KINEMATICS OF THE CONTRALATERAL AND IPSILATERSHoulder: A POSSIBLE RELATIONSHIP WITH POST-STROKE SHOULDERPAIN

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Objective: Post-stroke shoulder pain is a common phenomenon in hemiplegia and impedes rehabilitation. The aim of this study was to identify a possible relationship between post-stroke shoulder pain, scapula resting position and shoulder motion.

Methods: Shoulder kinematics of 27 patients after stroke (17 men) were compared with 10 healthy age-matched control subjects. Using an electromagnetic tracking device, the kinematics of both the contralateral and ipsilateral (i.e. paretic and non-paretic) arm during active and passive abduction and forward flexion were measured and expressed in Euler angles.

Results: Scapular lateral rotation relative to the thorax was increased in patients with post-stroke shoulder pain compared with both patients without post-stroke shoulder pain and control subjects at rest as well as during arm abduction and forward flexion. Additionally, glenohumeral elevation was decreased in patients with post-stroke shoulder pain during passive abduction. No differences were found regarding scapula position (displacement relative to the thorax).

Conclusion: In patients with post-stroke shoulder pain a particular kinematical shoulder pattern was established, characterized by enhanced scapular lateral rotation and diminished glenohumeral mobility.

Key words: stroke, hemiplegia, shoulder pain, kinematics.


INTRODUCTION

Post-stroke shoulder pain (PSSP) is a common phenomenon in hemiplegia, with an estimated incidence of between 16\% and 84\% (1–5). PSSP impedes rehabilitation and may also interfere with walking, transfers, performance of self-care activities, and quality of life (3). The occurrence of PSSP is probably not related to age and gender and may be related to the severity of the paresis. However, within the group of patients with substantial upper limb deficits it is stated that factors such as soft tissue damage or spasticity have a greater effect on pain than the severity of the stroke deficit per se (4).

Among the various factors thought to be contributing to the occurrence of shoulder pain, some are related to the shoulder joint, such as rotator cuff injuries (6) or subluxation of the humeral head (7), whereas other factors are related to the neurological lesion, such as central post-stroke pain, lack of sensibility, unilateral neglect and spasticity (7, 8).

It has been shown that the kinematics of both shoulders are affected as result of a stroke (9, 10). To our knowledge, no study has evaluated a possible relation between the alterations in shoulder kinematics and the occurrence of PSSP. The goal of this study, therefore, was to identify a possible relationship between PSSP, shoulder-resting pose (i.e. position and orientation) and shoulder motion. We hypothesize that shoulder pain is related to a disturbed scapular and humeral resting pose and/or a deviating contribution of the scapula or humerus to the abduction and forward flexion of the arm. Then, chronic shoulder pain, whatever the initiating factor, may eventually be the consequence of a vicious circle of repetitive soft tissue damage caused by improper kinematics (11–13).

METHODS

Subjects

Twenty-seven patients in the subacute phase after stroke were recruited from the inpatient ward of a rehabilitation centre (Table I). All patients had experienced their first stroke and had no history of shoulder complaints prior to the stroke. They were able to perform all the required physical, cognitive and communicative tests for this study. Prior to the measurements, current shoulder pain, muscle tone and degree of paralysis were scored, using a 0–100-mm visual analogue scale (VAS) (14, 15) (0: no pain; 100: unbearable pain), a 0–5 scale modified Ashworth Scale (16, 17) (0: no increase in muscle tone; 5: limb rigid in flexion or extension) and 0–6 scale Brunnstrom stage (18) (0: complete hemiparesis; 6: no hemiparesis), respectively (Table I). Measurements were performed on the contralateral side (relative to the side of the lesion, i.e. “paretic”) as well as the ipsilateral side (relative to the side of the lesion, i.e. “non-paretic”).

Ten age-matched healthy subjects (Table I) with no history of shoulder complaints were used as a control group. The study was approved by the local Institutional Review Board and all subjects signed an informed consent statement before the start of the measurements.

