Shoulder Pain and Subluxation After Stroke: Correlation or Coincidence?

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Key Words: cerebrovascular disorders • hemiplegia • shoulder dislocation

Objective. Few studies have concomitantly examined shoulder subluxation and other potential causes of shoulder pain in persons who have had a stroke. This study explores whether shoulder pain after stroke is related to shoulder subluxation, age, limitations in shoulder range of motion, and upper extremity motor impairment.

Method. Shoulder pain was measured with a visual analog scale in 20 subjects admitted to a rehabilitation hospital within 6 weeks of onset of their first stroke. Degree of shoulder pain was correlated with vertical, horizontal, and total asymmetries of glenohumeral subluxation; age; shoulder flexion, abduction, and external rotation; and the upper extremity subscore of the Fugl-Meyer Motor Assessment.

Results. Shoulder pain after stroke was not correlated with age ($r = .019, p = .916$); vertical ($r = .081, p = .324$), horizontal ($r = .126, p = .241$), or total asymmetry ($r = -.098, p = .288$); shoulder flexion ($r = .049, p = .390$) or abduction ($r = -.074, p = .337$); or Fugl-Meyer scores ($r = -.123, p = .257$). In contrast, shoulder pain was strongly correlated with degree of shoulder external rotation ($r = -.457, p = .006$).

Conclusion. These results do not support a strong relationship between shoulder subluxation and pain after stroke. Appropriate precautions should be taken to prevent range of motion limitations that may result in shoulder pain.

Shoulder pain is a common complication after stroke, occurring in up to 84% of persons who have had a stroke (Braun et al., 1971; Hurd, Farrell, & Waylonis, 1974; Najenson, Yacubovich, & Pikielny, 1971; van Ouwenaller, Laplace, & Chantraine, 1986). The pain may be accompanied by decreased motivation, mood changes, poor motor recovery, and failure to respond to rehabilitation (Roy, 1988). Commonly reported etiologies of poststroke shoulder pain include adhesive capsulitis (i.e., “frozen shoulder”) and shoulder subluxation (Roy, 1988; Totta & Beneck, 1991). Other causes of shoulder pain include brachial plexus injuries (Kaplan, Meridith, Taft, & Betts, 1977; Moskowitz & Porter, 1963), impingement syndromes (Griffin, 1986), and rotator cuff injuries (Najenson et al., 1971). Persistent or untreated shoulder pain may result in reflex sympathetic dystrophy, which may make the pain more difficult to treat (Tepperman, Greyson, Hilbert, Jimenez, & Williams, 1984; van Ouwenaller et al., 1986).

Similarly, subluxation of the humeral head from the glenoid fossa after stroke has not been disputed. Reported incidences in persons with shoulder subluxation after stroke vary from 17% to 66% (Fitzgerald-Finch & Gibson, 1975; Najenson & Pikielny, 1965; Smith, Cruik-
shank, Dunbar, & Akhtar, 1982). However, the correlation between shoulder pain and subluxation after stroke has remained a controversial topic. Studies have reported subluxation in the hemiplegic shoulder resulting in dis
tension of the normal joint capsule and stretching of the surrounding muscles (Moskowitz, Goodman, Smith, Balthazar, & Melfins, 1969; Taketomi, 1975). Sometimes pain was caused by rotator cuff injuries that were accompanied by subluxation of the humeral head (Najenson et al., 1971); other times, pain and inferior subluxation were caused by traumatic tendinitis or rupture of the coracohumeral ligament (Jensen, 1980). Many researchers advocated the use of shoulder supports or electrical stimulation to prevent or reduce pain caused by shoulder subluxation, although no causal relationship between shoulder pain and subluxation was shown (Brudny, 1985; Faghihi et al., 1994; Smith et al., 1982; Smith & Okamoto, 1981; Varghese, 1981).

