{start}=30  
N_{end}=30 | E: Triamcinolone acetonide (40mg) + physical therapy  
C: Physical therapy | - FIM (-)  
- Brunnstrom (-)  
- Modified Ashworth Scale (-)  
- Pain: during rest, activity, at night (-)  
- Rotation: Internal (-); External (-)  
- Flexion (-)  
- Abduction (-) |
| **Lakse et al. (2009)**  
RCT (4)  
N=38 | E: Triamcinolone acetonide  
C: No injection | - Pain: during rest, activity, at night (+)  
- Range of Motion (+)  
- Brunnstrom (-) |

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

**Discussion**

Various studies have investigated the effects of steroid injections for the treatment of hemiplegic shoulder pain. The results of these heterogeneous studies provide mixed evidence, with four studies showing some positive effect of using steroid injections (Lakse et al., 2009; Rah et al., 2012; Baykal et al. 2013; Yasar et al. 2011). When compared to lidocaine, triamcinolone acetonide was found to be superior at reducing pain during the day and at night (Rah et al., 2012) however, not when compared against Botox (Lim et al., 2008), placebo (Snels et al., 2000) or nerve block injections despite a decrease in pain reported in both treatment groups (Yasar et al., 2011). Baykal et la. (2013) also found that pain reported during rest, activity and at night decreased in both patient groups (i.e those treated with triamcinolone and those receiving physical therapy only) but no significant difference between the groups was found after 1 week or 7 weeks of therapy. Conversely, Lakse et al. (2009) found that treatment with triamcinolone reduced pain during rest, activity and at night to a significantly greater extent compared to the control group which received no injection. This effect was found after 1 week and after 4 weeks post-treatment.

Further research is required to investigate the efficacy of steroid use for the treatment of shoulder pain versus alternative treatments as these studies provided limited and conflicting evidence. Furthermore, steroid injections were not found to be superior at improving motor function, spasticity, and disability.
It is encouraged however that the results are to be interpreted with caution since the power of the studies included is fairly low (i.e. N<100) and only 3 RCTs were of good methodological quality (i.e. PEDro≥6).

**Conclusions Regarding Steroid Injections for Shoulder Pain**

*There is conflicting level 1a and level 2 evidence regarding the effect of intra-articular Triamcinolone acetonide injections on hemiplegic shoulder pain.*

More studies of greater methodological quality and higher power are needed to determine the efficacy of Triamcinolone acetonide injections at reducing hemiplegic shoulder pain.

**11.7.9 Aromatherapy/Acupressure Treatment for Shoulder Pain**

Aromatherapy has been shown to help reduce pain and anxiety associated with chronic pain conditions (Buckle, 1999). The use of aromatherapy plus acupressure in the treatment of hemiplegic shoulder pain has been investigated in a single RCT (Table 11.7.9.1).

**Table 11.7.9.1 Summary of RCTs Evaluating Aromatherapy Acupressure**

<table>
<thead>
<tr>
<th>Author, Year PEDro Score</th>
<th>Intervention</th>
<th>Main Outcome(s)</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shin &amp; Lee (2007) RCT (6) N=30</td>
<td>E: Acupressure with essential oils C: Dry acupressure</td>
<td>• Pain (+)</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**

The authors speculated that the improvement in shoulder pain might result from an enhancement of the parasympathetic response through the effects of smell and touch that encourage relaxation (Shin & Lee, 2007). Relaxation has been shown previously to alter the perception of pain. More research is needed to determine the role of aromatherapy and acupressure on relieving hemiplegic shoulder pain.

**Conclusions Regarding Aromatherapy Acupressure**

*There is level 1b evidence that aromatherapy combined with acupressure may reduce pain associated with painful hemiplegic shoulder.*

Further research is needed to determine the benefits of aromatherapy in combination with acupressure regarding its effects on reducing pain caused by shoulder hemiplegia.

