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DOCTORAL DISSERTATION

Movement and muscle activation patterns of the shoulder girdle after stroke

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Doctor of Rehabilitation Sciences and Physiotherapy, to be defended by

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List of abbreviations

3D	Three-dimensional
AC	Acromioclavicular
AI	Acromial index
ClinScaP	Clinical scapular protocol
FF	Forward flexion
GH	Glenohumeral
IwS	Individuals with stroke
PAF	Proximal arm function
PMI	Pectoralis minor index
ROM	Range of motion
SC	Sternoclavicular
SDT	Scapular distance test
ST	Scapulothoracic
TR	Trunk
WHO	World Health Organization

General introduction

1. About this work

A proper and pain free shoulder function is essential for accurate performance of daily activities and contributes to daily life autonomy and quality of life. The brain damage underlying a stroke results in several motor impairments such as muscle weakness, increased muscle tone, pathological muscle synergies and altered temporal muscle activity.[1-4] At the level of the shoulder complex, these impairments may specifically hamper scapulohumeral control, i.e. the adaptation of scapular position and movement according to the humeral position. Reduced scapulohumeral control is known to contribute to the difficulties individuals with stroke (IwS) experience when moving their paretic arm.[5] Upper limb rehabilitation after stroke could benefit from specific training to enhance scapular positioning and movement control. However, such therapy planning firstly requires an extensive evaluation of the scapulothoracic joint. Currently available clinical measurement scales for stroke are typically limited to a global upper limb assessment.[6] The specific assessment of scapulohumeral control has thus far not been covered. This doctoral project aims to enhance current knowledge on scapulohumeral control in IwS by developing a quantitative assessment protocol and a clinical scapular assessment protocol to objectively evaluate scapular movement patterns and muscle activity in IwS. As such, we want to provide more insights into altered scapulohumeral control in IwS and thereby pave the road for improved upper limb rehabilitation management.

In the first part of this general introduction, we provide information about the stroke event, how a stroke might influence normal motor control from a neurophysiological viewpoint and how this leads to post-stroke pain syndromes. Then we go deeper into the main topic of this thesis, the post-stroke shoulder. We explain the anatomy and kinesiology of the shoulder complex during arm elevation and how this is guided through specific muscular actions. Subsequently we describe what is already known on scapular movement patterns after stroke. At the end of this introduction, we formulate the different aims of the current doctoral project and outline the several chapters of this thesis.

2. Stroke

2.1. What is stroke?

The World Health Organization's (WHO) defines stroke as "rapidly developing clinical signs of focal or global disturbance of cerebral function, lasting more than 24 hours or leading to dead, with no apparent cause other than that of vascular origin".[7] However, the tremendous advances in knowledge on the epidemiology and clinical symptoms of stroke, and in medical imaging technologies have led to an updated version of the definition: "central nervous system infarction is defined as brain, spinal cord, or retinal cell death attributable to ischemia, based on (1) pathological, imaging, or other objective evidence of cerebral, spinal cord, retinal focal ischemic injury in a defined vascular distribution; or (2) clinical evidence of cerebral, spinal cord, retinal focal ischemic injury based on symptoms persisting ≥ 24 hours or until death, and other etiologies excluded".[8] Based on the different causes of stroke, i.e. hemorrhagic or ischemic, several sub-definitions have also been formulated. *Hemorrhagic stroke* is least common and is caused by a leak in a weakened blood vessel or by a brain aneurism burst and most often results in death. *Ischemic stroke* specifically refers to central nervous system infarction accompanied by overt symptoms, while silent infarction by definition causes no known symptoms. Such ischemic stroke occurs in approximately 80% of all stroke cases, and is caused by a clot blocking the blood transport to the brain. It can occur in two ways: an embolic stroke, which is caused by a clot formed somewhere in the body that travels to the brain and blocks a blood vessel there. This type of ischemic stroke is often seen in IwS with cardiac diseases like atrial fibrillation. In contrast, when the clot is formed into an artery that supplies blood directly to the brain, we call it a thrombotic stroke, linked to high cholesterol levels and arthrosclerosis.

Stroke is a major issue we face in the modern world, as it is one of the leading causes of morbidity and mortality worldwide.[9] In America, it is the fourth leading cause of death and the leading cause of adult disability. In Europe, stroke is the most important cause of morbidity and long-term disability. Current demographic evolutions will eventually lead to an increase in both the incidence and prevalence of stroke.[10] Within Europe, the large differences in incidence, prevalence and mortality have been ascribed to distinct risk factors in different regions. More severe strokes are noted in Eastern Europe, which is related to higher levels of hypertension and other risk factors over there.[11] Notable regional variations have also been found within Western Europe. In Belgium, the annual incidence of stroke is between 185-230 per 100.000 inhabitants.[12] Six percent of these IwS die within 24 hours, and 29% within the first month post-stroke. After one year, 47% is deceased.[12,13] This means that in Belgium, each year about 9000 deaths are due to stroke.

The severity of stroke correlates well with the degree of functional recovery, the total length of the hospital stay and secondary complications such as shoulder hand syndrome or pain after stroke. A major problem

accompanying stroke is the loss of function that prevents IwS from living an independent life. It is estimated that 30 to 66% of IwS are permanently disabled and dependent on the help of close relatives.[14,15] For Belgium, this means that each year approximately 19.000 families are directly confronted with the negative consequences of stroke. Moreover, over one third of IwS develop a depression and cognitive disorders.[16-18] Stroke is also the second most common cause of dementia and the most frequent cause of epilepsy in elderly.[19,20] The severity of the motor and cognitive dysfunctions, along with the social support a patient might rely on, and his own coping strategies, all strongly influence the quality of life after a stroke.[21]
In addition to the dramatic impact on personal and family level, stroke also has massive socio-economic consequences. This does not only include direct costs from hospitalization and rehabilitation, but also refers to general costs due to decreased productivity and costs for the social health care system. In Belgium, the estimated cost per patient in the acute setting lays around 44.600 euros. In industrialized countries, 2 to 4% of the national health budget would be spent on treatment of IwS, with a longer hospital stay and greater initial stroke severity as two major determinants for higher costs.[22,23]

2.2. Impact of stroke on movement control

Hemiparesis is a common residual motor impairment resulting from stroke and is characterized by unilateral (contralateral to the side of the lesion) weakness, increased muscle tone and/or (partial) loss of movement coordination. Apart from these motor impairments, many stroke patients also experience sensory deficiencies such as loss of tactile or movement sense. Together, these impairments result in a loss of voluntary movement control,[14,24] which in turn impacts on the functional abilities of the patient. In this section, we will concisely go into the normal cortical processing and neurophysiological pathways to control movement.
Controlled movement between body segments is complex, and much more than a linear sum of the multimodal input. We integrate our incoming sensory information, and create an appropriate motor output. However, when these modalities are mismatched, this leads to aberrant control. Movement control starts with 'the decision to move', occurring within the frontal brain areas, like the premotor cortex and the prefrontal cortex.[25] The decision to make a motor action is based on, or a result of, the input that these areas get from the somatosensory cortex about the location and the movement of body segments, as well as from brain areas responsible for vestibular, auditory and visual information, e.g. the posterior partial cortex.[25] The parietal cortex in turn is also highly connected with the prefrontal cortex. By integrating information on various modalities, the frontal brain areas make the decision to move and send their output to the supplementary motor area and premotor cortex. The latter two brain regions decide how the movement will be executed and also receive important information from subcortical regions (thalamus, the basal ganglia) and from

the cerebellum to filter and fine-tune movement execution.[25] The subcortical input ensures that only proper movement will be executed, and that inadequate motor actions are inhibited. Cerebellar input is responsible for coordinated movement execution via its input to the premotor cortex on correct timing of muscle contractions. The supplementary motor area and premotor cortex give input to the primary motor cortex that then initiates the movement via the descending neural pathways.[25]

Descending lateral and medial neural pathways receive their input mainly from the primary motor cortex, though the supplementary motor area, premotor cortex, several subcortical structures and areas in the brainstem also provide input. These descending pathways send a motor command toward the muscles.[25]

Within the lateral pathway, axons of the *corticospinal tract* arise from the primary motor cortex, premotor cortex and supplementary motor area, pass through the posterior limb of the internal capsule, the midbrain and pons, and the medulla oblongata before reaching the spinal cord. Ninety percent of the axons cross to the contralateral side before they descend in the lateral column of the spinal cord, where they form synapses with mostly contralateral alpha motor neurons (lateral corticospinal tract). Ten percent of the neurons of the corticospinal tract do not cross and terminate in the anterior ipsilateral spinal cord (anterior corticospinal tract). The lateral corticospinal tract is the major motor output pathway to control fractionated voluntary movement of the distal limbs.[25] A lesion in the primary motor cortex or along this tract will thus negatively influence skillful movement of arm and hand. The anterior corticospinal tract is important for neck, shoulder and trunk muscle control, though little is known on the exact innervation pattern of the proximal muscles. It seems that control of proximal muscles is more related to a muscle's function than to its anatomical location. Even though proximal muscles might have a similar cortical anatomical representation, a proximal muscle with an axial function is under bilateral cortical control, e.g. upper trapezius functioning as a cervical spine extensor; whereas a proximal muscle without an axial function is mainly contralaterally innervated, e.g. serratus anterior.[26] As such, the latter muscle is more vulnerable for a tract lesion that cannot be compensated by uncrossed fibers from the undamaged hemisphere.

The medial pathway consists of two main tracts: the medial and lateral vestibulospinal tract, and the reticulospinal tract. In general, the medial tracts are involved in control of posture and proximal movements. The *medial and lateral vestibulo-spinal tract* arises in the medulla oblongata. The medial tract is important for movement and control of the head and upper back muscles in reaction to vestibular information from the ear.[25] The lateral tract is important to maintain the center of gravity over the base of support, by facilitating extensor muscles in the mid and lower back, and in the lower limbs. Finally, the *reticulospinal* tract originates from the reticular formation in the brainstem and receives input from various brain areas. It is responsible for optimizing reflexes against gravity via axial and proximal muscle contractures to create tone and posture.[25] This tract is thus involved in gross movements such as reaching and locomotion. Although secondary to the corticospinal tract, there is emerging evidence assuming that the reticulospinal tract can also exert influence over hand movements.[27] This is important for example following corticospinal damage due to stroke. In these cases, the reticulospinal tract could provide some recovery of hand function.[27]

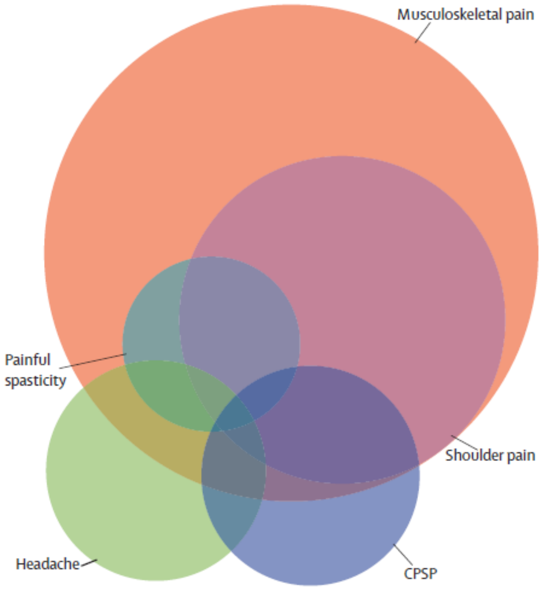
2.3. Pain after stroke

Apart from muscle weakness, increased muscle tone, loss of movement coordination and sensory deficiencies, many IwS also experience pain post-stroke, which vastly contributes to a reduced quality of life. Figure 1 clearly illustrates common types of pain after stroke. Pain due to spasticity (increased muscle tone) accounts for 7% of pain after stroke, headache for 10%, central post-stroke pain for 10%, shoulder pain for 20% and musculoskeletal pain in general for 40%.[28] However, many IwS present with a combination of several of the abovementioned pain types (as shown by the overlapping areas).

Interestingly, pain due to spasticity and shoulder pain are partially overlapping, and both are completely overlapped by musculoskeletal pain. This suggests that a painful shoulder and pain caused by spasticity are fully attributable to painful underlying musculoskeletal structures. Although this might provide a somewhat one-sided viewpoint, it is accepted that abnormal joint torques caused by spastic muscles indeed induce abnormal joint forces, leading to pain within the musculoskeletal system. The onset of hemiparesis after stroke can also adversely affect normal joint alignment, movement patterns and muscle activation patterns of the shoulder complex through mechanisms including muscle weakness, muscle spasticity and loss of voluntary motor control.[29] These changes negatively influence the stability of the shoulder complex and thereby contribute to the development of pathologies leading to musculoskeletal shoulder pain (e.g. tendinopathy of the rotator cuff, subluxation of the humeral head, adhesive capsulitis).[29]

Given the high reported prevalence of acute post-stroke upper limb dysfunctions and shoulder pain,[30-32] and the fact that shoulder pain is mainly attributed to musculoskeletal problems (as depicted in Figure 1), the next section describes the musculoskeletal characteristics of post-stroke shoulder (dys)functions and pain in further detail.

Figure 1. Different types of pain after stroke.



CPSP: central post-stroke pain;
With permission of Klit et al. Lancet Neurol 2009:8:857-68.[28]

3. The post-stroke shoulder

Adequate handling and treatment of the shoulder immediately post-stroke and during rehabilitation, are crucial to prevent or treat pain and secondary complaints such as shoulder-hand syndrome. Important factors associated with shoulder pain post-stroke are decreased range of motion of glenohumeral abduction and external rotation, poor scapulothoracic position and aberrant scapulohumeral motion, spasticity of the elbow flexors, and sensory deficits. The association of shoulder pain with restrictive passive range of motion, poor scapular control and signs of impaired sensory input may implicate a vicious circle of repetitive trauma to subacromial structures, which in turn induces shoulder pain. Other risk factors in the development post-stroke shoulder pain include trophic changes, diabetes mellitus type 2 and impaired voluntary motor control.[33-38] Lastly, left-side hemiparesis, shoulder pain or limited passive abduction range of motion at 4 months post-stroke have also been put forward as important predictors to persistent shoulder pain 1 year post-stroke.[35]

Apart from shoulder management, regaining hand function also constitutes a major goal of stroke rehabilitation. Functional use of the hand requires, in addition to intact finger control and manipulation, the ability to move the upper limb freely in all dimensions.[39-41] Positioning and orienting the hand thereby relies on the ability to control movements of the shoulder and thus on a proper and pain free shoulder function. Moreover, it has already been shown that 88% of the variance in hand function is explained by changes in active range of shoulder motion.[42] Important preconditions for optimal active range of motion of the shoulder are trunk stability, correct scapular movement, and the ability to selectively recruit muscles.[43-45] As such, optimal conditions for the external rotators of the glenohumeral joint are created, which is essential for reaching and grasping.

3.1. What is normal shoulder movement and how is it established?

It is well accepted that control of the shoulder complex relies on the appropriate passive support, supplemented with muscle forces that are coordinated by the nervous system.

3.1.1. Anatomy

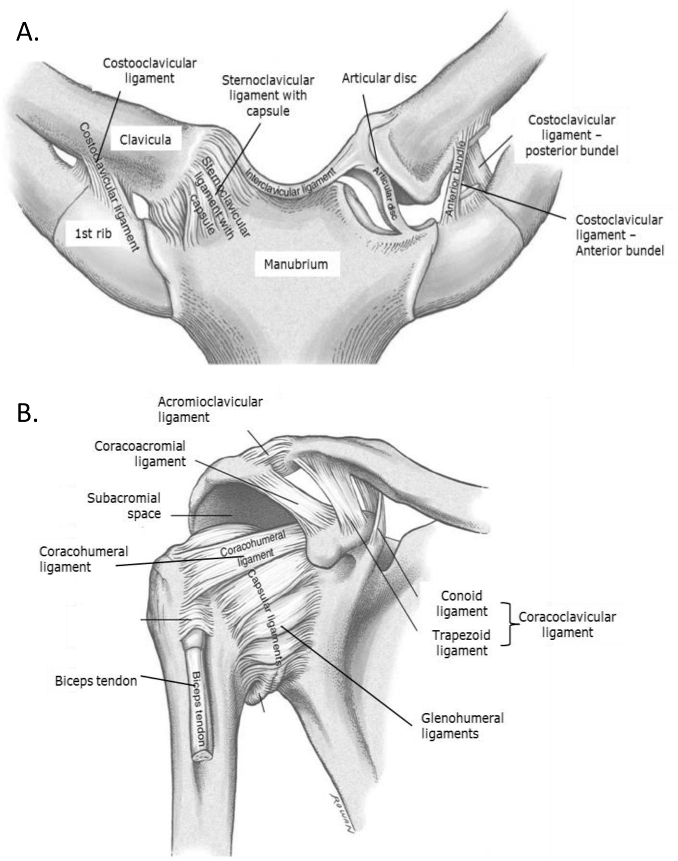
Of all joints in the human body, the shoulder complex has the largest degree of freedom in movement. This is due to the specific construction of its four different articulations, i.e. the glenohumeral, scapulothoracic, acromioclavicular and sternoclavicular joint (Figure 2).

The most proximal articulation within the shoulder complex is the *sternoclavicular joint*. This joint is the only bony connection between the whole upper limb and the axial skeleton, meaning it must be firmly attached but at the same time allow enough range of movement. A capsule, reinforced by sternoclavicular ligaments, the costoclavicular ligament and active muscles, i.e. sternocleidomastoideus, subclavius, sternohyoid and sternothyroid, add to the stability of the joint. The sternoclavicular joint is finally strengthened by an articular disc.[46]

The lateral end of the clavicula articulates with the acromion in the *acromioclavicular joint*, making the connection between clavicula and scapula.[46] Due to predominantly flat joint surfaces, the stability in this joint is ensured via a joint capsule and superior and inferior acromioclavicular ligaments.[47] Extrinsic stabilization of the acromioclavicular joint is provided by the strong coracoclavicular ligament,[48] which also functions as a transporter of movement from the scapula to the clavicula.

The scapula resting against the ribcage forms the *scapulothoracic joint*.[46] The scapula is stabilized on the thorax purely on a muscular basis, and is hence considered a pseudo-articulation. Among the 14 muscles surrounding and attaching to the scapula, upper trapezius, lower trapezius and serratus anterior are the greatest contributors to scapular stability on the thorax.[49-51] Such a stable scapulothoracic position is essential for the static stability of the glenohumeral joint, which is the most distal and most mobile link of the shoulder complex.

Figure 2. Anatomy of the shoulder complex



Anatomy of the sternoclavicular (A), acromioclavicular and glenohumeral (B) joint. With permission from Neumann D, Mosby, Elsevier[46]

At the *glenohumeral joint*, the surface of the humeral head is three to four times larger than the surface of the glenoid fossa of the scapula.[46] To further increase the depth of the fossa, the glenoid is surrounded by a labrum. A relatively thin capsule surrounds the articulation, which is reinforced by the superior, middle and inferior glenohumeral ligaments and the coracohumeral ligament to further provide passive stabilization. The stability of this joint can only be ensured via the rotator cuff muscles, i.e. supraspinatus, infraspinatus, teres minor and subscapularis, which provide an active stabilization at any glenohumeral position. It is known that the strength of this rotator cuff increases with 13 to 24% when the scapula is stabilized in a neutral position.[52,53] As such, an optimal scapulothoracic interaction is crucial for proper dynamic glenohumeral joint stabilization. Lastly, due to its course over the glenohumeral joint, the long head of the biceps brachii also contributes to the dynamic stability of the glenohumeral joint.[46]

A clinically very interesting but vulnerable area is the subacromial space, located between the coracoacromial arch (formed by the acromion and coracoacromial ligament) and the humeral head. This space contains the supraspinatus tendon, the subacromial bursa, and the long head of the biceps. Their specific location makes these structures highly vulnerable to damage or inflammation.

All joints of the shoulder complex are linked and work together to allow maximal upper limb movement. Moreover, in order to achieve complex coordinated actions of the multiple joints, muscles around the shoulder complex act mostly in "teams", instead of isolated work of a single muscle. This also means that weakness, pain or damage in one specific structure or muscle might disrupt the kinematic chain and decrease the effectiveness of the entire shoulder complex.

3.1.2. Kinematics

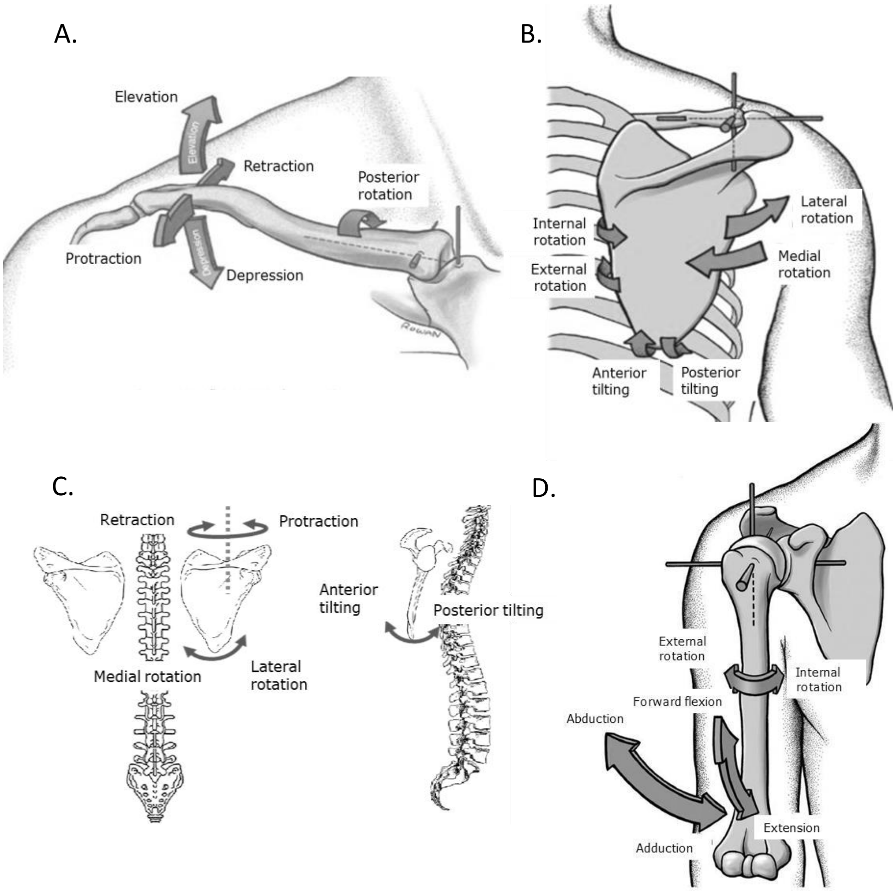
The clavicula rotates in the sternoclavicular joint in three directions or planes. During arm elevation, the clavicula rotates posterior, retracts and undergoes elevation (Figure 3A) to get the scapula in the optimal position on the thorax.[54-57] As such, the sternoclavicular joint provides the general path of the scapula through the movement of the clavicula.

The rotations in the acromioclavicular joint are more subtle, though very important to optimize and fine-tune the fit and movement between the scapula and the thorax.[54] The scapula tilts posterior, and rotates lateral and internal in the acromioclavicular joint during arm elevation (Figure 3B).[54,57,58]

The combination of movement in the sternoclavicular and acromioclavicular joint result in movements of the scapulothoracic joint. Scapulothoracic lateral rotation at full arm elevation is the combination of clavicular elevation in the sternoclavicular joint and lateral rotation of the scapula in the acromioclavicular joint.[54] Scapulothoracic posterior tilting is predominantly a tilting movement in the acromioclavicular joint, with only little contribution from retraction in the sternoclavicular joint.[54,59] Late in the elevation range, the clavicula also rotates posteriorly due to a tensioned coracoclavicular ligament,[46] which furthermore contributes to the posterior tilting of the scapula in the scapulothoracic joint.[57] No consensus is reached in literature on scapulothoracic protraction or retraction during arm elevation. The sternoclavicular retraction and acromioclavicular internal rotation are contrary motions, that have no consistent net change in the scapulothoracic joint (protraction or retraction) among individuals (Figure 3C).[55] Scapulothoracic protraction/retraction is furthermore minimal prior to 100 degrees of arm elevation.[54,60] This nomenclature for the scapulothoracic rotations is consistent with the terminology used by the International society of Biomechanics (ISB).

In the glenohumeral joint, abduction/adduction, forward flexion/extension, and internal/external rotation are the three degrees of freedom. During arm elevation, the humerus rotates externally (Figure 3D).[54]

Figure 3. Kinematics of the shoulder complex



Kinematics of the sternoclavicular (A), acromioclavicular (B), scapulothoracic (C) and glenohumeral (D) joint. With permission from Neumann D, Mosby, Elsevier[46]

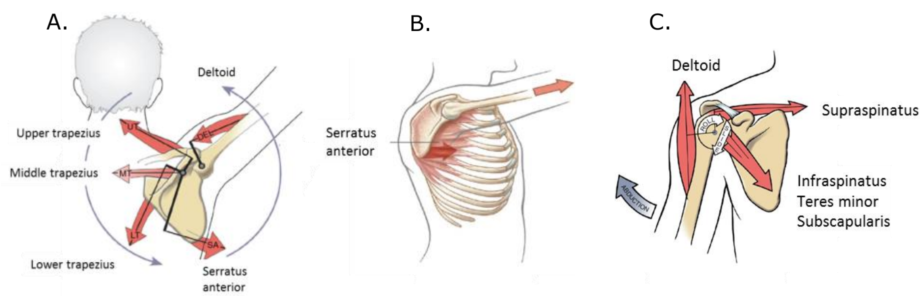
Angular positions of the clavicula and scapula at resting posture, with the arm at the side have also been described. In the sternoclavicular joint, retraction, elevation and very little posterior rotation is reported. In the acromioclavicular as well as scapulothoracic joint, the scapular resting posture is in protraction, lateral rotation and anterior tilt.[54]
Although an agreement on the direction of movement during arm elevation is reached for most joints of the shoulder complex, large variations in absolute degrees or in normal ranges of movement have been reported.

These variations are attributable to differences in measurement techniques, population samples, addition of external load, type of data processing, etc.

3.1.3. Muscles that elevate the arm

Optimal function of the shoulder complex is created by the balanced action between proximal stabilizers, i.e. muscles with a proximal origin on the axial skeleton and insertion on the scapula or clavicula, and distal movers, i.e. muscles originating on the scapula or clavicula and inserting on the humerus or more distally. Proximal stabilizers are serratus anterior and the trapezius muscle, distal movers include the deltoid and biceps brachii muscle.[46] Arm elevation (i.e. forward flexion and abduction) requires actions from muscles that elevate the humerus at the glenohumeral joint (i.e. deltoid, biceps brachii, supraspinatus), combined with activation of muscles that control the movements of the scapulothoracic joint (i.e. trapezius, serratus anterior) and muscles controlling the dynamic stability of the glenohumeral joint (i.e. rotator cuff). Indeed, to laterally rotate the scapula during arm elevation, the upper and lower trapezius work together in a force couple with the serratus anterior,[51,61-63] and the serratus anterior posteriorly tilts and externally rotates the scapula in the acromioclavicular joint during arm elevation (Figure 4A, 4B).[51,61,64,65] This in turn creates a stable base for the rotator cuff to stabilize the glenohumeral joint. To neutralize the strong protraction moment of the serratus anterior, middle trapezius and rhomboids are also active during arm elevation.[66]
The rotator cuff excels in its capacity to stabilize the glenohumeral joint.[67-72] Its distal attachment blends into the glenohumeral joint capsule before it attaches to the humerus. As such, this cuff is very rigid and produces forces

Figure 4. Muscle of the shoulder complex



Scapulothoracic force couple for lateral scapular rotation (A), serratus anterior ensuring posterior scapular tilting (B), and inferior directed force of infraspinatus, teres major and subscapularis during arm abduction or forward flexion (C). With permission from Neumann D, Mosby, Elsevier[46] not only to rotate the humeral head, but also to centralize the humeral head on the fossa[73,74] and to allow for correct kinematics of the glenohumeral joint.[75-78] The supraspinatus allows for a superior roll of the humeral head, and compresses the head in the fossa. Meanwhile, the subscapularis, teres minor and infraspinatus pull the humeral head downward and provide an inferiorly directed translation to counterbalance excessive superior translation by the deltoid. Teres minor and infraspinatus externally rotate the humerus, which is essential for full range arm elevation (Figure 4C).

It is important to notice that other intrinsic muscles (e.g. pectoralis minor), and extrinsic muscles (e.g. latissimus dorsi, pectoralis major), play important but not primary roles. Pectoralis minor for example assists serratus anterior in lower levels of elevation to protract the scapula.[79]

The activity of the main stabilizers of the shoulder complex depends not only on the production of force. Neuromuscular control in terms of a precise coordinated activity that occurs at the right moment and that creates the right amount of force is also crucial.[80,81]

3.2. What is known about shoulder kinematics after stroke?

Given that post-stroke impairments such as muscle weakness, spasticity, and/or loss of voluntary motor control adversely affect the normal position and movement of the shoulder complex, this will inadvertently impact on scapular kinematics and muscle control in IwS.