Several studies have found no relationship between shoulder pain and subluxation without investigating other likely causes. One study reported no significant relation between shoulder pain and subluxation while identifying diagnostic evidence of subluxation on radiographs of subjects with hemiplegia (Arsenault, Silodeau, Duril, & Riley, 1991). A second study found no significant difference in the degree of shoulder pain between subjects with and without subluxation and no correlation between grade of subluxation and degree of pain (Van Langenberghe & Hogan, 1988). Additionally, a third study did not support the association of shoulder subluxation with shoulder pain in subjects averaging 71 ± 100 days (range 11 days–511 days) after stroke (Bohannon & Andrews, 1990).

Other studies have rejected a causal relationship between shoulder subluxation and pain after stroke because other etiologies have been identified. One study principally supported the close association of shoulder pain with spasticity (van Ouwenaller et al., 1986). Although subluxation was observed in 50% of the subjects, the presence of spasticity in almost all the subjects with subluxation (n = 219) led the authors to the conclusion that "spasticity...is the prime factor and most frequently encountered in the genesis of shoulder pain in the hemiplegic patient" (van Ouwenaller et al., 1986, p. 26). Another study described subjects with shoulder pain after stroke who had significantly more spasticity of the affected limb, less sensitivity to pinprick of the upper paralyzed extremity, and a more severe subluxation of the affected shoulder than those who had no shoulder pain (Poulin de Courval et al., 1990). However, no shoulder range of motion measurements were reported in any of these studies.

Few studies have concomitantly explored shoulder subluxation and other potential causes of shoulder pain in persons after stroke. The present study was conducted to determine whether shoulder pain after stroke is related to shoulder subluxation, age, limitations in shoulder range of motion, and severity of upper extremity motor impairment. The results may clarify not only the etiologies, but also possible treatment rationales of shoulder pain after stroke.

Method

Subjects

Two hundred nineteen patients consecutively admitted to the East Orange Facility of the Kessler Institute for Rehabilitation in New Jersey during a 10-month period for rehabilitation of first thromboembolic or hemorrhagic stroke were screened for inclusion in this study. Inclusion criteria included clinical evidence of shoulder subluxation of the affected extremity. Exclusion criteria included time from onset of stroke to enrollment in the study of more than 6 weeks, history of neurologic condition resulting in unilateral or bilateral hemiparesis, presence of clinical stroke syndrome undetected by computed tomography (CT) or magnetic resonance imaging (MRI), neuroanatomic lesions resulting in bilateral hemiparesis, and history of neurologic or orthopedic conditions that possibly cause shoulder subluxation. The presence of a pure unilateral lesion ensured that the unaffected shoulder could serve as an accurate baseline measure for each subject when the degree of shoulder subluxation of the affected shoulder was calculated. Twenty-six (12%) of the 219 patients admitted met the inclusion criteria.

Informed consent was obtained before enrollment. The neuroanatomic location of the stroke was recorded by the first author from the acute care hospital's reports of CT or MRI. The clinical stroke syndrome was recorded from the admission physical examination. Data were collected using Part J of the National Institute of Neurological Diseases and Stroke (NINDS) Stroke Data Bank (Kunitz et al., 1984).

Instrument

Each subject was interviewed to obtain demographic information and descriptions of shoulder pain. Subjects also were clinically evaluated to determine the degree of upper extremity motor function, shoulder range of motion, and shoulder subluxation.

Shoulder pain. A visual analog scale (Huskisson, 1974), which is commonly interpreted as a valid report of subjective pain, was used to record degree of pain. Each
subject was asked to rate the presence and degree of pain in the affected shoulder as 0 (no pain experienced) to 10 (worst pain imaginable) during normal daily activity. No pain testing was performed during the evaluation.

**Upper extremity motor function.** Motor impairment of the affected extremity was assessed with the sum of the upper extremity, wrist, and hand scores of the Fugl-Meyer Motor Assessment (Fugl-Meyer, Jääskö, Leyman, Olsson, & Steglund, 1975). The Fugl-Meyer assessment is a reliable quantitative application of the Brunnstrom Scale of Motor Recovery (Brunnstrom, 1970). An ordinal scale was used to rate various stereotypical movements: 0 for unable to perform, 1 for able to perform partially, and 2 for able to perform completely. The highest possible attainable score was 66.