**11.7.10 Massage Therapy**

There are numerous theories about how massage therapy may affect the body. For example, the "gate control theory" suggests that massage may provide stimulation that helps to block pain signals sent to the brain. Other theories suggest that massage might stimulate the release of certain chemicals in the body, such as serotonin or endorphins, or cause beneficial mechanical changes in the body.
Table 11.7.10.1 Summary of RCTs Evaluating Massage Therapy

<table>
<thead>
<tr>
<th>Author, Year PEDro Score</th>
<th>Intervention</th>
<th>Main Outcome(s) Result</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Li et al.</strong> (2012) RCT (6) N=120</td>
<td>E: Massage + electric acupuncture C: Rehabilitation Therapy</td>
<td>• Pain (+)  • Fugl Meyer (+)  • Functional hand activity (-)</td>
</tr>
<tr>
<td><strong>Mok &amp; Woo</strong> (2004) RCT (5) N=102</td>
<td>E: Slow-stoke back massage C: No 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
Two RCTs examined the use of massage in improving should pain post stroke (Table 11.7.10.1). Mok and Woo (2004) conducted an RCT evaluating the healing effects of slow-stroke back massage for elderly stroke patients with shoulder pain and anxiety. All participants reported beneficial effects from the massage intervention, which included relief of pain, relaxation and ability to sleep better. Patients receiving massage therapy had significantly lower blood pressure, lower levels of anxiety, lower pain and heart rate scores when compared to the control group. In the second RCT, Li et al. (2012) delivered a treatment combining massage therapy with acupuncture. The study found significantly lower pain among individuals receiving treatment compared to those in the standard treatment group for up to 12 weeks post treatment. Due to the nature of the treatment however, it is unknown whether the benefits were a result of the acupuncture, the massage or the combination. Motor function also improved post treatment; however it was not maintained over the 12 week period. A higher frequency prescription of massage therapy may be required to maintain improved outcomes.

Conclusions Regarding Massage Therapy

There is level 1b and limited level 2 evidence that massage therapy by itself or in combination with acupuncture may reduce hemiplegic shoulder pain. Evidence also suggests improvements in anxiety, heart rate, blood pressure, and general motor function. Despite the positive findings regarding the use of massage therapy, further research is still warranted.

Massage therapy may reduce hemiplegic shoulder pain, anxiety, and other physiological functions (i.e. blood pressure, and heart rate) however, more research is still warranted.

11.7.11 Suprascapular Nerve Block for the Treatment for Shoulder Pain

Thus far, three studies have investigated the effect of suprascapular nerve block on hemiplegic shoulder pain (Table 11.7.11.1).

Table 11.7.11.1 Summary of RCTs Evaluating Subscapular Nerve Treatment

<table>
<thead>
<tr>
<th>Author, Year PEDro Score</th>
<th>Intervention</th>
<th>Main Outcome(s) Result</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Adey-Wakeling et al.</strong> (2013) RCT (9)</td>
<td>E: Suprascapular nerve block C: Saline injection</td>
<td>• Pain (+)</td>
</tr>
</tbody>
</table>
Yasar et al. (2011)  
RCT (5)  
N=26  
E1: Suprascapular nerve block  
E2: Single injection of intra-articular steroid  
• Pain (-)

Boonsong et al. (2009)  
RCT (4)  
N=10  
E1: Suprascapular nerve block + lidocaine  
E2: Ultrasound therapy  
• Pain (+)  
• Range of Motion: Abduction (-); Flexion (-)

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

Discussion
A total of three RCTs have investigated the effect of suprascapular nerve block therapy for the treatment of shoulder pain. In several studies, nerve block injections improved pain scores when compared to the control saline injection (Adey-Wakeling et al., 2013). Similarly, when combined with lidocaine, suprascapular nerve block reduced pain scores but it had no effect on motor function involving abduction or flexion, relative to ultrasound therapy (Boonsong et al., 2009). Lastly, although no difference was found between steroid or nerve block injections regarding their effect on reducing shoulder pain, there was an overall reduction in pain (Yasar et al., 2011). More research is needed to discern the benefits of nerve block therapy on shoulder pain post-stroke.

Conclusions Regarding Suprascapular Nerve Block

There is level 1b and limited level 2 evidence that nerve block injections relative to saline injections or ultrasound therapy, may improve shoulder pain but not range of motion.

There is limited level 2 evidence that nerve block therapy may not be superior over intra-articular steroid injections at reducing shoulder pain.

Although nerve block injections appear to be superior over saline injections or ultrasound therapy, more research is needed.

11.7.12 Segmental Neuromyotherapy for the Treatment for Shoulder Pain
The segmental neuromyotherapy (SNMT) approach aims to diagnose the precise segments involved in the sensitization process. The treatment includes a combination of modalities including therapy, injections with local anaesthetics, the application of heat and electrical stimulation.