Kinematics

An electromagnetic tracking device (MotionMonitor™; Innovative Sports Training, Inc., Chicago, IL, USA) was used to quantify shoulder kinematics. This setup consists of a transmitter creating a weak magnetic
with arm elevation as a within factor and group (control, patients with
Scapular and humeral poses of the shoulders of patients after stroke with
Statistics
the movements of the humerus relative to the scapula.
relative to the thorax. Glenohumeral poses were obtained by expressing
90
subjects' arm was elevated approximately 60
°
and sagittal plane for both shoulders. during these measurements, the
and external rotations of the humerus were determined in the frontal
planes. Movements were standardized by aligning the elevation plane to an
the sensor on the acromion are questionable above this angle (20). The
this was expected to be feasible for most patients (9), and readings of
the right) (19). The orientations of these LCSs were decomposed and
expressed in Euler angles using the decomposition order as proposed
by the ISG (19), i.e. Y, X', Z'' (pro/retraction, medial/lateral rotation,
and the elbow was in
°
and 120
°
of flexion. Movements of the scapula and humerus were expressed
relative to the thorax. Glenohumeral poses were obtained by expressing
the movements of the humerus relative to the scapula.
Statistics
Scapular and humeral poses of the shoulders of patients after stroke with
and without PSSP and control subjects were compared using a General
Linear Model analysis of variance (ANOVA) with repeated measures,
with arm elevation as a within factor and group (control, patients with
and without PSSP) as a between factor. One-way ANOVA was used to
compare the poses of the scapula and humerus at rest and to evaluate
maximal internal and external humeral rotations. Results were considered
significant at p < 0.05. When a significant difference was found, a Tukey
honestly significant difference post-hoc test was used to determine which
groups were different. An a priori power analysis was conducted based
on previous studies (9, 13, 22–24). The effect size was 0.7 based on an
inter-individual variability of scapular rotations of 7°. With an alpha of
0.05 and a desired smallest detectable difference between the groups of
3.5°, a sample size of n = 8 per group results in a power of 0.8.

RESULTS
As only 2 patients were able to reach the target elevation angle of 120° with their contralateral (“paretic”) arm, only the scapular and humeral poses in rest and at 30°, 60° and 90° of arm elevation were used for analysis. For patients with PSSP it appeared to be very difficult to perform any active arm elevation with their contralateral arm. With their ipsilateral (“non-paretic”) arm, all patients reached 90° during active and passive abduction and forward flexion.

Resting pose
Fig. 1 shows a schematic representation of scapular medial/lateral rotation at rest of all subjects. At rest, the contralateral shoulder of patients with PSSP showed more scapular lateral rotation than both patients without PSSP and control subjects (p = 0.03 and p < 4830.01; Figs 1 and 2). Scapula laterorotation of patients with PSSP at the ipsilateral side was enhanced compared with control subjects but not to patients without PSSP (p = 0.01 and p = 0.25 respectively, Figs 1 and 2). No differences in scapular anterior/posterior tilt or pro/retraction were found. All differences in poses between the groups

Table 1. Characteristics of subjects with and without post-stroke shoulder pain (PSSP) and controls

<table>
<thead>
<tr>
<th></th>
<th>Patients with PSSP</th>
<th>Patients without PSSP</th>
<th>Control subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of subjects (male/female)</td>
<td>13 (10/3)</td>
<td>14 (7/7)</td>
<td>10 (4/6)</td>
</tr>
<tr>
<td>Age, years (mean (SD))</td>
<td>59.3 (11.1)</td>
<td>57.0 (9.5)</td>
<td>49.3 (7.2)</td>
</tr>
<tr>
<td>VAS score, mm (mean (SD))</td>
<td>49.7 (24.7)</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Range Brunnstrom stage (degree of paralysis)</td>
<td>1–5</td>
<td>2–5</td>
<td>NA</td>
</tr>
<tr>
<td>Range Modified Ashworth scale (muscle tone)</td>
<td>0–2</td>
<td>0–3</td>
<td>NA</td>
</tr>
<tr>
<td>Time since stroke, weeks (mean (SD))</td>
<td>14.4 (9.3)</td>
<td>13.0 (7.6)</td>
<td>NA</td>
</tr>
<tr>
<td>Side of brain lesion: left/right</td>
<td>10/3</td>
<td>5/9</td>
<td>NA</td>
</tr>
<tr>
<td>Lesion location: cortical/sub-cortical</td>
<td>8/5</td>
<td>9/5</td>
<td>NA</td>
</tr>
</tbody>
</table>

NA: not applicable; VAS: visual analogue scale; SD: standard deviation.

