

SUMMARY

Shoulder pain after stroke.

The occurrence of shoulder pain after stroke is quite common in hemiplegia with an estimated incidence between 16% and 84%. This so-called Post Stroke Shoulder Pain, or PSSP can impede rehabilitation and interfere with both function and quality of life. It may not only interfere with shoulder function, but also with balance, walking, transfers and performance of self-care activities.

PSSP is a general term that is used in relation to the occurrence of shoulder pain after stroke and it could be caused by known afflictions such as adhesive capsulitis, impingement due to rotator cuff tears and subluxation of the glenohumeral joint. What exactly causes PSSP is as yet unclear. Most likely, PSSP is caused by damage to the structures around the glenohumeral joint due to a muscular imbalance (rotator cuff tears, spasticity, and paralysis), a disturbance in positioning of the GH joint (impingement, or upward migration of the humeral head, or inferior subluxation) or damages due to a disturbed sensibility, possibly related to unilateral neglect. Damage to the upper trunk of the brachial plexus has also been cited as a possible cause of pain and subluxation in the hemiplegic shoulder. Downward subluxation may produce traction on the axillary nerve as it winds round the surgical neck of the humeral shaft. Also entrapment of the suprascapular nerve could cause shoulder pain, but some reports contradict this finding. Some authors have described shoulder pain after stroke as a special form of Reflex Sympathetic Dystrophy (RSD), possibly caused by subluxation of the glenohumeral joint but also other precipitating factors, including immobilization of the upper extremity, trauma to the joint structures, rotator cuff tears and spasticity of the shoulder musculature have been mentioned as a possible cause.

This thesis was an attempt to get a better grip on the causes of PSSP by focusing on the relationship between stroke and shoulder kinematics and shoulder musculoskeletal proprioception. Starting from the assumption that stroke will lead to a disturbed motion pattern in the upper extremity, it was attempted to quantify the magnitude of this disturbance relate it to the existence (and on the longer term the development) of PSSP. Additionally, the disturbed motion pattern will probably lead to length changes of the shoulder muscles. Therefore muscles lengths during rest and shoulder movements are quantified and related to known muscle-related causes of shoulder pain.

Disturbances in shoulder kinematics are only one (assumed) aspect, or better, effect, of stroke. Since the musculoskeletal balance in the shoulder is essentially maintained by fine-tuned muscle function, deviations in shoulder kinematics may be caused by the effects of stroke on the level of the motor function of muscles (paralysis, spasticity), but also by disturbances in the tuning of these muscles due to the de-

organization of control. To quantify one aspect of the likely occurring de-organization, this thesis also includes experimental studies related to the proprioceptive status of the arm after stroke.

In Chapter 2 the method used to measure proprioception in the experiments included in this thesis was validated. To this end, it was investigated whether passive and active reproduction of joint position, as well as detection of passive motion (as measures of a subject's proprioception) of the shoulder differs while sitting compared to lying supine.

Shoulder proprioception of twenty-eight healthy subjects (age: 22.2 ± 1.7 years, 15 males) was tested. To test proprioception, angular motion (in degrees) for Threshold to Detection of Passive Motion (TDPM) and absolute matching error (in degrees) for Passive and Active Reproduction of Joint Position (PRJP and ARJP) were measured using an Isokinetic dynamometer. As a measure of consistency, the standard deviation per subject and test (TDPM, PRJP and ARJP) was measured over three trials.

It was found that body orientation had no effect on TDPM, PRJP and ARJP scores. Significantly larger errors were found during ARJP compared to PRJP and subjects were also significantly less accurate during ARJP.

It was concluded that body orientation does not significantly influence proprioceptive errors or consistency while movement mode (active or passive) does. The significance of these findings is that, depending on the situation or the patient's ability, proprioception tests can be conducted while either lying or sitting, but movement modes can not interchangeably be used.

A possible disturbance of shoulder proprioception after stroke could influence the stability of the shoulder and thus the rehabilitation process. In chapter 3 shoulder proprioception of both shoulders of stroke patients was compared to that of healthy control subjects. The goal of this study was to investigate whether the proprioception of both shoulders is affected after stroke.

Angular displacement (in degrees) for threshold to detection of passive motion (TDPM) tests and absolute error (in degrees) for passive reproduction of joint position tests of a total of 22 inpatients with stroke and 10 healthy control subjects was measured.

It was found that the TDPM for internal and external rotation was significantly higher for both the patient's contralateral (paretic) side and their ipsilateral (nonparetic) side as compared with the control group. Also, for internal rotation, TDPM was significantly higher on the patient's contralateral side compared with the ipsilateral side. For passive reproduction of joint position tests, no differences were

found so it was concluded that both the contralateral and ipsilateral shoulder of stroke patients showed impaired threshold to detection of passive motion while passive reproduction of joint position does not seem to be affected as a result of a stroke.

Disturbed shoulder kinematics influence shoulder (in-)stability. Shoulder pain could be related to a disturbed scapular and humeral resting pose and/or a deviating contribution of the scapula or humerus to the movements of the shoulder. The goal of the study described in chapter 4, was to identify a possible relationship between PSSP, shoulder-resting pose (i.e. position and orientation) and shoulder motion.

Shoulder kinematics of 27 stroke patients (17 male) were compared to 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.