Niessen et al. (2008) have reported increased scapulothoracic lateral rotation at the hemiplegic side in IwS with shoulder pain compared to controls, during both active and passive abduction and forward flexion. When compared to IwS without shoulder pain, this increased scapulothoracic lateral rotation was only found during passive abduction.[37] In contrast, Hardwick and Lang (2011) found that those IwS with more shoulder pain had less scapulothoracic scapular lateral rotation during scapular plane elevation.[82] These authors also reported a significant decreased scapulothoracic lateral rotation in IwS without shoulder pain in comparison to controls during a person-assisted forward flexion task.[38] Finally, Robertson et al. (2012) showed less scapulothoracic protraction in IwS without shoulder pain compared to controls during various reaching tasks.[83]

Remarkably, none of the studies thus far has linked the alterations in movement of the shoulder complex to changes in muscle activation patterns. It is however known that in non-stroke persons with musculoskeletal shoulder pain or pathology, all muscles of the scapulothoracic force couple for lateral rotation show a delayed muscle onset at the affected shoulder.[81] Persons with multidirectional instability also show an altered recruitment pattern of the deltoid, major pectoral muscle, supraspinatus and infraspinatus, as well as biceps and triceps brachii during arm elevation.[84,85]

The ability to actively control movements and position the scapula is essential for optimal upper limb function. Compared to other joints, most pathologies or functional impairments around the shoulder do not originate from bony disorders, but rather from soft tissue disturbances. These exists in the form of inflexibility, e.g. shorted muscles or intrinsic muscle pathology, e.g. supraspinatus tendinopathy.[79] Accurate assessment of scapular movements, and key scapulothoracic and scapulohumeral musculature (m. trapezius, m. serratus anterior, rotator cuff) is crucial to gain a deeper understanding of pathologies and impairments of the shoulder in IwS. These insights are the foundation to design or optimize treatment strategies for enhancing shoulder and upper limb function in IwS.

4. Objectives and thesis outline of the doctoral project

4.1. Objectives

The scope of this doctoral project was to contribute to the understanding of impaired scapular control in IwS. The inconsistent results in kinematics post-stroke, together with the lack of combined assessment of scapular movement patterns and muscle activation patterns, as well as the absence of a clinical scapular assessment in IwS, have led to the formulation of different objectives:

- To develop both an instrumented and a clinical measurement method to assess scapular position, movement patterns, and muscle activation patterns in IwS and to evaluate the psychometric properties.
- To study characteristics of impaired scapular control in IwS to gain insights into deficits in scapular positioning, movement and muscle activation patterns.
- To investigate the association between objective and clinical measures of scapular position and movement.

The following research questions were addressed:

1. How can we reliably assess the scapulothoracic joint and its function in IwS in a laboratory and clinical setting?
2. Can instrumented and clinical measures of scapular control identify deficits in scapular positioning, movement and muscle activation patterns in IwS?
3. What is the relationship between three-dimensional (3D) and clinical measures of scapular position and movement control?

These objectives and research questions were addressed in several studies, which are described in detail in each of the chapters of this doctoral thesis. A schematic overview of all studies is given in Figure 4. Table 1 additionally provides an overview of the study design, participants and outcome parameters per study.

4.2. Thesis outline

Chapter 1 is a systematic review of 3D movement patterns and muscle activity of the scapulothoracic joint in healthy persons, persons with primary shoulder pain and IwS. Little was found on scapulothoracic kinematics post-stroke, and no results were available on scapular muscle activity post-stroke. The results of this systematic search formed the basis for the development of a protocol for 3D movement analysis in IwS, which is presented in **Chapter 2**. Moreover, its feasibility and reliability in IwS and healthy controls is outlined in this second chapter. Since our systematic search revealed that there was no literature on scapular muscle timing post-stroke, a first exploration of muscular timing characteristics in IwS was performed and is described in **Chapter 3**. This chapter outlines the alterations found in scapular muscle timing characteristics between IwS with and without PSSP and healthy controls. **Chapter 4** additionally gives the results of the comparison between IwS and healthy controls regarding 3D scapular kinematics and associated muscle timing strategies. Given that 3D movement analysis is furthermore difficult to use in clinical stroke practice, scapulohumeral control was also assessed by means of clinical scapular measures in IwS. **Chapter 5** introduces a clinical scapular measurement protocol and reports the reliability results of the protocol, along with the differences between controls and IwS, and between IwS with various degrees of arm function. **Chapter 6** describes the association between 3D scapulothoracic kinematics and the outcomes of the different tests of the clinical scapular protocol.

This doctoral thesis is concluded by a general discussion, in which main findings on differences between IwS and controls are further discussed and interpreted. The assessment methods used in the several studies are critically reviewed. Lastly, suggestions are made regarding the integration of these results in the upper limb examination and treatment planning of IwS.

Figure 4. Overview of the different studies of this doctoral project

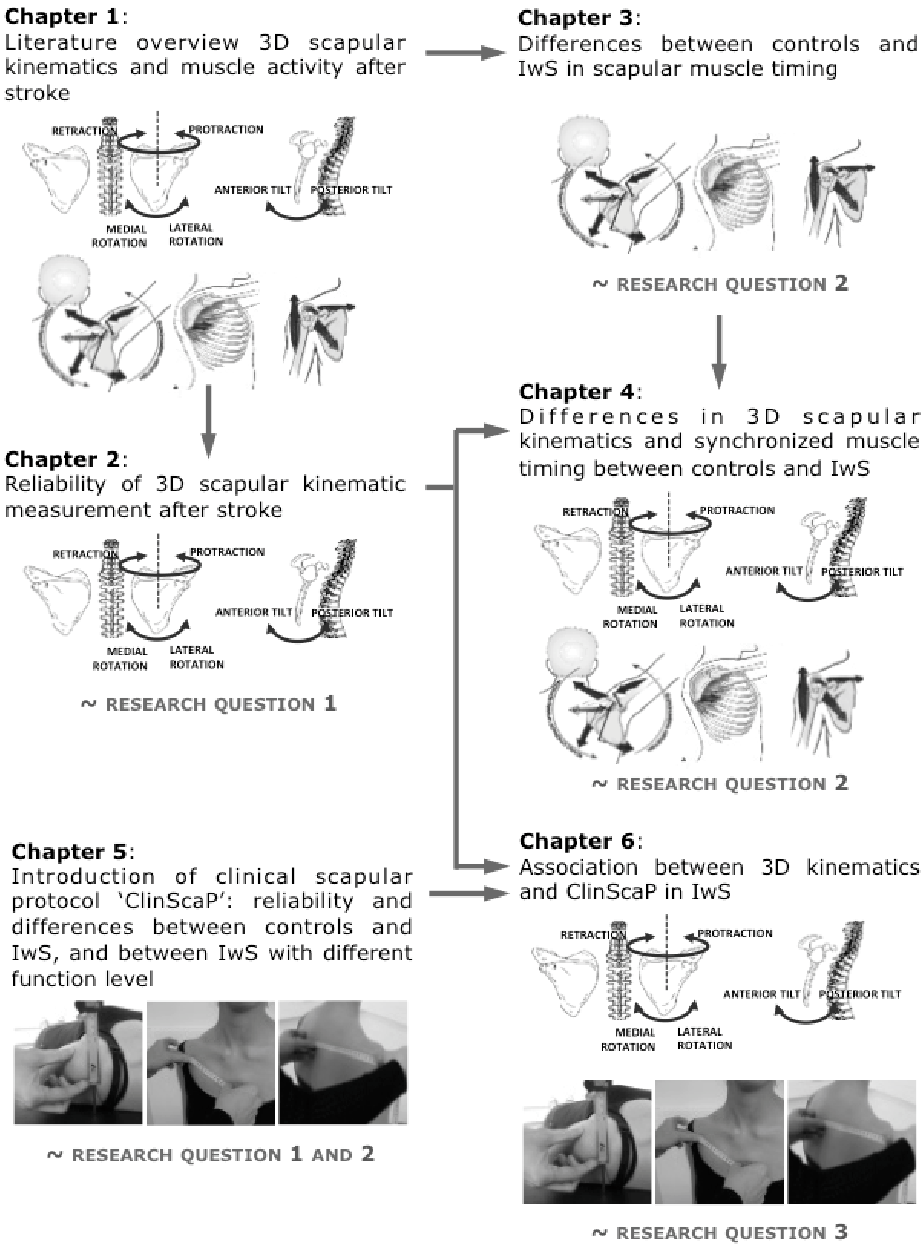


Table 1. Overview of study design and participant characteristics of each of the studies performed within the doctoral thesis

	Study design	Participants							Outcome parameter
		Type	Age	wks PS	N	Inclusion criteria	Exclusion criteria	FM	
Chapter 1	Systematic review	-			-	-	-	-	3D ST & muscle timing
Chapter 2	Feasibility - Reliability	IwS	18-69	5-39	10	· 1-12 months PS · First time stroke, (sub)cortical lesion · No shoulder complaints prior to stroke · Ability to perform 60° of HT elevation · Able to understand instructions	· Reduced communicative or cognitive abilities	35 - 64	3D ST
		HC	18-70		10		· Current shoulder dysfunctions or treatment		
Chapter 3	Cross-sectional	IwS Painful shoulder	62±21	22±7	10	· At least 6 weeks PS · First time stroke, (sub)subcortical lesion · Score of ≥ 30 on the FM · Ability to perform 45° of HT elevation · Anterolateral shoulder pain during ADL · Positive Neer impingement test	· BMI < 28 · Inability to understand instructions · History of shoulder and/or neck pain or discomfort in the last 6 months prior to stroke · Event of shoulder dislocation, fracture or surgery during life time · Other systemic and/or neurologic diseases	51 ± 7	ST muscle timing
		IwS Pain free shoulder	59±11	24±18	20	· At least 6 weeks PS · First time stroke, (sub)cortical lesion · Score of ≥ 30 on the FM · Ability to perform 45° of HT elevation		53 ± 7	
		HC	61±11		16	· No self-reported shoulder pain			
Chapter 4*,¥	Cross-sectional	IwS Pain free shoulder	52±13	16±10	18	Idem Chapter 3	Idem Chapter 3	45 - 63	3D ST & muscle timing
		HC	52±13		15	Idem Chapter 3			
Chapter 5¥	Reliability Cross-sectional	IwS Pain free shoulder	69±9 62±14	18±7 25±29	57	· First time stroke, (sub)cortical lesion · Sit independently with low back support · Ability to perform 45° active and 90° passive HT elevation	· Inability to understand instructions · Event of shoulder dislocation, fracture or surgery during life time · Other systemic and/or neurologic diseases	44 ± 16	Clinical ST measures
		HC	52±13		15	Idem chapter 3			
Chapter 6*	Cross-sectional	IwS Pain free shoulder	52±13	16±10	18	Idem Chapter 3	Idem Chapter 3	45 - 63	3D & clinical ST measures

IwS: individuals with stroke; wks PS: weeks post-stroke; HC: healthy controls; 3D: three-dimensional kinematics; ST: scapulothoracic; HT: humerothoracic; ADL: activities of daily living; PS: post-stroke; BMI: Body Mass Index; FM: Fugl-Meyer upper limb motor part (max 66); *: IwS were included in Chapter 4 and 6; ¥: HC were included in Chapter 4 and 5

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Chapter 1

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A systematic review of 3D scapular kinematics and muscle activity during elevation in stroke subjects and controls

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1. Abstract

Through the onset of post-stroke motor disorders, the normal scapular function is compromised. As a result, shoulder pain and associated upper limb dysfunctions frequently arise after stroke.

This review aimed to provide a systematic overview of available literature on scapular function, i.e. scapular three-dimensional (3D) kinematics and muscle activity during elevation, in healthy persons, persons with primary shoulder disorders and post-stroke patients. 3D scapular kinematics have been widely reported in healthy persons and persons with primary shoulder disorders, whereby a general pattern of lateral rotation and posterior tilt during elevation has been agreed upon. Results on scapular internal/external rotation are inconsistent. In a post-stroke population, 3D scapular kinematics are less frequently reported. Scapular muscle activity has thus far been studied to very limited extend and firm conclusions could not be drawn.

Although 3D scapular kinematics and muscle activity registrations are being increasingly used, some general methodological aspects should be considered. While the International Society of Biomechanics already proposed recommendations on the definition of upper limb joint coordinate systems and rotation sequences, proper result comparison necessitates further guidelines on other methodological aspects, i.e. data collection, processing, analyzing, and reporting.

2. Introduction

Shoulder pain and associated upper limb dysfunctions are common complications after stroke.[1-3] Depending on the time since stroke, the study design, but also the definition of shoulder pain, the reported incidence of post-stroke shoulder pain (PSSP) varies between 16% and 84%.[4-5]

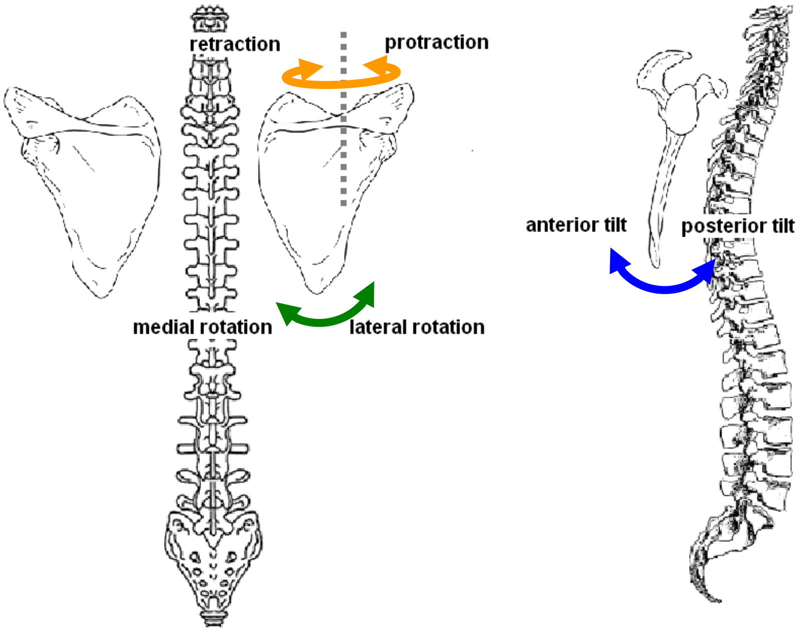
This shoulder pain, together with the upper limb dysfunctions, result in reduced self-care and functional autonomy[6] and have a negative influence on rehabilitation outcome.[3] In the long run, failure to substantially recover arm function and reduce shoulder pain will lead to a decreased quality of life.[5,7,8] Restoration of a pain free upper limb function should therefore be one of the main objectives in stroke rehabilitation.[9,10]

Adequate treatment requires a good understanding of the underlying mechanisms of PSSP, based on an accurate, reliable and valid assessment. Given that frequent post-stroke impairments such as paralysis, spasticity, or loss of motor control adversely affect the normal position and movement of the shoulder complex,[11] assessment of the scapulothoracic movement pattern is crucial. Scapular kinematics with respect to the thorax are described in 3 rotations during humerothoracic elevation: protraction/retraction, medial/lateral rotation and posterior/anterior tilt (Figure 1). Two-dimensional movement analysis and clinical scales do not fully capture the three-dimensional (3D) nature of scapular movement. As such, a 3D movement analysis seems most appropriate to assess scapular movement.[12,13]

Given that movement of the scapula is induced by well-coordinated scapular muscle activity,[14-18] knowledge of the muscle activity, mainly on recruitment patterns and muscle latencies, is particularly important.[19] The objective assessment of this activity, by means of electromyographic measurements (EMG) will add to our understanding of the effects of altered muscle activity on scapular movement.[20,21]

In other patient groups with shoulder disorders, alterations in 3D scapular kinematics and muscle activity have been put forward as important factors in the development of shoulder pain.[22,23] Since humerothoracic elevation is a sensitive task to provide valuable information on these scapular changes, knowledge of 3D scapular kinematics and associated muscle activity during this elevation in healthy persons and in persons with primary shoulder disorders is of utmost importance for a sound interpretation of results found in persons with PSSP. Despite existing literature on 3D scapular kinematics and muscle activity during humerothoracic elevation, a systematic review, bundling existing knowledge to attain a general consensus has thus far been lacking in literature. Therefore, the objective for this review is to describe scapular kinematics and muscle activity during humerothoracic elevation in healthy persons, in order to get a clear view on scapular kinematics and muscle activity during this well-

Figure 1. Scapular rotations in the frontal, sagittal and transverse plane



defined movement. Additionally, studies on scapular kinematics during that same analytical movement in persons with shoulder pain, and post stroke patients, are included, to achieve a straightforward comparison between the different groups (healthy, primary shoulder pain, stroke). This will give the opportunity to determine causes of pain due to alterations in scapular kinematics or due to changed muscle activity.

3. Literature search

Papers were selected based on a systematic search using following electronic databases: PubMed, Cochrane Library and Web of Science. Keywords for this search were shoulder (upper extremity, shoulder complex, shoulder girdle, scapula), muscle activity (muscle activity, EMG) and movement patterns (kinematics, movement patterns). To specify results, these terms were combined with a search for stroke (stroke, hemiplegia) and shoulder disorder (impingement, instability, frozen shoulder). Only full text papers published between 2000 and 2010 were retained. Measurement methods, analyzed tasks and outcomes were evaluated based on the abstracts. Papers were included in case they described (1) 3D scapular kinematics (joint angles) and/or scapular muscle activity (recruitment patterns or latencies) (2) during active humerothoracic elevation, (3) in healthy persons, persons with primary shoulder disorders or stroke patients. Papers were excluded if they were not published in English, or in case of cadaver studies. Studies inspecting only spatiotemporal movement characteristics (e.g. movement velocity, endpoint error, trajectory length) or 3D scapular kinematics during reaching or grasping tasks were also excluded. Reference lists of the selected papers were screened to ensure no paper was missed. Title and abstract of all selected papers were subsequently checked by a second independent researcher, and planned to be discussed in case of disagreement. However, no disagreement raised during the selection process.

Database search identified 166 articles, and subsequent screening of the reference lists identified another 19 articles. Based on the proposed inclusion and exclusion criteria, 30 papers describing 3D scapular kinematics during humerothoracic elevation were selected: 29 papers reported 3D scapular kinematics with respect to the thorax, one paper reported 3D scapular kinematics with respect to the acromioclavicular joint (Table 1 and 2).[24] Furthermore, six papers were selected on scapular muscle activity during elevation (Table 3 and 4).

4. Assessment of quality

All included studies were additionally assessed by two independent researchers for methodological quality. Criteria for assessment of methodological quality were adapted from different tools or checklists (CASP tool for qualitative and case control studies, STROBE Statement for case control studies), as no validated scoring tool with regard to our research objectives was found. The agreement between both researchers on the individual criteria and the total scores was evaluated with Kappa statistics (κ), and the intraclass correlation coefficient (ICC), respectively.[25,26] Seven different criteria were selected to assess the most relevant methodological issues concerning our research scope. The scoring tool was expected to score whether the following criteria were described in sufficient detail to permit replication: (1) inclusion/exclusion criteria, (2) manner of participant recruitment, (3) definition of scapular rotations or EMG data processing, (4) the methods of task performance, including sensor and electrode placement, (5) the reliability of the applied methodology, (6) the statistical power (justification of sample size) and (7) the statistical procedure. For papers on scapular kinematics, absolute agreement between both researchers (κ 1.00) was found for all criteria, except ‘inclusion-exclusion’ (κ 0.46) and ‘manner of recruitment’ (κ 0.87); the ICC for the total score was also very high (0.96). For the papers on scapular muscle activity, only the ‘inclusion-exclusion’ criteria did not show absolute agreement between both researchers (κ 0.67); the ICC for the total score was very high (0.93).

Table 1. Description of the study population for the selected papers on scapular kinematics

	Study	Subjects (n)	Age*	Gender
A. Healthy population				
A1	Ludewig et al, 2009	healthy (n=12)	22-41	M/F
	McClure et al, 2001	healthy (n=8)	27-37	M/F
	Teece et al, 2008	healthy (n=30)	18-40	M/F
A2	Crosbie et al, 2008	healthy (n=32)	19-74	F
	Ebaugh et al, 2005	healthy (n=17)	18-30	M/F
	Fayad et al, 2006	healthy (n=30)	24.7 (4.7)	M/F
	Finley & Lee, 2003	healthy (n=16)	21.6 (3.9)	M/F
	Price et al, 2000	healthy (n=10)	17-78	M/F
	Yano et al, 2010	healthy (n=21)	18-32	M/F
A3	Borstad & Ludewig, 2005	healthy (n=50) [a]	25.8; 28.6	M/F
	Dayanidhi et al, 2005	healthy (n=29) [b]	6.7 (1.5); 28.8 (4.3)	M/F
	Myers et al, 2005	healthy (n=42) [c]	21.6 (1.8); 24.7 (4)	M
A4	Borstad et al, 2009	healthy (n=28)	25.2 (4.3)	M/F
	Ebaugh et al, 2006a	healthy (n=20)	22.0 (3.4)	M/F
	Ebaugh et al, 2006b	healthy (n=20)	18-30	M/F
	Tsai et al, 2003	healthy (n=30)	28.0 (6.0)	M/F
B. Primary Shoulder Disorders				
B1	Borstad & Ludewig, 2002	controls (n=26) [d] impingement (n=26) [d]	20-71	M
	Hébert et al, 2002	controls (n=10) impingement (n=41)	30-60	M/F
	Laudner et al, 2006	controls (n=11) [e] impingement (n=11) [e]	18-30	M
	Ludewig & Cook, 2000	controls (n=26) [d] impingement (n=26) [d]	20-71	M
	McClure et al, 2006	controls (n=45) impingement (n=45)	24-74	M/F
	Fayad et al, 2008	controls (n=16) [f] frozen shoulder (n=16)	41-86	M/F
	Ostgon & Ludewig, 2007	controls (n=29) multidirectional instability (n=29)	15-45	M/F
B2	Lin et al, 2006	anterior shoulder tightness (n=6) [g] posterior shoulder tightness (n=6) [g]	40-69	-
	Yang et al, 2009	anterior shoulder tightness (n=12) [g] posterior shoulder tightness (n=12) [g]	34-72	M
B3	Matias & Pascoal, 2006	glenohumeral instability (n=6)	37.0 (8.5)	M/F
B4	McClure et al, 2004	impingement (n=39)	26-78	M/F
B5	Yang et al, 2008	frozen shoulder (n=34)	41-65	M/F
C. Stroke patients				
	Meskers et al, 2005	controls (n=10) stroke (n=10) [h]	60.8 (12.4) 53.4 (10.3)	M/F
	Niessen et al, 2008	controls (n=10) stroke (n=27) [i]	49.3 (7.2) 59.3 (11.1); 57 (9.5)	M/F

* either age range or mean (standard deviation) is reported; M: male; F: female; PSSP: post stroke shoulder pain; -: not reported; [a] short or long pectoralis minor resting length; [b] children and adults; [c] throwers and controls; [d] overhead workers; [e] throwers; [f] controls have osteoarthritis; [g] tightness due to impingement, frozen shoulder or rotator cuff tears; [h] ipsilateral shoulder is investigated, [i] PSSP: n=13, without PSSP: n=14.

A1: studies on scapular kinematics, A2: additionally comparing both sides and several test conditions or A3: between different populations. A4: studies reporting the effect of exercise protocol on scapular kinematics.

B1: studies comparing to healthy controls or non-affected side, B2: studies comparing shoulders with anterior and posterior tightness, B3: comparing to estimation of normality, B4: reporting effect of intervention, B5: use of kinematics to predict the clinical course of frozen shoulder syndrome.

Table 2. Task and measurement methods for the selected papers on scapular kinematics

Study	Task	Motion capture system	Scapular kinematics measuring technique		Euler decomposition	Scapular landmarks	HT elevation
A. HEALTHY POPULATION							
Ludewig et al, 2009	A, F, Sc	Electromagnetic	Receiver on scapular spine pins	Bone-fixed	Pro/lat/tilt	TS, AC, AI	Full range*
McClure et al, 2001	Sc, A	Electromagnetic	Receiver on scapular spine pins	Bone-fixed	Pro/lat/tilt	TS, AC, AI	Full range
Teece et al, 2008	Sc	Electromagnetic	Acromial receiver	Skin-fixed	Pro/lat/tilt	TS, AC, AI	90°
Crosbie et al, 2008	A, F, Sc	Electromagnetic	Acromial receiver	Skin-fixed	Pro/lat/tilt	TS, AA, AI	Full range
Ebaugh et al, 2005	Sc	Electromagnetic	Scapular spine-acromial receiver	Skin-fixed	Pro/lat/tilt	TS, AC, AI	Full range
Fayad et al, 2006	A, S	Electromagnetic	Acromial receiver	Skin-fixed	Pro/lat/tilt	TS, AA, AI	> 120° *
Finley & Lee, 2003	Sc	Electromagnetic	Acromial receiver	Skin-fixed	-	-	Full range*
Price et al, 2000	A	Electromagnetic	Scapula locator	Manually positioned	-	TS, AC, AI	50°
Yano et al, 2010	Sc	Opto-electronic	Markers on different scapular points	Skin-fixed	-	TS, AA, AI	Full range
Borstad & Ludewig, 2005	A, F, Sc	Electromagnetic	Acromial receiver	Skin-fixed	-	TS, AC, AI	Full range*
Dayanidhi et al, 2005	Sc	Electromagnetic	Acromial receiver	Skin-fixed	Pro/lat/tilt	TS, AC, AI	125°
Myers et al, 2005	Sc	Electromagnetic	Acromial receiver	Skin-fixed	Pro/lat/tilt	TS, AA, AI	Full range*
Borstad et al, 2009	Sc	Electromagnetic	Acromial receiver	Skin-fixed	Pro/lat/tilt	TS, AA, AI	120°
Ebaugh et al, 2006a	Sc	Electromagnetic	Scapular spine-acromial receiver	Skin-fixed	Pro/lat/tilt	TS, AC, AI	Full range
Ebaugh et al, 2006b	Sc	Electromagnetic	Scapular spine-acromial receiver	Skin-fixed	Pro/lat/tilt	TS, AC, AI	Full range*
Tsai et al, 2003	Sc	Electromagnetic	Scapular spine-acromial receiver	Skin-fixed	Pro/lat/tilt	TS, AC, AI	Full range

Continued

Table 2. Continued

B. Primary Shoulder Disorders							
Borstad & Ludewig, 2002	Sc	Electromagnetic	Acromial receiver	Skin-fixed	Pro/lat/tilt	TS, AC, AI	Full range*
Hébert et al, 2002	A, F	-	-	-	Lat/tilt/pro	-	110°
Laudner et al, 2006	Sc	Electromagnetic	Acromial receiver	Skin-fixed	Pro/lat/tilt	TS, AA, AI	Full range*
Ludewig & Cook, 2000	Sc	Electromagnetic	Acromial receiver	Skin-fixed	Pro/lat/tilt	TS, AC, AI	Full range*
McClure et al, 2006	F, Sc	Electromagnetic	Scapular receiver	Skin-fixed	Pro/lat/tilt	TS, AC, AI	Full range
Fayad et al, 2008	A, S	Electromagnetic	Acromial receiver	Skin-fixed	Pro/lat/tilt	TS, AA, AI	30° - 60°
Ostgon & Ludewig, 2007	A, Sc	Electromagnetic	Acromial receiver	Skin-fixed	Pro/lat/tilt	TS, AC, AI	Full range*
Lin et al, 2006	A, F, Sc	Electromagnetic	Acromial receiver	Skin-fixed	Pro/lat/tilt	TS, AC, AI	<120°
Yang et al, 2009	Sc	Electromagnetic	Acromial receiver	Skin-fixed	Pro/lat/tilt	TS, AC, AI	90°
Matias & Pascoal, 2006	F	Electromagnetic	Scapular locator	Manually positioned	-	TS, AA, AI	140°
McClure et al, 2004	F, Sc	Electromagnetic	Scapular receiver	Skin-fixed	Pro/lat/tilt	TS, AC, AI	Full range
Yang et al, 2008	Sc	Electromagnetic	Acromial receiver	Skin-fixed	Pro/lat/tilt	TS, AC, AI	Full range
C. Stroke patients							
Meskers et al, 2005	A, F	Electromagnetic	Scapula locator	Manually positioned	Pro/lat/tilt	TS, AA, AI	130° *
Niessen et al, 2008	A, F	Electromagnetic	Acromial receiver	Skin-fixed	Pro/lat/tilt	-	120° *

A: Abduction (frontal plane elevation); F: Forward flexion (sagittal plane elevation); Sc: Scaption (Scapular plane elevation); Pro: Scapular protraction; Lat: Scapular lateral rotation; Tilt: Scapular tilt; TS: Trigonum spina scapulae; AA: Angulus acromialis scapulae; AI: Angulus inferior scapulae; AC: Posterior acromioclavicular joint; -: Not reported; HT: Humerothoracic; *: Scapular rotation analyzed up to 120° of humerothoracic elevation or less; >: Higher than; <: Lower than.