Fig. 1. Schematic representation of scapular medial/lateral rotation in degrees (dorsal view) during rest of patients with and without post-stroke shoulder pain (PSSP), and of control subjects. *Significant differences. Positive values: medial rotation; negative values: lateral rotation.

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were found to be regarding scapular orientation, not position (displacement relative to the thorax).

**Contralateral shoulder movements**

During passive abduction a significant group-angle interaction effect for glenohumeral elevation was found ($p = 0.04$), indicating a larger increase in glenohumeral elevation from rest to 30° arm elevation for control subjects than the paretic shoulder of all patients.

Patients with PSSP showed enhanced scapular lateral rotation during active and passive abduction and forward flexion when compared with control subjects. When compared with patients without PSSP, scapular lateral rotation was only found to be enhanced during passive abduction, while no significant differences were found during active and passive forward flexion and active abduction (Table II). No differences were found between patients without PSSP and controls in shoulder kinematics during all performed movements.

**DISCUSSION**

The goal of this study was to identify a possible relationship between PSSP and deviations in shoulder kinematical patterns.
Table III. p-values of maximal internal and external glenohumeral rotations

<table>
<thead>
<tr>
<th>Plane of rotation</th>
<th>Movement direction</th>
<th>PSSP + vs PSSP – Controls</th>
<th>PSSP + vs Controls</th>
<th>PSSP – vs Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Contralateral</td>
<td>Frontal</td>
<td>0.82</td>
<td>0.06</td>
<td>0.16</td>
</tr>
<tr>
<td></td>
<td>Internal</td>
<td>0.02</td>
<td>&lt;0.00</td>
<td>0.12</td>
</tr>
<tr>
<td>Sagittal</td>
<td>Internal</td>
<td>0.36</td>
<td>0.02</td>
<td>0.28</td>
</tr>
<tr>
<td></td>
<td>External</td>
<td>0.08</td>
<td>0.00</td>
<td>0.22</td>
</tr>
<tr>
<td>Ispilateral</td>
<td>Frontal</td>
<td>0.08</td>
<td>0.46</td>
<td>0.66</td>
</tr>
<tr>
<td></td>
<td>Internal</td>
<td>0.68</td>
<td>0.12</td>
<td>0.42</td>
</tr>
<tr>
<td>Sagittal</td>
<td>Internal</td>
<td>0.50</td>
<td>0.10</td>
<td>0.42</td>
</tr>
<tr>
<td></td>
<td>External</td>
<td>0.97</td>
<td>0.09</td>
<td>0.75</td>
</tr>
</tbody>
</table>

PSSP: patients with (+) and without (–) post-stroke shoulder pain.

This relation could indeed be established. We therefore state that shoulder pain is related to a disturbed scapular and humeral resting position and/or a deviating contribution of the scapula or humerus to the abduction and forward flexion of the arm.

The most remarkable findings in this study are the clear differences in scapular lateral rotation in patients with PSSP in both the contralateral (“paretic”) and the ipsilateral (“non-parietic”) shoulder when compared with patients without PSSP and control subjects. Price et al. (25) also found a relation between a more laterally rotated scapula (scapular lead) on the contralateral side and shoulder pain. However, they only compared the contralateral to the ipsilateral side and not to a control group. Therefore, changes in kinematics of the contralateral shoulder could have been underestimated and possible changes on the ipsilateral side were not noticed.

From Table II it seems that patients with PSSP predominantly have a left hemispheric lesion, while this seems to be inversed in patients without PSSP. However, this effect is not significant and is probably due to the small group sizes.

Based on the data from the present study, it is not possible to make comments about the origin of shoulder pain. There are several possible relations between PSSP and changes in shoulder kinematics.