Table 11.7.12.1 Summary of RCTs Evaluating Segmental Neuromyotherapy Nerve Block for HSP

<table>
<thead>
<tr>
<th>Author, Year PEDro Score</th>
<th>Intervention</th>
<th>Main Outcome(s) Result</th>
</tr>
</thead>
</table>
| Ratmanský et al. (2012) RCT (7) N=24 | E: Segmental neuromyotherapy + standard therapy  
C: Oral pain medication + standard therapy | • Pain (-)  
• Fugl-Meyer (+)  
• Neer Test (+) |
Discussion
In one recent RCT, Ratmansky et al. (2012) used segmental neuromyotherapy (SNMT) in addition to standard therapy to identify its effects on pain, upper limb motor function and spasticity (Table 11.7.12.1). The results suggest that although patients receiving SNMT demonstrated a significant reduction in perceived pain, the difference between the control group receiving just standard therapy and the SNMT group was not significant. On the other hand, participants treated with SNMT showed greater improvements in motor function at the end of the treatment compared to the control group.

Conclusions Regarding Segmental Neuromyotherapy

There is level 1b evidence that segmental neuromyotherapy may improve hemiplegic upper limb motor function however it may not be more efficient than oral pain medication at reducing hemiplegic shoulder pain.

Further research is needed to determine the effect of segmental neuromyotherapy on hemiplegic shoulder pain.

11.8 Complex Regional Pain Syndrome (CRPS)

11.8.1 Stages and Symptoms of CRPS
CRPS can be of type I or II. Type I is more common and is associated with hemiplegia. It is also referred to as shoulder hand syndrome or reflex sympathetic dystrophy. Type II CRPS is also referred to as causalgia, and it is less common and associated with traumatic injury. CRPS-1 is characterized by numerous peripheral and central nervous system changes in the absence of obvious nerve injury. Peripheral changes include vasomotor tone with associated hand pain and swelling, exquisite tenderness or hyperaesthesia, protective immobility, trophic skin changes and vasomotor instability of the involved upper extremity. Central changes include a disruption of sensory cortical processing, disinhibition of the motor cortex and disrupted body schema (Moseley, 2004; Lorimer Moseley, 2006).

The condition is not unique to patients recovering from stroke, but is also associated with phantom limb pain, and is prevalent among patients with head injury, spinal cord injury and even mild injury to the extremities.

Table 11.8.1.1 Iwata et al. (2002) empirically describe 3 stages of CRPS/ Stages and Characteristics of CRPS

<table>
<thead>
<tr>
<th>Stage</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Persistent pain, described as burning, or aching and aggravated by movement</td>
</tr>
<tr>
<td></td>
<td>The extremity is edematous, warm and hyperesthetic</td>
</tr>
<tr>
<td></td>
<td>Lasting 3-6 months</td>
</tr>
<tr>
<td>2</td>
<td>Early dystrophic changes in the limb present</td>
</tr>
<tr>
<td></td>
<td>Atrophy of the muscle and skin</td>
</tr>
<tr>
<td></td>
<td>Vasospasm with hyperhidrosis</td>
</tr>
<tr>
<td>3</td>
<td>Soft-tissue dystrophy</td>
</tr>
<tr>
<td></td>
<td>Contractures which produce “frozen shoulder”</td>
</tr>
<tr>
<td></td>
<td>Pain and vasomotor changes are infrequent</td>
</tr>
</tbody>
</table>
CRPS generally presents initially with pain in the shoulder followed by a painful, edematous hand and wrist. There is frequently decreased range of motion at the shoulder and hand while the elbow joint is spared (Davis et al., 1977). Passive flexion of the wrist, MCP and PIP 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 often the edema subsides spontaneously after a few weeks.

CRPS is often regarded as a form of sympathetically mediated pain involving the hemiplegic upper extremity. The relationship between the sympathetic nervous system and pain remains hypothetical and has yet to be proven. The incidence of HSP is known to be higher among patients with spasticity (Wanklyn, 1994). CRPS develops in about one in four hemiplegics. It is associated with involvement of the premotor region and spasticity in the involved upper extremity. Typically patients with post stroke CRPS present with pain, hyperalgesia, joint stiffness and swelling and autonomic abnormalities. While recovery is largely spontaneous, conditions that persist for greater than 6 months are often difficult to treat.