Table 3. Description of the study population for the selected papers on scapular muscle activity

Study	Subjects (n)	Age*	Gender
A. HEALTHY POPULATION			
Cools et al, 2002	healthy (n=30)	18-26	M/F
B. PRIMARY SHOULDER DISORDERS			
Cools et al, 2003	controls (n=30) [a]	16-36	M/F
	impingement (n=39) [a]		
Moraes et al, 2008	controls (n=10)	30-38	M/F
	impingement (n=10)		
Barden et al, 2005	controls (n=11)	29.6 (9.7)	M
	multidirectional instability (n=7)	25.0 (9.5)	
Illyés & Kiss, 2006	controls (n=15)	F 24.6 (6.1) ; M 28.1 (5.1)	M/F
	multidirectional instability (n=15)	F 24.5 (4.6) ; M 19.2 (3.1)	
Morris et al, 2004	controls (n=7)	19-35	-
	multidirectional instability (n=6)		
	multidirectional laxity (n=4)		

* either age range or mean (standard deviation) is reported
M: male; F: female; -: not reported
[a] overhead athletes

Table 4. Task and measurement methods for the selected papers on scapular muscle activity in healthy persons and persons with primary shoulder disorders

Study	Task	M. Trapezius pars descendens	M. Trapezius pars transversa	M. Trapezius pars ascendens	M. Deltoideus pars clavicularis	M. Deltoideus pars acromialis	M. Deltoideus pars spinalis	M. Serr Ant	M. Latt Dorsi	M. Pect Major	Rotator Cuff M. Ssp	Rotator Cuff M. Isp	Rotator Cuff M. Ssc	M. Bic Brachii	M. Tric Brachii
A. Healthy population															
Cools et al, 2002	A^1	O	O	O		O									
B. Primary Shoulder Disorders															
Cools et al, 2003	A^1	O	O	O		O									
Moraes et al, 2008	Sc	O	O	O				O							
Barden et al, 2005	A^2, F^2				ø	ø	ø		ø	ø	æ	æ			
Illyés and Kiss, 2006	Sc			X	X	X	X	X	X	X	X	X		X	X
Morris et al, 2004	^{A2}, F^2				æ	æ	æ				æ	æ	æ		

A: Abduction (frontal plane elevation); F: Forward flexion (sagittal plane elevation); Sc: Scaption (Scapular plane elevation);

X: Monopolar surface electrode; O: Bipolar surface electrode; ø: Double differential surface electrode; æ: Intramuscular fine-wire electrode;

M.: Musculus; Ssp: Supraspinatus; Isp: Infraspinatus; Ssc: Subscapularis; Serr Ant: Serratus anterior; Latt Dorsi: Lattisimus Dorsi; Pect Major: Pectoralis Major; Bic Brachii: Biceps Brachii; Tric Brachii: Triceps Brachii

[1]reaction on sudden falling of the arm, [2]on an isokinetic dynamometer

Information on methodological quality of the selected papers can be found in Table 5 and 6.
The quality and credibility of the study results were guaranteed, as in all selected papers the results were related to the research question and the purpose of the current study. All papers also used informative and appropriate graphics to present their results.
All papers were retained for inclusion despite methodological quality, in order to provide an all-inclusive overview of available literature on scapular kinematics and muscle activity.

5. Results

5.1 Methodological aspects

5.1.1 Scapular kinematics

Studies described 3D scapular kinematics during elevation in *healthy persons* (Table 1 - A1), with additional reporting on the comparison between the dominant and non-dominant side, between different test conditions (Table 1 - A2) or between different populations (Table 1 - A3). Four studies described the effect of an exercise protocol on 3D scapular kinematics (Table 1 - A4).
Twelve studies reported on 3D scapular kinematics in *persons with primary shoulder disorders* (Table 1 and 2). Seven studies compared between healthy persons and persons with primary shoulder disorders (Table 1 - B1) of which two papers additionally compared the affected and the non-affected side.[22,27] Persons with anterior shoulder tightness were compared to persons with posterior shoulder tightness in two papers (Table 1 - B2). One paper compared scapular position between disordered shoulders and an estimation of normality (Table 1 - B3), and one paper reported on the effect of intervention (Table 1 - B4). Finally, one paper used 3D scapular kinematics to predict the clinical course of frozen shoulder syndrome (Table 1 - B5).
Two papers were selected comparing 3D scapular kinematics of *stroke patients* to healthy persons (Table 1 and 2).

5.1.2 Scapular muscle activity

All but one manuscript, wherein scapular muscle activity in healthy persons is studied, investigate scapular muscle activity in persons with primary shoulder disorders. All these papers reported on muscle activity during elevation by comparing persons with primary shoulder disorders and healthy controls. One paper additionally assessed scapular muscle activity between the affected and non-affected side (Table 3 and 4).[28]

Table 5. Methodological quality of the selected papers on scapular kinematics

Study	Inclusion-exclusion	Manner of recruitment	Methods for task performance	Definition of scapular rotations	Reliability of methodology	Justification for sample size	Statistical procedure	Final score (max. 14)
A. Healthy population								
Ludewig et al, 2009	1	0	2	2	2	0	2	9
McClure et al, 2001	1	0	2	2	0	0	0	5
Teece et al, 2008	1	0	2	2	2	0	2	9
Crosbie et al, 2008	1	2	2	2	2	0	2	11
Ebaugh et al, 2005	2	0	2	2	2	0	2	10
Fayad et al, 2006	1	0	2	2	0	0	2	7
Finley & Lee, 2003	1	0	2	2	2	0	2	9
Price et al, 2000	1	2	1	2	0	0	2	8
Yano et al, 2010	1	0	2	2	2	0	1	8
Borstad & Ludewig, 2005	2	2	2	2	2	2	2	14
Dayanidhi et al, 2005	2	2	2	2	0	0	2	10
Myers et al, 2005	2	0	2	2	2	0	1	11
Borstad et al, 2009	1	0	2	2	0	2	2	9
Ebaugh et al, 2006a	2	0	2	2	2	0	2	10
Ebaugh et al, 2006b	2	0	2	2	2	0	2	10
Tsai et al, 2003	1	0	2	2	2	2	2	11
B. Primary Shoulder Disorders								
Borstad & Ludewig, 2002	2	2	2	2	2	0	2	12
Hébert et al, 2002	2	2	1	1	0	0	2	8
Laudner et al, 2006	2	2	2	2	2	0	2	12
Ludewig & Cook, 2000	2	2	2	2	2	2	2	14
McClure et al, 2006	2	2	2	2	2	0	2	12
Fayad et al, 2008	1	0	1	2	2	0	2	8
Ostgon & Ludewig, 2007	2	2	2	2	2	2	2	14
Lin et al, 2006	1	0	2	2	2	0	2	9
Yang et al, 2009	2	2	2	2	2	2	2	14
Matias & Pascoal, 2006	1	0	1	0	0	0	2	4
McClure et al, 2004	2	2	2	2	2	0	2	12
Yang et al, 2008	2	2	2	2	2	0	2	12
C. Stroke patients								
Meskers et al, 2005	1	2	2	2	0	2	2	11
Niessen et al, 2008	1	2	2	2	0	2	2	11

2 : Good description; 1 : Moderate description; 0 : No description

Table 6. Methodological quality of the selected papers on scapular muscle activity

Study	Inclusion-exclusion	Manner of recruitment	Methods for task performance	Description of signal processing	Reliability of methodology	Justification for sample size	Statistical procedures	Final score (max. 14)
A. Healthy population								
Cools et al, 2002	2	0	2	2	2	2	2	12
B. Primary Shoulder Disorders								
Cools et al, 2003	2	0	2	2	2	2	2	12
Moraes et al, 2008	2	0	2	2	0	0	2	8
Barden et al, 2005	2	0	2	2	0	0	2	8
Illyés and Kiss, 2006	2	2	2	2	0	0	1	9
Morris et al, 2004	1	0	2	2	0	0	2	7

2 : Good description; 1 : Moderate description; 0 : No description

5.2 Study results

5.2.1 3D scapular kinematics

During humerothoracic elevation in the frontal (abduction), sagittal (forward flexion) or scapular plane (scaption), a general pattern of increased scapular lateral rotation (range 25°- 47° at 120° of elevation) and posterior tilt (range 3°-22° at 120° of elevation) is reported in *healthy persons*. However, Ebaugh et al. (2005, 2006a, 2006b) described an increased posterior tilt only until 60° to 90° of elevation, changing into increased anterior tilt afterwards.[29-31] Results on protraction/retraction were inconsistent. While some reported an increase in retraction (range 2°- 6° at 120° of elevation) during active elevation,[29-36] others found an increase in protraction (range 8°-35° at 120° of elevation).[24,37-41] Fayad et al. (2006) specifically reported an increase in protraction up to 90° of sagittal plane elevation (forward flexion), changing in an increased retraction until 120°.[42] This pattern was almost inversed during frontal plane elevation (abduction). Results for the comparison between dominant and non-dominant side, different test conditions, and different populations and results on the effect of an exercise protocol, are given in Table 7.

In *persons with primary shoulder disorders*, the same general pattern of scapular lateral rotation and posterior tilt was reported, although three papers did report scapular anterior tilting in persons with shoulder disorders during elevation.[23,43,44] Results on protraction/retraction during elevation were less consistent, whereby both a pattern of retraction[45-47] and protraction[22,23,43,44,48] was reported.

When comparing the affected shoulder of persons with primary shoulder disorders to the non-affected arm or to healthy controls, several significant results were found. A reduced lateral rotation in persons with impingement or multidirectional instability was reported.[23,44] In contrast, McClure et al. (2006) found an increased lateral rotation in persons with impingement.[47] A similar pattern of increased lateral rotation has also been reported for persons with frozen shoulder.[22] Two papers reported an increased posterior tilt in persons with impingement.[47,48] In persons with multidirectional instability, a decrease in anterior tilt was described.[44] Persons with impingement or multidirectional instability showed an increased protraction,[23,43,44] while frozen shoulders resulted in less protraction.[22]

In *stroke patients*, arm elevation at the non-hemiplegic side resulted in the general pattern of lateral rotation, posterior tilt and protraction.[49] Results for the hemiplegic side also showed a pattern of lateral rotation, though the pattern of tilting and protraction/retraction left unspoken.[50] Details on the differences between controls and stroke patients are summarized in Table 8.

Table 7. Papers on scapular kinematics in healthy persons: results

Study		Significant results
COMPARISON BETWEEN DOMINANT AND NON DOMINANT SIDE		
Crosbie et al, 2008		Increased LAT on non-dominant side during unilateral and bilateral elevation (±7°)
Price et al, 2000		-
Yano et al, 2010		-
COMPARISON BETWEEN DIFFERENT CONDITIONS		
Fayad et al, 2006	Comparison between static and dynamic humerothoracic elevation	Decrease in LAT in static elevation
	Comparison between slow and fast velocity of humerothoracic elevation	-
Ebaugh et al, 2005	Comparison between passive and active humerothoracic elevation	Increase in LAT (20.6°) and RETR (5.7°) in active elevation
Price et al, 2000	Comparison between passive and active humerothoracic elevation	-
Finley & Lee, 2003	Comparison between slouched and upright sitting position	Decreased P-TILT, decreased RETR in slouched position
Crosbie et al, 2008	Comparison between unilateral and bilateral humerothoracic elevation	Increased RETR in unilateral elevation (4° to 9°, dependent on plane of elevation)
COMPARISON BETWEEN DIFFERENT POPULATIONS		
Borstad & Ludewig, 2005	Comparison between short and long pectoralis minor resting length	Less P-TILT (6°-10°) and more PRO (7°-9°) in short pectoralis minor resting length
Dayanidhi et al, 2005	Comparison between children and adults	More LAT in children (±15°)
Myers et al, 2005	Comparison between throwers and controls	More LAT (±7°) and PRO (±9°) in throwers
EFFECT OF EXERCISE PROTOCOL		
Ebaugh et al, 2006a	Effect of shoulder elevation fatigue protocol	Increased LAT (±22°), increased RETR (±19.8°) after fatigue protocol
Ebaugh et al, 2006b	Effect of shoulder external rotation fatigue protocol	Increased LAT (circa 6°), decreased P-TILT (circa 3°) after fatigue protocol
Tsai et al, 2003	Effect of shoulder external rotation fatigue protocol	Decreased LAT (2.5°), decreased P-TILT (4°), decreased RETR (2.4°) after fatigue protocol
Borstad et al, 2009	Effect of a m. serratus anterior fatigue protocol	Decreased P-TILT (15.2°), increased PRO (10.9°) after fatigue protocol

PRO: Scapular protraction; RETR: Scapular retraction; LAT: Scapular lateral rotation; P-TILT: Scapular posterior tilt; -: No significant results

Table 8. Papers on scapular kinematics in persons after stroke: results

Study		Significant results
Meskers et al, 2005	Comparison between the non-hemiplegic shoulder of persons after stroke and healthy controls	Decreased PRO (±13°) in persons after stroke during sagittal plane elevation
Niessen et al, 2008	Comparison between persons with(out) PSSP and healthy controls	Increased LAT at non-hemiplegic side in persons with PSSP compared to persons without PSSP (±5°) and controls (±6°) Increased LAT at hemiplegic side in persons with PSSP compared to controls (±10°)

PSSP: Post Stroke Shoulder Pain; LAT: Scapular lateral rotation; PRO: Scapular protraction

5.2.2 Scapular muscle activity

Cools et al. (2002) reported m. deltoideus pars acromialis to be active prior to all parts of m. trapezius as a reaction to sudden falling of the arm.[51] A fatigue exercise did not alter this pattern, though all muscles showed an increased latency time, except for m. trapezius pars ascendens. In persons with impingement, an increased latency time was found in m. trapezius pars transversa and m. trapezius pars ascendens for the same task.[52] During humerothoracic elevation, a consistent recruitment pattern was found in healthy persons and persons with unilateral impingement, whereby m. trapezius pars descendens was activated first, followed by m. serratus anterior, m. trapezius pars transversa and m. trapezius pars ascendens. However, all muscles of the affected shoulder showed an increased latency time in the persons with impingement.[28] Persons with multidirectional instability showed an earlier activation of m. deltoideus pars spinalis and a longer activation of m. pectoralis major (later termination).[53] In contrast, Illyés and Kiss (2006) reported in these patients a decreased length of activation for the three parts of m. deltoideus and m. pectoralis major, and an increased length of activation of m. supraspinatus, m.infraspinatus, m. biceps brachii and m. triceps brachii during elevation.[54]

6. Discussion

Coordinated humeral and scapular movement, induced by fine-tuned muscle activity, contribute to optimal and pain free shoulder motion.[16] In stroke patients, an optimal active range of motion of the shoulder is reported as a prerequisite for good hand function,[55] whereby 88% of the variance in hand function is explained by changes in active range of shoulder motion. This confirms the importance of adequate shoulder and thus scapular kinematics and muscle activity post-stroke.

To attain a sound interpretation of the scapular function in persons with PSSP, knowledge of 3D scapular kinematics and associated muscle activity in healthy persons and persons with primary shoulder disorders is crucial. Alterations in scapular kinematics and the presence of shoulder muscle imbalance have been put forward as important contributors to shoulder pain in the latter group.[22,23] If the alterations seen in persons with PSSP resemble those found in persons with primary shoulder dysfunctions, further research towards PSSP rehabilitation could benefit from the strategies used in persons with primary shoulder dysfunctions.

6.1 Methodological considerations

Before results on 3D scapular kinematics can be discussed, several methodological aspects, i.e. joint angles calculation, measurement methods, and tracking systems need to be taken into account. To facilitate result comparison and the communication among researchers, the International Society of Biomechanics (ISB) has advised to use the angulus acromialis, the trigonum spina and the angulus inferior (AA, TS, AI) to define the scapular joint coordinate system and to use the protraction/medial rotation/posterior tilt Euler decomposition to calculate joint angles.[13] This sequence is preferred as it is consistent with the 2D description of scapular motion whereby the rotation axis for lateral rotation falls into the horizontal plane.[56] Karduna et al. (2000) additionally reported differences of up to 50° of scapular protraction/retraction when choosing different rotation sequences around the anatomical axes. The proposed scapular landmarks (AA, TS, AI) decrease the risk for singular positions during scapular lateral rotation, typically occurring from 70° of lateral rotation, and thus avoid the occurrence of gimbal lock.[57] Furthermore, Ludewig et al. (2010) also discussed the impact of choosing a different origin to construct the scapular coordinate systems, i.e. using the AA (instead of the acromioclavicular landmark (AC), as proposed by ISB) resulted in reduced protraction and lateral rotation and more posterior tilt during arm elevation in the scapular plane (scaption). This can be linked to the results of two selected papers,[29,41] both investigating scapular kinematics during maximal scapular plane elevation (scaption), but using different scapular landmarks. At 120° of humerothoracic elevation, circa 7° less protraction, 37° less lateral rotation and 20° more posterior tilt were reported when the AA[41] instead of the AC landmark[29] was chosen as origin.

Finally, when interpreting 3D kinematics, the tracking method should also be considered. The ISB already recommended digitizing anatomical landmarks with reference to a technical receiver, instead of using skin-mounted markers during movement registration.[13] The impact of such skin-motion artifacts has recently been quantified by Lempereur et al. (2010),[58] thereby supporting the ISB recommendation. Although a bone-fixed receiver is highly accurate, this method is invasive and not suitable for clinical use. Using a skin-fixed scapular receiver is a valid alternative, though only for humerothoracic elevation up to 120°.[59,60] When using an acromion marker cluster, elevation of the humerus should not even exceed 100°.[61] The majority of the studies included in this review used a skin-fixed receiver, though 10 studies did analyze scapular movements during elevations above 120° (Table 2).

Methodological aspects affecting the *EMG signal,* e.g. placement of electrodes, choice of EMG parameters, definition of muscle activity, etc. should also be considered prior to result interpretation. In the selected papers, different guidelines for electrode placement were followed, enquiring a careful data-interpretation.[20,62] Furthermore, EMG parameters should be carefully selected. Absolute intensities of electrical activity are not believed to represent functional muscle activity, whilst recruitment patterns and muscle latencies are considered more valuable in identifying causes of shoulder dysfunction.[19] Different determination methods for onset of muscle activity were reported: (1) surpassing a threshold EMG activity with respect to the maximum voluntary contraction (10%[51,52] or 20 %[54]); (2) surpassing the main baseline activity with two[28] or three standard deviations.[53]

6.2 Integrated result interpretation

In view of the abovementioned impact of various methodological aspects, the wide variability in population demographics (age, hand dominance, gender, occupational activities) across the different studies, the variability in reported results is not surprising. The amount of lateral rotation and posterior tilt differed vastly among the different studies, and no consistent pattern of protraction/retraction could be determined. Moreover, while studies generally reported altered kinematics in persons with primary shoulder pain and PSSP compared to healthy controls, no consensus on the type of alterations could be reached. This is confirmed by the results of a recent meta-analysis of Timmons et al. (2012).[63] These authors investigated the influence of population characteristics and plane and degree of elevation on scapular kinematics in subacromial impingement syndrome. With this in mind, results of 3D scapular kinematics and muscle activity in persons with primary shoulder disorders and PSSP should be considered from different points of view.

At first, results can be discussed in light of the pain adaptation model.[64] In this model, a reorganization in scapular muscle activity and scapular kinematics occurs in response to pain in order to minimize disturbances, e.g. impingement of rotator cuff, during the dynamic task. The increased lateral rotation[47-50,65] and posterior tilt seen in persons with impingement and instability[44,47,48] can be interpreted according to this model, as these rotations move the acromion away from the humeral head to avoid impingement.[23] The longer activation period of the rotator cuff in persons with glenohumeral instability (described by Illyés and Kiss (2006) is also considered as a pain adaptation strategy for the underlying joint instability.[54] Apart from the prolonged rotator cuff activity, m. teres major is also believed to be more active in instable shoulders in order to pull down the humeral head. Such contraction of m. teres major further increases scapular lateral rotation during elevation. This in turn contributes to pain avoidance or reduction.

In contrast, reduced lateral rotation[23,44] and a pattern of anterior tilt[23] in persons with shoulder disorders, should be interpreted as a primary cause for shoulder

disorders. These alterations do not preserve the optimal kinematics and thereby contribute to the development of pain and pathology. The reduced lateral rotation and pattern of anterior tilt can be viewed as the result of a preferred slouched sitting position[33] and a short pectoralis minor resting length.[37] The alterations can also be caused by a delayed, an inhibited or a less active m. serratus anterior and m. trapezius,[28,38,52] inducing muscle imbalances and subsequent pathology around the shoulder complex.[23,66]
Finally, in patients with frozen shoulder pathology, the increased scapular lateral rotation should be interpreted as a compensation for the reduced elevation in the glenohumeral joint.[22] Shortened internal humeral rotators, e.g. m. teres major or m. pectoralis major, further add to the enlarged scapular lateral rotation by pulling the scapula laterally even during little humerothoracic elevation.[17] The latter could also explain the increased lateral rotation in persons with anterior shoulder tightness,[65] who often show shortened internal glenohumeral rotators.

6.3 Future directions in scapular assessment

Currently, the measurement of 3D scapular kinematics and muscle activity during elevation is an accepted method for the description and comparison of scapular function between different groups. It provides an understanding of the deficits underlying the impaired scapular function in persons with primary shoulder disorders or post-stroke shoulder pain. Such understanding is required for the evaluation of treatment efficacy aimed at optimizing scapular control and for the characterization of the re-education process in primary shoulder pain and PSSP. Nevertheless, methodological concerns should always be clarified to the reader and taken into account prior to result interpretation. An important number of studies included in this review failed to give complete methodological information, especially on the items concerning participant selection, manner of recruitment, reliability of the applied methodology and justification for sample size (Table 5 and 6).
The elevation task is easy to use in clinical practice, has an important amount of comparable data, and is the first dynamic movement in the management of musculoskeletal shoulder disorders.[67] Knowledge on the normal scapular function during the simple elevation task is also an absolute prerequisite to understand scapular kinematics during more complex and functional movements. For future research and clinical applications, the evolution towards scapular assessments during functional tasks should be considered. Especially for the stroke population, whereby rehabilitation is focused mainly on regaining functional abilities, the scapular assessment during a set of predefined tasks covering an overall range of functional tasks will have added value compared to a mere elevation task.[68] However, the tasks should take movement direction, speed and distance into account and data analysis/reporting should always follow the ISB recommendations.[69-71]

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Chapter 2

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Dynamic scapular movement analysis: is it feasible and reliable in stroke patients during arm elevation?

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1. Abstract

Knowledge of three-dimensional scapular movements is essential to understand post-stroke shoulder pain. The goal of the present work is to determine the feasibility and the within and between session reliability of a movement protocol for three-dimensional scapular movement analysis in stroke patients with mild to moderate impairment, using an optoelectronic measurement system. Scapular kinematics of 10 stroke patients and 10 healthy controls was recorded on two occasions during active forward flexion and abduction from 0° to 60° and from 0° to 120°. All tasks were executed unilaterally and bilaterally. The protocol's feasibility was first assessed, followed by within and between session reliability of scapular total range of motion (ROM), joint angles at start position and of angular waveforms. Additionally, measurement errors were calculated for all parameters. Results indicated that the protocol was generally feasible for this group of patients and assessors. Within session reliability was very good for all tasks. Between sessions, scapular angles at start position were measured reliably for most tasks, while scapular ROM was more reliable during the 120° tasks. In general, scapular angles showed higher reliability during forward flexion compared to abduction, especially for protraction. Scapular lateral rotations resulted in smallest measurement errors. This study indicates that scapular kinematics can be measured reliably and with precision within one measurement session. In case of multiple test sessions, further methodological optimization is required for this protocol to be suitable for clinical decision-making and evaluation of treatment efficacy.

2. Introduction

Shoulder pain is a common and disabling complication after stroke, affecting one-third of the stroke patients in general.[1] Moreover, bicipital tenderness, supraspinatus tenderness, and a positive Neer impingement sign are described in 54%, 48% and 30% of the stroke patients, respectively.[2] These problems negatively affect functional arm recovery and thereby decrease daily life autonomy and quality of life.[3-5]

Careful assessment of the shoulder is thus essential in stroke patients. Motor scales used in routine clinical practice are typically limited to a global upper limb assessment.[6] Hence, key information on the isolated shoulder function and the more specific scapulothoracic movement is missed. However, given that adequate scapular behavior is a prerequisite for pain free shoulder movement, assessment of this scapulothoracic joint should be considered in stroke patients at risk to develop shoulder pathology and/or pain. Correct scapular movements are established by scapular muscles working in specific activation patterns.[7] Several neurological impairments (e.g. lack of muscle tone, spasticity and loss of motor control) will induce scapular muscular imbalances, which in turn will influence scapular position and movements. This altered scapular behavior is suggested to contribute to the development of rotator cuff impingement, and consequently to the development of shoulder pain.[8] Shoulder pathology has already been related to three-dimensional (3D) scapular movements (Figure 1A) during a humerothoracic elevation task. Despite the simplicity of this task, it has been shown sensitive enough to detect changes in 3D scapular movements associated with shoulder pathology.[9-11] More recently, measuring 3D scapular movements during humerothoracic elevation has also been introduced in stroke patients.[12-15] However, the value of such kinematic analysis in clinical decision-making or to evaluate treatment efficacy firstly requires the establishment of its feasibility and reliability.[16]

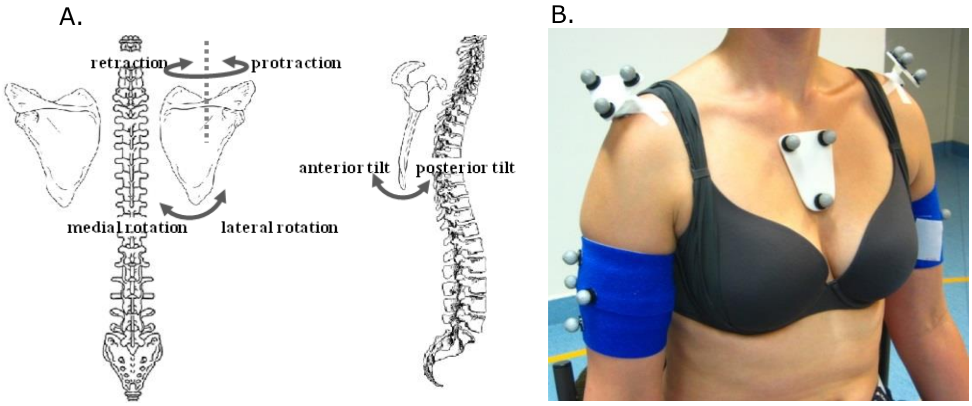
The goal of the present work was to assess the feasibility and reliability of a specific humerothoracic elevation protocol to measure 3D scapular movements in stroke and in healthy controls. We furthermore aimed to formulate recommendations regarding parameter selection when using 3D scapular movements for clinical decision-making or to evaluate treatment efficacy.

3. Methods

3.1. Participants

An overview of the participants' characteristics is given in Table 1. All stroke patients were hospitalized in the University Hospital Pellenberg and eligible for participation when they (1) were between 1 and 12 month(s) after a first time stroke; (2) had no shoulder complaints prior to stroke; (3) could perform 60° of humerothoracic elevation and (4) were able to understand the instructions. Stroke patients with an occipital, brainstem or cerebellar lesion or with reduced communicative or cognitive abilities were not considered for inclusion. Controls were recruited via family and colleagues and were excluded in case of current shoulder dysfunctions or treatment.

Figure 1. Overview of scapular rotations and marker



Three-dimensional scapular rotations (A) and marker cluster placement

3.2. Ethical Statement

This study was approved by the Ethical Committee of the University Hospital Pellenberg. All participants provided written informed consent to participate in this study, as approved by the Ethical Committee. The person on the photograph in Figure 1 and in Online Supplement 2 has given written informed consent (as outlined in PLOS consent form) to publish her photographs.

Table 1. Participants' characteristics

	Stroke patients	Healthy controls
Number of subject (men/women)	10 (7/3)	10 (4/6)
Age (years), range	18-69	18-70
Shoulder pain, yes/no	2/8	0/10
Hand dominance, left/right	0/10	0/10
Side of hemiplegia, left/right	2/8	NA
Time since stroke (weeks), range	5-39	NA
Lesion location	Cortical*	NA
Type of stroke, ischaemic/hemorhagic	7/3	NA
Fugl-Meyer score** (0-66), range	35-64	NA

*: One patient had an addition lesion in the basal ganglia; **: Upper extremity motor section; NA: not applicable

3.3. Kinematic Data Acquisition

Bilateral 3D kinematic data were captured with 15 infrared cameras sampling at 100 Hz (Vicon, Oxford Metrics, UK) and filtered with spline-interpolation.[17] Clusters of three or four markers, mounted on tripods or cuffs, were placed on the sternum, scapula (flat part of the acromion) and upper arm (proximal, lateral) (Figure 1B). Anatomical landmarks were digitized during static trials, using a pointer with four linear markers. Anatomical landmarks were defined within their respective segmental marker cluster (CAST-procedure),[18] and subsequently used to construct anatomical coordinate systems and calculate joint kinematics. To ensure correct and accurate location of all anatomical landmarks, we adhered to specific palpation guidelines.[19] All kinematic calculations were done according to the ISB-guidelines.[20] Scapular kinematics were described for following three rotations: protraction/retraction, medial/lateral rotation, anterior/posterior tilting (Figure 1A).

3.4. Measurement Procedure

Each participant was measured on two occasions, 5 to 10 days apart, by the same assessor. This assessor was trained to correctly conduct the measurement procedure and to perform the anatomical palpation properly. In this way, a repeatable and accurate placement of the marker clusters and palpation of anatomical landmarks was ensured. All measurements took place at the clinical motion analysis laboratory of the University Hospital Pellenberg. Marker clusters were mounted on the participant's upper body, who was then seated on a chair with low back support. Next, static calibration trials were collected to digitize anatomical landmarks and participants were subsequently asked to execute the movement protocol (Table S1 and Figure S1): humerothoracic elevation in the frontal (abduction) and sagittal (forward flexion) plane, executed from 0° to 60° and from 0° to 120°. Each elevation task was done unilaterally and bilaterally at self-selected speed. Elevation height was marked on a pole to maximize standardization. Participants were given a practice trial prior to recording and each movement was demonstrated by an assessor seated in front of the participant. Three dynamic trials consisting of four repetitions each were recorded for every elevation task.

