Published in final edited form as:

Int J Ther Rehabil. 2011 April 6; 18(4): 210–220.

Scapula and humeral movement patterns and their relationship with pain: A preliminary investigation

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Abstract

Purpose—Altered movement patterns may contribute to this shoulder pain following stroke. The purpose of this study was to examine scapular and humeral movement patterns in people with hemiparetic shoulder pain (HSP), and to explore the relationships between pain and these movement patterns.

Methods—Kinematic data from nine participants with HSP and twelve matched controls were collected as they performed scapular plane shoulder elevation. Correlation analyses were used to examine relationships between participants' ratings of pain and kinematic data.

Results—The HSP group had decreased humeral external rotation compared to the control group. Despite a trend toward decreased scapular upward rotation, there was considerable variation in scapular upward rotation movement patterns across the participants with HSP. Scapular tilt was not different between groups. There was an inverse relationship between pain and scapular upward rotation, indicating that those with more pain had less scapular upward rotation.

Conclusions—People with HSP demonstrated scapular and humeral movement patterns that differed from controls, where some of the altered movement patterns were related to reported pain levels. Rehabilitation management of people with HSP may be improved by careful assessment of scapulohumeral movement patterns and treatments aimed at normalizing these patterns.

Keywords

stroke; hemiparesis; pain; shoulder; biomechanics; rehabilitation; scapula; humerus; kinematics; movement

Introduction

Shoulder pain following stroke is a prevalent condition that affects up to 72% of persons with hemiparesis (Van Ouwenaller et al., 1986; Joynt, 1992; Wanklyn et al., 1996). Hemiparetic shoulder pain (HSP) has been associated with increased length of stay in the hospital, increased likelihood of requiring long-term hospital care, and inability to move from a bed to chair (Roy et al., 1994; Roy et al., 1995; Wanklyn et al., 1996). Much of the early research and prevailing thought focused on shoulder subluxation being the cause of the pain. There is still much controversy however, as to whether HSP is related to shoulder

subluxation (Roy et al., 1994; Wanklyn et al., 1996; Price et al., 2001). Others have found relationships between HSP and several factors, including loss of passive range of motion, reduced shoulder shrugging and pain during movement (Bohannon et al., 1986; Joynt, 1992; Wanklyn et al., 1996). Research has also found that HSP is associated with subacromial impingement and rotator cuff pathology (Bohannon et al., 1986; Joynt, 1992; Shah et al., 2008).

Normal shoulder elevation involves a coupling between the scapula and the humerus, termed the scapulohumeral rhythm (Poppen, 1976; Levangie and Norkin, 2005). Despite clinical perceptions of a fixed 2:1 ratio of humeral to scapular movement and minimal scapular movement in the first 30° of humeral elevation, recent literature utilizing bone pins has suggested that during the first 30° of humeral elevation, the humeral to scapular movement is linear with a steeper slope than in the later portions of the movement (McClure et al., 2001; Braman et al., 2009). As the humerus is elevated, the scapula must upwardly rotate and posteriorly tilt (Poppen, 1976; McClure et al., 2001; Levangie and Norkin, 2005; Braman et al., 2009). This serves to maintain congruency of the glenohumeral joint to prevent impingement upon the tendons of the rotator cuff between the humerus and the acromion (Poppen, 1976; Levangie and Norkin, 2005). For proper upward rotation and posterior tilt of the scapula to occur during humeral elevation, there must be timely activation of the serratus anterior, and trapezius muscles, hereafter referred to as the scapulothoracic muscles (Kamkar et al., 1993; Decker et al., 1999; Levangie and Norkin, 2005). Proper scapular motion maintains the length-tension relationship of the rotator cuff muscles, allowing them to externally rotate the humerus as the arm is elevated and keeping the humerus centered in the glenoid (Poppen, 1976; Levangie and Norkin, 2005).