11.8.2 Pathophysiology of CRPS

CRPS has been associated with lesions of the pre-motor area of the brain. The etiology of CRPS is unknown; the sympathetic nervous system has often been implicated largely because of the associated vasomotor changes. Theoretical peripheral and central etiologies have been proposed. Peripheral etiological theories postulate a role for trauma to the peripheral nerves. One of these theories postulates ephaptic conduction between efferent sympathetic nerves and afferent somatic nerves with the latter depolarization being perceived as pain. Numerous central etiological theories have also been proposed. For instance, it has been postulated that there is a disruption of autonomic nervous control from higher CNS centres, which 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 shoulder subluxation, may stimulate the internuncial pool of the spinal cord resulting in an abnormal sympathetic response. A link between the abnormal sympathetic nervous system and pain has also been postulated but never proven.

Geurts et al. (2000) systematically reviewed the etiology and treatments of post stroke hand edema and shoulder-hand syndrome. The authors identified 5 etiological studies and 6 therapeutic studies. Based on their systematic review of the literature, the authors concluded that the shoulder was involved in only half the cases although all of the cases were characterized by painful swelling of the wrist and hand, thereby suggesting a “wrist-hand syndrome” in half the cases. Furthermore, they noted that the hand edema was not a lymphoedema and that CRPS usually coincided with increased arterial blood flow.

Iwata et al. (2002) suggested that CRPS might be due to paresis following stroke, mediated by disruption of homeostasis and the balance between intracellular and extracellular fluid. Three possible mechanisms include: i) an increase in capillary blood pressure, caused by a decrease in peripheral venous return and lymph flow; ii) a drop in the colloidal osmotic pressure in the early stages of stroke due to an acute phase response; iii) enhanced permeability of capillary walls which may result from synovial inflammation, brought about by rough management of the affected arm and hand.

Conclusions Regarding CRPS

*The pathophysiology of complex regional pain syndrome is not fully understood. Most cases appear to improve with time.*


The pathophysiology of complex regional pain syndrome is poorly understood.

11.8.3 Incidence of CRPS
There is a wide range of reported prevalence of CRPS following stroke. Part of variability can be attributed to timing and form of assessment used. While the incidence of CRPS appears to range between 12% and 32%, Petchkrua et al. (2000) suggested that the incidence of CRPS is over-estimated and the results from previous studies were obtained before patients routinely received early intensive inpatient rehabilitation. At admission to hospital and once a week until discharge, patients admitted to an acute rehabilitation facility were evaluated for shoulder pain, decreased passive range of motion of the shoulder, wrist/hand pain, edema, and skin changes. If three of these five criteria were positive, patients underwent a triple-phase bone scan (TPBS). Bone scan findings that were consistent with CRPS type 1 pathophysiology were considered a confirmation of the diagnosis. Of 64 subjects, 13 underwent bone scans, with only one (1.56%) positive result. The authors noted it was possible that patients were discharged before they developed symptoms of CRPS. However, the results from several newer studies suggest the incidence is higher. Since both studies originated in the same country, the results may not be generalizable. The authors themselves speculate that the poor economic conditions with limited patient care capabilities likely contributed to the high prevalence (Kocabas et al., 2007).

Conclusions Regarding the Incidence of CRPS Post Stroke

The incidence of complex regional pain syndrome post stroke ranges from 12%-48% and may be influenced by the timing as well as the type of assessment.

The incidence rate of complex regional pain syndrome varies greatly among post-stroke individuals depending on the timing and the type of assessment used.

11.8.4 Diagnostic Tests of CRPS
Several approaches to diagnose CRPS have been used, although no one single test will identify all persons with CRPS. Three sets of criteria are used routinely: International Association for the Study of Pain (IASP) 1994 consensus criteria (Stanton-Hicks et al., 1995), Bruehl’s (1999) criteria and Veldman’s (1993) criteria. The sensitivities and specificities of these sets of criteria range from 70% to 100% and 36% to 94%, respectively. Common features among these criteria include: pain, allodynia, hyperalgesia, edema, sweating changes, and limitations in range of motion. Quisel et al. (2005) have suggested that although diagnosis through instrumentation, bone scans (scintigraphy) and radiography are common, there is limited evidence that these techniques add to the accuracy of the diagnosis.

Routine radiographs of the involved upper extremity may demonstrate a patchy, periarticular demineralization (Sudek’s atrophy) as early as 3-6 months after the onset of clinical signs. The most sensitive diagnostic test is the technetium diphosphonate bone scan which demonstrates increased periarticular uptake (mostly at the shoulder and wrist) in the affected upper extremity. Bone scan abnormalities appear earlier than the x-ray changes. Tepperman et al. (1984) found 25% of hemiplegic patients demonstrated evidence of CRPS in the involved upper extremity although only two-thirds went on to develop the clinical syndrome. 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, however, has improved with image guided injections (e.g. ultrasound guided stellate block). Thermography, in controlled studies, has failed to consistently diagnose reflex sympathetic
dystrophy and is not considered a valid test. However, Kozin et al. (1981) suggested that clinical measurements such as grip strength, tenderness and ring size were more accurate diagnostic indicator of CRPS. Iwata et al. (2002) have suggested that a ratio of the circumference of the middle finger (affected:unaffected) greater than 1.06 at four weeks post stroke was predictive of CRPS.