3.5. Data Analysis

From the recorded trials, only the second and third repetition were selected for data analysis (as these were not corrupted by initiation/completion strategies), resulting in six cycles per elevation task per session. Movement cycles were visually defined from start to highest arm position. Data was further processed with Matlab®, using BodyMech (http://www.bodymech.nl) and custom-written routines. Each movement cycle was time-normalized and joint angles were visualized as function of time to check for erroneous signals. Discrete parameters of interest were (1) scapular range of motion (ROM) expressed for each scapular rotation in every elevation task, and (2) 3D scapular joint angles at start position. ROM was defined as the absolute difference between highest and lowest recorded joint angle per movement cycle.

3.6. Statistical Analysis

Reliability of discrete parameters was calculated with the intraclass correlation coefficient (ICC) and the standard error of measurement (SEM) based on the square root of the mean square error term from the two-way ANOVA.[16] Single data from the first session was used to calculate within session reliability (ICCw(2,1); SEMw), averaged data from both sessions was used for between session reliability assessment (ICCb(2,k); SEMb). ICCs >0.80 were considered very high, 0.60-0.79 moderately high, 0.40-0.59 moderate and <0.40 low.[21] Percentage SEM (%SEM, i.e. (SEM/mean)*100) was additionally calculated for ROM[22] to indicate the preciseness[16] per rotation for each elevation task, relative to the total amount of ROM.

Within and between session reliability of angular waveforms was assessed with the adjusted coefficient of multiple correlation (CMCw;CMCb) and group means were calculated.[23] CMCs >0.90 were considered excellent, 0.80-0.89 good, 0.60-0.79 moderate and <0.60 poor. Waveform measurement errors (σ) were calculated and the ratio of between (σb) to within session errors (σw) was also reported.[24]

4. Results

Figures 2 and 3 show within and between session ICCs and CMCs (see also Table S2 and S3) and measurement errors are listed in Table 2 and 3.

Three patients were not able to perform the 120° elevation tasks. Analysis of these tasks was therefore performed on seven patients.

4.1. Range of Motion

Reliability results are given in Figure 2A and 2B, in Table 2 and 3, and in Table S2.

Within session reliability was moderately high to very high in patients and controls (ICCw 0.63-0.99), except for few scapular ROM during 60° abduction (non-dominant side tilt; hemiplegic side lateral rotation; non-hemiplegic side protraction).

Between session reliability for scapular ROM at the dominant side (controls) was in general moderately high to very highly reliable for lateral rotation and tilt for the 120° tasks (ICCb 0.63-0.83), protraction showed low to moderately high reliability results (ICCb 0.21-0.78). Results for the 60° tasks at the dominant side and for both the 60° and 120° tasks at the non-dominant side were less reliable. Patients showed more variable results between sessions. In general, all scapular ROM during the 120° tasks were moderately high to very highly reliable at the hemiplegic and non-hemiplegic side and lowest ICCb-values were found for lateral rotation at the non-hemiplegic side (ICCb 0.02-0.17). Reliability of scapular ROM during 60° elevation tasks was inconsistent in patients, with ICCs

Table 2. Within session mean, standard deviation (SD) and standard error of measurement (SEM) for scapular range of motion (ROM).

	Forward flexion 60°			Forward flexion 120°		
	Mean (SD)	SEM_w	$\%SEM_w$	Mean (SD)	SEM_w	$\%SEM_w$
Controls dominant side						
Protraction	13.2 (4.0)	1.5	**11.3**	17.1 (4.7)	2.3	**13.1**
Lateral rotation	10.6 (3.3)	2.1	19.4	37.6 (6.4)	3.4	**9.1**
Tilt	5.6 (3.1)	0.9	16.6	14.5 (4.9)	2.9	19.7
Controls non-dominant side						
Protraction	14.0 (3.6)	1.3	**9.1**	18.9 (4.2)	2.1	**11.0**
Lateral rotation	15.9 (9.6)	2.0	**12.7**	42.5 (10.1)	1.9	**4.5**
Tilt	5.9 (3.3)	0.9	15.9	10.9 (6.7)	2.4	21.8
Stroke hemiplegic side						
Protraction	12.0 (7.3)	3.6	30.1	20.8 (12.8)	1.1	**5.5**
Lateral rotation	17.6 (8.7)	2.6	**14.7**	43.0 (10.8)	1.9	**4.5**
Tilt	8.5 (4.1)	1.8	20.9	16.1 (10.4)	0.3	**2.1**
Stroke non-hemiplegic side						
Protraction	14.2 (5.1)	2.5	17.6	21.4 (10.8)	0.9	**4.3**
Lateral rotation	15.4 (8.0)	2.1	**13.9**	45.5 (12.2)	1.4	**3.0**
Tilt	9.9 (5.2)	2.7	27.0	15.6 (9.0)	1.8	**11.5**

	Abduction 60°			Abduction 120°		
	Mean (SD)	SEM_w	$\%SEM_w$	Mean (SD)	SEM_w	$\%SEM_w$
Controls dominant side						
Protraction	4.4 (2.6)	1.0	21.7	13.8 (8.3)	1.8	**12.9**
Lateral rotation	13.4 (5.2)	1.8	**13.7**	41.0 (6.4)	3.6	**8.7**
Tilt	5.3 (3.8)	1.2	22.2	18.8 (9.1)	1.3	**7.1**
Controls non-dominant side						
Protraction	5.3 (3.8)	1.7	31.1	12.5 (8.8)	5.2	41.6
Lateral rotation	18.6 (7.9)	1.7	**9.3**	44.8 (13.7)	6.1	13.6
Tilt	4.6 (2.3)	1.1	25.0	15.5 (6.8)	5.7	36.7
Stroke hemiplegic side						
Protraction	6.3 (4.5)	1.4	22.7	15.2 (9.4)	1.1	**7.2**
Lateral rotation	20.1 (7.2)	4.3	21.6	47.2 (10.5)	2.5	**5.3**
Tilt	6.6 (4.8)	0.4	**6.0**	15.0 (11.8)	3.8	25.4
Stroke non-hemiplegic side						
Protraction	5.4 (2.4)	1.8	33.8	12.4 (3.6)	1.2	**10.0**
Lateral rotation	17.0 (7.8)	6.1	36.0	48.5 (9.2)	4.1	**8.4**
Tilt	6.6 (4.4)	2.5	38.3	20.5 (8.8)	2.4	**11.7**

Continued

Table 2. Continued

	Bilateral forward flexion 60°			Bilateral forward flexion 120°			
	Mean (SD)	SEM_w	$\%SEM_w$	Mean	(SD)	SEM_w	$\%SEM_w$
Controls dominant side							
Protraction	13.8 (3.8)	1.5	**10.9**	20.4	(5.8)	1.2	**6.0**
Lateral rotation	13.2 (3.9)	2.2	16.4	41.3	(7.6)	2.8	**6.9**
Tilt	5.0 (2.3)	0.7	**13.0**	13.1	(5.3)	2.3	17.2
Controls non-dominant side							
Protraction	15.2 (3.7)	2.0	**13.5**	21.2	(5.9)	2.7	**12.5**
Lateral rotation	15.8 (7.4)	1.9	**12.0**	45.4	(8.7)	2.7	**6.0**
Tilt	6.8 (3.8)	0.8	**12.2**	12.7	(5.5)	1.0	**7.7**
Stroke hemiplegic side							
Protraction	13.4 (8.5)	1.3	**9.8**	17.6	(9.1)	0.8	**4.4**
Lateral rotation	19.3 (8.4)	0.9	**4.9**	48.6	(8.9)	3.0	**6.1**
Tilt	8.9 (4.8)	1.8	20.2	17.7	(8.2)	2.4	**13.6**
Stroke non-hemiplegic side							
Protraction	18.1 (8.4)	3.3	18.4	23.7	(9.5)	3.8	15.8
Lateral rotation	22.2 (9.0)	2.0	**9.2**	49.9	(11.2)	2.8	**5.6**
Tilt	10.5 (6.6)	1.2	**11.7**	16.1	(7.3)	1.5	**9.1**
	Bilateral abduction 60°			Bilateral abduction 120°			
	Mean (SD)	Mean	(SD)	Mean	(SD)	SEM_w	$\%SEM_w$
Controls dominant side							
Protraction	5.6 (3.8)	1.8	32.7	15.4	(8.4)	3.2	21.1
Lateral rotation	23.4 (10.0)	1.8	**7.6**	49.3	(7.3)	3.4	**6.9**
Tilt	5.4 (4.2)	0.7	**12.8**	18.0	(7.3)	4.9	27.1
Controls non-dominant side							
Protraction	8.2 (5.5)	1.7	21.3	13.0	(7.9)	2.3	17.7
Lateral rotation	22.6 (8.9)	1.9	**8.3**	49.9	(14.7)	1.9	**3.8**
Tilt	5.4 (2.3)	1.3	24.5	13.6	(7.3)	1.2	**8.7**
Stroke hemiplegic side							
Protraction	8.3 (6.7)	6.4	77.0	23.8	(14.9)	6.7	28.0
Lateral rotation	23.7 (7.7)	5.0	21.2	54.8	(8.0)	1.6	**2.9**
Tilt	6.5 (4.4)	1.9	29.6	24.5	(13.1)	3.8	15.7
Stroke non-hemiplegic side							
Protraction	8.5 (4.1)	2.2	26.4	15.1	(5.1)	2.7	17.6
Lateral rotation	24.2 (7.7)	3.6	**14.9**	54.0	(11.1)	3.1	**5.7**
Tilt	7.5 (4.7)	3.1	41.4	16.9	(10.5)	2.5	**14.6**

Mean, SD and SEM are presented in degrees; %SEM represents the percentage SEM with respect to the mean; %SEMs lower than 15% are marked in bold

Table 3. Between session mean, standard deviation (SD) and standard error of measurement (SEM) for scapular range of motion (ROM).

	Forward flexion 60°			Forward flexion 120°		
	Mean (SD)	SEM_b	$\%SEM_b$	Mean (SD)	SEM_b	$\%SEM_b$
Controls dominant side						
Protraction	13.5 (3.2)	3.1	23.0	17.8 (5.4)	5.4	30.3
Lateral rotation	11.0 (3.0)	2.6	23.8	37.1 (5.5)	3.9	**10.4**
Tilt	5.9 (3.5)	3.3	55.2	15.2 (6.8)	4.9	32.1
Controls non-dominant side						
Protraction	12.7 (3.9)	2.8	21.9	18.9 (5.8)	3.5	18.5
Lateral rotation	14.9 (7.4)	5.0	33.5	39.1 (10.3)	7.0	17.8
Tilt	6.2 (3.5)	2.7	43.0	11.7 (6.5)	4.6	39.5
Stroke hemiplegic side						
Protraction	10.9 (6.1)	6.1	56.2	19.7 (10.4)	3.4	17.2
Lateral rotation	16.8 (8.2)	6.2	36.9	42.3 (12.4)	5.0	**11.8**
Tilt	8.1 (4.0)	5.1	62.9	15.1 (8.5)	3.0	19.9
Stroke non-hemiplegic side						
Protraction	13.8 (4.6)	2.4	17.1	23.0 (9.2)	7.8	33.7
Lateral rotation	14.0 (7.2)	10.7	76.4	42.2 (11.2)	13.1	30.9
Tilt	9.6 (5.4)	2.8	29.1	14.6 (8.7)	4.3	29.6

	Abduction 60°			Abduction 120°		
	Mean (SD)	SEM_b	$\%SEM_b$	Mean (SD)	SEM_b	$\%SEM_b$
Controls dominant side						
Protraction	4.1 (2.2)	1.9	47.3	11.8 (7.5)	3.7	31.5
Lateral rotation	14.1 (4.9)	3.8	26.9	40.5 (8.2)	6.4	15.7
Tilt	5.3 (3.4)	3.1	57.3	18.8 (9.1)	6.3	33.6
Controls non-dominant side						
Protraction	4.0 (2.9)	3.7	91.7	10.0 (7.0)	7.5	74.7
Lateral rotation	16.1 (6.7)	7.6	47.1	41.0 (13.0)	10.6	25.7
Tilt	4.8 (3.0)	2.3	48.1	14.7 (6.9)	5.0	34.0
Stroke hemiplegic side						
Protraction	4.7 (3.6)	0.4	**7.5**	13.3 (7.5)	4.7	36.1
Lateral rotation	20.4 (6.4)	3.9	19.1	46.0 (9.8)	5.4	**11.6**
Tilt	5.7 (4.1)	1.7	29.3	14.5 (10.3)	0.9	**6.3**
Stroke non-hemiplegic side						
Protraction	4.6 (3.0)	1.5	32.0	10.5 (3.5)	1.5	28.7
Lateral rotation	17.0 (6.8)	3.7	21.6	46.7 (10.0)	11.5	24.7
Tilt	6.0 (3.4)	1.8	30.2	18.2 (9.5)	5.8	31.7

Continued

Table 3. Continued

	Bilateral forward flexion 60°				Bilateral forward flexion 120°			
	Mean	(SD)	SEM_b	$\%SEM_b$	Mean	(SD)	SEM_b	$\%SEM_b$
Controls dominant side								
Protraction	14.4	3.8	3.0	20.8	19.5	6.3	4.8	24.5
Lateral rotation	14.1	4.0	2.2	15.3	40.8	7.2	4.4	**10.8**
Tilt	6.1	3.3	2.9	46.8	13.3	6.2	5.3	39.8
Controls non-dominant side								
Protraction	13.9	3.4	1.6	**11.3**	20.9	7.0	5.7	27.2
Lateral rotation	16.4	7.0	5.1	31.3	43.2	9.6	6.8	15.7
Tilt	6.9	3.8	2.8	40.6	12.7	5.9	3.2	24.8
Stroke hemiplegic side								
Protraction	12.5	6.9	1.6	**12.5**	17.2	11.1	3.9	22.8
Lateral rotation	19.3	8.5	7.5	38.9	47.4	10.0	5.2	**10.9**
Tilt	8.9	4.3	1.9	20.9	15.8	7.6	3.3	20.9
Stroke non-hemiplegic side								
Protraction	16.8	6.9	5.1	30.5	25.6	8.1	5.2	20.2
Lateral rotation	21.3	7.9	10.7	50.3	48.6	8.7	10.8	22.3
Tilt	10.3	5.8	2.4	23.0	14.7	7.3	4.8	32.3
	Bilateral abduction 60°				**Bilateral abduction 120°**			
	Mean	(SD)	SEM_b	$\%SEM_b$	Mean	(SD)	SEM_b	$\%SEM_b$
Controls dominant side								
Protraction	4.9	3.2	2.8	56.4	12.5	7.2	5.3	42.4
Lateral rotation	22.5	8.0	6.4	28.4	50.6	9.0	6.3	**12.5**
Tilt	5.3	3.8	2.7	50.2	16.0	6.8	4.9	30.4
Controls non-dominant side								
Protraction	6.8	4.3	4.3	63.2	11.6	7.8	7.3	62.9
Lateral rotation	20.7	7.4	8.7	40.3	48.5	11.9	10.0	20.6
Tilt	5.4	2.3	1.7	30.7	13.5	6.8	6.3	46.4
Stroke hemiplegic side								
Protraction	7.5	6.1	5.3	70.9	19.9	13.0	10.0	50.5
Lateral rotation	23.2	5.7	4.2	18.2	53.7	7.6	1.9	**3.6**
Tilt	5.3	3.5	1.8	33.6	20.4	12.8	7.1	34.8
Stroke non-hemiplegic side								
Protraction	6.7	3.7	3.0	44.3	12.0	4.7	3.7	30.4
Lateral rotation	23.8	8.1	8.0	33.8	52.4	11.8	12.4	23.7
Tilt	6.6	3.5	4.1	62.2	15.9	10.5	4.9	30.6

Mean, SD and SEM are presented in degrees; %SEM represents the percentage SEM with respect to the mean; %SEMs lower than 15% are marked in bold ranging from very low to very high for both sides (ICCb 0.05-0.88). In summary, we conclude for scapular ROM that 120° tasks are more reliable than 60° tasks, and that the dominant and hemiplegic side are slightly more reliably measureable than the non-dominant and non-hemiplegic side. No marked differences in reliability of scapular ROM were furthermore found for unilateral versus bilateral tasks.

%SEM-values depended not only on the task (60° vs. 120°) and side ((non)dominant vs. (non)hemiplegic), but also clearly differed for the three scapular rotations. In agreement with the ICCs, %SEM below 15% (higher precision) was found for the 120° tasks, especially at the dominant side (controls) and hemiplegic side (patients).

In general, lateral rotation showed lower %SEM (<15% for all tasks within session and for 120° tasks at the dominant and hemiplegic side between sessions) than protraction (within sessions %SEM 4.3%-77%; between sessions %SEM 7.5%-91.7%) and tilt (within sessions %SEM 0.3%-41.4%; between sessions %SEM 6.3%-62.9%), especially between sessions.

4.2. Joint angles at start position

Only the results of joint angles at start for the following tasks are reported: unilateral 120° forward flexion and unilateral 120° abduction (Figure 2C, Table 4 and Table S2). Results for the other tasks were comparable.

Controls and patients showed very high within session reliability for all scapular rotations at start for both tasks (ICCw>0.84). Between session reliability of scapular angles at start was also moderately high to very high for both sides in controls (ICCb 0.69-0.93), apart from non-dominant side tilt (ICCb 0.04). In patients, highest between session reliability was found for tilt at start position of both sides and during both tasks (ICCb 0.75-0.98), followed by protraction at start (ICCb 0.46-0.76). For both tasks, start position of lateral rotation was also highly reliable at the non-hemiplegic side (ICCb>0.90), though was poorly reliable at the hemiplegic side (ICCb<0.45).

In patients and controls, within session SEM was below 3° for all scapular angles at start, for all tasks and at both sides; between session SEM was higher, with values ranging from 0.8° to 8.6°. Patients generally showed slightly higher SEMs at the hemiplegic side compared to the non-hemiplegic side and to controls.

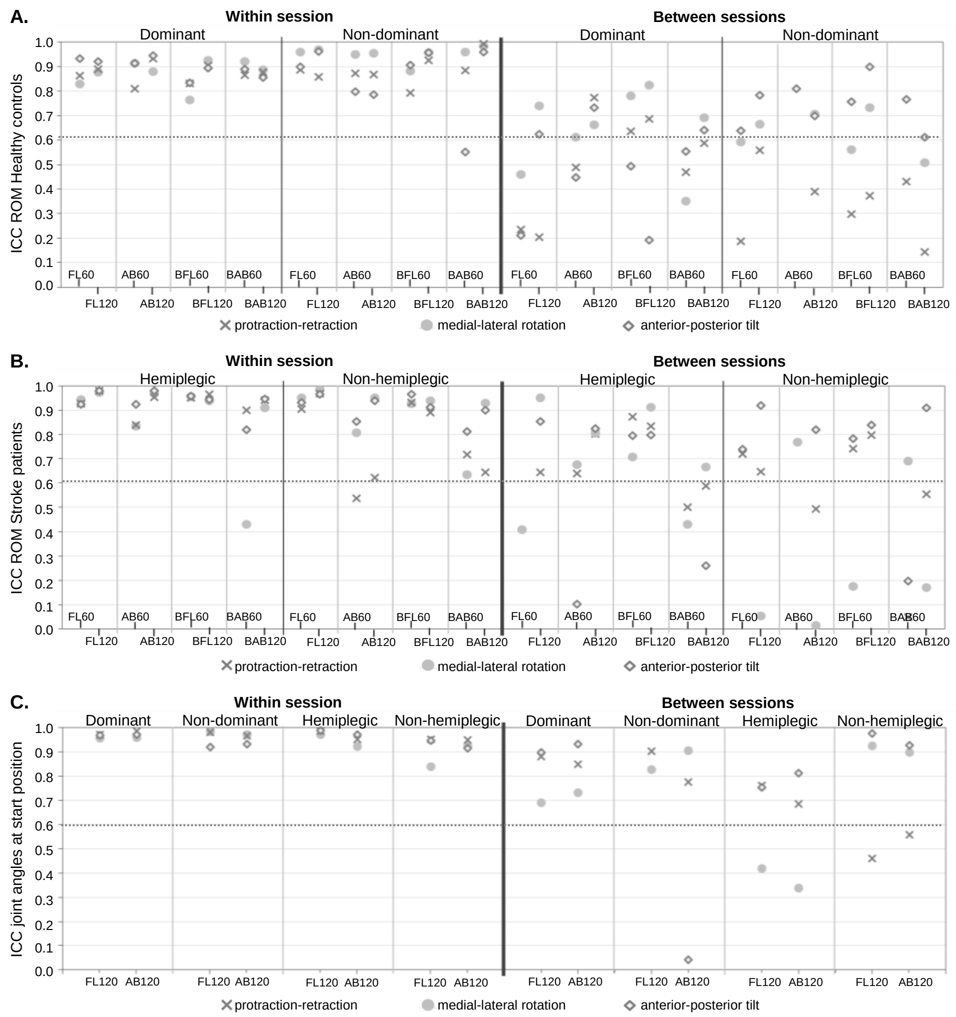
4.3. Angular waveforms

Results are given in Figure 3, Table 5 and Table S3.

Within session reliability of angular waveforms was excellent for lateral rotation and moderate to excellent for protraction and tilt for all tasks and both sides in patients and controls. Between session reliability of angular waveforms was excellent for lateral rotation in 120° tasks and good to moderate in 60° tasks. For protraction and tilt, between session reliability ranged from poor to excellent, whereby higher values were found for forward flexion than abduction tasks.

Waveform measurement errors (σw-σb) were generally lower for forward flexion tasks compared to abduction tasks. Error ratio's (σb/σw) ranged from 2.2 to 4.9 for protraction, from 1.4 to 3.8 for lateral rotation and from 1.4 to 3.6 for tilt.

Figure 2. Within and between session reliability of parameters of interest



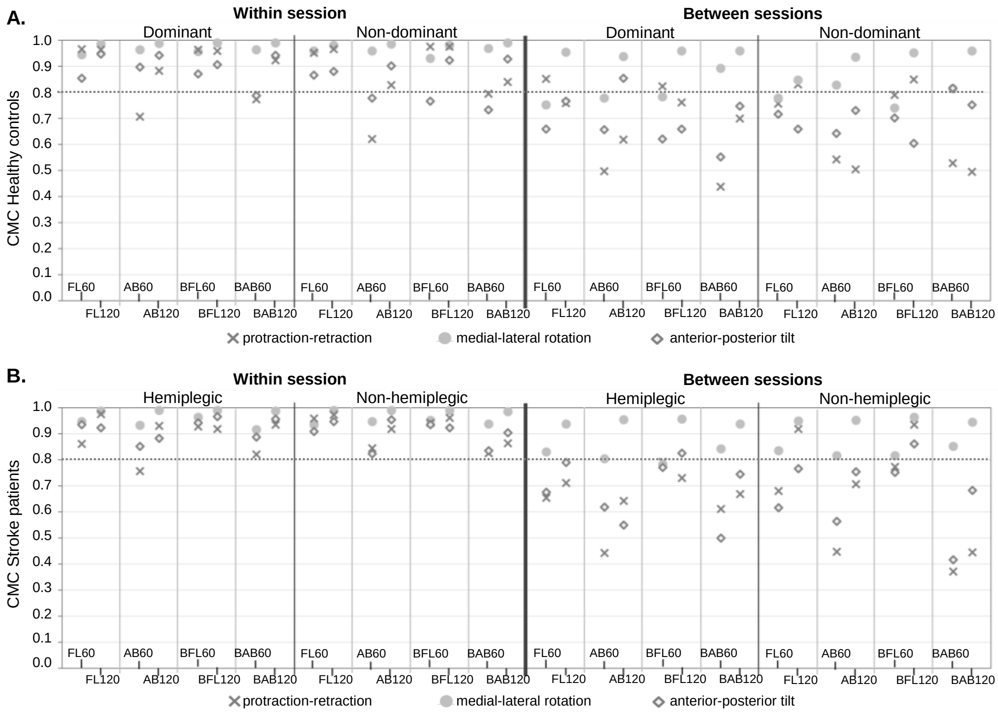
ICCs of range of motion in healthy controls (A), ICCs of range of motion in stroke patients (B) and ICCs of start position in healthy controls and stroke patients (C); FL: forward flexion; AB: abduction; BFL: bilateral forward flexion; BAB: bilateral abduction; No symbol is shown in case of calculation errors.

Table 4. Within and between session mean, standard deviation (SD) and standard error of measurement (SEM) for scapular joint angles at start position.

	WITHIN SESSION					
	Forward flexion 120°			Abduction 120°		
	Mean	(SD)	SEM_w	Mean	(SD)	SEM_w
Controls dominant side						
Protraction	28.0	(8.0)	2.7	26.9	(8.5)	2.1
Lateral rotation	9.1	(7.0)	1.2	6.6	(7.2)	1.2
Tilt	8.0	(4.5)	0.6	8.5	(5.4)	0.4
Controls non-dominant side						
Protraction	26.6	(7.5)	0.9	27.0	(9.9)	2.3
Lateral rotation	6.9	(10.4)	1.9	3.2	(9.0)	2.2
Tilt	5.8	(3.4)	0.8	6.4	(4.5)	0.7
Stroke hemiplegic side						
Protraction	26.8	(8.0)	1.4	27.7	(6.4)	1.6
Lateral rotation	1.8	(6.3)	0.9	1.3	(4.6)	1.3
Tilt	10.3	(7.4)	1.1	9.0	(6.2)	1.9
Stroke non-hemiplegic side						
Protraction	34.6	(6.3)	1.9	32.7	(6.9)	2.0
Lateral rotation	0.8[a]	(2.9)	1.3	1.8[a]	(4.8)	1.6
Tilt	11.7	(5.1)	0.8	14.0	(4.4)	0.4
	BETWEEN SESSIONS					
	Forward flexion 120°			Abduction 120°		
	Mean	(SD)	SEM_b	Mean	(SD)	SEM_b
Controls dominant side						
Protraction	28.7	(7.0)	3.8	27.5	(7.5)	4.0
Lateral rotation	7.6	(6.5)	3.6	5.7	(6.9)	4.1
Tilt	7.3	(5.6)	2.8	8.1	(6.4)	2.7
Controls non-dominant side						
Protraction	25.9	(6.9)	3.5	24.9	(8.9)	3.5
Lateral rotation	4.1	(9.5)	3.5	4.0	(8.8)	3.6
Tilt	6.3	(4.9)	3.8	7.5	(5.5)	5.4
Stroke hemiplegic side						
Protraction	29.4	(8.1)	5.1	29.5	(6.6)	6.0
Lateral rotation	2.5	(6.8)	8.6	1.5	(6.1)	7.5
Tilt	9.2	(6.4)	4.5	9.7	(5.6)	3.9
Stroke non-hemiplegic side						
Protraction	32.1	(5.9)	3.9	30.7	(6.7)	5.6
Lateral rotation	0.8[a]	(2.8)	1.4	2.2[a]	(4.4)	1.4
Tilt	11.4	(5.0)	0.8	14.1	(4.4)	0.8

Mean, SD and SEM are presented in degrees; Bilat: Bilateral; [a]: Medial instead of lateral rotation

Figure 3. Within and between session reliability of angular waveforms



CMCs in healthy controls (A) and CMCs in stroke patients (B); FL: forward flexion; AB: abduction; BFL: bilateral forward flexion; BAB: bilateral abduction; No symbol is shown in case of calculation

5. Discussion

This study investigated the feasibility and reliability of a protocol to measure 3D scapular kinematics. Such assessment is believed to provide additional information on the 3D character of scapular movements that is not captured with a two-dimensional analysis or the available clinical scales.

The discussion on feasibility of the applied methodology is twofold. Firstly, to ensure adequate assessment of scapular kinematics, patients should be able to perform the protocol as requested. Movements in the frontal plane were executed less accurately, especially by those patients with impaired arm proprioception, i.e. patients with more dysmetria during the Finger-to-Nose test of the Fugl-Meyer scale. As patients were instructed to look forward during task performance, they could not rely on visual feedback of their performance. Better guidance by means of e.g. a mirror or auditory signals is thus proposed.