Following a stroke, damage to the corticospinal system decreases the neural input to the muscles contralateral to the lesion (Twitchell, 1951). Decreased neural input may disrupt the scapulohumeral rhythm in the affected arm. Decreased activation of the scapulothoracic muscles could result in less upward rotation and less posterior tipping of the scapula, which could be even more pronounced with spasticity of the scapular downward rotators. Alternatively, the scapula may compensate for a loss of humeral motion post stroke by increasing scapular upward rotation. Either abnormal movement pattern may lead to decreased congruency of the glenohumeral joint and could impair the length-tension relationship of the rotator cuff muscles. Furthermore, the rotator cuff muscles could be directly affected by the decreased neural input. Without adequate neural input to the rotator cuff muscles, the humerus may translate superiorly and anteriorly to contact the glenoid fossa during humeral elevation (Levangie and Norkin, 2005). Thus, based on data from orthopedic populations, abnormal scapular and humeral movement patterns may lead to impingement of the rotator cuff between the acromion and humerus, resulting in the clinical experience of HSP (Poppen and Walker, 1976; Ludewig and Cook, 2000).

The etiology and contributing factors of shoulder pain following are multifactorial and not fully understood (Hakuno et al., 1984; Roy et al., 1995; Wanklyn et al., 1996; Lo et al., 2003; Aras et al., 2004; Niessen et al., 2009). These factors include shoulder subluxation, reflex sympathetic dystrophy, and adhesive capsulitis. It is likely that for a given person, more than 1 factor may be present. Irrespective of diagnosis and contributing factors, the resultant disruptions in movement patterns can lead to strain and tearing of rotator cuff muscles as well as impingement of the rotator cuff tendons (Hakuno et al., 1984; Roy et al., 1995; Wanklyn et al., 1996; Lo et al., 2003; Aras et al., 2004; Niessen et al., 2009).

Movement patterns of the scapula and humerus in people with HSP have been investigated in four studies to date. Several studies of the kinematics of humeral elevation have found that scapular upward rotation is increased in participants with HSP (Price et al., 1999;

Niessen et al., 2008; Niessen et al., 2009). A fourth study, however, found that scapular upward rotation was variable in this population (Price et al., 2001). Scapular tilt does not appear to be abnormal in people with HSP compared to controls (Niessen et al., 2008). Humeral external rotation has been examined passively (Andrew and Bohannon, 1989; Lo et al., 2003; Rajaratnam et al., 2007; Niessen et al., 2008), but has not been examined dynamically during active movement in people with HSP. Because reports of pain are greater with movement than at rest (Roy et al., 1994; Roy et al., 1995; Wanklyn et al., 1996), it is critical to examine humeral external rotation dynamically during shoulder elevation for a complete picture of movement patterns in people with HSP.

The first purpose of this study was to examine the scapular and humeral movement patterns in people with hemiparetic shoulder pain, particularly humeral external rotation. The second purpose of this study was to explore relationship between reports of pain and abnormal scapular and humeral movement. We studied participants with HSP and age-matched neurologically intact control participants kinematically as they elevated their affected, contralesional arm. We hypothesized that participants with HSP would have abnormal scapular and humeral movement patterns compared to controls. Specifically we expected to see increased scapular upward rotation (as been found previously) and decreased humeral external rotation. We also anticipated that severity of pain would be related to the extent of the abnormal movement patterns.

Methods

Participants

A convenience sample of nine participants with HSP, 5 male and 4 female was recruited from a local rehabilitation hospital (see Table 1). Participants with HSP were included if they: 1) had a diagnosis of stroke, 2) onset of shoulder pain following their stroke, and 3) were able to move the affected limb against gravity. Participants were excluded if they: 1) had a history of shoulder pain and pathology prior to stroke, 2) they were unable to follow 2-step commands, 3) showed signs of hemineglect, 4) showed symptoms consistent with referral from cervical or thoracic spine, 5) had any serious medical complications that would prevent them from participating, and 6) were unable to sign informed consent.

Twelve healthy participants, 7 male and 5 female, were recruited from the community (see Table 1). The age and gender composition of the control group was selected to match the age and gender composition of the HSP group. Control participants were excluded if they 1) had a history of a diagnosis of stroke, 2) history or current complaints of shoulder pain or history of diagnosed shoulder pathology, 3) if they had any serious medical conditions that would prevent them from participating, and 4) if they failed to provide informed consent. The study was approved by the Human Research Protection office of Washington University prior to recruitment and testing. All participants signed informed consent prior to participating.