**Conclusions Regarding Diagnostic Tests of CRPS**

*Several CRPS diagnostic tests exist however, none will identify all patients with CRPS. Most sensitive is the technetium diphosphonate bone scan.*

**No one diagnostic test is ideal for the identification of CRPS. Further research is required.**

11.8.5 Treatment of CRPS

Prevention of shoulder problems and aggressive early treatment are 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 either in the form of stellate ganglion blocks or guanethedine local venous blocks may be tried in persistent disabling cases. Constraint-induced movement therapy and sensory discrimination training have also been proposed as possible treatments for CRPS (Acerra et al., 2007). A surgical sympathectomy may be considered if stellate ganglion sympathetic blocks are consistently effective but symptoms recur. However, there is no evidence that surgical sympathectomy alter outcomes.

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 that many of the commonly-used treatments are effective (Pertoldi & Di Benedetto, 2005; Quisel et al., 2005). CRPS, which presents for greater than 6 months without appropriate treatment, has a poor prognosis (Lieberman, 1986).

Despite a limited number of trials, a review by Geurts et al. (2000) concluded that oral corticosteroids were the most effective treatment for CRPS. While a single controlled trial found that calcitonin treatment effectively treated pain associated with CRPS, it is not widely used clinically.

11.8.6 Pharmacological Treatment of CRPS

Four studies evaluated the use of corticosteroids for the treatment of CRPS associated with stroke. The results are presented in Table 11.8.6.1.

**Table 11.8.6.1 Summary of RCTs Evaluating Corticosteroid Treatment for CRPS**

<table>
<thead>
<tr>
<th>Author, Year PEDro Score</th>
<th>Intervention</th>
<th>Main Outcome(s) Result</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Kalita et al.</strong> (2006) RCT (7) N=60</td>
<td>E: Prednisolne (40mg) C: Piroxicam (20mg)</td>
<td>• Complex Regional Pain Syndrome (CRPS) score (+)</td>
</tr>
<tr>
<td><strong>Rah et al.</strong> (2012) RCT (6) N=58</td>
<td>E: Triamcinolone (40mg) C: Lidocaine</td>
<td>• Pain (+) • Shoulder Disability Questionnaire (+)</td>
</tr>
<tr>
<td><strong>Braus et al.</strong> (1994) RCT (5) N=36</td>
<td>E: Methylprednisolone (8mg) C: Placebo</td>
<td>• Pain (+)</td>
</tr>
</tbody>
</table>
Discussion

The efficacy of corticosteroids for the treatment of CRPS-1 has not been well-studied. Three RCTs were identified, in which two studies used a placebo and one study used a NSAID as the control treatment. Braus et al. (1994) reported that oral corticosteroids improved CRPS-1 symptoms for at least 4 weeks, and Kalita et al. (2006) indicated a significant decrease in symptoms with steroids. Rah et al. (2012) found improvements in both pain and disability measures up to 8 weeks post treatment.

**Conclusions Regarding Oral Corticosteroids in CRPS**

*There is level 1a evidence that oral corticosteroids may improve pain and potentially shoulder disability however, further investigations in the efficiency of corticosteroids for reducing complex regional pain syndrome are need.*

**Oral corticosteroids appear to improve complex regional pain syndrome; however, more research is needed.**

11.8.7 Mirror Imagery as a Treatment for CRPS

Although mirror imagery is more commonly associated with the treatment of phantom limb pain it may also be useful in the treatment of CRPS-1 post stroke. It is believed that mirror imagery helps to create a movement illusion of the affected arm with the brain. These mirror illusions are believed to compensate for a reduced or absent proprioceptive input and to re-establish the normal pain free relationship between sensory feedback and motor intention, resolving the pain state. The patient is seated in such a manner so that the unaffected limb may be viewed in the mirror (or mirror box), while the affected limb is concealed behind the mirror while performing a variety of movements with the upper limb. Table 11.8.7.1 summarize several RCTs that evaluate the efficacy of mirror on CRPS.