Table 5. Within and between session measurement errors of the angular waveforms (expressed in degrees) and the ratio of between to within errors

	Forward flexion 60°			Forward flexion 120°		
	σ_w	σ_b	r	σ_w	σ_b	r
Controls dominant side						
Protraction	1.3	2.9	2.2	1.3	4.3	3.4
Lateral rotation	1.1	2.9	2.6	1.8	3.7	2.1
Tilt	0.9	2.1	2.4	1.0	2.8	2.8
Controls non-dominant side						
Protraction	1.1	4.1	3.7	1.3	5.2	4.1
Lateral rotation	1.1	3.8	3.4	1.9	7.4	3.8
Tilt	0.8	2.6	3.3	1.0	3.6	3.6
Stroke hemiplegic side						
Protraction	1.2	4.3	3.7	1.2	5.2	4.2
Lateral rotation	1.6	5.2	3.3	2.0	5.4	2.7
Tilt	0.9	2.5	2.8	1.3	2.6	2.0
Stroke non-hemiplegic side						
Protraction	1.3	5.1	3.8	1.4	4.5	3.3
Lateral rotation	1.5	2.6	1.8	1.9	4.6	2.5
Tilt	1.0	2.8	2.8	1.2	2.7	2.2
	Abduction 60°			Abduction 120°		
	σ_w	σ_b	r	σ_w	σ_b	r
Controls dominant side						
Protraction	1.0	3.4	3.3	1.5	4.1	2.7
Lateral rotation	1.3	3.4	2.6	2.1	4.8	2.3
Tilt	0.9	2.3	2.7	1.5	3.4	2.3
Controls non-dominant side						
Protraction	0.9	4.6	4.9	1.8	5.3	2.9
Lateral rotation	1.6	3.8	2.4	2.4	5.1	2.1
Tilt	1.1	3.2	2.8	1.7	3.9	2.3
Stroke hemiplegic side						
Protraction	1.5	3.6	2.4	1.5	5.2	3.6
Lateral rotation	2.1	5.2	2.5	1.9	4.5	2.4
Tilt	1.1	2.2	2.0	1.5	3.2	2.1
Stroke non-hemiplegic side						
Protraction	1.0	4.2	4.3	1.4	4.0	2.9
Lateral rotation	1.9	3.2	1.6	3.0	4.3	1.4
Tilt	1.1	2.1	1.9	1.4	2.4	1.7

Continued

Table 5. Continued

	Bilat Forward flexion 60°			Bilat Forward flexion 120°		
	σ_w	σ_b	r	σ_w	σ_b	r
Controls dominant side						
Protraction	1.2	3.4	2.9	1.6	4.6	2.9
Lateral rotation	1.3	3.0	2.4	1.8	3.6	2.0
Tilt	0.9	2.3	2.6	1.2	3.4	2.8
Controls non-dominant side						
Protraction	1.1	4.4	4.1	1.5	3.5	2.4
Lateral rotation	1.8	3.6	2.0	2.2	4.5	2.0
Tilt	1.2	2.6	2.2	1.1	3.4	3.2
Stroke hemiplegic side						
Protraction	1.2	3.6	2.9	1.3	5.1	4.0
Lateral rotation	1.4	4.9	3.4	2.0	4.8	2.4
Tilt	0.9	2.7	3.0	1.5	2.6	1.8
Stroke non-hemiplegic side						
Protraction	1.8	4.3	2.4	1.8	4.2	2.3
Lateral rotation	1.7	4.3	2.5	2.2	4.7	2.2
Tilt	1.0	2.3	2.3	1.6	2.3	1.4
	Bilat Abduction 60°			**Bilat Abduction 120°**		
	σ_w	σ_b	r	σ_w	σ_b	r
Controls dominant side						
Protraction	1.1	3.8	3.5	1.7	4.4	2.5
Lateral rotation	1.7	3.7	2.1	2.1	4.8	2.2
Tilt	0.9	2.5	2.7	1.7	3.5	2.1
Controls non-dominant side						
Protraction	1.5	4.1	2.8	1.5	4.6	3.0
Lateral rotation	1.7	4.4	2.5	2.0	5.1	2.6
Tilt	1.1	3.4	3.1	1.2	3.9	3.1
Stroke hemiplegic side						
Protraction	2.0	5.4	2.7	1.8	6.8	3.8
Lateral rotation	3.3	4.7	1.4	2.7	5.9	2.2
Tilt	1.4	3.1	2.3	1.9	4.5	2.4
Stroke non-hemiplegic side						
Protraction	1.4	4.0	2.9	2.3	5.4	2.4
Lateral rotation	3.0	4.3	1.4	3.0	5.9	1.9
Tilt	1.4	2.4	1.7	2.2	3.4	1.6

σ_w: within session error; σ_b: between session error; r: ratio of σ_b / σ_w; Bilat: Bilateral

Furthermore, this protocol is specifically designed for stroke patients who are already relatively high functioning and thus at higher risk to develop shoulder pathology, and who would benefit most from scapular stabilization training in the prevention of or to treat e.g. shoulder pain. An active humerothoracic elevation of at least 60° is an absolute prerequisite to measure scapular behavior, and patients were selected accordingly in this study. The second feasibility issue focuses on the assessor. Although the use of the acromion marker cluster to measure scapular joint angles is validated by van Andel et al. (2009),[25] the assessor should be adequately trained to place the marker cluster and to perform the anatomical palpation in a repeatable manner. Therefore, a trained assessor with high knowledge in anatomical palpation performed all measurements in this study.

Reliability results of the current study showed that angles at start position were measured reliably in patients (hemiplegic side) and controls (dominant side) (ICC>0.60; SEM<8.6°), except lateral rotation at the hemiplegic side. As shoulder movement dysfunctions often find their origin in altered scapular start positions, these angles are highly relevant from a clinical viewpoint. The proposed method is thus a promising tool to measure the effect of scapular stabilization training. ROM was also generally more reliable at the dominant (controls) and hemiplegic arm (patients). For both groups, highest within session %SEMs were found for protraction in 60° abduction, suggesting a high natural intra-subject variability.[24] Since this variability cannot be controlled, 60° abduction tasks are considered less suitable to measure true changes in scapular protraction. Between sessions, tilt showed poorest ICCs and %SEMs, especially in 60° tasks. This indicates that a significant amount of methodological errors is introduced when measuring scapular tilt during 60° of sagittal or frontal plane elevation.[24] Hence, the effect of cluster placement on the acromion and palpation inaccuracies on scapular tilt should be further explored using advanced processing and analyzing techniques. Meskers et al. (2007) already proposed combining cluster recordings with recordings from a scapula locator at the beginning of every measurement.[26] This could serve as a check or correction for possible orientation changes of the cluster. ROM of scapular lateral rotation showed a high preciseness (lowest %SEM) within and between sessions, stressing the value of this angle in shoulder assessment, clinical decision-making and evaluation of treatment efficacy. The better %SEM-values for lateral rotation were however not reflected in overall higher ICCs. This inconsistency could be explained by the lower heterogeneity in results for lateral rotation, typically resulting in lower ICCs.[16]

Apart from discrete joint angles, the similarity and measurement errors of angular waveforms were also assessed (CMCs and σ).[23,24] Higher CMCs were found for 120° elevation compared to 60° elevation, and forward flexion tasks showed better results than abduction tasks. Lateral rotation had highest CMCs for all tasks, in both patients and controls. For controls, lateral rotation was followed by protraction and tilting was least reliable during forward flexion. In

contrast, tilting was more reliable than protraction during abduction. The scapula at the patients' non-hemiplegic side performed similarly, while all rotations showed similar reliability at the hemiplegic side. The apparent differences in reliability between the three scapular rotations, based on the CMC, do not correspond to the waveforms' measurement errors. Forward flexion did result in systematically lower waveform errors compared to abduction, in both patients and controls. This discrepancy can be explained by the inherent dependency of the CMC on the amplitude of the waveforms. Rotations with small amplitude typically result in lower CMCs. For instance, the 60° tasks optimally require a scapular setting, i.e. stabilization and not movement, resulting in small amplitudes and hence lower CMCs. Movement amplitudes for scapular lateral rotation are twice those of tilting and protraction, resulting in higher CMCs. Forward flexion also elicits more scapular protraction compared to abduction, again resulting in higher CMCs for protraction in the former task.
A summary of the major clinical implications according to these reliability results is given in Table 6.
In literature, scapular joint angles during elevation have already been described in stroke patients. Meskers et al. reported less protraction at the non-hemiplegic side compared to the dominant side of controls at different degrees of forward flexion.[15] Niessen et al. further found increased lateral rotation in rest at the non-hemiplegic side and during elevation at the hemiplegic side in stroke patients with shoulder pain compared to those without shoulder pain or

Table 6. Implications for clinical use

- The proposed measurement protocol allows the reliable assessment of scapular angles at start position in healthy controls and stroke patients
- 120° tasks are most valuable for assessments of the full range of motion of scapular angles
- Forward flexion tasks are more reliable compared to abduction tasks for discrete joint angles and waveforms, especially for protraction, and are thus preferred to use for clinical interpretation and decision-making
- Measurement errors were lowest for lateral rotation, stressing the importance of this angle to assess i.e. treatment efficacy
- Scapular angles at the patients' hemiplegic arm and the controls' dominant arm show slightly higher reliability, and should preferably be used in future

controls.[14] Conversely, Hardwick and Lang described less lateral rotation at the hemiplegic side during active-assisted elevation.[12] Comparable literature on the reliability of scapular kinematics in stroke patients is however scarce. The few available reliability studies in stroke did not report both within and between session measurement errors, thereby failing to discriminate natural variation from methodological errors.[22,27] Moreover, the lack of consensus in applied methodology hinders proper result comparison. Van Andel et al. applied a similar marker set-up and protocol in healthy young adults and reported somewhat worse ICCs and SEMs of scapular rotations at 120° elevation compared to those reported in this study.[25] Additionally, Thigpen et al. also reported highest CMCs during sagittal plane elevation for all scapular rotations in healthy adults.[28] Roy et al. reported slightly higher ICCs and SEMs compared to our results,[29] though scapular angles were assessed during static arm positions in adults with and without impingement. Jaspers et al. reported higher ICCs and lower SEMs in children with cerebral palsy during reaching tasks, which might be explained by the rigorous standardization of the test set-up and task execution.[30]

The proposed measurement protocol allows the reliable assessment of scapular angles at start position in healthy controls and stroke patients. These angles are particularly interesting from a clinical viewpoint as arm movement dysfunctions often find their origin in altered scapular start positions. However, pain free shoulder movements additionally require adequate scapular behavior throughout task execution. The 120° tasks were most valuable for assessment of the full ROM of scapular angles. Whilst this restricts the protocol's applicability, it also helps identifying those stroke patients at risk to develop shoulder pathology and/or pain. Forward flexion tasks also resulted in higher reliability compared to abduction tasks, especially for protraction, and are thus preferably used for clinical interpretation and decision-making. Furthermore, measurement errors were lowest for lateral rotation, stressing the importance of this angle to assess i.e. treatment efficacy. Scapular angles at the patients' hemiplegic arm and the controls' dominant arm were slightly better reliable, probably due to the reduced degrees of freedom in hemiplegic arms and the more controlled performance of dominant arms.

However, the results of this feasibility and reliability study should be interpreted with care due to the limited sample size, together with the stroke patients' heterogeneity. Furthermore, this protocol is specifically designed for stroke patients with the ability to perform an active arm elevation. Therefore, this measurement method is limited to those patients with a moderate to mild motor impairment. A necessary future step in the analysis of scapular kinematics post stroke is the assessment of the discriminative ability of the proposed movement protocol. Factors such as age, pre-stroke hand dominance and time since stroke have been reported to impact on motor recovery and should therefore be taken into account.[31,32]

In conclusion, with the recommendations for task selection in mind, the measurement protocol is a valuable tool to assess scapular behavior and thereby contributes to the evaluation of arm dysfunction, the clinical decision-making and treatment planning in stroke patients.

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Table S1. Movement protocol: elevation tasks in order of performance

	Side		Elevation tasks
	Healthy Controls	Stroke patients	
Unilaterally	Dominant	Non-hemiplegic	Forward flexion 60°
	Non-dominant	Hemiplegic	Forward flexion 60°
	Dominant	Non-hemiplegic	Forward flexion 120°
	Non-dominant	Hemiplegic	Forward flexion 120°
	Dominant	Non-hemiplegic	Abduction 60°
	Non-dominant	Hemiplegic	Abduction 60°
	Dominant	Non-hemiplegic	Abduction 120°
	Non-dominant	Hemiplegic	Abduction 120°
Bilaterally	Both sides		Forward flexion 60°
	Both sides		Forward flexion 120
	Both sides		Abduction 60°
	Both sides		Abduction 120°

Table S2. Intraclass correlation coefficients for scapular angle range of motion (ROM) and for start position of the 120° forward flexion and abduction tasks.

	Forward flexion 60°		Forward flexion 120°				Abduction 60°		Abduction 120°			
	ROM		ROM		Start position		ROM		ROM		Start position	
	ICC_w	ICC_b	ICC_w	ICC_b	ICC_w	ICC_b	ICC_w	ICC_b	ICC_w	ICC_b	ICC_w	ICC_b
Controls dominant side												
Protraction	**0.87**	0.24	**0.89**	0.21	**0.97**	**0.88**	**0.81**	0.49	**0.94**	**0.78**	**0.99**	**0.85**
Lateral rotation	**0.83**	0.46	**0.88**	**0.74**	**0.96**	**0.69**	**0.92**	**0.62**	**0.88**	**0.67**	**0.96**	**0.73**
Tilt	**0.94**	0.21	**0.92**	**0.63**	**0.97**	**0.90**	**0.92**	0.45	**0.95**	**0.73**	**0.97**	**0.93**
Controls non-dominant side												
Protraction	**0.89**	0.19	**0.86**	0.56	**0.98**	**0.90**	**0.87**	Nan	**0.87**	0.39	**0.97**	**0.78**
Lateral rotation	**0.96**	**0.60**	**0.97**	**0.67**	**0.99**	**0.83**	**0.95**	Nan	**0.96**	**0.71**	**0.97**	**0.91**
Tilt	**0.90**	**0.64**	**0.96**	**0.79**	**0.92**	Nan	**0.80**	**0.81**	**0.79**	**0.70**	**0.93**	0.04
Stroke hemiplegic side												
Protraction	**0.93**	Nan	**0.98**	**0.65**	**0.99**	**0.76**	**0.84**	**0.64**	**0.96**	**0.80**	**0.95**	**0.69**
Lateral rotation	**0.95**	0.41	**0.98**	**0.95**	**0.97**	0.42	**0.84**	**0.68**	**0.97**	**0.81**	**0.92**	0.34
Tilt	**0.93**	Nan	**0.98**	**0.86**	**0.99**	**0.75**	**0.93**	0.11	**0.98**	**0.83**	**0.97**	**0.81**
Stroke non-hemiplegic side												
Protraction	**0.91**	**0.72**	**0.97**	**0.65**	**0.95**	0.46	0.54	Nan	**0.63**	0.50	**0.95**	0.56
Lateral rotation	**0.95**	Nan	**0.99**	0.06	**0.84**	**0.93**	**0.81**	**0.77**	**0.95**	0.02	**0.93**	**0.90**
Tilt	**0.93**	**0.74**	**0.97**	**0.92**	**0.95**	**0.98**	**0.86**	Nan	**0.94**	**0.82**	**0.92**	**0.93**

Continued

Table S2. Continued

	Bilat forward flexion 60° ROM		Bilat forward flexion 120° ROM		Bilat abduction 60° ROM		Bilat abduction 120° ROM	
	ICC_w	ICC_b	ICC_w	ICC_b	ICC_w	ICC_b	ICC_w	ICC_b
Controls dominant side								
Protraction	**0.83**	**0.64**	**0.91**	**0.69**	**0.87**	0.47	**0.88**	0.59
Lateral rotation	**0.77**	**0.78**	**0.93**	**0.83**	**0.92**	0.35	**0.89**	**0.69**
Tilt	**0.84**	0.50	**0.90**	0.20	**0.89**	0.56	**0.86**	**0.64**
Controls non-dominant side								
Protraction	**0.80**	0.30	**0.93**	0.37	**0.89**	0.43	**0.99**	0.15
Lateral rotation	**0.89**	0.56	**0.96**	**0.74**	**0.96**	Nan	**0.99**	0.51
Tilt	**0.91**	**0.76**	**0.96**	**0.90**	0.55	**0.77**	**0.96**	**0.62**
Stroke hemiplegic side								
Protraction	**0.95**	**0.88**	**0.97**	**0.84**	**0.90**	0.50	**0.94**	0.59
Lateral rotation	**0.96**	**0.71**	**0.94**	**0.91**	0.43	0.43	**0.91**	**0.67**
Tilt	**0.96**	**0.80**	**0.95**	**0.80**	**0.82**	Nan	**0.95**	0.26
Stroke non-hemiplegic side								
Protraction	**0.93**	**0.75**	**0.89**	**0.80**	**0.72**	0.05	**0.65**	0.56
Lateral rotation	**0.93**	0.18	**0.94**	Nan	**0.64**	**0.69**	**0.93**	0.17
Tilt	**0.97**	**0.79**	**0.91**	**0.84**	**0.81**	0.20	**0.90**	**0.91**

ICC_w: Intraclass correlation coefficients within session; ICC_b: Intraclass correlation coefficients between sessions; Bilat: Bilateral; Nan: Could not be calculated; ICCs higher than 0.60 are marked in bold

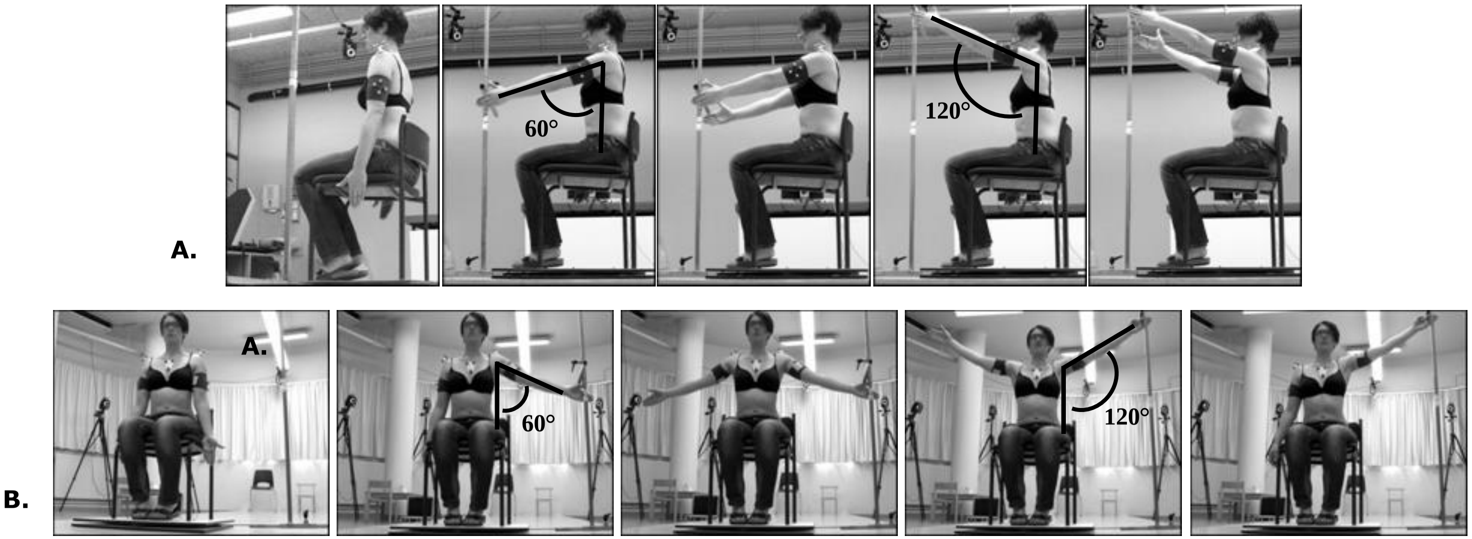
Table S3. The adjusted coefficient of multiple correlation for scapular waveforms.

	Forward flexion 60°		Forward flexion 120°		Abduction 60°		Abduction 120°	
	CMC_w	CMC_b	CMC_w	CMC_b	CMC_w	CMC_b	CMC_w	CMC_b
Controls dominant side								
Protraction	**0.97**	**0.85**	**0.97**	**0.76**	**0.71**	0.50	**0.89**	**0.62**
Lateral rotation	**0.95**	**0.75**	**0.99**	**0.96**	**0.96**	**0.78**	**0.99**	**0.94**
Tilt	**0.86**	**0.66**	**0.95**	**0.77**	**0.90**	**0.66**	**0.94**	**0.86**
Controls non-dominant side								
Protraction	**0.95**	**0.76**	**0.97**	**0.83**	**0.62**	0.54	**0.83**	0.51
Lateral rotation	**0.96**	**0.78**	**0.98**	**0.85**	**0.96**	**0.83**	**0.99**	**0.94**
Tilt	**0.87**	**0.72**	**0.88**	**0.66**	**0.78**	**0.64**	**0.90**	**0.73**
Stroke hemiplegic side								
Protraction	**0.86**	**0.66**	**0.98**	**0.71**	**0.76**	0.44	**0.93**	**0.65**
Lateral rotation	**0.95**	**0.83**	**0.99**	**0.94**	**0.94**	**0.80**	**0.99**	**0.96**
Tilt	**0.94**	**0.68**	**0.93**	**0.79**	**0.85**	**0.62**	**0.89**	0.55
Stroke non-hemiplegic side								
Protraction	**0.96**	**0.68**	**0.97**	**0.92**	**0.85**	0.45	**0.92**	**0.71**
Lateral rotation	**0.94**	**0.84**	**0.99**	**0.95**	**0.95**	**0.82**	**0.99**	**0.95**
Tilt	**0.91**	**0.62**	**0.95**	**0.77**	**0.83**	0.57	**0.96**	**0.75**

	Bilat forward flexion 60°		Bilat forward flexion 120°		Bilat abduction 60°		Bilat abduction 120°	
	CMC_w	CMC_b	CMC_w	CMC_b	CMC_w	CMC_b	CMC_w	CMC_b
Controls dominant side								
Protraction	**0.97**	**0.82**	**0.96**	**0.76**	**0.78**	0.44	**0.92**	**0.70**
Lateral rotation	**0.96**	**0.78**	**0.99**	**0.96**	**0.96**	**0.89**	**0.99**	**0.96**
Tilt	**0.87**	**0.62**	**0.91**	**0.66**	**0.79**	0.55	**0.94**	**0.75**
Controls non-dominant side								
Protraction	**0.98**	**0.79**	**0.98**	**0.85**	**0.80**	0.53	**0.84**	0.50
Lateral rotation	**0.93**	**0.74**	**0.98**	**0.95**	**0.97**	**0.81**	**0.99**	**0.96**
Tilt	**0.77**	**0.70**	**0.92**	**0.61**	**0.73**	**0.82**	**0.93**	**0.75**
Stroke hemiplegic side								
Protraction	**0.93**	**0.80**	**0.92**	**0.73**	**0.82**	**0.61**	**0.94**	**0.67**
Lateral rotation	**0.96**	**0.78**	**0.99**	**0.96**	**0.92**	**0.84**	**0.99**	**0.94**
Tilt	**0.94**	**0.77**	**0.97**	**0.83**	**0.89**	0.50	**0.96**	**0.75**
Stroke non-hemiplegic side								
Protraction	**0.94**	**0.78**	**0.96**	**0.94**	**0.83**	0.37	**0.86**	0.45
Lateral rotation	**0.95**	**0.82**	**0.99**	**0.97**	**0.94**	**0.85**	**0.99**	**0.95**
Tilt	**0.94**	**0.75**	**0.93**	**0.86**	**0.84**	0.42	**0.91**	**0.68**

CMC_w : adjusted coefficient of multiple correlation within session; CMC_b : adjusted coefficient of multiple correlation between sessions; Bilat: Bilateral; CMCs higher than 0.60 are marked in bold

Figure S1. Start position and execution of the different elevation tasks



Humerothoracic elevation (A) in the sagittal plane (forward flexion tasks) and (B) in the frontal plane (abduction tasks), executed from 0° to 60° and from 0° to 120°. Each elevation task was done unilaterally and bilaterally.

Chapter 3

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Characteristics of neuromuscular control of the scapula after stroke: a first exploration

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1. Abstract

This study aimed to characterize scapular muscle timing in stroke patients with and without shoulder pain. Muscle activity of upper trapezius, lower trapezius, serratus anterior, infraspinatus and anterior deltoid was measured (Delsys Trigno surface EMG system, US) in 14 healthy controls (dominant side) and 30 stroke patients (hemiplegic side) of whom 10 had impingement-like shoulder pain. Participants performed 45° and full range forward flexion, in two load conditions. The impact of group, forward flexion height, load condition and muscle was assessed for onset and offset of the different muscles relative to the onset and offset of anterior deltoid, using a 3 (group)* 2 (height)* 2 (load)* 4 (muscle) mixed model design. Recruitment patterns were additionally described. Across all load conditions and groups, serratus anterior had a significantly earlier onset and, together with lower trapezius, a significantly later offset in 45° compared to full range forward flexion tasks ($p<.001$). In stroke patients without pain, lower trapezius had furthermore a significantly earlier onset in comparison to stroke patients with shoulder pain (all tasks, $p=.04$). Serratus anterior also showed a significantly earlier offset in stroke patients with shoulder pain in comparison to controls ($p=.01$) and stroke patients without pain ($p<.001$). Analysis of muscle recruitment patterns indicated that for full range tasks, stroke patients without pain used early and prolonged activity of infraspinatus. In stroke patients with shoulder pain, recruitment patterns were characterized by delayed activation and early inactivity of serratus anterior. These timing results can serve as a reference frame for scapular muscle timing post-stroke, and when designing upper limb treatment protocols and clinical guidelines for shoulder pain after stroke.

2. Introduction

Shoulder pain is a common complication after stroke, affecting one-third of stroke patients in general. It not only negatively impacts on a patient's independency level and functional ability, but also impedes a successful rehabilitation.[1]

Apart from decreased glenohumeral motion, spasticity, subluxation and somatosensory impairments,[2-4] poor scapulothoracic position and aberrant scapulohumeral motion are also considered risk factors in the development of shoulder dysfunction and pain after stroke.[5,6] The ability to adapt the scapulothoracic position to the degree of humerothoracic movement during arm forward flexion relies upon adequate timing of specific scapular musculature. A stable scapulohumeral joint requires the synchronized control of the scapulothoracic and glenohumeral stabilizing muscles relative to prime mover muscle activity.[7,8] The association between altered scapular muscle activity, and the occurrence of impingement pain has been extensively studied in non-stroke subjects during arm elevation.[7,9-15] However, results of these studies are not conclusive. For example, Moraes et al. found a delayed onset of lower trapezius in patients with impingement,[14] whereas Worsley et al. additionally reported a delayed onset of serratus anterior, and an earlier offset of serratus anterior and lower trapezius,[15] and Padke and Ludewig furthermore reported an earlier activation of upper trapezius.[7] In contrast, Larsen et al. did not find any changes in onset time of these muscles in persons with impingement.[9] Moreover, apart from the discrepancies regarding muscle timing, results on shoulder muscle recruitment patterns are also highly variable. While several authors have reported a comparable pattern of muscle activation (upper trapezius activity, followed by serratus anterior and lower trapezius) in persons with and without shoulder pathology,[13,14,16] others did find alterations in recruitment patterns.[7] More specifically, in pain free persons, the latter authors reported activity of anterior deltoid, followed by serratus anterior, upper trapezius and lower trapezius during unloaded arm forward flexion.[7] In persons with impingement pain, upper trapezius activity was followed by anterior deltoid, serratus anterior, and lower trapezius.[7]

To our knowledge, no evidence currently exists on how residual motor impairments in stroke patients, who have developed the ability to perform isolated and selective arm movements, might create imbalances in scapulothoracic and scapulohumeral muscle coordination during upper limb tasks.[12] Such information could be of interest when studying the impact of the scapulothoracic and glenohumeral joints as contributing factors to upper limb (dys)functions (i.e. decreased range of motion) and pain after stroke. There is furthermore a lack of evidence regarding the influence of external load and forward flexion height on the activation patterns of scapular muscles post-stroke. An earlier activation and a delayed deactivation of lower trapezius and serratus anterior were already detected in loaded conditions during raising and lowering of the arm, respectively, in healthy persons and persons with impingement.[7]

Knowledge of typical temporal patterns of shoulder muscle activity in stroke patients with and without shoulder pain, and the influence of specific parameters, can be of value for physical therapists designing upper limb treatment programs. Therefore the goal of this study is to characterize the typical temporal patterns of scapular muscle activity in stroke patients with and without shoulder pain as compared to healthy controls, by means of electromyography during low and high, unloaded and loaded forward flexion tasks. We hypothesize altered recruitment patterns and muscle timing of stabilizing musculature in stroke patients with shoulder pain as compared to healthy controls and patients without shoulder pain. Furthermore, an earlier activation, and later deactivation of scapulothoracic musculature in high versus low forward flexion tasks and in loaded versus unloaded tasks, is hypothesized.

3. Methods

3.1. Participants

Stroke patients were recruited from different rehabilitation centers in Flanders (Belgium) and were considered eligible for participation in case they: (1) were at least 6 weeks after a first time stroke (cortical or subcortical lesion); (2) had mild to moderate upper limb motor impairment (score of ≥ 30 on the Fugl-Meyer upper limb motor part);[17] and (3) were able to perform 45° of active humerothoracic forward flexion (measured with goniometry). Stroke patients with shoulder pain were included when they: (1) experienced anterolateral shoulder pain during daily activities with a painful arc during 60 to 120° of arm forward flexion for at least four weeks since stroke onset; and (2) had a positive Neer impingement test, i.e. reported pain when the humeral greater tuberosity was impacted against the inferior acromion.[18] Healthy controls without self-reported shoulder pain were recruited via family and relatives. For all participants, following exclusion criteria were applied: (1) body mass index higher than 28; (2) inability to understand the instructions; (3) known history of shoulder and/or neck pain or discomfort in the last six months prior to stroke; (4) an event of shoulder dislocation, fracture or surgery during life time; or (5) other systemic and/or neurologic diseases. All stroke patients received standard care and physiotherapy, attuned to their specific needs. An overview of participants' characteristics is given in Table 1.

All participants gave informed consent, as approved by the Ethical Committee of the University Hospital Leuven (Belgium), prior to study participation.