Kinematic Measurement

Computer-based kinematic techniques were used to quantify movement of the shoulder, arm and thorax (Wu et al., 2005). Three-dimensional movements of the involved upper extremity were captured using an electromagnetic tracking system (The Motion Monitor, Innovative Sports Training Inc, Chicago IL). Four sensors (Figure 1) were attached to the 1) trunk: mid-sternum, 2) the upper arm: proximal to the lateral epicondyle, bisecting the upper arm mass, 3) the forearm: proximal to the mid-point between the radial and ulnar styloids on the dorsum of the forearm, and 4) the scapula: distal flat aspect of the acromion (Ludewig and Cook, 2000). This noninvasive technique has shown excellent consistency with invasive

technique using fixed bone pin markers (Braman et al., 2009b). All sensors and trailing wires were taped down and secured with Coban (3M, St. Paul MN) to prevent slippage and arbitrary sensor movement. Bony landmarks, proximal and distal landmarks for each segment as well as points on and above the plane, were digitized on the thorax, scapula, and humerus at rest with the arms relaxed at the side. This permitted transformation of sensor data into local segment coordinates using the accepted order of internal/external rotation, upward/downward rotation, and posterior/anterior tilting, according to the protocol recommended by the International Society of Biomechanics, Shoulder Group (Wu et al., 2005). The sternum marker was used as the reference marker for the scapula and for the humerus. The hardware manufacturer reports a root mean square accuracy of 0.5° for orientation and 1.8 mm for position for the sensors used. Error in scapular measurements with this system have been reported by others to be 2-3 mm with good repeatability within testing sessions (Meskers et al., 1999; Shimomura et al., 2000; Thigpen et al., 2005).

The starting position for each movement was seated in wooden chair with a back support with the upper extremity hanging down at the side. Care was taken to ensure that the tested upper extremity and scapula did not contact or were otherwise obstructed by the chair. Participants were asked to perform 3 trials of scapular plane shoulder elevation, which was defined as a plane 30° anterior to the frontal plane. Scapular plane shoulder elevation was chosen because it is often used in literature examining shoulder motion in healthy controls and patient populations (Ludewig et al., 1996; Lukasiewicz et al., 1999; Ludewig and Cook, 2000; McClure et al., 2001; McClure et al., 2006). Participants were asked to move at a self-selected speed. Participants were given the option to take rest breaks as needed. The examiner made sure that the shoulder elevation movement was in the scapular plane, and participants were given a target, the tester's hand, to aid in staying in the scapular plane. We note that this target did not provide any support or assistance to subject during movement. Participants were instructed to keep the elbow straight, and the movement was corrected and repeated if attempted with a bent elbow. All participants were able to perform the scapular plane shoulder elevation movement as instructed, although some required several practice trials before movements were recorded. The electromagnetic tracking system with the Motion Monitor software used permitted accurate measurement of scapular and humeral movements irrespective of how the participant moved, i.e. scapular rotation was captured whether it occurred during humeral elevation or during a shoulder shrug.

Kinematic data were low-pass filtered at 6 Hz using a second-order Butterworth filter. Motion Monitor (The Motion Monitor, Innovative Sports Training Inc, Chicago IL) software was used to calculate and extract segmental position and angle data from the sensor data using standard rigid body methodology (Wu et al., 2005). The angular data extracted were humeral elevation, scapular upward rotation, scapular tilt, and humeral external rotation. Scapular internal/external rotation data were also extracted but are not included in this report due to lack of consensus on what constitutes normal scapular internal and external rotation during humeral elevation with some studies reporting scapular external rotation as the arm is elevated (Ludewig et al., 1996; Lukasiewicz et al., 1999; McClure et al., 2001) and some studies demonstrating scapular internal rotation as the arm is elevated (Borstad and Ludewig, 2002; Braman et al., 2009a; Ludewig and Reynolds, 2009). Anatomical variations in the shape and size of the thorax and ribs could also impact the relative internal and external rotation of the scapula as it slides along the thorax. Scapular upward rotation was rotation of the scapula in frontal plane about an anterior-posterior axis in which the inferior angle moves laterally. Scapular tilt was rotation of the scapula in the sagittal plane about a lateral axis in which the superior border of the scapula moves posteriorly. Humeral external rotation is defined as spinning of the humerus on the glenoid laterally. All angular data were calculated according to the protocol recommended by the International Society of Biomechanics, Shoulder Group (Wu et al., 2005). For ease of

communication, increasing humeral external rotation was represented as positive on the y-axis. Custom-written software in MATLAB (The Mathworks, Natick MA) was then used for subsequent analysis to find humeral elevation, scapular upward rotation, scapular tilt, and humeral external rotation at the start of movement and at, 30°, 60°, 90°, and 120° of humeral elevation.