**Table 11.8.7.1 Summary of RCTs Evaluating Mirror Therapy for CRPS**

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>PEDro Score</th>
<th>Intervention</th>
<th>Main Outcome(s)</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cacchio et al. (2009)</td>
<td>RCT (7)</td>
<td>N=48</td>
<td>E: Mirror therapy</td>
<td>• Pain (+)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C: Control</td>
<td>• Wolf Motor Function (+)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Motor Activity Log (+)</td>
</tr>
<tr>
<td>Moseley (2006)</td>
<td>RCT (6)</td>
<td>N=51</td>
<td>E: Mirror therapy</td>
<td>• Pain (+)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>C: Standard therapy</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

Moseley (2006) included mirror therapy as one component of a three-part treatment Motor Imagery Program (MIP; hand laterality recognition, hand imaged based on picture, adoption of posture in the picture). The mechanisms underlying motor imagery remain unclear, although it is known that both real and imagined movements activate similar cortical networks. Proposed mechanisms include reconciliation
of motor output and sensory feedback, activation of mirror neurons and graded activation of cortical motor networks (Acerra et al., 2007).

Mirror therapy, when provided as a single intervention was found to be more effective in reducing pain than either sham mirror therapy or mental practice. The effects were also reported to be long lasting. (Cacchio et al., 2009).

**Conclusions Regarding Mirror Therapy Post Stroke**

*There is level 1b evidence that mirror therapy may be superior over placebo treatments at improving upper limb motor function.*

*There is level 1b evidence that mirror therapy may help reduce pain due to hemiplegic shoulder.*

*A mirror therapy program appears to improve shoulder-hand syndrome.*

**11.8.8 Passive Range of Motion Exercises for the Prevention of CRPS**

In addition to treatment of CRPS-1, there are also strategies to prevent its development. Although physiotherapy is regarded as the cornerstone of integrated treatment, no controlled trials have been conducted to evaluate its effect in preventing the development of CRPS.

A single trial using historical controls evaluated the benefit of an exercise program to reduce the development of CRPS following stroke (Kondo et al., 2001). The set protocol developed by the authors was in response to the observation that patients were inappropriately performing excessive passive range of motion exercises, which may have contributed to the development of CRPS. They hypothesized that by limiting inappropriate exercise and replacing it with a set protocol performed by therapists that they could reduce the incidence of CRPS. The results demonstrate that the incidence of RSD was lower (18%) in patients following protocol compared to those not following the protocol (32.4%).

**Conclusions Regarding Physical Therapy to Prevent CRPS**

*There is limited level 2 evidence that passive range of motion exercises may prevent the development of complex regional pain syndrome.*

*There is not enough evidence to understand the effect of motion exercises on complex regional pain syndrome.*

**11.8.9 Calcitonin for the Prevention of CRPS**

Although calcitonin has been used successfully to treat osteoporosis, as well as a variety of pain conditions following surgery and trauma, its use in the treatment of CRPS-1 following stroke has not been examined previously. Its mechanism of action is not well-understood and no site of action has been identified.

A single historically controlled trial was identified examining the benefit of intramuscular injections of calcitonin, in addition to regular rehabilitation (Matayoshi et al., 2009). An incidence rate of 8.2% was found among patients with post-stroke hemiplegia. Of the patients with severe stroke that received calcitonin however, the incidence rate of CRPS was significantly lower (12.5%) compared to the control
group (57.1%). The study suggested that early treatment with calcitonin occurring within the first 4 weeks was capable of preventing CRPS however not when the treatment was started after 6 weeks.

**Conclusions Regarding Calcitonin to Prevent CRPS**

*There is limited level 2 evidence that intramuscular injections of calcitonin may prevent the development of complex regional pain syndrome.*

*Although early treatment (<4wks post-stroke) with calcitonin is found to prevent CRPS, more research is need to understand its effect in the long term and across various stroke severities.*
Summary

1. The reported incidence of post stroke shoulder pain varies from 9% to 73%.

2. Causes and risk factors of hemiplegic shoulder pain include muscle imbalance and poor arm function, pectoralis/subscapularis spasticity, glenohumeral subluxation, bursitis, tendonitis/tear, adhesive capsulitis, and shoulder-hand syndrome.

3. Shoulder subluxation may occur early on in the hemiplegic arm due to flaccid supporting shoulder musculature.

4. Shoulder subluxation may be a cause of shoulder pain; however, patients with shoulder subluxation do not necessarily experience pain and not all cases of hemiplegic shoulder pain suffer from subluxation.