Table 1. Participants' characteristics

		HEALTHY CONTROLS	SP WITHOUT SHOULDER PAIN	SP WITH SHOULDER PAIN
Subjects	number	14	20	10
	male/female	10/4	14/6	9/1
Age (yrs)	mean ± SD	61 ±11	59 ± 11	62 ± 21
Body Mass Index	mean ± SD	24 ± 2	25 ± 2	25 ± 2
Dominant side	left/right	2/12	1/19	1/9
Hemiplegic side	left/right	-	9/11	5/5
Time since stroke (wks)	mean ± SD	-	24 ± 18	22 ± 7
Lesion location	cortical/subcortical	-	15/5	8/2
Fugl-Meyer score*	mean ± SD	-	53 ± 7	51 ± 7

SP: stroke patients; *: upper limb motor section, maximal 66

3.2. Measurement Procedure

Scapular muscle activity was recorded using surface electromyography (Delsys Trigno EMG system, Boston, US) at the hemiplegic/dominant side. Following muscles were measured: upper trapezius (midpoint of the line between angulus acromialis and C7 processus spinosus), lower trapezius (at one third of the line between trigonum scapula and T8 processus spinosus), serratus anterior (anterior to the latissimus dorsi at the level of the scapular inferior angle), infraspinatus (approximately 4cm below the spine, over the infrascapular fossa, parallel to the scapular spine), and anterior deltoid (2-4cm below the lateral clavicula, parallel to the muscle fibers).[19,20] To ensure consistency of EMG-sensor position, these were placed by the same investigator for all participants. Prior to sensor placement, the skin over the muscle of interest was prepared and cleaned with alcohol. Muscle activity was recorded with a sampling rate of 2000 Hz. Correct sensor positioning and signal quality was verified by visual inspection of the EMG-signals during muscle specific movements.

The movement protocol consisted of forward flexion tasks from 0° to 45° and from 0° to maximal forward flexion, all executed while seated. These forward flexion tasks were performed under an unloaded condition and a loaded condition. Every task consisted of 12 consecutive repetitions. For the loaded conditions, a dumbbell weighting 1 to 1.25kg (calculated relative to body weight) was attached to the participants' wrist (average weight 1.15kg). A bar was placed in front of the patient at 45° of forward flexion to give visual information about the correct forward flexion height for this task. Participants were instructed to start with the elbow fully extended and thumb pointing upward, and to maintain this positioning during the forward flexion. To ensure correct task performance and a proper pace of task execution (1s up, 1s down, 3s rest for 45° tasks; 3s up, 3s down, 4s rest for full range tasks), each participant was given some practice trials.

3.3. Data-analysis

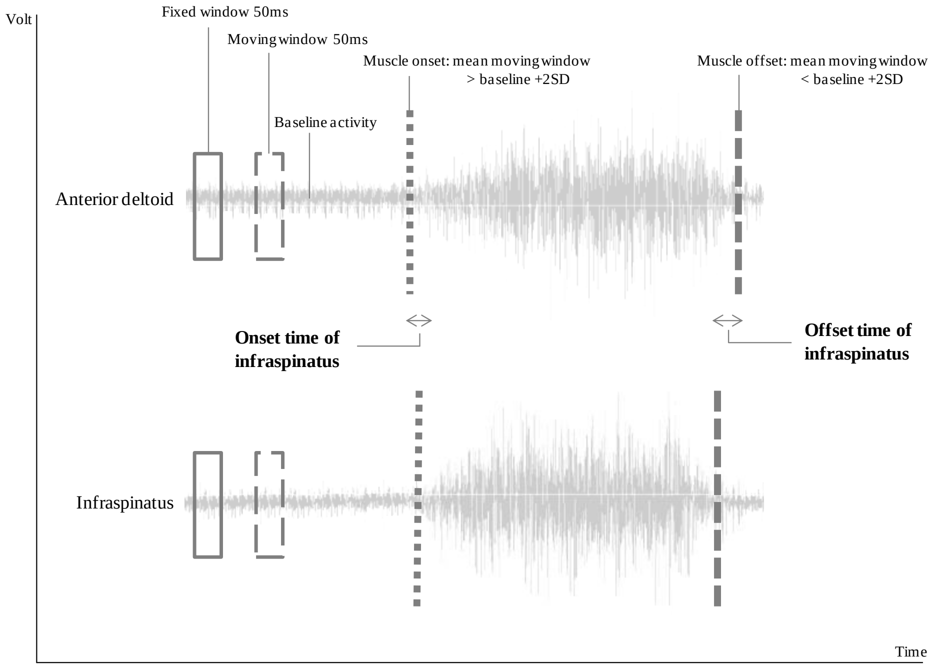
From the recorded trials, only the middle 10 repetitions were selected for data-analysis, as these were considered free from initiation or completion strategies. Raw EMG-data were first high-pass filtered with a 6th-order Butterworth filter of 20Hz to avoid movement and cardiac artifacts, and subsequently rectified and filtered with a low-pass filter (cut-off frequency of 45Hz) to smooth the data. Both filters were implemented as bidirectional filters to reduce the phase error.

Onset of muscle activity was defined as an increase in muscle activity of more than 2 standard deviations on top of the mean baseline activity (as recorded for 10s prior to movement start) for at least 50ms. Muscular offset was reached when the recorded activity was lower than the mean baseline activity plus two standard deviations for at least 50ms.[21] Each calculated onset and offset was visually checked for erroneous signals due to cardiac or other motion artifacts.[22]

Parameters of interest were: (1) time of muscular onset, and (2) time of muscular offset of stabilizing musculature (upper trapezius, lower trapezius, serratus anterior and infraspinatus) relative to time of the onset and offset respectively of the prime mover for forward flexion (anterior deltoid) (Figure 1). Data of these two timing parameters were compared between the different groups, height and load conditions, and muscles. A positive latency time (ms) indicated activity prior to anterior deltoid onset and inactivity prior to anterior deltoid offset.

Lastly, the sequence in time of onset and time of offset of the different muscles was analyzed per group (based on average group data). Afterwards, sequences of recruitment were further explored for each task at the level of the individual subject within each group. Given that the sampling rate of the EMG-signal was set at 2000 Hz (which implies a time resolution of 0.5ms), time intervals could theoretically be measured with an accuracy of 1ms. Even though bidirectional filters were used to reduce timing errors, an uncertainty error of 4ms was taken into account. Therefore, the accuracy for determining the onset time was estimated at 10ms, meaning that the difference in timing between two muscles within one individual had to exceed 10ms to represent real difference in recruitment timing.

Figure 1. Visual representation of the calculation of muscle onset and offset



In this example, infraspinatus was active after the onset of anterior deltoid, resulting in a negative onset time, and inactive before the offset of the anterior deltoid, resulting in a positive offset time.

3.4. Statistical analysis

Differences in onset and offset timing between groups (stroke patients without shoulder pain, stroke patients with shoulder pain and controls), forward flexion heights (45°, full range), load conditions (unloaded, loaded) and muscles (infraspinatus, upper trapezius, lower trapezius and serratus anterior) were analysed using a 3 (group) x 2 (height) x 2 (load) x 4 (muscle) Mixed Model.[23] As such, four main effects and six interaction effects were calculated for onset timing and offset timing separately. The use of mixed model analysis was preferred as this analysis is robust in analysing semi-normally distributed data,[24] allows for repeated measures analysis, and can handle missing data (which was the case for on/offset data of some participants). All statistics were done in SAS software version 9.4 Foundation and enterprise guide.

4. Results

4.1. Timing parameters of stabilizing muscles relative to anterior deltoid timing

In three stroke patients with shoulder pain, no onset/offset could be detected for serratus anterior and lower trapezius in 45° forward flexion tasks and data for these muscles in this task was limited to 7 patients only.

A significant main effect for height ($p<.001$) and muscle ($p<.0001$), and a significant interaction effect for height*muscle ($p=.02$) were observed for onset and offset.

Post-hoc analysis of the height*muscle interaction effect for onset indicated that, for all load conditions and groups, all muscles had a significantly different onset (all $p<.005$). Only serratus anterior and lower trapezius (both heights), and serratus anterior and infraspinatus (45° task) did not differ significantly from each other . Post-hoc analysis further indicated that only serratus anterior had a significant earlier onset in 45° compared to full range tasks ($p<.0001$).

Post-hoc analysis of the height*muscle interaction effect for offset indicated that, over all load conditions and groups, all muscles had a significantly different offset ($p<.005$) except for infraspinatus and upper trapezius (both heights). This analysis also showed that only serratus anterior and lower trapezius had a significant earlier offset in full range compared to 45° forward flexion tasks ($p<.001$).

A significant group*muscle interaction effect was also found for onset and offset. Post-hoc analysis for onset firstly indicated that, over all tasks, lower trapezius had a significant earlier onset in stroke patients without pain than in stroke patients with shoulder pain ($p=.04$). Secondly, for each group, all muscles showed a significantly different onset ($p<.001$), except for (1) infraspinatus and serratus anterior in controls, (2) infraspinatus and lower trapezius, and serratus anterior and lower trapezius in stroke patients without shoulder pain, and (3) serratus anterior and lower trapezius in stroke patients with shoulder pain.

Post-hoc analysis for offset indicated that, over all tasks, serratus anterior had a significant earlier offset in stroke patients with shoulder pain compared to controls ($p=.01$) and stroke patients without shoulder pain ($p<.001$). Furthermore, all muscles showed a significantly different offset for every group ($p<.001$), except for (1) infraspinatus and serratus anterior, and infraspinatus and upper trapezius in controls, (2) infraspinatus and upper trapezius in stroke patients without shoulder pain, and (3) serratus anterior and lower trapezius, and infraspinatus and upper trapezius in stroke patients with shoulder pain.

4.2. Recruitment patterns

4.2.1. Recruitment patterns based on average group data

In controls and stroke patients with shoulder pain, upper trapezius was activated first during each task, while lower trapezius was activated last. In stroke patients without shoulder pain, upper trapezius was also active first, but lower trapezius was activated last only during the 45°, unloaded task. In all other

tasks, serratus anterior was activated last in this group. The difference in onset between upper trapezius and lower trapezius, and upper trapezius and serratus anterior was significant across all groups and tasks (all p<.001).
In every group, lower trapezius was also the first muscle that was inactive and upper trapezius the last. Only in stroke patients with shoulder pain, during the 45° tasks, serratus anterior was inactive first (in the loaded condition together with lower trapezius), while in the full range loaded task infraspinatus was inactive last. The difference in offset between upper trapezius and lower trapezius, upper trapezius and serratus anterior, and lower trapezius and infraspinatus was significant across all groups and tasks (all p<.0001). Recruitment patterns based on average group data are visualized for every task in Supplementary material (appendix 1).

4.2.2. Muscle recruitment patterns based on individual data

Due to large standard deviations of group onset/offset data (Table 2), recruitment patterns were further explored based on individual muscle activation patterns per task. This was accomplished by categorizing each muscle's onset/offset timing as activity/inactivity before, after or together with the onset/offset of anterior deltoid. Results of this individual data exploration are found in Table 3. As group differences in average onset and offset recruitment patterns were mainly found in the sequence of infraspinatus, serratus anterior and anterior deltoid, we will focus on these muscles only.
Concerning onset timing for the 45° forward flexion tasks, serratus anterior activity prior to or together with anterior deltoid was seen in 67% (unloaded) and 73% (loaded) of controls, in 67% (unloaded) and 31% (loaded) of stroke patients without shoulder pain, and in 38% (unloaded) and 22% (loaded) of patients with shoulder pain. Infraspinatus was active before or together with anterior deltoid in 45% (unloaded) and 31% (loaded) of controls, in 65% (unloaded) and 45% (loaded) of stroke patients without shoulder pain, and in 80% (unloaded) and 60% (loaded) of stroke patients with shoulder pain.
For full range forward flexion tasks, serratus anterior activated before or together with the anterior deltoid in 39% (unloaded and loaded) of controls, in 17% (unloaded) and 33% (loaded) of stroke patients without shoulder pain, and in 10% (unloaded and loaded) of patients with shoulder pain. Infraspinatus was active prior to or together with anterior deltoid in 36% (unloaded) and 21% (loaded) of controls, in 70% (unloaded) and 42% (loaded) of stroke patients without shoulder pain and in 50% (unloaded) and 30% (loaded) of stroke patients with shoulder pain.
For offset timing during the 45° forward flexion tasks, serratus anterior was inactive before or together with the offset of anterior deltoid in 55% (unloaded and loaded) of controls, in 28% (unloaded) and 35% (loaded) of stroke patients without shoulder pain, and in 50% (unloaded) and 67% (loaded) of patients with shoulder pain. Infraspinatus was inactive before or together with anterior deltoid in 66% (unloaded) and 54% (loaded) of controls, in 32% (unloaded) and 45%

Table 2. Mean (X) with standard deviation (SD) of timing parameters for the all forward flexion tasks, expressed in milliseconds

		controls		Stroke No pain		Stroke Pain	
		45° FORWARD FLEXION, UNLOADED					
		controls		Stroke No pain		Stroke Pain	
		ON	OFF	ON	OFF	ON	OFF
UT	X (SD)	115(219)	-70(341)	75(168)	-371(557)	152(218)	-151(415)
IF	X (SD)	-63(143)	80(351)	8(89)	-231(402)	-8(241)	-145(444)
LT	X (SD)	-236(201)	419(318)	-35(152)	290(440)	-135(134)	338(388)
SA	X (SD)	48(144)	114(437)	-7(162)	-307(495)	-5(255)	380(639)
		FULL RANGE FORWARD FLEXION, UNLOADED					
		controls		Stroke No pain		Stroke Pain	
		ON	OFF	ON	OFF	ON	OFF
UT	X (SD)	254(336)	-151(733)	115(252)	-351(257)	114(245)	-74(163)
IF	X (SD)	-38(297)	-29(546)	5(204)	-276(460)	-19(205)	20(284)
LT	X (SD)	-265(281)	789(794)	-140(173)	843(614)	-398(704)	889(884)
SA	X (SD)	164(553)	421(906)	-321(335)	240(717)	-279(836)	836(502)
		45° FORWARD FLEXION, LOADED					
		controls		Stroke No pain		Stroke Pain	
		ON	OFF	ON	OFF	ON	OFF
UT	X (SD)	79(133)	-97(271)	35(128)	-162(502)	178(229)	-235(474)
IF	X (SD)	-80(121)	26(260)	-36(143)	-128(368)	10(201)	-99(503)
LT	X (SD)	-172(169)	305(280)	-92(175)	419(326)	-246(254)	436(551)
SA	X (SD)	29(133)	15(382)	-143(179)	-108(550)	-119(249)	429(206)
		45° FORWARD FLEXION, LOADED					
		controls		Stroke No pain		Stroke Pain	
		ON	OFF	ON	OFF	ON	OFF
UT	X (SD)	56(370)	-49(654)	-24(146)	-224(647)	122(280)	-364(491)
IF	X (SD)	-69(290)	-30(460)	-151(499)	-261(439)	-34(205)	-12(560)
LT	X (SD)	-206(296)	666(617)	-96(142)	639(682)	-249(500)	985(801)
SA	X (SD)	-84(385)	237(680)	-184(253)	236(743)	-352(432)	766(748)

UT: Upper trapezius; IF: Infraspinatus; LT: Lower trapezius; SA: Serratus anterior

Table 3. Group-specific classifications of onset and offset time relative to anterior deltoid (AD) onset and offset time per task

		45° FORWARD FLEXION UNLOADED			45° FORWARD FLEXION LOADED		
		Controls	Stroke No pain	Stroke Pain	Controls	Stroke No pain	Stroke Pain
ONSET							
UT	Before AD	75%	67%	80%	67%	56%	80%
	After AD	25%	33%	20%	33%	44%	20%
	Together with AD	-	-	-	-	-	-
LT	Before AD	10%	43%	10%	8%	31%	50%
	After AD	90%	57%	60%	92%	69%	20%
	Together with AD	-	-	-	-	-	-
	No onset	-	-	30%	-	-	30%
SA	Before AD	67%	67%	38%	73%	25%	22%
	After AD	33%	33%	25%	27%	69%	45%
	Together with AD	-	-	-	-	6%	-
	No onset	-	-	37%	-	-	33%
IF	Before AD	45%	53%	50%	31%	35%	40%
	After AD	55%	35%	20%	69%	55%	40%
	Together with AD	-	12%	30%	-	10%	20%
OFFSET							
UT	Before AD	42%	26%	40%	42%	31%	30%
	After AD	58%	74%	60%	58%	69%	70%
	Together with AD	-	-	-	-	-	-
LT	Before AD	90%	71%	40%	75%	89%	50%
	After AD	10%	21%	30%	25%	11%	20%
	Together with AD	-	8%	-	-	-	-
	No onset	-	-	30%	-	-	30%
SA	Before AD	55%	28%	50%	55%	35%	67%
	After AD	45%	72%	13%	45%	65%	-
	Together with AD	-	-	-	-	-	-
	No onset	-	-	37%	-	-	33%
IF	Before AD	66%	32%	50%	54%	45%	50%
	After AD	34%	68%	50%	46%	55%	40%
	Together with AD	-	-	-	-	-	10%

Continued

Table 3. Continued

		FULL FORWARD FLEXION UNLOADED			FULL FORWARD FLEXION LOADED		
		Controls	Stroke No pain	Stroke Pain	Controls	Stroke No pain	Stroke Pain
ONSET							
UT	Before AD	71%	71%	30%	43%	61%	60%
	After AD	29%	29%	70%	50%	39%	40%
	Together with AD	-	-	-	7%	-	-
LT	Before AD	14%	22%	30%	21%	22%	30%
	After AD	86%	72%	70%	79%	78%	70%
	Together with AD	-	6%	-	-	-	-
	No onset	-	-	-	-	-	-
SA	Before AD	39%	17%	10%	39%	22%	10%
	After AD	61%	83%	90%	61%	67%	90%
	Together with AD	-	-	-	-	11%	-
	No onset	-	-	-	-	-	-
IF	Before AD	21%	60%	20%	14%	37%	20%
	After AD	64%	30%	50%	79%	58%	70%
	Together with AD	15%	10%	30%	7%	5%	10%
OFFSET							
UT	Before AD	43%	6%	30%	50%	37%	30%
	After AD	57%	94%	70%	50%	63%	70%
	Together with AD	-	-	-	-	-	-
LT	Before AD	93%	94%	90%	86%	84%	100%
	After AD	7%	6%	10%	14%	16%	-
	Together with AD	-	-	-	-	-	-
	No onset	-	-	-	-	-	-
SA	Before AD	77%	69%	100%	69%	58%	90%
	After AD	23%	31%	-	31%	42%	10%
	Together with AD	-	-	-	-	-	-
	No onset	-	-	-	-	-	-
IF	Before AD	43%	26%	70%	43%	39%	70%
	After AD	50%	74%	20%	50%	61%	30%
	Together with AD	7%	-	10%	7%	-	-

UT: Upper trapezius; IF: Infraspinatus; LT: Lower trapezius; SA: Serratus anterior

(loaded) of stroke patients without shoulder pain and in 50% (unloaded) and 60% (loaded) of stroke patients with shoulder pain.

In full range tasks, serratus anterior was inactive before or together with the offset of anterior deltoid in 77% (unloaded) and 69% (loaded) of controls, in 69% (unloaded) and 58% (loaded) of stroke patients without shoulder pain, and in 100% (unloaded) and 90% (loaded) of patients with shoulder pain. Infraspinatus stopped before or together with anterior deltoid in 50% (unloaded and loaded) of controls, in 26% (unloaded) and 39% (loaded) of stroke patients without shoulder pain and in 80% (unloaded) and 70% (loaded) of stroke patients with shoulder pain.

5. Discussion

When moving the arm, our muscles exhibit a feed-forward or anticipatory control activity to ensure that the scapular position is adapted to the humeral position. As such, the scapula serves as a stable base for arm forward flexion. Motor control of the shoulder relies on a synchronized activation of the upper trapezius, lower trapezius and serratus anterior to upwardly rotate the scapula.[25,26] Next, this scapulothoracic force couple works in coordination with the humeral elevators (anterior deltoid) and thereby offers an optimal tension-force relation for glenohumeral muscles such as infraspinatus. Infraspinatus activity counterbalances the upward force of the anterior deltoid on the humerus, and thus centers the humeral head in the glenoid fossa. As post-stroke hemiparesis might provide inadequate conditions for selective muscle activation, this study wanted to assess alterations in this feed-forward control in stroke patients and investigate their relation to the development of shoulder pain by means of muscular timing assessments (EMG). Additionally, the impact of forward flexion height and load was assessed. Tasks were chosen based on the clinical evaluation of the shoulder and their relevance in daily life.

Across all tasks, we found a delayed onset for lower trapezius in stroke patients with shoulder pain as compared to stroke patients without shoulder pain. Inspection of the individual recruitment patterns further indicated that, especially in the low forward flexion tasks, a high percentage of stroke patients with pain had no or a delayed serratus anterior onset. This was not seen in controls or stroke patients without pain. Serratus anterior was moreover earlier inactive in stroke patients with shoulder pain compared to controls and stroke patients without shoulder pain, across all tasks. This was also confirmed in the inspection of individual recruitment patterns, i.e. more stroke patients with shoulder pain showed inactivity of serratus anterior before the offset of anterior deltoid, compared to the other two groups. The alterations in stroke patients with shoulder pain are quite similar to the delayed serratus anterior and lower trapezius onset,[14,15] and earlier serratus anterior offset seen in persons with impingement.[15] The pull of serratus anterior and lower trapezius ensures a stable scapulothoracic joint and seems necessary to reduce the risk for subacromial impingement, probably by providing adequate scapular lateral rotation and posterior tilting.[11] This pull might furthermore allow for proper muscle activation of the rotator cuff. It appears that stroke patients without shoulder pain use a strategy of early infraspinatus activity, in comparison to stroke patients with shoulder pain and healthy controls. They also showed a longer activity of the infraspinatus relative to anterior deltoid's offset, as compared to stroke patients with shoulder pain. These results point towards the role of the timing of the infraspinatus to adequately stabilize the shoulder and prevent shoulder pain in stroke patients. The stereotyped pattern of early and prolonged infraspinatus activation is thus likely to guarantee sufficient subacromial space, probably by pulling the humerus downward during forward flexion, and this knowledge should be taken into account during rehabilitation to prevent impingement.

In contrast to our hypothesis and other studies who reported alterations in timing of lower trapezius and serratus anterior during loaded arm forward flexion in healthy persons and persons with impingement,[7] our study could not identify significant effects of the loading. However, we did show effects of forward flexion height. Across all groups, serratus anterior was earlier active, and, together with lower trapezius, later inactive in low versus high tasks. These results additionally stress the importance of the serratus anterior and lower trapezius, especially during low forward flexion tasks, to keep the scapula still on the thorax, i.e. to ensure scapular setting. Since subacromial impingement is already possible at low forward flexion angles,[27] this setting is essential to avoid compression of soft tissue structures in the subacromial space. The early activation of serratus anterior activates and its prolonged activity in low versus high forward flexion tasks might counteract the passive scapular movement caused by posterior glenohumeral structures (e.g. joint capsular structures, teres major, lattisimus dorsi, infraspinatus) when the humerus moves, and thereby keep the scapula still on the thorax.

The lack of uniform data on muscle recruitment patterns in healthy controls and persons with impingement in previous literature[12,13] and the high inter-subject variability we found within each study group clearly show that it is not recommended to describe typical recruitment patterns based on average data. We therefore considered the individual-specific patterns of recruitment to further interpret our data and compare this to previous literature. Such approach has been used recently to describe scapulothoracic kinematics in healthy controls.[28]

A limitation of the current study is that we only included stroke patients who had developed the ability to move outside the synergetic pattern and to perform analytical forward flexion movements. As a consequence, results should not be generalized to patients who move in the abnormal, stereotypical pattern of elbow flexion with shoulder abduction-extension-external rotation, or elbow extension with adduction-forward flexion-internal rotation.[29] Furthermore, we did not account for the side and type of stroke or the amount of brain damage. Lastly, the use of surface EMG is prone to signal noise by motion or cardiac artifacts, which resulted in some data loss, i.e. 15% lost trials for upper trapezius, 16% for lower trapezius, 21% for serratus anterior, and 7% for infraspinatus. Combining EMG data with scapular kinematics is indispensable to further increase our knowledge of upper limb recovery post-stroke, and to clarify why some patients do and others do not develop compensations and/or shoulder pain.

Current study results paved the road to gain a deeper understanding on alterations in recruitment and the emergence of compensatory strategies in the scapular and glenohumeral stabilizers post-stroke. We identified the presence of compensatory motor control of infraspinatus during arm forward flexion in stroke patients without shoulder pain. Our results furthermore indicated that

stroke patients with shoulder pain should relearn scapular motor control, mainly of the serratus anterior and lower trapezius to address the scapular setting and to restore dynamic stability while doing arm movements. Future treatment guidelines for scapular motor control and shoulder muscle strengthening should moreover consider the impact of a task demand, i.e. reaching height. As important stabilizers are already active in low forward flexion tasks to perform a scapular setting, this task is deemed highly suited for the training of temporal muscle timing early after stroke when patients may still have mobility problems in higher degrees. Motivating patients to perform arm forward flexion tasks in higher degrees in occupational or physical therapy should be done cautiously and only after a good control of scapular setting and rotation in lower forward flexion tasks has been established.
This knowledge has the potential to offer a useful way for clinicians to prescribe appropriate therapeutic management strategies and for researchers to enhance knowledge in relation to this clinical challenge.