**Shoulder range of motion.** Passive range of motion of the affected shoulder was measured in the sitting position with standard goniometric technique (Norkin & White, 1985). Range of motion included shoulder flexion, abduction, and external rotation. Each plane was measured once, but if a notable amount of tone was encountered, one of the other investigators helped to support the extremity while the measure was repeated.

**Shoulder subluxation.** The degree of shoulder subluxation was measured with anteroposterior radiographs of the subject's unaffected and affected shoulders in the sitting position. Radiographs are the accepted standard of measuring shoulder subluxation (Arsenault et al., 1991; Boyd et al., 1993; Hall, Dudgeon, & Guthrie, 1995; Prevost, Arsenault, Dutil, & Drouin, 1987). A 7-mm lead marker was placed over the distal aspect of the clavicle to correct any variability resulting from magnification of the X ray.

Radiographs were analyzed on a standard viewing box, in random order, and with identifying information masked. Three reference points (see Figure 1) were identified, as described by Prevost and colleagues (1987): the central point of the glenoid fossa (G), the central point of the humeral head (C), and the most inferolateral point of the acromion (A). The central point of the glenoid fossa was the intersection of a horizontal line connecting points that produced the greatest width and a vertical line connecting points that produced the greatest height. The central point of the humeral head was the bisection point of the line that measured the greatest distance horizontally across the humeral head. This line was parallel to the line within the glenoid fossa.

The vertical (V) component of the glenohumeral position was determined by measuring the distance between the point on the acromion and a perpendicular horizontal line through the central point of the humeral head. The horizontal component (H) was determined by measuring the distance between the central point of the glenoid fossa and a perpendicular vertical line through the central point of the humeral head.

Asymmetries between the unaffected and affected shoulders were calculated with the vertical and horizontal components to gauge the degree of subluxation in the affected shoulder. **Vertical asymmetry** was calculated by subtracting the vertical displacement of the affected shoulder from the vertical position of the unaffected shoulder. A negative value indicated the presence of inferior subluxation. Similarly, **horizontal asymmetry** was calculated by subtracting the horizontal displacement of the affected shoulder from the horizontal position of the unaffected shoulder. A negative value indicated lateral displacement of the humeral head.

The best single measure of degree of correction should summarize as much of the data on different aspects of error as possible. Vertical and horizontal asymmetry data were summarized by computing **total asymmetry** along the hypotenuse of the horizontal and vertical asymmetries:

\[
\text{Total asymmetry} = \sqrt{\text{(vertical asymmetry)}^2 + \text{(horizontal asymmetry)}^2}
\]

Total asymmetry reflects absolute error, not direction of error, because squaring yields the same result regardless of whether asymmetry values are positive or negative. Therefore, mean total asymmetry values were calculated only from individual total asymmetry values, not from means of raw or absolute vertical and horizontal asymmetry values.

**Data Analysis**

Data were analyzed with the Statistical Package for the Social Sciences (SPSS) for Windows, version 6.1 (Norusis, 1993). Kendall's tau correlation coefficients (\(\tau\)) were calculated to explore the relationships among the vertical, horizontal, and total asymmetries and the visual analog pain scores; amount of shoulder flexion, abduction, and external rotation; Fugl-Meyer subscore; and age.

**Results**

Of the 26 subjects enrolled in the study, radiographs of 6 unexpectedly demonstrated no subluxation (i.e., no difference in vertical and horizontal components between the unaffected and affected shoulders). Therefore, these data were excluded, and 20 subjects completed the study.

Table 1 outlines the characteristics of the subjects. The mean age of the subjects was 63 years (range 42 years–83 years). Fourteen (70%) of the subjects were men, and 6 (30%) were women. Thirteen (65%) subjects had nonhemorrhagic strokes. Six (30%) had lesions in the
left hemisphere, 13 (65%) had lesions in the right hemisphere, and 1 (5%) had a lesion in the brainstem. Eleven (55%) subjects had cortical lesions, whereas 8 (40%) had subcortical lesions.