5. The incidence of contractures in hemiplegic shoulder pain range from 54.6% to 76.7%.

6. The development of painful hemiplegic shoulder may be associated with poorer functional outcomes, reduced motor ability, and stroke severity.

7. There is level 1a evidence that shoulder positioning may not reduce pain, motor function, range of motion, or spasticity.

8. There are a wide variety of shoulder slings/treatment options available; however, there is no consensus regarding which is the most efficacious at reducing subluxation.

9. There is level 1a evidence that shoulder strapping/taping may reduce hemiplegic shoulder pain; however, it may not improve range of motion, spasticity, disability, or upper limb motor function.

10. There is level 1b evidence that supplementing range of motion activities with ultrasound or positioning exercises may not be more effective than when performing range of motion exercises alone.

11. There is level 2 evidence that aggressive range of motion therapies, using overhead pullies may result in increased rates of shoulder pain.

12. There is level 1b evidence that continuous passive range of motion exercises are not superior over self-range of motion exercises at improving joint stability, spasticity, or pain.

13. There is limited level 2 evidence that stretching and joint stabilizing therapies are more effective at improving motor arm function compared to normal development therapies.

14. There is limited level 2 evidence that Bobath therapy for the hemiplegic shoulder may be associated with greater pain reduction than passive cryotherapy (application of local cold therapy).

15. There is level 1a and level 2 evidence that surface neuromuscular electrical stimulation (NMES) delivered prior to 6 months post-stroke may be more effective than conventional therapy at preventing/reducing shoulder subluxation but not shoulder pain. Treatment delivered after 6 months may not be more effective than conventional therapy at reducing shoulder subluxation. The evidence for the effect of NMES on hemiplegic shoulder pain after 6 months post-stroke is currently limited.

16. There is level 1a evidence that intramuscular NMES however, may be an effective treatment of hemiplegic shoulder pain that has lasting effects (up to 12 months post-treatment).

17. There is level 1b evidence that interferential electrical stimulation (IES) is beneficial at reducing pain during range of motion and at rest in patients suffering from shoulder hemiplegia.
18. There is limited level 2 evidence that high voltage pulsed galvanic stimulation (HVPGS) is superior to conventional therapy at reducing subluxation and improving shoulder joint displacement.

19. There is limited level 2 evidence that transcutaneous electrical nerve stimulation (TENS) may only improve passive range of motion when delivered at a high intensity.

20. There is limited level 4 evidence that surgically resecting the subscapularis and pectoralis muscle tendons improves range of motion in stroke patients with a painful hemiplegic shoulder. Further research is needed to confirm these findings.

21. There is level 1a evidence that low doses of Botox (100-150U) may not improve pain, range of motion or spasticity.

22. There is level 1b evidence that 500U of Botox in combination with or without TENS may not reduce shoulder spasticity. The evidence for its use on reducing pain and improving range of motion is currently conflicting.

23. There is conflicting level 1a and level 2 evidence regarding the effect of intra-articular Triamcinolone acetonide injections on hemiplegic shoulder pain.

24. There is level 1b evidence that aromatherapy combined with acupressure may reduce pain associated with painful hemiplegic shoulder.

25. There is level 1b and limited level 2 evidence that massage therapy by itself or in combination with acupuncture may reduce hemiplegic shoulder pain. Evidence also suggests improvements in anxiety, heart rate, blood pressure, and general motor function. Despite the positive findings regarding the use of massage therapy, further research is still warranted.

26. There is level 1b and limited level 2 evidence that nerve block injections relative to saline injections or ultrasound therapy, may improve shoulder pain but not range of motion.

27. There is limited level 2 evidence that nerve block therapy may not be superior over intra-articular steroid injections at reducing shoulder pain.

28. There is level 1b evidence that segmental neuromyotherapy may improve hemiplegic upper limb motor function however it may not be more efficient than oral pain medication at reducing hemiplegic shoulder pain.

29. The pathophysiology of complex regional pain syndrome is not fully understood. Most cases appear to improve with time.

30. The incidence of complex regional pain syndrome post stroke ranges from 12%-48% and may be influenced by the timing as well as the type of assessment.

31. Several CRPS diagnostic tests exist however, none will identify all patients with CRPS. Most sensitive is the technetium diphosphonate bone scan.

32. There is level 1a evidence that oral corticosteroids may improve pain and potentially shoulder disability however, further investigations in the efficiency of corticosteroids for reducing complex regional pain syndrome are need.