Scapular lateral rotation relative to the thorax was increased in patients with PSSP compared to both patients without PSSP and control subjects in rest as well as during arm abduction and forward flexion. Additionally, glenohumeral elevation was decreased in patients with PSSP during passive abduction. No differences were found regarding scapula position (displacement relative to the thorax). Therefore it was concluded that in patients with PSSP a particular kinematical shoulder pattern was established, characterized by enhanced scapular lateral rotation and diminished glenohumeral mobility.

In chapter 5 a possible relation among disturbed proprioception, altered kinematics and shoulder pain after stroke was discussed. If both the shoulder kinematics and shoulder proprioception are affected after stroke, chronic shoulder pain, whatever the initiating factor, may eventually be the consequence of a vicious circle of repetitive soft tissue damage.

A total of 21 inpatients with stroke and 10 healthy control subjects were included and the orientations of both the contralateral and ipsilateral (i.e. paretic and non- paretic) shoulders during rest in degrees, as well as angular displacement (degrees) for threshold to detection of passive motion (TDPM) tests and absolute error (degrees) for passive reproduction of joint position (PRJP) tests were measured.

The contralateral shoulder of patients with PSSP showed more scapular lateral rotation and larger TDPM and PRJP scores compared to both patients without PSSP and control subjects. Additionally, the contralateral shoulder of patients with deteriorated proprioception showed more scapular lateral rotation compared to control subjects whereas their ipsilateral shoulder showed more scapular lateral rotation when compared to both control subjects and patients with good proprioception.

A clear relation between affected shoulder kinematics, affected proprioception and PSSP was found. In determining the risk of developing PSSP, attention should be paid to a patient's shoulder proprioception and kinematics. If both are altered after stroke, this could worsen the initial pathology or cause secondary pathologies and thus initiate a vicious circle of repetitive soft tissue damage leading to chronic PSSP. Additionally, more attention should be paid to the ipsilateral (i.e. non-paretic) shoulder since it could be used in determining the risk of developing PSSP in the contralateral (i.e. paretic) shoulder.

Alterations in shoulder kinematics inevitably lead to length changes in the muscles surrounding the shoulder. Different shoulder muscles have been mentioned in literature in combination with shoulder pain and the goal of chapter 6 was to investigate whether during arm elevation the glenohumeral (GH) reaction force and its location inside the GH joint as well as muscle lengths of the shoulder muscles differed between stroke patients with and without post stroke shoulder pain (PSSP) and control subjects.

A biomechanical model of the shoulder was used to calculate the GH reaction force, its location within the GH joint and muscle lengths of patients with and without PSSP and control subjects. Inputs to the model were the position of the Incisura Jugularis (IJ) and the rotations of the thorax, clavicle, scapula, humerus and forearm obtained with an electromagnetic tracking device during active arm abduction and forward flexion.

It was found that the reaction force within the GH joint remained more centered on the glenoid fossa when comparing stroke patients to control subjects but no clear differences were found between patients with and without PSSP. The mean GH reaction force was lower in both patient groups when compared to controls during forward flexion. Patients with PSSP showed a smaller increase in GH reaction forces at higher elevation angles. When looking at muscle lengths, only small differences were found (5-10 %).

In general we can conclude that stroke patients use a different movement pattern compared to healthy control subjects, apparently causing the GH joint reaction force to be centered more on the glenoid fossa. This could act as a mechanism counteracting the decreased scapulohumeral balance after stroke and can mainly be attributed to the differences in humeral rotations (i.e. plane of elevation and axial rotation) and not to differences in scapular rotations. No relevant differences were found between patients with and without PSSP regarding GH joint reaction force, its location on the glenoid fossa and muscle lengths, suggesting that shoulder pain can not be explained by these factors.

Finally, in chapter 7, the main results of this thesis were summarized and implications for future research and practice were discussed. Future studies investigating the causal relationship between proprioception, shoulder kinematics and the development of PSSP should incorporate a longitudinal design to make valid comments about the development of PSSP and the possible role of shoulder kinematics and proprioception in this development. The first measurement should be taken as soon as possible after the stroke and patients should be followed over the course of at least two years.

Intervention studies can also provide valuable information and should be aimed at improving shoulder kinematics and shoulder proprioception. As data from this thesis suggest that deteriorated shoulder kinematics are related to shoulder pain, attention should be paid to improving shoulder mobility. Dundar *et al* showed that continuous passive motion (CPM) can provide good pain control in patients with adhesive capsulitis. CPM is a treatment method designed to aid in the recovery of joints carried out by a CPM device, which constantly moves the joint through a controlled range of motion (ROM), which is in most cases increased over time and robot-assisted therapy can potentially be used to achieve these goals. With forced-use therapy patients are forced to use the contralateral (i.e. paretic) arm, while the ipsilateral (i.e. non-paretic) arm is immobilized and this method could also be used to improve shoulder mobility. We have also shown a relationship between affected proprioception and shoulder pain so interventions could also be aimed at improving proprioception. There is still some controversy however whether this is actually possible, or that these interventions train motor performance rather than proprioception.

Additionally, more research could be done on the possibility of using measurements of the ipsilateral shoulder as an indicator for the development of PSSP on the contralateral side. Measuring the kinematics and proprioception of the ipsilateral arm is less invasive for the patients and it can provide information of the status of the contralateral side, even if due to practical limitations this side is difficult to measure.