Clinical Measures

Shoulder pain at rest and during movement trials was recorded using a numeric pain rating scale ranging from 0 to 10, with 0 representing no pain and 10 representing the most severe pain imaginable. Participants were asked to rate their pain on this scale prior to testing and after each trial. They were specifically asked to only report pain in the immediate shoulder and upper arm area. The scale has been shown to be reliable and sensitive for use in older populations (Von Korf et al., 2000). It has good reliability in participants with orthopedic shoulder conditions (Mintken et al., 2009) as well as participants with HSP (Rajaratnam et al., 2007). This scale served as our primary pain measure in subsequent analyses correlating kinematics and pain.

Other clinical measures were used to provide descriptive information about the participants. Severity of shoulder pain during daily life was measured using the Shoulder Pain and Disability Index pain subscale (Williams et al., 1995; Cloke et al., 2005; MacDermid et al., 2006; Ekeberg et al., 2008). This is a self report measure of pain ratings during normal daily tasks such as reaching to a shelf, worst pain in the previous week, or touching their head, with higher ratings indicating more pain. Only data from the Pain subscale was reported since the Disability subscale would be influenced by the direct disability from their hemiparesis. The Neer's impingement test was used as a clinical indicator of involvement of the rotator cuff tendons (Neer, 1983; Calis et al., 2000; MacDonald et al., 2000; Johansson and Ivarson, 2009). This test has been shown to be reliable (Dromerick et al., 2006) as well as sensitive (Rajaratnam et al., 2007) in persons with stroke. Palpation to the long head of the biceps and the supraspinatus tendon was used as clinical measures of impingement and the involvement of either or both tendons (Hoppenfeld and Hutton, 1976; Dromerick et al., 2006). The Stroke Impact Scale, Hand Function subscale was used to capture the upper extremity functional deficits in the sample (Duncan et al., 2003). Scores on this scale range from 0 to 100 with 100 indicating normal. This quick measure agrees well with the more time-consuming Fugl-Meyer Upper Extremity Motor subscale (Duncan et al., 1999). Lastly, spasticity in the elbow and shoulder was assessed using the Modified Ashworth Scale, and while painful guarding could impact the score, a trained tester is able to distinguish between active resistance due to guarding and velocity-dependent resistance to passive movement (Bohannon and Smith, 1987).

Data analysis

A statistical package (Statistica v6.0, StatSoft, Tulsa OK) was used and the criterion for statistical significance was set at $p < 0.05$. T-tests were used to test for significant differences in scapular upward rotation, scapular tilt, and humeral external rotation between the HSP and control groups at start of movement ($n=9$ in the HSP group) and 30° ($n = 6$ in the HSP group) of humeral elevation. Descriptive statistics were calculated for scapular upward rotation, scapular tilt, and humeral external rotation at 60°, 90°, and 120° of humeral elevation; no statistical tests were done on these because of the small number of subjects achieving these humeral elevation angles ($n = 5$ in the HSP group). Data from participants with HSP were compared to data from an age-matched control group's right extremity and not to the participant with HSP's less involved side. The less involved extremity in participants with HSP may not be a good control, because it has been shown to have kinematic alterations (Meskers et al., 2005).

A paired samples T-test was used to compare pain at rest (prior to performing any movement) and pain during movement, quantified by the average of numeric pain rating given during the 3 trials. Spearman Rho correlations were used to assess the relationship between pain during movement and kinematic variables. Our small sample size limited the likelihood of obtaining statistically significant correlation coefficients. We therefore used the following criteria to interpret the magnitude of the coefficients. Coefficients of 0.25 or below were considered low, coefficients ranging from 0.26 – 0.50 were considered fair, coefficients ranging from 0.51 – 0.75 were considered good, and those greater than 0.75 were considered strong (Portney and Watkins, 1993).