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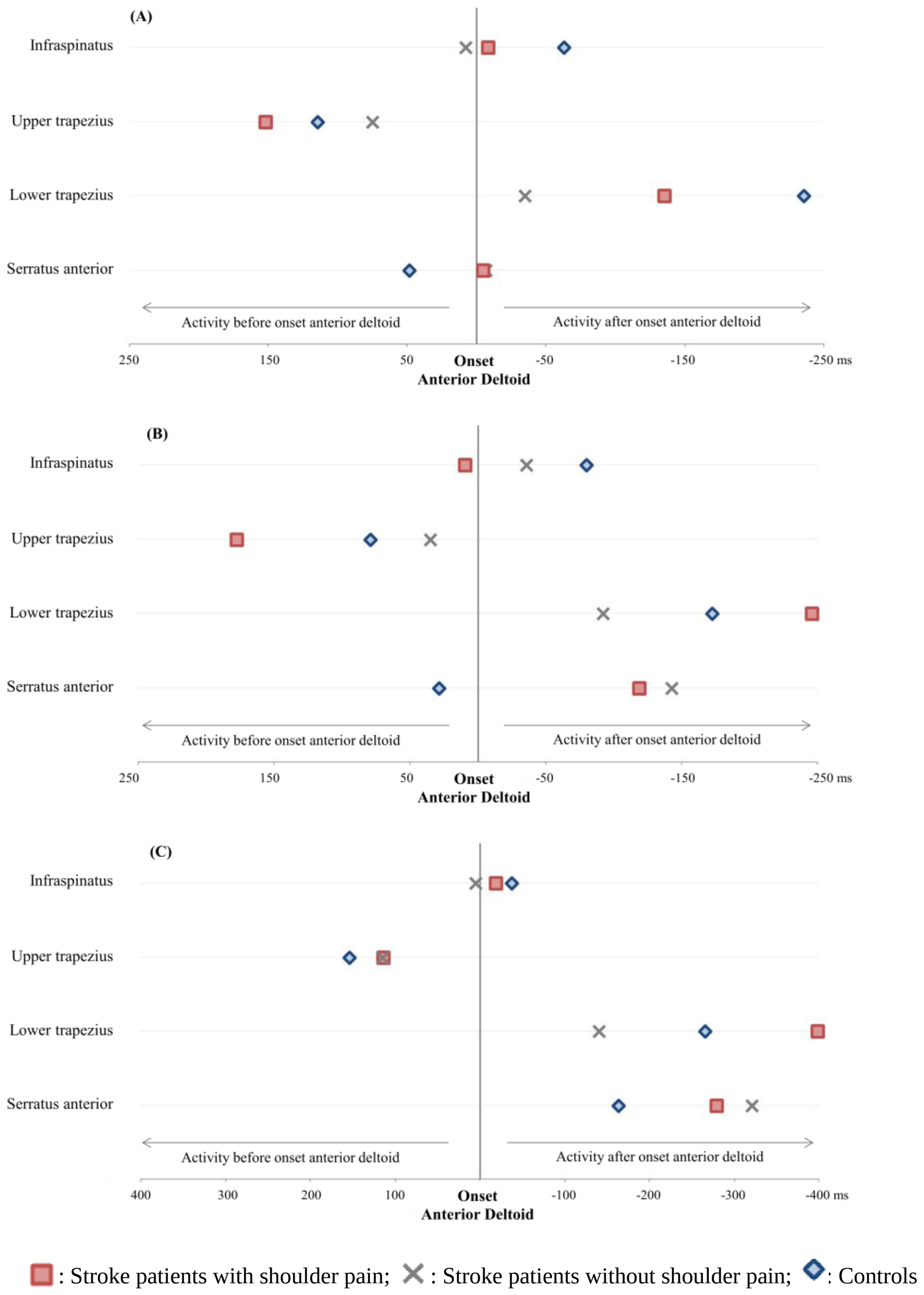
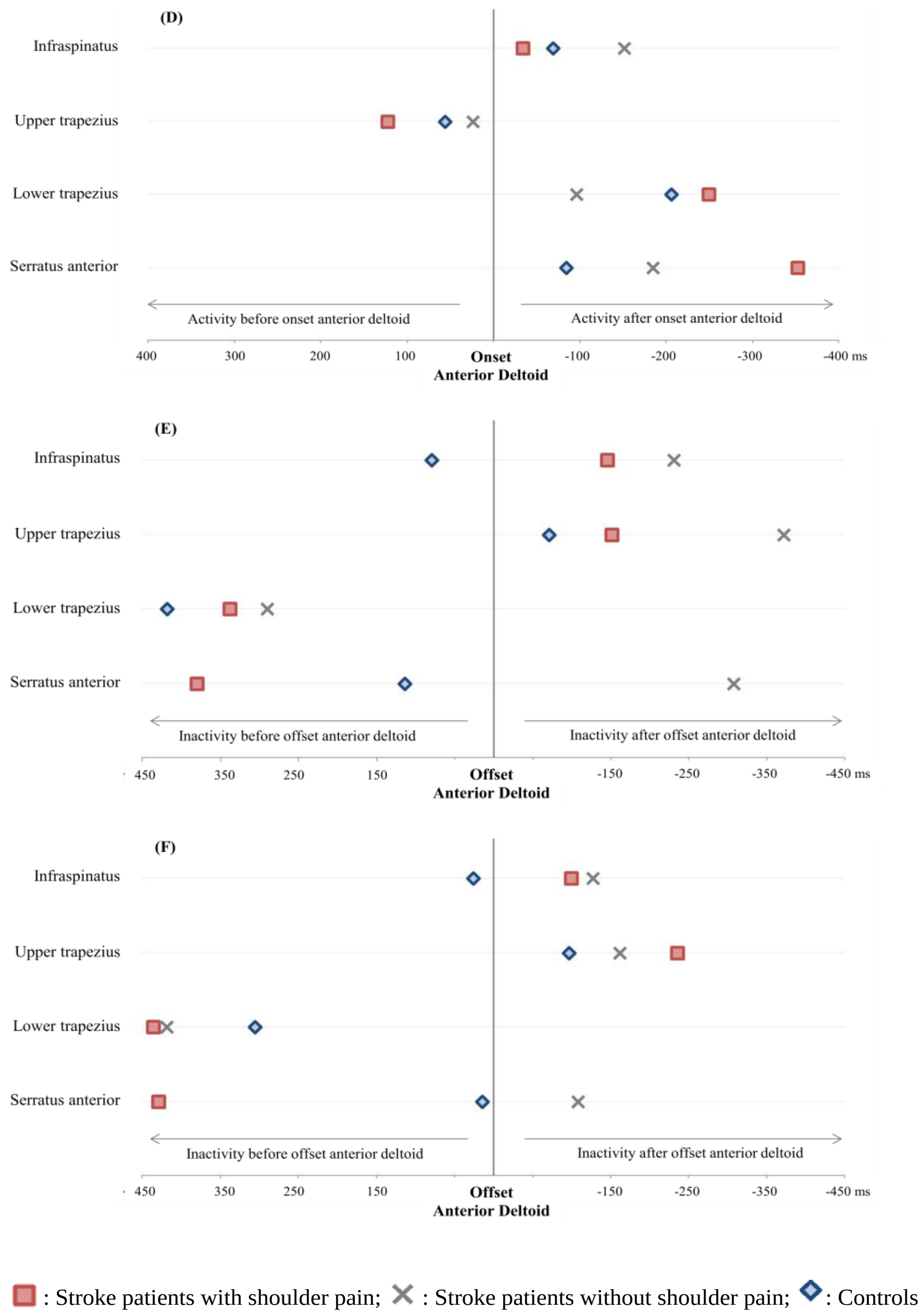
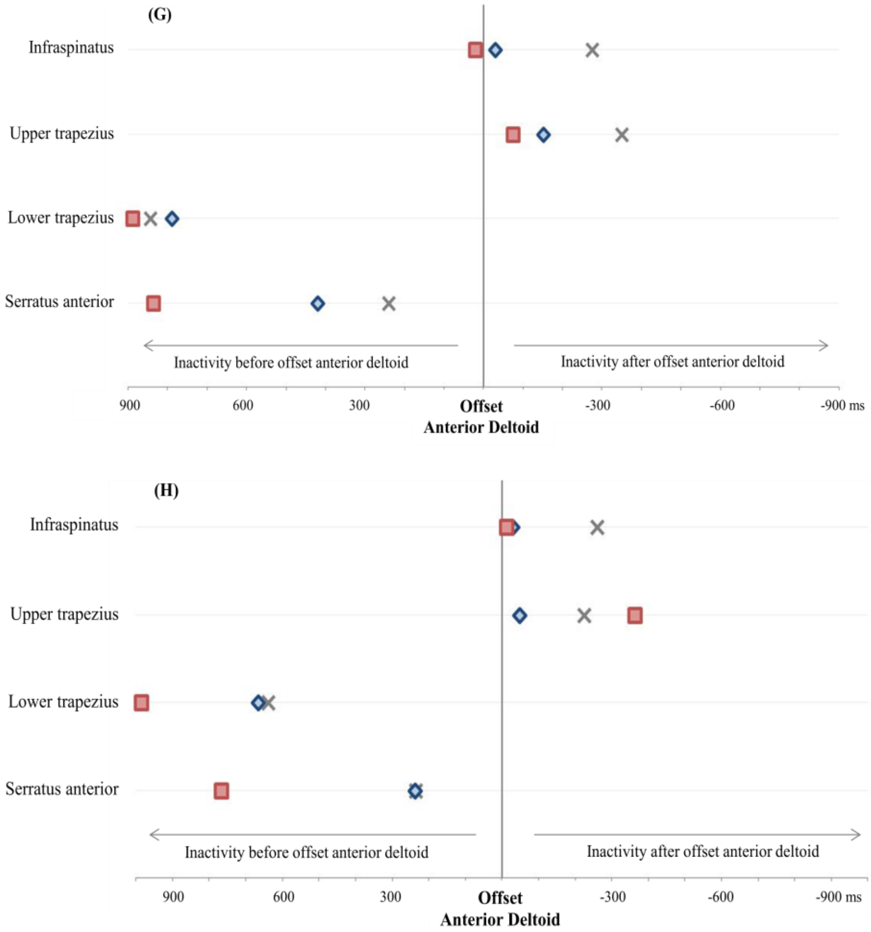
Appendix 1. Average onset (A-D) and offset (E-H) relative to anterior deltoid onset and offset in 45°, unloaded forward flexion (A, E); 45°, loaded forward flexion (B, F); full range, unloaded forward flexion (C, G); full range, loaded forward flexion (D, H)




(D)
Infraspinatus
Upper trapezius
Lower trapezius
Serratus anterior
Activity before onset anterior deltoid
Activity after onset anterior deltoid
400 300 200 100
Onset
Anterior Deltoid
-100 -200 -300 -400 ms
(E)
Infraspinatus
Upper trapezius
Lower trapezius
Serratus anterior
Inactivity before offset anterior deltoid
Inactivity after offset anterior deltoid
450 350 250 150
Offset
Anterior Deltoid
-150 -250 -350 -450 ms
(F)
Infraspinatus
Upper trapezius
Lower trapezius
Serratus anterior
Inactivity before offset anterior deltoid
Inactivity after offset anterior deltoid
450 350 250 150
Offset
Anterior Deltoid
-150 -250 -350 -450 ms
: Stroke patients with shoulder pain; : Stroke patients without shoulder pain; : Controls



: Stroke patients with shoulder pain; : Stroke patients without shoulder pain; : Controls

Chapter 4

In preparation for Clinical Biomechanics

Three-dimensional kinematics of the scapula and trunk, and associated scapular muscle timing in stroke patients

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1. Abstract

This study characterizes three-dimensional (3D) kinematics of the trunk and scapulothoracic joint, and scapular muscle timing in individuals with stroke (IwS). Trunk and scapular 3D kinematics and surface muscle activity of upper and lower trapezius, serratus anterior, infraspinatus and anterior deltoid were measured in 15 healthy controls (dominant side) and 18 IwS (hemiplegic side). Participants performed a low and high forward flexion (FF) task. Group differences in range of motion (ROM) of the scapula and trunk were assessed at 45° (low FF task) and 90° (high FF task) using a t-test for independent samples. Differences in muscle onset and offset time relative to movement start (both FF tasks) were determined using a mixed model taking into account the different groups and muscles. Recruitment patterns per group and task were furthermore described based on significant differences between muscles. In IwS, earlier lower trapezius and longer infraspinatus activity was found in the low task, as well as a later onset and earlier offset of the serratus anterior. For the low FF task, we also found significantly more trunk axial internal rotation in IwS during both the elevation and lowering phase. In the high FF task, IwS showed significantly less scapular posterior tilt during elevation and significantly more scapular lateral rotation during arm lowering. IwS also demonstrated adaptive muscle timing with earlier initiation and prolonged activation of lower trapezius and infraspinatus. These timing results together with the alterations in kinematic characteristics can serve as a reference for scapular behavior post-stroke.

2. Introduction

Individuals with stroke (IwS) frequently experience loss of voluntary motor control, reducing their ability for selective muscle recruitment. Shoulder dysfunctions are, with a reported prevalence between 48% and 77%,[1,2] commonly associated with stroke and strongly impact on daily life functioning of these patients.[3] From early stages in rehabilitation, adequate strategies to prevent or reduce shoulder function losses are thus imperative. However, adequate treatment can only be attained via a meaningful and comprehensive assessment.

Since correct shoulder function is dependent on proper scapulothoracic functioning, shoulder assessment should include the scapulothoracic joint. During arm elevation in healthy persons, the scapula moves toward lateral rotation and posterior tilting, and depending on the elevation plane, both scapulothoracic protraction and retraction have been reported, (Figure 1A).[4] To achieve such a 3D character of scapulothoracic movement, coordinated muscle activation of scapulothoracic stabilizers is required, i.e. serratus anterior, upper and lower trapezius.[5] Furthermore, it is known that a proper spinal posture also contributes to correct scapulothoracic motion and muscle activity during humeral elevation.[6] Hence, advanced assessment methods like 3D movement analysis with synchronized electromyography (EMG) might offer further insights into the scapulothoracic joint in IwS.

Studies thus far reported discrete but inconclusive alterations in 3D scapulothoracic kinematics post-stroke.[7-12] Niessen et al. (2008) reported increased lateral rotation in IwS with shoulder pain compared to controls, whereas Hardwick et al. (2011) reported only a trend toward a decreased scapular lateral rotation in IwS.[9,11] One study reported early activity of lower trapezius and delayed inactivity of serratus anterior in IwS without shoulder pain, compared to those with shoulder pain.[13] Unfortunately, no evidence exists on the combined assessment of 3D scapular kinematics and muscle timing post-stroke, nor on the effect of spinal posture hereon. Such information is essential when studying the role of the scapulothoracic joint with respect to upper limb (dys)functions (i.e. decreased range of motion) in IwS. Here, we investigated trunk and scapula kinematics, and synchronized scapular muscle timing, in IwS compared to a group of age-matched healthy controls.

3. Methods

3.1. Participants

IwS without shoulder pain were recruited from the University Hospital Pellenberg (Belgium) and were considered eligible for participation in case they: (1) were at least 6 weeks after a first time stroke (cortical or subcortical lesion); (2) had mild to moderate upper limb motor impairment (score of ≥ 30/66 on the Fugl-Meyer upper limb motor part);[14] and (3) were able to perform 45° of active humerothoracic forward flexion (FF) (measured with goniometry). Healthy controls without self-reported shoulder pain were recruited via family and relatives. For all participants, following exclusion criteria were applied: (1) body mass index (kg/m²) > 28; (2) inability to understand the instructions; (3) known history of shoulder and/or neck pain or discomfort in the last six months prior to stroke; (4) an event of shoulder dislocation, fracture or surgery during life time; (5) other systemic and/or neurologic diseases. All IwS received standard care and physiotherapy, attuned to their specific needs. An overview of participants' characteristics is given in Table 1. All participants gave informed consent prior to study participation, as approved by the Ethical Committee of the University Hospital Leuven (Belgium).

Table 1. Participants' characteristics

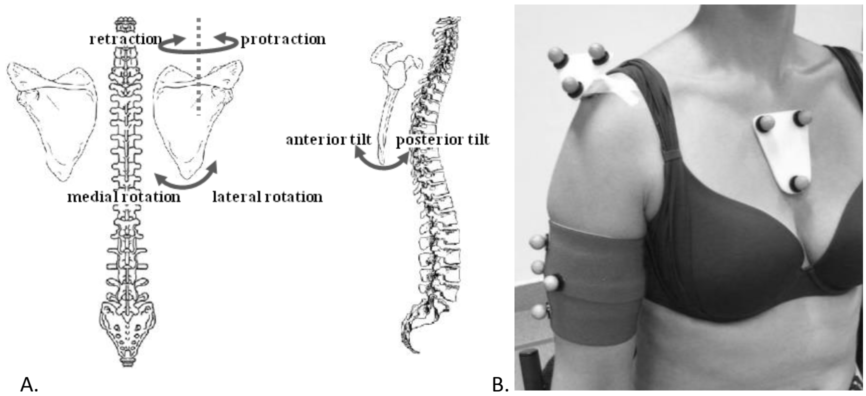
		IwS	Controls
Participants	N (male/female)	18 (12/6)	15 (8/7)
Age (years)	mean ± SD	51.8 ± 13.3	51.7 ± 13.1
Body mass index	mean ± SD	23.7 ± 2.1	23.6 ± 2.1
Dominant side	right/left	18/0	17/1
Hemiplegic side	right/left	10/8	-
Time since stroke (weeks)	mean ± SD	15.6 ± 10.0	-
Lesion location	cortical/subcortical	14/4	-
Fugl-Meyer upper limb[¥]	range	45-63	-
Fugl-meyer proximal*	range	24-35	-

IwS: Individuals with stroke; [¥]: Fugl-Meyer upper limb motor scale, maximum score 66; *: Shoulder and elbow parts of the Fugl-Meyer upper limb motor scale, maximum score 36

3.2. Kinematic and electromyographic (EMG) data acquisition

3D kinematic data were captured with 15 infrared cameras sampling at 100 Hz (Vicon, Oxford Metrics, UK) and filtered with a spline-interpolation.[15] Clusters of three or four markers, mounted on tripods or cuffs, were placed on the sternum, scapula (flat part of the acromion) and upper arm (proximal, lateral) (Figure 1B) at the hemiplegic (IwS) or dominant (controls) side. Anatomical landmarks were digitized during static trials, using a pointer with four linear markers. Anatomical landmarks were defined within their respective segmental marker cluster (CAST-procedure),[16] and subsequently used to construct anatomical coordinate systems and to calculate joint kinematics. All kinematic calculations were done according to the ISB-guidelines.[17] Trunk kinematics were described in following rotations: flexion/extension, homolateral (toward the moving arm side)/hererolateral (away from the moving arm side) lateral flexion, internal (toward the moving arm side)/external (away from the moving arm side) axial rotation. Scapulothoracic kinematics were described in following three rotations: protraction/retraction, medial/lateral rotation, anterior/posterior tilting (Figure 1A).

Figure 1. Scapulothoracic kinematics (A) and cluster placement at the sternum, acromion and upper arm (B)



Scapular muscle activity was recorded using surface electromyography (wireless Zerowire system, Cometa, Milan, IT) at the hemiplegic (IwS) or dominant (controls) side: upper trapezius (midpoint of the line between angulus acromialis and C7 processus spinosus), lower trapezius (at one third of the line between trigonum scapula and T8 processus spinosus), serratus anterior (anterior to the latissimus dorsi at the level of the scapular inferior angle), infraspinatus (approximately 4cm below the spine, over the infrascapular fossa, parallel to the scapular spine), and anterior deltoid (2-4cm below the lateral clavicula, parallel to the muscle fibers).[18,19] To ensure consistency of EMG-sensor position, these were placed by the same investigator for all participants. Prior to sensor placement, the skin over the muscle of interest was prepared and cleaned with alcohol. Muscle activity was recorded at a sampling rate of 2000 Hz. Correct sensor positioning and signal quality was verified in real-time through visual inspection of the EMG-signals during muscle specific movements.

3.3. Measurement procedure

All measurements took place at the Clinical Motion Analysis Laboratory of the University Hospital Pellenberg. While seated on a chair with low back support, marker clusters were mounted on the participant's upper body. Static calibration trials were first collected to digitize the anatomical landmarks. Participants were subsequently instructed to perform a low and high unilateral humerothoracic elevation in the sagittal plane (forward flexion, FF). A bar was placed in front of the participant at ±60° and ±120° of FF height (determined with goniometry) to indicate the appropriate level of elevation for the low and high FF task, respectively. Participants were instructed to start with the elbow fully extended and thumb pointing upward, and to maintain this positioning during FF. To ensure correct task performance and a proper pace of task execution (1s up, 1s down, 3s rest for low FF; 3s up, 3s down, 4s rest for high FF), each participant was given some practice trials. After these practice trials, eight repetitions per task were recorded.

3.4. Kinematic and EMG Data Analysis

From the eight recorded trials, only the middle six repetitions per FF task were selected for data analysis (as these were considered free from initiation/completion strategies). Movement cycles were visually defined from movement start to highest arm position (elevation phase), and from highest arm position to movement stop (lowering phase). Kinematic data was further processed with Matlab®, using U.L.E.M.A..[20] Each movement cycle was time-normalized and joint angles were visualized as a function of time to check for erroneous signals. Discrete parameters of interest were (1) trunk and scapula joint angles at start position, and (2) trunk and scapula range of motion (ROM) at 45° (low FF) and 90° (high FF) of shoulder forward flexion. ROM was defined as the absolute difference between the highest and lowest recorded joint angle between start and 45° or 90° of shoulder forward flexion (elevation phase), and between 45° or 90° of shoulder forward flexion and the stop of movement (lowering phase). All participants were able to attain 45° and 90° of FF.

Raw EMG-data were first high-pass filtered with a 6th-order Butterworth filter with a cut-off frequency of 20Hz to filter out movement and cardiac artifacts, and subsequently rectified and filtered with a low-pass filter (cut-off frequency of 45Hz) to smooth the data. Both filters were implemented as zero-phase shift filters. Onset and offset of muscle activity was defined according to the method of Staude, which applies an approximated generalized likelihood principle by detecting statistically optimal changes throughout the signal for automatic onset detection.[21] Each calculated onset and offset was visually checked for erroneous signals (e.g. cardiac or other motion artifacts). Parameters of interest from the EMG-analysis were: (1) time of muscular onset, and (2) time of muscular offset relative to movement start/stop. These parameters were expressed in percentage of total movement time. A positive latency time (%) indicated a muscle onset after movement start and a latency (offset) time above 100% indicated muscle offset after movement stop.

Lastly, the sequence in time of onset and time of offset of the different muscles relative to the movement start was analyzed per group and per task, based on average group data.

3.5. Statistical analysis

Distribution of data and its residual errors was verified with the Kolmogorov-Smirnov test.

Differences between both groups for 3D trunk and scapulothoracic kinematics were analyzed using t-tests for independent samples. The level of significance was set at α-level 0.05.

Differences in onset and offset timing between groups and muscles were determined using a 2 (group)*5 (muscle) mixed model. As such, two main effects and one interaction effect was calculated per task for muscle onset and muscle offset separately. The use of mixed model analysis was preferred as this analysis allows for repeated measures analysis.[22] The level of significance was set at α-level 0.05 for the main effects, with post-hoc Bonferoni correction for multiple testing (α-level of 0.025 for group differences and 0.01 for differences in muscle timing). All statistics were done in SAS (software version 9.4 Foundation and enterprise guide).

4. Results

4.1. Participants

Eighteen IwS (12 men, 51.8±13.3 years) and 15 age-matched healthy controls (8 men, 51.7±13.1 years) were included in the study. An overview of participants' characteristics is given in Table 1.

4.2. Kinematic analysis

Mean and standard deviation of all kinematic outcomes are given in Table 2.

4.2.1. Joint angles at start position

Lateral bending and axial rotation of the trunk significantly differed between IwS and controls ($p = 0.009$ and $p = 0.029$, respectively). IwS started with their trunk bend towards the non-hemiplegic arm (heterolateral lateral bending) and rotated toward their hemiplegic arm (internal rotation), while controls' trunk was bend toward their dominant arm (homolateral lateral bending) and rotated toward the non-dominant arm (external rotation). No significant differences in scapulothoracic joint angles were found between both groups.

4.2.2. ROM at 45°FF in low FF

During elevation and lowering, IwS showed significantly more ROM in internal axial trunk rotation than controls ($p = 0.04$ and $p = 0.01$, respectively). No significant differences in scapulothoracic joint ROM were found between IwS and controls.

4.2.3. ROM at 90°FF in high FF

No significant differences in trunk joint ROM were found between IwS and controls. During elevation, IwS showed significantly less ROM in posterior tilting than controls ($p = 0.049$). During arm lowering, IwS showed significantly more ROM in lateral rotation compared to controls ($p = 0.008$).

Table 2. Mean and SD of 3D scapular joint angles, expressed in degrees

TRUNK		**Flexion/extension**				**Lateral bending**				**Axial rotation**			
		IwS		Controls		IwS		Controls		IwS		Controls	
		Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)
Start FF†		2.7	(5.3)	-0.6	(4.6)	-0.8	(1.9)	0.8	(1.5)*	1.8	(1.4)	-0.7	(2.7)*
ROM 45°	- elevation phase	1.1	(0.5)	0.7	(0.6)	0.7	(0.6)	0.5	(0.5)	2.6	(2.0)	1.3	(1.4)*
	- lowering phase	1.0	(0.7)	0.9	(0.8)	0.8	(0.7)	0.6	(0.7)	2.2	(1.4)	1.1	(1.1)*
ROM 90°	- elevation phase	1.9	(1.1)	1.8	(1.3)	3.5	(1.8)	4.4	(2.4)	3.7	(2.1)	2.5	(1.5)
	- lowering phase	2.4	(1.4)	1.6	(1.3)	4.3	(2.2)	3.3	(1.7)	3.2	(1.8)	2.1	(1.4)
SCAPULA		**Pro/retraction**				**Medial/lateral rotation**				**Tilt**			
		IwS		Controls		IwS		Controls		IwS		Controls	
		Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)
Start FF†		29.4	(7.2)	30.9	(10.1)	1.1	(7.0)	0.5	(4.8)	-9.0	(6.4)	-10.9	(5.4)
ROM 45°	- elevation phase	8.0	(4.1)	6.7	(3.3)	9.0	(3.8)	7.8	(4.6)	6.0	(2.8)	5.8	(4.0)
	- lowering phase	8.8	(4.0)	6.9	(3.5)	9.2	(4.8)	5.8	(4.1)	5.8	(3.0)	5.8	(4.1)
ROM 90°	- elevation phase	17.2	(6.5)	14.0	(5.9)	34.5	(10.1)	29.5	(8.7)	8.6	(4.4)	13.5	(8.1)*
	- lowering phase	16.8	(6.4)	13.5	(6.3)	38.3	(9.1)	29.3	(9.0)*	10.1	(6.9)	13.6	(7.7)

IwS: Stroke patients; FF: forward flexion;
†: Trunk flexion (+) and extension (-); Trunk lateral bending homolateral (+) and heterolateral (-); Trunk axial rotation internal (+) and external (-); Scapula protraction (+) and retraction (-); Scapular medial (+) and lateral (-) rotation; Scapula posterior (+) and anterior (-) tilt;
ROM: Range of motion, positive value when movement toward trunk flexion, trunk homolateral lateral bending, trunk internal axial rotation, scapular protraction, scapular lateral rotation, scapular posterior tilt.
*: significant difference between IwS and controls ($p < 0.05$)

4.3. EMG analysis

Mean and standard deviation of all timing parameters are given in Table 3.

Table 3. Mean and SD of muscle timing parameters, expressed in percentage of total movement time

ONSET	**Low FF**				**High FF**			
	IwS		Controls		IwS		Controls	
	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)
Upper trapezius	3.2	(3.0)	2.5	(3.0)	2.8	(3.4)	3.3	(2.4)
Lower trapezius	8.5	(3.7)	12.5	(5.8)*	6.9	(2.7)	6.5	(4.5)
Serratus anterior	9.0	(7.5)	3.7	(4.8)*	9.6	(3.9)	6.7	(4.4)
Infraspinatus	5.6	(3.8)	5.3	(3.6)	5.2	(3.9)	2.7	(2.7)
Anterior Deltoid	3.4	(1.8)	3.8	(3.8)	3.0	(1.5)	3.9	(3.9)
OFFSET	**Low FF**				**High FF**			
	IwS		Controls		IwS		Controls	
	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)
Upper trapezius	75.7	(12.4)	72.7	(11.1)	82.6	(9.4)	81.4	(4.3)
Lower trapezius	65.1	(6.7)	63.2	(6.0)	70.4	(8.6)	75.6	(8.6)
Serratus anterior	68.4	(12.1)	76.8	(17.2)*	75.7	(9.3)	74.6	(14.2)
Infraspinatus	79.2	(12.1)	69.4	(7.3)*	80.6	(6.2)	82.4	(6.9)
Anterior Deltoid	79.1	(11.1)	76.0	(6.2)	82.5	(5.6)	85.1	(6.6)

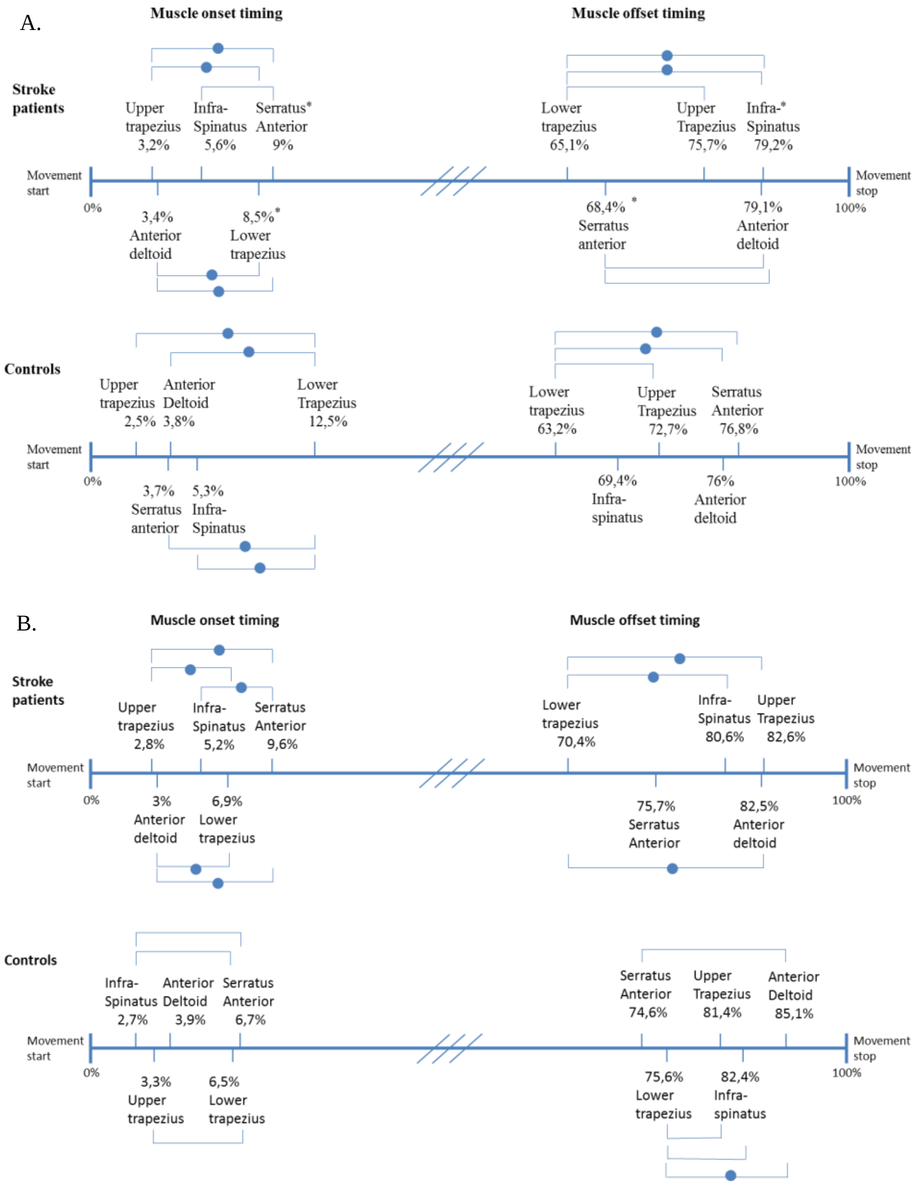
FF: Forward flexion; IwS: Individuals with stroke;

*: Significant difference between IwS and controls ($p < 0.05$)

4.3.1. Muscle onset and offset timing

During low FF, a significant main effect for muscle ($p < 0.0001$) and a significant interaction effect for group*muscle (onset $p = 0.0015$; offset $p = 0.006$) was found. Post-hoc analyses showed a significantly earlier onset of lower trapezius ($p = 0.017$) and a significantly later offset of infraspinatus ($p = 0.012$) in IwS than controls. In contrast, serratus anterior had a delayed onset and earlier offset in IwS compared to controls ($p= 0.0012$; $p = 0.02$, respectively). For onset and offset timing during high FF, only a significant main effect for muscle ($p < 0.0001$) was found. Significant post-hoc differences between the different muscles for onset and offset timing per group and task, together with the significant differences between groups, are visualized in Figure 2.

Figure 2. Recruitment patterns during a low forward flexion task (A) and high forward flexion task (B)



: Significantly different muscle onset/offset timing; p <.01; : Significantly different muscle onset/offset timing; p <.001;
*: Significantly different from controls

4.3.2. Recruitment patterns

Recruitment patterns based on average data are also visualized in Figure 2. In both groups, all muscles were active after the start of the moment and inactive before the stop of the movement for both FF tasks. Onset in low and high FF in IwS was characterized by following recruitment pattern: upper trapezius, anterior deltoid and infraspinatus were significantly earlier active than lower trapezius and serratus anterior. Controls showed the same sequence in the low FF, except for serratus anterior, which was also significantly earlier active than lower trapezius. In the high FF, controls showed earlier activity in infraspinatus and upper trapezius than in serratus anterior, and in infraspinatus relative to lower trapezius.