33. There is level 1b evidence that mirror therapy may be superior over placebo treatments at improving upper limb motor function.

34. There is level 1b evidence that mirror therapy may help reduce pain due to hemiplegic shoulder.

35. There is limited level 2 evidence that passive range of motion exercises may prevent the development of complex regional pain syndrome.
36. There is limited level 2 evidence that intramuscular injections of calcitonin may prevent the development of complex regional pain syndrome.
References


Appendix

**Acute Stroke**

1. There is level 1a evidence that static positioning of hemiplegic shoulder does not improve motor function, range of motion, pain, spasticity, or disability when compared to conventional rehabilitation.

2. There is level 1a evidence that strapping/taping of hemiplegic shoulder reduces pain when compared to conventional rehabilitation, but does not improve motor function, range of motion, spasticity, or disability when compared to conventional rehabilitation.

3. There is level 1b evidence that continuous passive range of motion exercises for hemiplegic shoulder do not reduce pain, spasticity, or joint instability when compared to self-range of motion exercises.

4. There is limited level 2 evidence that aggressive range of motion exercises for hemiplegic shoulder result in increased pain, but not increased subluxation, when compared to conventional exercises.

5. There is level 1a and level 2 evidence that surface neuromuscular electrical stimulation (NMES) for hemiplegic shoulder reduces subluxation when compared to conventional therapy; however there is conflicting evidence as to whether it reduces pain and improves motor function.

6. There is limited level 2 evidence that high voltage pulsed galvanic stimulation (HVPGS) for hemiplegic shoulder reduces subluxation and dislocation when compared to conventional therapy.

7. There is limited level 2 evidence that triamcinolone acetonide injection reduces pain and improves range of motion in hemiplegic shoulder.

8. There is level 1b evidence that acupressure combined with aromatherapy reduces pain in hemiplegic shoulder when compared to acupressure alone.

9. There is level 1b and level 2 evidence that corticosteroids reduce pain in CRPS when compared to NSAIDs or placebo.

**Subacute Stroke**

1. There is level 1b and level 2 evidence that static positioning of hemiplegic shoulder does not improve motor function, range of motion, pain, or spasticity when compared to conventional rehabilitation.

2. There is level 1b evidence that range of motion exercises for hemiplegic shoulder supplemented with ultrasound or positioning do not improve range of motion when compared to the exercises alone.

3. There is limited level 2 evidence that joint stabilizing exercises for hemiplegic shoulder improve range of motion when compared to conventional exercises.

4. There is level 1b evidence that interferential electrical stimulation (IES) for hemiplegic shoulder reduces pain at rest and during range of motion.

5. There is limited level 2 evidence that transcutaneous electrical nerve stimulation (TENS) for hemiplegic shoulder improves passive range of motion when delivered at a high intensity.

6. There is level 1b evidence that suprascapular nerve block reduces pain in hemiplegic shoulder when compared to saline injection.

7. There is level 1b evidence that segmental neuromyotherapy improves motor function in hemiplegic shoulder, but does not reduce pain, when compared to oral pain medication.

8. There is level 1b evidence that mirror therapy reduces pain and improves motor function in CRPS when compared to control.
Chronic Stroke
1. There is limited level 2 evidence that Bobath therapy for hemiplegic shoulder reduces pain when compared to cryotherapy.

2. There is limited level 2 evidence that surface NMES improves range of motion and reduces shoulder subluxation and spasticity in hemiplegic shoulder when compared to conventional therapy.

3. There is level 1a evidence that intramuscular NMES for hemiplegic shoulder reduces pain for up to 12 months when compared to conventional therapy.

4. There is level 1a evidence that lower doses of Botox (100-150U) do not reduce pain or spasticity nor improve range of motion in hemiplegic shoulder when compared to placebo.

5. There is level 1a evidence that higher doses of Botox (500U) do not reduce spasticity in hemiplegic shoulder when compared to placebo; however there is conflicting evidence as to whether it reduces pain or improves range of motion.

6. There is conflicting level 1a and level 2 evidence as to whether triamcinolone acetonide injection improves motor function, range of motion, or recovery and reduces pain, spasticity, or disability.

7. There is limited level 2 evidence that suprascapular nerve block does not reduce pain in hemiplegic shoulder when compared to triamcinolone acetonide.

8. There is level 1b evidence that corticosteroids reduce pain and disability in CRPS when compared to placebo.

9. There is level 1b evidence that mirror therapy reduces pain in CRPS when compared to standard care.