Because previous studies had reported contrasting results with respect to scapular upward rotation, and because the time since stroke varied in these studies compared to ours (Price et al., 1999; Niessen et al., 2008), we also divided the HSP sample into two subgroups: (1) 0-3 months and (2) greater than 3 months post-stroke. For each subgroup, we generated descriptive information in the form of the grand means of scapular upward rotation angles across all humeral elevation angles.

Results

Sample characteristics of the 9 participants with HSP and 12 controls are provided in Table 1. Participants with HSP and controls were closely matched in mean age and gender. All participants had at least 120° passive shoulder elevation. The number of participants who could actively achieve each shoulder elevation angle was n=9 at 0°, n=6 at 30°, n=5 at 60°, n=4 at 90°, and n=2 at 120°. Time since stroke for participants with HSP was variable, ranging from 1 month to 2 years. Pain on movement, with an average rating of 4.8/10, was significantly increased compared to resting pain, with an average of 2.2/10 ($p = 0.02$). Shoulder pain disrupted daily life as indicated by moderate ratings (mean of 44.9) on the Pain subscale of the Shoulder Pain and Disability Index. Six of nine participants had positive results for both the Neer's test and palpation to the biceps and supraspinatus tendons, with 2 participants refusing for fear of pain. This is consistent with shoulder impingement. As expected, upper extremity function was decreased, as indicated by a means score of 34 on the Hand Function subscale of the Stroke Impact Scale. Overall, participants with HSP had modest increases in spasticity at the elbow and shoulder, as measured by the Modified Ashworth Scale.

Kinematic Data

The main finding of this study is that participants with HSP had decreased humeral external rotation (Figure 2A) during elevation of the humerus in the scapular plane. External rotation was significantly decreased in the HSP group compared to the control group at 0° ($p = 0.003$) and at 30° ($p = 0.003$) of humeral elevation. Means and standard deviations for external rotation angles at 60°, 90°, and 120° of humeral elevation were $49 \pm 14^\circ$, $52 \pm 14^\circ$, $53 \pm 15^\circ$ for the control group and $36 \pm 13^\circ$, $36 \pm 18^\circ$, $27 \pm 5^\circ$ for the HSP group. All participants had full passive range of motion for humeral external rotation.

Scapular upward rotation may be problematic in some people with HSP (Figure 2B and Figure 3). Scapular upward rotation in the HSP group was not significantly different from the control group at 0° of humeral elevation ($p = 0.162$) but was different at 30° ($p = 0.009$). Means and standard deviations for scapular upward rotation angles at 60°, 90°, and 120° of humeral elevation were $26 \pm 6^\circ$, $34 \pm 7^\circ$, $41 \pm 9^\circ$ for the control group and $19 \pm 12^\circ$, $29 \pm 14^\circ$, $39 \pm 11^\circ$ for the HSP group. When looking at individual participants however (Figure 3), a variety of patterns of scapular upward rotation were evident. Figures 3A and 3B show examples of scapular upward rotation in control participants. The control participant in 3B had more scapular upward rotation (steeper slope) than the control participant in 3A,

showing the variability present in the neurologically-intact population. Participants with HSP were much more variable (Figure 3C-E). S3 (3C) started in marked downward rotation and upwardly rotated very little. Conversely, S6 (3D) started in 10° of upward rotation and showed a relatively linear increase in upward rotation with increasing humeral elevation. S5 (3E) showed a very steep slope over the small humeral elevation excursion. Finally, S7 (3F) showed a lag between the start of humeral elevation and when the scapula begins to upwardly rotate, as indicated by the flat slope until approximately 20° of humeral elevation. Figure 3 demonstrates that there are a variety of ways in which the scapula upwardly rotates in people with HSP. These variations are hidden when data are collapsed across participants. In the exploratory subgrouping where participants were divided into those who were more acute versus more chronic, scapular upward rotation across all humeral elevation angles averaged 14.7° in the more acute subgroup and 2.4° in the more chronic subgroup.

Scapular tilt was not different between the participants with HSP and controls (Figure 2C). No differences in scapular tilt were found at 0° ($p = 0.74$) or 30° ($p = 0.26$) of humeral elevation. Means and standard deviations for scapular tilt angles at 60°, 90°, and 120° of humeral elevation were $-10 \pm 9^\circ$, $-10 \pm 11^\circ$, $-10 \pm 14^\circ$ for the control group and $-13 \pm 5^\circ$, $-12 \pm 7^\circ$, $-16 \pm 3^\circ$ for the HSP group.