Table 2 shows the individual measurements obtained from each subject. The average Fugl-Meyer score was 8.7 points (95% confidence interval 4.8, 12.6). Eight (40%) subjects were classified in Brunnstrom stage II (early synergy), 11 (55%) in stage III (full synergy), and 1 (5%) in stage IV (some movement out of synergy) (Brunnstrom, 1970).

Nine (45%) subjects had pain scores between 4 and 10, and the remainder had no (0) or minimal (1) pain. There was a strong correlation between shoulder pain and shoulder external rotation ($r_h = -0.457, p = 0.006$) (see Figure 2). In contrast, shoulder pain was not correlated with degree of shoulder flexion ($r_h = 0.049, p = 0.390$), shoulder abduction ($r_h = 0.074, p = 0.337$), vertical asymmetry ($r_h = 0.081, p = 0.324$), horizontal asymmetry ($r_h = 0.126, p = 0.241$), total asymmetry ($r_h = 0.098, p = 0.288$), age ($r_h = 0.019, p = 0.916$), or Fugl-Meyer score ($r_h = -0.123, p = 0.257$).

Discussion

This study showed that limitations in shoulder external rotation play an important role in shoulder pain after stroke, whereas shoulder subluxation does not. The pathophysiologies of these abnormalities are quite different (Caillet, 1991). Shoulder subluxation is associated with flaccid hemiplegia and may occur because of impairments in the angulation of the glenoid fossa, the tone of the supraspinatus muscle, the reflex contraction of the rotator cuff muscles during humeral abduction, isometric and isotonic contractions of the deltoid muscle, or the integrity of the superior aspect of the gleno-humeral joint. Upon development of spasticity, the affected extremity rests in a predominant pattern of shoulder adduction and internal rotation. Without active or passive ranging of the shoulder, contracture of the shoulder ensues, and pain follows.

All subjects had evidence of both shoulder subluxation (documented radiographically) and spasticity. Spasticity, although not formally evaluated, was "developing but may not be very marked" (Brunnstrom, 1970, p. 43) because all subjects met the criteria for Brunnstrom stage II or greater. However, despite the presence of subluxation and spasticity in each subject, shoulder pain was highly correlated with external rotation and not with subluxation. Therefore, it is likely that the external rotation explains the etiology of shoulder pain in these subjects.

Two investigations also found important relationships between shoulder pain and limitations in external rotation. The first did not find correlations among shoulder pain, spasticity, and Fugl-Meyer scores (Bohannon, Larkin, Smith, & Horton, 1986). The second found correlations between shoulder pain and strength of the shoulder external rotators and abductors but found no correlation among shoulder pain, age, and body weight (Bohannon, 1988). In summary, limited shoulder range of motion appears to be a more likely cause of shoulder pain than subluxation. Onset of shoulder pain after surgical treatment of subarachnoid hemorrhage appeared to be related to age and limitations in mean range of shoulder external rotation (Bruckner & Nye, 1981). Subjects with painful, stiff shoulder joints were found to have a pathogenesis similar to that of idiopathic adhesive capsulitis (Rizk, Christopher, Pinals, Salazar, & Higgins, 1984).

The present findings suggest that attention should be focused on maintaining shoulder range of motion. Persons who have had a stroke should learn range of motion exercises to prevent limitations that may cause pain and subsequent complications (Jensen, 1980; Varghese, 1981). Medications such as nonsteroidal antiinflamma-
**Table 1**