First, changes in shoulder kinematics could lead to shoulder pain. Stroke patients might not be able to compensate for the gravitational pull on the scapula due to the hemiparesis. Muscles such as the m. trapezius, m. levator scapulae, m. rhomboid minor, major and serratus anterior are responsible for maintaining scapula position (26), and if the strength of these muscles is diminished, the positioning of the scapula can be affected. Since the scapula only articulates with the clavicle (at the acromioclavicular joint), the inability to compensate for the gravitational pull could cause a lateral rotation of the scapula with the acromioclavicular joint acting as a pivot, as found in this study. This, in turn, could initiate or worsen pathologies such as subluxation, impingement and capsulitis, resulting in PSSP. However, not all patients after stroke have PSSP. It might be that patients who do not suffer from PSSP have more control over their shoulder muscles and are thus better able to compensate for the mechanisms that cause PSSP. It must be noted that the alterations found in shoulder kinematics of the ipsilateral shoulder did not cause PSSP in that shoulder, which indicates that the movement alteration itself does not cause pain. However, the ipsilateral shoulder is much less affected by the stroke and therefore this shoulder is probably better able to cope with, and compensate for, mechanisms leading to PSSP.

Secondly, PSSP could cause the changes in shoulder kinematics found. If PSSP starts to develop, shoulder kinematics could be altered as pain relief. In a normal, healthy shoulder, next to stabilization of the shoulder by the rotator cuff, lateral (upward) rotation of the scapula prevents impingement by rotating the major tubercle of the humerus away from the coraco-acromial arch, thus preventing the supraspinatus outlet from being narrowed (4, 13). This mechanism can be used by patients suffering from PSSP. The fact that both shoulders of patients with PSSP show altered kinematics, with only the contralateral shoulder being painful, indicates that a central compensating mechanism for pain relief affecting both shoulders could be involved. However, this bilateral effect can also be explained by the fact that both hemispheres control both sides of the body (27–29), so a lesion in one-half of the brain also affects the other half. It has been shown before that the ipsilateral side of stroke patients is also affected after stroke (9, 30).

Thirdly, an additional factor causes both PSSP and changes in kinematics. Impingement syndrome is often considered as a cause of shoulder pain in hemiplegic stroke patients and could also result in changes in kinematics (4, 31). Adhesive capsulitis (frozen shoulder), is characterized by diminished range of shoulder motion (especially exorotation) and shoulder pain (32) and the term is used quite generally in relation to PSSP (4). The shoulders of our patients with PSSP show these characteristics but this does not mean that these patients suffer from capsulitis. It has been shown that the scapula of patients with capsulitis is oriented normally during rest, but during arm elevation, the scapula shows larger lateral rotation (33). It must be noted, however, that the patients used in that study were not stroke patients. A common complication in stroke patients is build up of bilateral muscle tension around the head and neck and the restriction of blood flow to those areas as a result. This could eventually lead to tension-type headaches and could also explain differences in scapular kinematics of the ipsilateral shoulder found in the present study (34–36).

Based on the data from the present study, it is difficult to draw conclusions about the origin of shoulder pain. A longitudinal study, in which patients are measured multiple times during the course of rehabilitation, starting as soon as possible after the stroke, could provide more insight into the process. If pain occurs before alterations in scapular kinematics are observed, then the alterations of the lateral rotation of the scapula could act as a compensatory mechanism. If the scapular lateral rotations are already altered before the pain occurs, then the effect of the alteration itself could cause PSSP. Also, the effects of pain relief by means of injections (e.g. marcaine, lidocaine, bupivacaine) on the scapular kinematics of patients with PSSP could be of great value.

The clinical implications of the observed kinematic differences in the shoulders of stroke patients with and without PSSP remain speculative. Many pathologies could lead to
and what the relation is between PSSP and the altered shoulder kinematic alterations worsen the initial pathology or cause secondary pathologies and thus initiate a vicious circle of repetitive soft tissue damage leading to chronic PSSP.

In general we can conclude that patients with PSSP show a particular kinematical shoulder pattern, characterized by enhanced scapular lateral rotation and diminished glenohumeral mobility. The question remains as to what causes these changes and what the relation is between PSSP and the altered shoulder kinematics. It could be that PSSP causes the kinematic differences found, but it could also be the other way around. It is also possible that an additional factor could cause both PSSP and changes in shoulder kinematics.

REFERENCES