Relationships between Pain and Kinematics

There were minimal relationships using Spearman Rho correlations between pain at rest ($r = 0.12$) or pain on movement ($r = 0.20$) and how far HSP participants could elevate their arm (maximal humeral elevation). Pain on movement had minimal relationships with humeral external rotation at 30° and 60° of humeral elevation ($r = -0.15$ and -0.21 , respectively). Pain on movement had a strong, negative relationship with scapular upward rotation at 30° of humeral elevation ($r = -0.84$) and good relationships at 60° ($r = -0.72$). Pain on movement was minimally related to scapular tilt at the humeral elevation angles tested (r values -0.06 – 0.35).

Discussion

When elevating their arm in the scapular plane, participants with HSP had decreased humeral external rotation compared to controls. Scapular upward rotation may be reduced at some humeral elevation angles in some people with HSP, however there were a variety of scapular upward rotation movement patterns seen in the HSP group. No difference between groups was found for scapular tilt.

The main finding of this study was that humeral external rotation was decreased in participants with HSP compared to controls. The results of this study build upon earlier findings associating reduced passive humeral external rotation and HSP (Andrew and Bohannon, 1989; Lo et al., 2003; Rajaratnam et al., 2007; Niessen et al., 2008). Our data add important information to the current understanding of HSP by showing that dynamically, during elevation of the humerus, a pattern of reduced humeral external rotation is present. Biomechanical conditions that decrease humeral external rotation increase rotator cuff compression particularly against the greater tuberosity; the compression increases as the humerus is elevated (An et al., 1991; Flatow et al., 1994; Brossmann et al., 1996). The compression can be eliminated with external rotation of the humerus (Brossmann et al., 1996). Clinically, data from the present study point to the need to assess loss of dynamic humeral external rotation in people with HSP. This is especially important when no passive loss of external rotation is noted. Furthermore, it highlights the need for treating clinicians to give appropriate treatment to restore proper humeral external rotation dynamically during arm elevation tasks.

Decreased scapular upward rotation was found at 30° of humeral elevation for participants with HSP versus controls. This finding is in contrast to other reports of increased scapular upward rotation in people with HSP (Price et al., 1999; Niessen et al., 2008; Niessen et al., 2009). Differences in samples may be at least partially responsible for the inconsistency between our data and the aforementioned studies. Niessen et al.'s sample averaged approximately 3 months post-stroke (Niessen et al., 2008), while our sample averaged approximately 7 months post-stroke. When examining the present data individually, substantial variation existed in scapular upward rotation movement patterns. Some of the HSP participants had decreased scapular upward rotation while others had increased scapular upward rotation. The variability we found between individual HSP participants here is consistent with the variability that other researchers have found, albeit with a different protocol and different measurement device (Price et al., 2001). Our additional exploratory subgrouping leaves open the possibility that participants who had more recently had a stroke may have increased scapular upward rotation, while participants who were later post-stroke may have had decreased scapular upward rotation. Longitudinal studies that follow participants with HSP at multiple time points are needed to investigate the time-dependent nature of scapular upward rotation.

Pain on movement was associated with decreased scapular upward rotation at 30° and 60° of humeral elevation. We found, however, only minimal correlations between pain on movement and humeral external rotation, scapular tilt, or maximal humeral elevation. Caution must be taken when interpreting the results of our correlation analysis. First, we have limited data points, particularly at the higher angles of humeral elevation. And second, these are only associations and cannot be used to imply causation.

Together, the present findings and the findings of others highlight the need for individual and frequent assessments of scapular movement patterns, and awareness that treatment interventions may need to be altered with time.