**Subject Age, Gender, Onset-to-Study Time, and Type and Site of Lesion**

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age</th>
<th>Gender</th>
<th>Onset-to-Study Time (Days)</th>
<th>Type</th>
<th>Lesion</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>76</td>
<td>M</td>
<td>36</td>
<td>Hem</td>
<td>Right thalamus</td>
</tr>
<tr>
<td>2</td>
<td>67</td>
<td>F</td>
<td>38</td>
<td>Inf</td>
<td>Right frontoparietal</td>
</tr>
<tr>
<td>3</td>
<td>55</td>
<td>M</td>
<td>41</td>
<td>Hem</td>
<td>Right thalamus</td>
</tr>
<tr>
<td>4</td>
<td>61</td>
<td>M</td>
<td>54</td>
<td>Inf</td>
<td>Right middle cerebral artery</td>
</tr>
<tr>
<td>5</td>
<td>47</td>
<td>M</td>
<td>29</td>
<td>Inf</td>
<td>Left temporoparietal</td>
</tr>
<tr>
<td>6</td>
<td>60</td>
<td>M</td>
<td>29</td>
<td>Inf</td>
<td>Right parietal</td>
</tr>
<tr>
<td>7</td>
<td>83</td>
<td>F</td>
<td>40</td>
<td>Hem</td>
<td>Right cerebral hemisphere</td>
</tr>
<tr>
<td>8</td>
<td>76</td>
<td>M</td>
<td>38</td>
<td>Inf</td>
<td>Left parietal</td>
</tr>
<tr>
<td>9</td>
<td>73</td>
<td>M</td>
<td>40</td>
<td>Inf</td>
<td>Right middle cerebral artery</td>
</tr>
<tr>
<td>10</td>
<td>79</td>
<td>M</td>
<td>36</td>
<td>Inf</td>
<td>Left frontotemporoparietal</td>
</tr>
<tr>
<td>11</td>
<td>50</td>
<td>M</td>
<td>25</td>
<td>Hem</td>
<td>Left basal ganglia</td>
</tr>
<tr>
<td>12</td>
<td>66</td>
<td>M</td>
<td>21</td>
<td>Inf</td>
<td>Midbrain or pons</td>
</tr>
<tr>
<td>13</td>
<td>57</td>
<td>M</td>
<td>20</td>
<td>Inf</td>
<td>Left middle cerebral artery</td>
</tr>
<tr>
<td>14</td>
<td>63</td>
<td>F</td>
<td>24</td>
<td>Inf</td>
<td>Right middle cerebral artery</td>
</tr>
<tr>
<td>15</td>
<td>47</td>
<td>M</td>
<td>24</td>
<td>Hem</td>
<td>Right basal ganglia</td>
</tr>
<tr>
<td>16</td>
<td>75</td>
<td>F</td>
<td>31</td>
<td>Inf</td>
<td>Right parietal</td>
</tr>
<tr>
<td>17</td>
<td>76</td>
<td>M</td>
<td>22</td>
<td>Inf</td>
<td>Left basal ganglia</td>
</tr>
<tr>
<td>18</td>
<td>69</td>
<td>F</td>
<td>31</td>
<td>Inf</td>
<td>Right caudate</td>
</tr>
<tr>
<td>19</td>
<td>42</td>
<td>M</td>
<td>13</td>
<td>Hem</td>
<td>Right basal ganglia</td>
</tr>
<tr>
<td>20</td>
<td>43</td>
<td>F</td>
<td>40</td>
<td>Hem</td>
<td>Right cerebral hemisphere</td>
</tr>
</tbody>
</table>

*Note:* Hem = Hemorrhagic infarct. Inf = Nonhemorrhagic infarct.

...tory drugs may be used as adjunct therapy to reduce pain during range of motion exercises (Poduri, 1993). Cold, heat, or nerve or motor point blocks may reduce spasticity (Caillet, 1991; Rizk, Christopher, Pinals, Higgins, & Frix, 1983). Surgical procedures may be considered if pain is not relieved by conservative means (Braun et al., 1971; Pinzur & Hopkins, 1986). Future research needs to explore whether a critical level of shoulder range exists that prevents or minimizes pain.

Our findings support a recommendation offered by Hurd and colleagues (1974) that shoulder supports should not be uniformly applied to all persons with shoulder subluxation after stroke because their use does not reduce shoulder pain. Supports can be useful for purposes other than the reduction of subluxation. For example, a patient with a flaccid upper extremity may benefit from a support to decrease traction forces or immobilize the extremity to prevent neurovascular or soft tissue injury. A patient with some volitional movement may use a support to distribute the weight of the affected limb to...

**Table 2**

**Subject Fugl-Meyer Scores, Visual Analog Pain Scores, Asymmetries of Affected Shoulder, and Shoulder Ranges of Motion**

<table>
<thead>
<tr>
<th>Subject</th>
<th>Pain Score</th>
<th>Fugl-Meyer Score</th>
<th>Vertical Asymmetry</th>
<th>Horizontal Asymmetry</th>
<th>Total Asymmetry</th>
<th>Flexion</th>
<th>Abduction</th>
<th>External Rotation</th>
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<td>-0.80</td>
<td>0.90</td>
<td>135</td>
<td>163</td>
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<tr>
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<td>4</td>
<td>-0.30</td>
<td>-1.10</td>
<td>-1.00</td>
<td>1.39</td>
<td>142</td>
<td>132</td>
</tr>
<tr>
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<td>-0.70</td>
<td>-0.30</td>
<td>0.76</td>
<td>137</td>
<td>134</td>
</tr>
<tr>
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<td>1.53</td>
<td>2.51</td>
<td>155</td>
<td>145</td>
</tr>
<tr>
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<td>4</td>
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<td>-1.00</td>
<td>-1.00</td>
<td>2.51</td>
<td>155</td>
<td>145</td>
</tr>
<tr>
<td>6</td>
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<td>6</td>
<td>-1.40</td>
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<td>0.60</td>
<td>1.52</td>
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<td>132</td>
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<td>0.82</td>
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<td>4</td>
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<td>0.60</td>
<td>0.60</td>
<td>1.80</td>
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<td>1.00</td>
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<td>17</td>
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<td>60</td>
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<td>19</td>
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<td>1.26</td>
<td>140</td>
<td>99</td>
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<tr>
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<td>-0.70</td>
<td>-0.70</td>
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</table>
another part of the body. Future studies need to address the benefits and complications of long-term use of shoulder supports to determine their purpose in stroke rehabilitation.

There are several unique aspects to this research. First, all subluxation measurements were calculated as asymmetries because they express the differences between measurements of the affected and unaffected shoulders more directly than raw values. Second, the measure of total asymmetry of the affected shoulder synthesizes vertical and horizontal asymmetry into a single measure. In future research, the measurement of total asymmetry in three dimensions could be a useful and powerful tool to compare asymmetries of shoulders with inferior and anterior subluxation. Such a calculation would require both anteroposterior and lateral radiographs of the shoulders. Third, the subjects were chosen within a homogenous period of 0 weeks to 6 weeks after stroke. Smith and colleagues (1982) found that the time-linked effects of the incidence of shoulder subluxation was 42.7% (47/110) 2 months after stroke. No other studies have examined the relationship between shoulder subluxation and pain at discreet times after stroke. The use of consistent onset-to-study times is needed to identify time-linked effects.

Limitations

There were three limitations to this study. First, the sample size is smaller than those of many of the studies previously cited. Although the correlation between shoulder pain and external rotation probably would not change, a small sample size can identify only major effects (Ottenbacher, 1995). Second, this study did not fully explore other causes of shoulder pain. For example, spasticity and sensation were not formally evaluated to determine their roles in causing shoulder pain. Arthrography or MRI of the shoulder were not performed to rule out the possibility of rotator cuff injuries. Future studies should test a larger number of concomitant etiologies of shoulder pain to better clarify their roles in causing the pain. Finally, this study describes relationships between shoulder pain and other variables only up to 6 weeks after stroke. Further research should address issues of shoulder pain in persons who live with long-term effects of stroke.
Conclusion
Like others, this study has found no association between shoulder pain and subluxation after stroke. The use of shoulder supports during rehabilitation after stroke may be beneficial to some persons, but these supports do not need to be used universally as a treatment of shoulder subluxation. Rather, limited passive shoulder range of motion, specifically external rotation, appears to cause shoulder pain. We recommend that persons who have had a stroke be taught to use a home range of motion exercise program that maintains the integrity of the shoulder joint and prevents complications such as pain. More research is needed to critically evaluate multiple causes of shoulder pain in persons after stroke so that the etiology and pathophysiology of the pain are clearer.

References


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