Study Limitations

Three main limitations should be taken into account when interpreting the results of this study. First, the sample size in this preliminary study was small with many participants not able to actively achieve the higher shoulder elevation angles. This limits the ability to generalize the findings. Additional studies with larger sample sizes are needed. Second, measurement error is always a factor when using surface markers for kinematic measurement. This measurement error occurs above > 110° of humeral elevation (Meskers et al., 2007), which most of our HSP participants did not achieve. Third, we studied only one movement: scapular plane shoulder elevation. While this is a commonly studied movement, it is only one of many movements that people make in daily life. Future investigations will be more informative if they study a variety of daily shoulder movements. Fourth, we only compared to a control sample of age and gender matched controls, and did not include a control sample of people with stroke without shoulder pain. Future studies that include a control group of people with stroke without pain would help to differentiate movement patterns in people with and without pain following stroke. Finally, we can only speculate about the causation of shoulder pain following stroke based on these data. Other factors not examined in this study could also be contributing to their perception of pain and their movement patterns. These include, but are not limited to: adhesive capsulitis (Turner-Stokes and Jackson, 2002), decreased proprioception (Niessen et al., 2009), and tension headaches affecting head and neck (Tentschert et al., 2005).

Conclusions

The main finding is that humeral external rotation was decreased in participants with HSP, as they dynamically elevated their arm in the scapular plane. The data also showed variable movement patterns of scapular upward rotation, which may change over the course of recovery. Third, we found associations between pain severity and decreased scapular upward rotation. Taken together, these data highlight the need for clinicians treating people with HSP to carefully and frequently monitor movement patterns and to provide treatment that encourages dynamic humeral external rotation and proper scapular upward rotation as they elevate their arm. Future studies with larger sample sizes that follow participants with HSP longitudinally and across a variety of movements are needed.

Acknowledgments

We would like to thank J. Beebe and S. DeJong for their assistance with data collection. This work was supported by an American Heart Association Predoctoral Fellowship Award, Foundation for Physical Therapy PODS I, NIH HD047669, and NIH HD007434.

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Key Findings

- Shoulder pain after stroke may result from abnormal movement.
- Dynamic external rotation of the humerus is decreased.
- Dynamic scapular upward rotation is variable.
- Reports of pain are greater in those with less scapular upward rotation.
- Frequent examination of dynamic scapular and humeral movement patterns are warranted.

Figure 1.
Sensor placement for testing. Note that although not shown in picture, sensors and trailing wires were secured with tape and Coban to prevent slippage and arbitrary sensor movement.

Figure 2. Group data for A: humeral external rotation; B: scapular upward rotation; C: scapular tilt. Values are means $\pm$ SE. (0° n = 9, 30° n = 6, 60° n = 5, 90° n = 4, 120° n = 2 for the HSP group)

Figure 3.

Examples of individual trials from selected controls and patients for humeral elevation versus scapular upward rotation. A & B: Control participants depicting observed variation in a healthy sample. C: HSP participant demonstrating a downwardly rotated start position and very little upward rotation. D: HSP participant demonstrating an upwardly rotated start position. E: HSP participant demonstrating a steep and abbreviated slope. F: HSP participant demonstrating a delayed upward rotation.

Table 1

Participant Characteristics.

	Hemiparetic Participants N=9	Control Participants N=12
Age (y)	56 ± 8 (49 - 65)	54 ± 9 (40 - 72)
Gender	5 male, 4 female	7 male, 5 female
Time since stroke (months)	7.2 ± 6.7 (1 - 24)	
Pain at rest	2.2 ± 2.3 (0 - 6)	
Pain on movement	4.8 ± 2.5 (2 - 6.2)	
SPADI Pain subscale	45 ± 19 (10 - 70)	
Neer's Impingement test	6 positive 1 negative 2 not tested due to fear of pain	
Tendon Palpation	6 positive 2 negative 1 not tested due to fear of pain	
Stroke Impact Scale Hand Function Subscale *	34 ± 27 (0-90)	
Modified Ashworth Scale †	Elbow: 0= 5 subjects 1= 1 subject 1+ = 1 subject 2= 1 subject 4 = 1 subject Shoulder: 0= 6 subjects 1= 1 subject 1+ = 1 subject 4 = 1 subject	

Values are means ± SD (range) or number, as appropriate.

SPADI: Shoulder Pain and Disability Index, higher scores indicate more pain.

* Self reported measure of upper extremity, scores range from 0 to 100, with 100 representing full hand function.

† Measure of spasticity using modified scale with grades: 0, 1, 1+, 2, 3, and 4. Higher scores indicate greater tone.