Following offset sequence was registered in IwS: lower trapezius (low and high FF) and serratus anterior (low FF) were earlier inactive then upper trapezius, anterior deltoid and infraspinatus. In contrast, controls showed a significantly earlier inactivity of lower trapezius than upper trapezius, serratus anterior and anterior deltoid in the low FF. During the high FF, controls showed significant earlier inactivity in serratus anterior relative to anterior deltoid, and in lower trapezius relative to upper trapezius, infraspinatus and anterior deltoid.

5. Discussion

Motor control of the scapulothoracic joint relies on a synchronized activation of the upper trapezius, lower trapezius and serratus anterior to upwardly rotate the scapula, and on a well-functioning serratus anterior to posteriorly tilt the scapula and provide a proper protraction movement during FF.[23,24] As such, a stable basis is offered for glenohumeral muscles such as infraspinatus to provide a stable glenohumeral joint. Post-stroke hemiparesis provides inadequate conditions for proper muscle activation, which might hamper proper trunk and scapulothoracic movement patterns and potentially lead to shoulder pathology and/or pain. This study wanted to assess alterations in 3D kinematics of trunk and scapula, and in synchronized shoulder muscle timing in SP and controls.

In low FF, SP had a delayed activation and early deactivation of serratus anterior compared to controls. Moreover, this muscle was active after and inactive before anterior deltoid in IwS, while in controls serratus anterior showed concurrent (in)activity with anterior deltoid. We identified adaptive activity of infraspinatus and lower trapezius in IwS during low FF. More specifically, IwS used early activity of lower trapezius and prolonged activation of infraspinatus to successfully establish the same pattern of scapulothoracic movement as shown by controls (lateral rotation, posterior tilt and protraction). In contrast to scapulothoracic kinematics, IwS did show altered trunk movements during this low task compared to controls, i.e. they used more internal trunk rotation. In literature, increased internal rotated trunk position has been related to a decreased scapulothoracic protraction and increased scapulothoracic lateral rotation,[6] which may account for the lack of significant group differences in scapulothoracic kinematics during low FF.

Late activity and early serratus anterior inactivity of serratus anterior is often described in persons with primary shoulder pain, i.e. due to subacromial impingement.[25] Additionally, early in elevation (30-60°) the supraspinatus tendon is in closest contact with the acromion. Taken both factors together, the development of subacromial complaints during repetitive low arm movement in IwS is to be expected. However, all IwS in the current study were pain-free, had mild to moderate impaired arm function, and were able to use their arm during daily activities. This might suggest that the combination of increased trunk internal rotation, early lower trapezius activity and prolonged infraspinatus activity serves as a protection against the development of shoulder pathology that would otherwise be caused by the serratus anterior timing dysfunction. This seems to be confirmed by previously reported results, indicating that pain-free IwS use an earlier activation of lower trapezius and prolonged activation of infraspinatus compared to IwS with subacromial shoulder pain.[13] However, one might also speculate that the altered trunk movements cause the altered serratus anterior timing. In this case, other post-stroke dysfunctions, like pectoralis major spasticity, might cause the increased trunk internal rotation. However, whether this dysfunction in serratus anterior timing is a cause or a compensation of alterations in trunk movements remains to be determined in future research.

During high FF, we did not observe significant differences in scapular muscle timing between IwS and controls. Inspection of the recruitment patterns did show that serratus anterior was significantly later active than anterior deltoid in IwS, while in controls serratus anterior showed concurrent activation with anterior deltoid. During this task, IwS also used less posterior tilting ROM (elevation phase) and more lateral rotation ROM (lowering phase). Given the limited differences in EMG muscle timing, the recorded alterations in scapulothoracic kinematics in IwS might be caused by shortened or inflexible structures. Reduced posterior tilting has been associated with a short pectoralis minor length,[26,27] and with subacromial pathologies. Also decreased mobility of posterior-inferior glenohumeral structures, i.e. capsular restrictions, requires specific attention since increased scapulothoracic lateral rotation has been specifically associated with capsular stiffness and subacromial disorders.[28,29] These restrictions and muscular inflexibilities should thus be prevented with appropriate manual therapy and stretching techniques.

The relevance of the reported differences in trunk rotation between groups in the low FF and in scapular tilting and lateral rotation in high FF should be discussed in view of the standard error of measurement (SEM). The difference in scapular tilting and lateral rotation between both groups (4.9 and 8.9 degrees, respectively) exceeds the previously reported between sessions percentage SEM of 1.71 and 4.52 degrees.[30] Unfortunately, SEMs for trunk rotations were not previously reported for this specific movement protocol in IwS. One might thus argue the clinical relevance of the reported statistically significant differences in

trunk rotation between groups in the low FF, since these differences are very small.
A limitation of the current study is that we did not account for hand dominance, side and type of stroke, or the amount of brain damage. Also, the use of surface EMG is prone to signal noise by motion or cardiac artifacts, which resulted in some data loss, i.e. 11% lost trials for upper trapezius, 10% for lower trapezius, 15% for serratus anterior, and 9% for infraspinatus and anterior deltoid. It was a distinctive choice to include IwS with a relatively high function. These individuals are at higher risk to develop shoulder pathology due to bad movement patterns in comparison to no or low functioning IwS, who will use their arm less in daily life. They would moreover benefit most from scapular stabilization training in the prevention of or to treat e.g. shoulder pain. However, it would be interesting to measure IwS with lower arm functioning and IwS with shoulder pain in future studies. As such, pathological movement patterns can be identified and potentially be detected early in motor recovery. Furthermore, this would allow for adaptation of therapy to the specific alterations in scapular movement or muscle timing. Additionally, further research should go beyond kinematic analysis of the shoulder complex by combining kinematics of the trunk and scapulothoracic joint with kinematic analysis of the glenohumeral and elbow joint to investigate whether distal compensations for proximal alterations can be identified.

6. Conclusion

Given that stroke is a leading cause of persistent physical disability in adults, it is important to understand underlying mechanisms influencing adequate motor control. Our results indicated that IwS with a well-preserved arm function demonstrate adaptive muscle timing with earlier initiation and prolonged activation of lower trapezius and infraspinatus respectively, to compensate for a late activation and early deactivation of serratus anterior in low FF. In high FF, it seems that passive structures hamper proper scapulothoracic movement patterns. In this way, 3D kinematic assessment and EMG muscle timing are believed to provide information on the compensations IwS use to preserve a pain-free shoulder function.

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Chapter 5

Disability and Rehabilitation; under review

Scapulohumeral control after stroke: reliability and discriminative ability of a clinical scapular protocol (ClinScaP)

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1. Abstract

Purpose: To develop a reliable protocol for the assessment of scapulohumeral control after stroke (ClinScaP). Secondly, the protocol's ability to distinguish between individuals with stroke (IwS) and healthy controls, and between IwS with different levels of proximal arm function (PAF) was investigated.

Method: Following scapular characteristics were clinically measured in 57 IwS (38 male, age 62±14 years, subdivided in a low, moderate, high PAF group) and in 15 healthy controls (8 male, 52±13 years): (1) observation of tilting and winging at rest and during movement; (2) shoulder girdle position upright and in supine (pectoralis minor index, acromial index, scapular distance test); (3) scapular lateral rotation at different forward flexion heights (inclinometry); (4) maximal active humeral elevation (goniometry); (5) scapular dynamic control in supine (medial rotation test). In 15 IwS, the ClinScaP was performed twice by the same assessor to determine intra-observer reliability. Differences between controls and IwS and between IwS with different levels of PAF were assessed using independent t-tests and one-way ANOVA (test 2, 3, and 4) or Mann-Whitney tests and Kruskal-Wallis (test 1 and 5), respectively.

Results: ICCs were very high for all tests (>0.80), except the pectoralis minor index (0.66). Weighted Kappas were high for observation and the medial rotation test (>0.70). Group differences between IwS and controls, and between IwS with different levels of PAF were mainly found for observation, inclinometry and maximal humeral elevation. IwS compared to controls, and IwS with lower compared to higher PAF generally showed (1) increased lateral rotation (p<.01); (2) decreased maximal active humeral elevation (p<.001); and (3) more often the presence of tilting and winging (p<.05).

Conclusions: Most tests of the ClinScaP can be reliably assessed, and three tests (observation, inclinometry and maximal humeral elevation) can differentiate between IwS and controls, and between IwS with different levels of PAF. The use of these tests in clinical settings will allow for easy and adequate identification of altered scapular characteristics, which will enhance efficient treatment planning to improve PAF post-stroke.

2. Introduction

The brain damage underlying a stroke results in several motor impairments such as muscle weakness, increased muscle tone, pathological muscle synergies and altered temporal muscle activity.[1-4] At the level of the shoulder complex, these motor impairments might specifically hamper scapulohumeral control, i.e. the adaptation of scapular position and movement according to the humeral position. Reduced scapulohumeral control is known to contribute to the difficulties individuals with stroke (IwS) experience when moving their paretic arm.[5] Upper limb rehabilitation after stroke could benefit from specific training to enhance scapular positioning and scapulohumeral movement control. However, such therapy firstly requires an extensive evaluation of the scapulothoracic joint.
Within the area of musculoskeletal research, there is a wide availability of tests or measurements to assess scapular position and scapulohumeral control in rest or during movement.[6-8] The reliability of these assessments has been verified,[7-9] and they are commonly used in cross-sectional or comparative studies and even in interventional research.[10,11] Such a clinical measurement approach, covering different aspects of static and dynamic scapulohumeral control, might prove valuable to assess the role of the scapula in upper limb and shoulder (dys)function in IwS. However, clinical scapulohumeral assessments have thus far been limited to the healthy population or to persons with musculoskeletal pathologies only.
Assessment of scapulohumeral control in IwS requires a clinical protocol that offers specific scapular information, which is not covered with currently available clinical measurement scales for the upper limb after stroke. Therefore, this study introduces a clinical scapular protocol (ClinScaP), in which tests are selected based on knowledge from musculoskeletal rehabilitation. Those tests that are expected to be associated with a specific scapular rotation (i.e. protraction, lateral rotation, tilting) are retained. As such, clinical observation of tilting and winging in rest and during movement is chosen to assess three-dimensional scapular protraction and tilting;[9-12] these scapular rotations are further assessed with the scapular distance test, the pectoralis minor index and the acromial index.[7,13-15] Scapular lateral rotation is assessed based on inclinometry at different forward flexion heights.[16] Dynamic scapulohumeral control is assessed based on maximal active humeral elevation and the medial rotation test.[8,17] However, prior to clinical implementation of any new protocol, its reliability and ability to discriminate between groups should be established. Therefore, this study assesses the protocol's intra-observer reliability, and investigates which test or battery of tests is able to differentiate between (1) IwS and healthy controls; and (2) IwS with different levels of proximal arm function (PAF).

3. Methods

3.1. Participants

A convenient sample of IwS was recruited via therapists from three rehabilitation centers in Flanders (Belgium). IwS were eligible for study-participation when (1) they had a first time stroke (cortical or subcortical area, verified using MRI); (2) had the ability to sit independently with low back support only; and (3) could perform 45° of active and 90° of passive humerothoracic forward flexion. Healthy controls were recruited via family and colleagues. Exclusion criteria for all participants were: (1) inability to understand the instructions; (2) anterolateral shoulder pain during daily activities with a painful arc between 60 and 120° of arm forward flexion for at least four weeks; (3) a positive Neer impingement test, i.e. reported pain when the humeral greater tuberosity was impacted against the inferior acromion;[18] (4) an event of shoulder dislocation, fracture or surgery during lifetime; or (5) other systemic and/or neurologic diseases.

IwS were divided in three groups based on their score on the shoulder and elbow parts of the upper limb motor part of the Fugl-Meyer (FM elbow-shoulder, max score 36),[19] i.e. low (score ≤ 16), moderate (score 17-26) or high (score 27-36) PAF. Similar to Lum et al. (2003),[20] these grouping criteria were based on the observed scores in our participants group, i.e. scores ranged between 6 and 36. As such, we divided this range of scores into even thirds in order to achieve three distinct groups.

Written informed consent, as approved by the Ethical Committee of the University Hospital Leuven and the local ethical committees of each of the rehabilitation centers, was obtained from all participants prior to study participation.

3.2. Clinical Scapular Protocol

One skilled physiotherapist with seven years of experience in manual therapy performed the Fugl-Meyer scale and all measures of the clinical scapular protocol (ClinScaP) at the hemiplegic (IwS)/dominant (controls) side of every participant. All participants were assessed in their respective rehabilitation centers. The full assessment protocol typically lasted for 20 minutes.

Fifteen IwS were additionally assessed on a second moment on the same day to assess intra-observer reliability. The time between both measurement sessions varied between 3 and 7 hours, depending on the person's availability. Every anatomical landmark was palpated and/or marked again during the second measurement session.

The ClinScaP consists of five tests, with several subtests, and was assessed in following order:

Test 1: Observation of tilting and winging

While seated upright in a chair with low back support, the presence (score 1) or absence (score 0) of scapular tilting and winging was scored by observing the participant's scapular position on the thorax. This scoring was done during rest (both arms alongside the body, thumbs pointing forward) and during active unloaded forward flexion. Participants were instructed to move bilaterally at a rate of 3 seconds up toward their maximal forward flexion, and 3 seconds down toward the rest position. Observation was done from a dorsal and lateral position. Presence of tilting or winging indicated a prominence of the inferior tip of the scapula dorsally or prominence of the medial scapular border, respectively.[9,12] Palpation was used to verify anatomical landmarks. A total score was calculated for observation at rest and for observation during movement. Score '0' indicated no presence of tilting or winging, score '1' the presence of tilting or winging and score '2' the presence of both tilting and winging.

Test 2: Shoulder girdle position

Three different measures were used to evaluate the participant's shoulder girdle position. Specific palpation guidelines were followed to ensure accurate palpation.[21]

Acromial index (AI): this index was assessed with the participant lying supine, the arms relaxed alongside the body with the palm placed on the table. The participant was instructed to stay relaxed during the measurement. In this position, the acromial angle was palpated and the vertical distance between this angle and the table (cm) was measured with a sliding carpenter. This distance was divided by the subject height (cm) and defined as the AI (no unit).[13]

Pectoralis minor index (PMI): this index was assessed with the participant seated upright in a chair with low back support and the arms relaxed alongside the body. The resting length of the pectoralis minor muscle was assessed by measuring the length (measurement tape) between the inferior medial tip of the coracoids process and the caudal edge of rib four (at its attachment to the sternum). Both reference points were first palpated and marked using a pen. Participants were instructed to exhale during the palpation, marking and measurement itself. The PMI (no unit) was defined as the pectoralis minor resting length (cm) divided by the subject height (cm).[7,14]

Scapular distance test (SDT): this test assesses the position of the scapula on the trunk in an upright-seated position with low back support with the arms relaxed alongside of the body. The SDT (no unit) was calculated by dividing the distance between the acromial angle and the spinous process of T3 (cm) by the distance between the acromial angle and the scapular trigonum (cm). Anatomical landmarks were first palpated and marked using a pen, and distances were subsequently measured with flexible measurement tape.[15]

Test 3: Scapular lateral rotation

Scapular lateral rotation was assessed with an inclinometer (Plurimeter -V gravity inclinometer, Dr Rippstein, Switserland), while participants were seated upright on a chair with low back support. The inclinometer was held manually on the scapular spine by the skilled physiotherapist, while an assisting physiotherapist passively elevated the participant's arm in the sagittal plane (forward flexion). The amount of lateral rotation (degrees) was read from the inclinometer at rest (arm alongside the body), and at 45°, 90° and 135° of passive forward flexion (determined by goniometry).[16] The elbow was extended and the thumb pointed upward during task performance.

Test 4: Maximal active humeral elevation

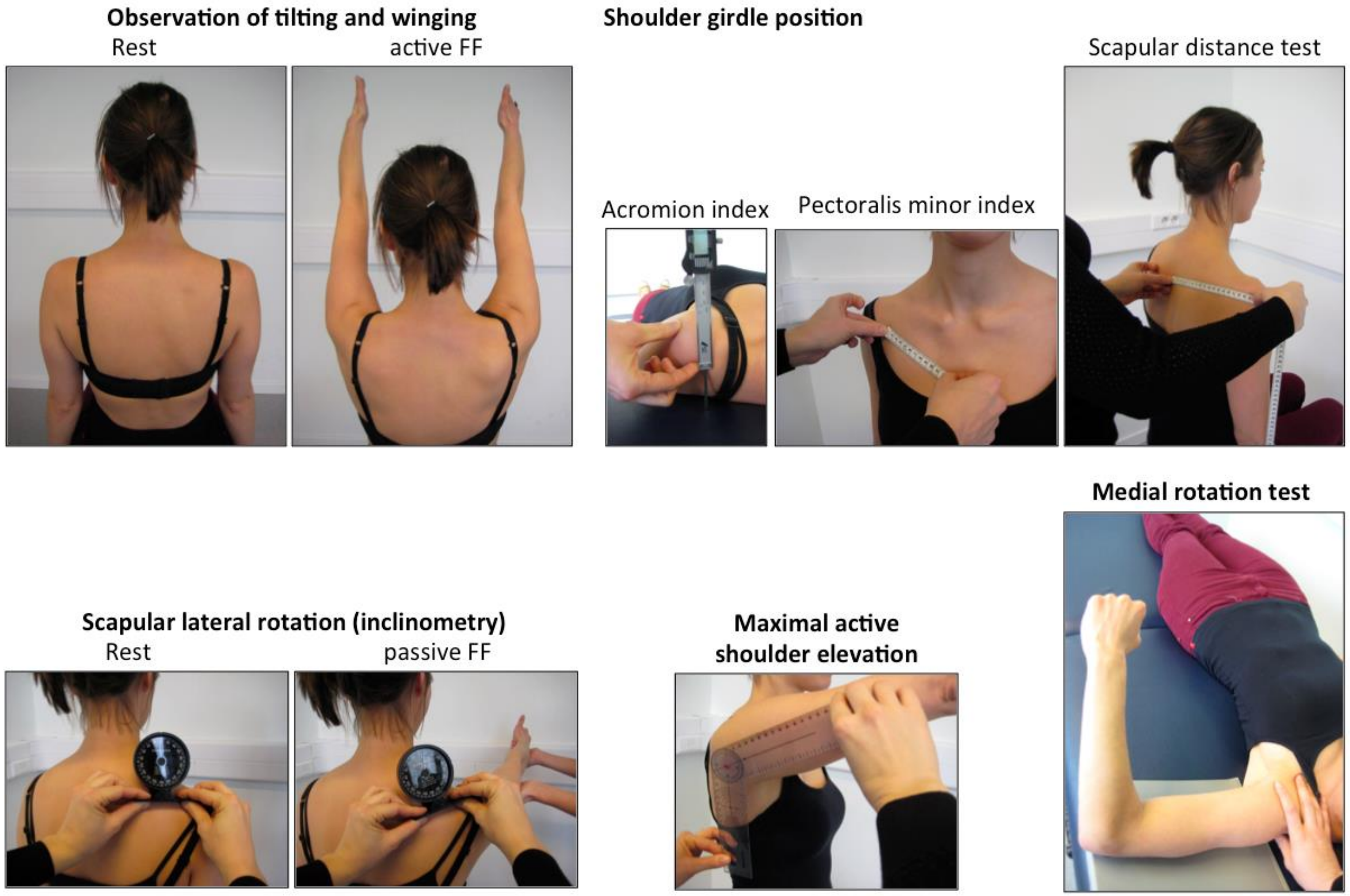
While seated upright with low back support, the maximal range of active humerothoracic elevation in the sagittal plane (forward flexion) was read from a goniometer (degrees). Participants were instructed to extend the elbow and to keep the thumb pointing upward during movement.

Test 5: Medial rotation test

This test assessed scapular dynamic control while participants laid supine with the upper arm passively supported by a wedge in 90° of humerothoracic scapular plane elevation (30° anterior to the frontal plane), and the elbow flexed. While actively performing a movement towards glenohumeral internal rotation, i.e. moving the forearm towards the table, the participant was instructed to keep the scapula still. Meanwhile, the assessor palpated the anterior humeral head and the coracoid process and judged the amount of anterior humeral translation and scapular movement. Aberrant dynamic control indicated excessive anterior humeral translation (more than 4 mm, judged by palpation) or scapular movement (more than 6 mm, judged by palpation in the direction of anterior tilt, downward rotation or scapular elevation) before 60° of internal glenohumeral rotation. A total score of '0' indicated correct humeral translation and scapular movement. A score of '1' meant aberrant humeral translation or aberrant scapular movement, and a score of '2' indicated aberrant humeral translation and aberrant scapular movement. Every participant received some practice trials to get familiarized with the test before the formal test was executed.[8,17]

The different tests of ClinScaP are visually presented in Figure 1. Further details for each of the different tests of ClinScaP can be found in Appendix 1.

Figure 1. ClinScaP



3.3. Statistical analysis

Descriptive statistics were used to document general characteristics for each participant group. A one-way ANOVA and post-hoc Tukey test were used to assess differences in age and time since stroke between the low, moderate and high PAF groups and the IwS included in the reliability assessment, and to assess differences in age between the low, moderate and high PAF groups and controls.

Bland-Altman plots were constructed for the measures of shoulder girdle position (AI, PMI, SDT – test 2), inclinometry (test 3) and maximal humeral elevation (test 4), to display the data graphically and to examine the distribution around the zero line. A 95% confidence interval was calculated (mean difference± 1,96* $SD_{\text{mean difference}}$) to identify systematic variance (i.e. zero line not included in the 95%CI) or outliers. To assess heteroscedasticity, correlations between the mean of the two test sessions and the difference between the two test sessions were calculated.

Intra-observer reliability of the measures of shoulder girdle position (AI, PMI, SDT – test 2), inclinometry (test 3) and maximal humeral elevation (test 4) was assessed with the intraclass correlation coefficient ($ICC_{2,1}$), with 95% confidence interval (CI). Standard error of measurement (SEM) based on the square root of the mean square error term from the two-way ANOVA,[22] and minimal detectable change (MDC, defined as SEM * 1.96 * √2) were also reported.[23] ICCs > 0.80 were considered very high, 0.60–0.79 moderately high, 0.40–0.59 moderate and <0.40 low.[24] Agreement of scoring between sessions for the observation of tilting and winging (test 1) and for the medial rotation test (test 5) was calculated by weighted Kappa (K). K < 0 reflected 'poor', 0 to 0.20 'slight', 0.21 to 0.4 'fair', 0.41 to .60 'moderate', 0.61 to 0.8 'substantial', and above 0.81 'almost perfect' agreement.[25]

An ICC or K value above 0.70 on a test was considered to indicate sufficient reliability or agreement for that specific test to be used in future clinical research.

Normal distribution of the data (test 2, 3, and 4) was verified with the Kolmogorov–Smirnov test. Group differences between controls and IwS were assessed using t-tests (test 2, 3, and 4) and Mann-Whitney tests (test 1 and 5) for independent samples. Subsequently, group differences between the low, moderate and high PAF group were assessed using a one-way ANOVA and post-hoc Tukey tests (test 2, 3, and 4), or a Kruskal-Wallis analysis and post-hoc Mann-Whitney tests (test 1 and 5). The level of significance was set at α-level 0.05 for the main effects, with post-hoc Bonferoni correction α-level of 0.0167. All statistics were done using SPSS version 22.

4. Results

4.1. Participants

Fifty-seven IwS (38 male, age 62±14 years) participated in the current study, and were categorized into the low PAF group (N=17, age 64±10; mean time after stroke 31±27 weeks; 6 right hemiplegia; 13/4 cortical/subcortical lesion; FM elbow-shoulder score 9±4), the moderate PAF group (N=19, age 66±17; mean time after stroke 25±16 weeks; 8 right hemiplegia; 13/6 cortical/subcortical lesion; FM elbow-shoulder score 23±2), and the high PAF group (N=21, age 58±12; mean time after stroke 22±38 weeks; 10 right hemiplegia; 19/2 cortical/subcortical lesion; FM elbow-shoulder score 32±3). Additionally, 15 healthy controls were measured (8 male, age 52±13 years).

Fifteen IwS were measured twice in the context of intra-observer reliability analysis (4 of the low, 6 of the moderate and 5 of the high PAF group; age 69±9 years; mean time after stroke 18±7 weeks; 6 right hemiplegia; 12/3 cortical/subcortical lesion; FM elbow-shoulder score 22±7).

No significant differences were found between the low, moderate and high PAF group and the reliability group for age or time since stroke.

4.2. Reliability

There were no missing values for the reliability analysis, except for inclinometry at 135°, with two out of four IwS of the low PAF group included. Reliability analyses for this measure is thus based on 13 IwS in total.

Bland-Altman plots are presented in Appendix 2. Visual inspection showed an equal distribution of data around the zero line for all test, except inclinometry at 45° and the PMI, i.e. no systematic variance was observed (zero was always included in the 95% CI). For inclinometry at 45° and the PMI, data points were more often distributed under or above the zero line, respectively. Furthermore, inspection of inclinometry at 45° indicated a trend toward heteroscedasticity.

However, no significant correlations were found between the mean of the two test sessions and the difference between both for any of the tests, indicating uniform variability across the mean outcome (no data-heteroscedasticity). Based on the 95% CI, one outlier was observed for every test. Therefore, reliability analyses are presented for the entire sample (n=15) as well as for the sample with the outlier excluded (n=14).

For the entire sample, ICCs were very high for test 2, 3 and 4 of the ClinScaP (>0.81), except for a moderately high ICC for the PMI (0.66). For the sample without outlier, all ICCs were very high (>0.85). Furthermore, almost perfect agreement was found for observation of winging and tilting during movement (0.89). Substantial agreement was found for observation at rest (0.77) and for the medial rotation test (0. 73). All ICCs and K-values, together with SEM and MDC values are presented in Table 1. Based on the data from the entire sample, the PMI was not considered reliable enough using the cut-off score of 0.70.

4.3. Assessment of group differences

Average values of the results for test 2, 3 and 4, and those for test 1 and 5 are presented for controls and every subgroup of IwS in Table 2.

Although a minimal range of 45° of active humeral elevation was required for inclusion in the current study, the presence of tilting and winging during movement could not be scored in six participants in the low PAF group due to insufficient active arm elevation. Furthermore, measuring lateral rotation by inclinometry at 135° of forward flexion was possible in only two participants in the low PAF group. As such, in part 1 of this section (*Differences between IwS and controls*), only 51/57 IwS were included for the analysis of group differences for observation during movement, and only 42/57 for the analysis of group differences in inclinometry at 135°. In part 2 of this section (*Differences between IwS with different levels of PAF*), only 11/17 participants of the low PAF group were included in the analysis of group differences for observation during movement. As measuring lateral rotation by inclinometry at 135° of elevation was possible in only two participants in the low PAF group, only differences between the moderate and high PAF group were analyzed for inclinometry at 135° of passive forward flexion.

4.3.1. Differences between IwS and controls

Presence of tilting and winging at rest (p=.012) and during movement (p=.007) was more often seen in IwS than in healthy controls. Controls had furthermore significantly less lateral rotation measured by inclinometry at 45° (p=.004), 90° (p=.001) and 135° of forward flexion (p=.01), and more active humeral elevation (p=.000) as compared to IwS. No differences for the different tests of shoulder girdle position and for the medial rotation test were found.

4.3.2. Differences between IwS with different levels of PAF

Significant differences between groups were found for presence of tilting and winging at rest (p=.012). Observation during movement (p=.055) did not significantly differ between groups. Post-hoc tests indicated that the occurrence of tilting and winging at rest was more often seen in participants with moderate compared to high PAF (p=.010).

Significant differences were found between the three groups for inclinometry at 45° and 90° of passive forward flexion (p=.004 and p=.001, respectively). Post-hoc analysis showed that participants with high PAF had significantly less scapular lateral rotation at 45°, compared to the moderate PAF group (p=.008) and at 90° compared to the low and moderate PAF group (p=.003 and p=.006, respectively). At 135° of passive forward flexion, the high PAF group showed significantly less scapular lateral rotation compared to the moderate PAF group (p=.002).

Significant differences were also found between the three groups for the amount of active humeral elevation (p<.001). Participants in the moderate and high PAF group had significantly more active humeral elevation than those in the low PAF group (p<.0001 and p<.0001); and participants with a high PAF had significantly more active elevation range than participants with a moderate PAF (p<.0001). No differences for the different tests of shoulder girdle position and for the medial rotation test were found.

Table 1. Reliability of the ClinScaP

		Complete sample (n=15)			Outlier excluded (n=14)			
		ICC (95%CI)	**SEM**	**MDC**	**ICC (95%CI)**	**SEM**	**MDC**	**K (95%CI)**
Test 1: Observation	At rest							0.77 (0.54-1.00)
	During movement							0.89 (0.79-1.00)
Test 2: Shoulder girdle position	Pectoralis minor index	0.66 (0.09-0.89)	0.39	1.08	0.85 (0.32-0.96)	0.31	0.87	
	Scapular distance test	0.81 (0.53-0.93)	0.05	0.15	0.94 (0.81-0.98)	0.05	0.13	
	Acromial index	0.86 (0.62-0.95)	0.42	1.18	0.97 (0.90-0.99)	0.24	0.67	
Test 3: Lateral rotation (inclinometry)	Start (°)	0.94 (0.83-0.98)	1.59	4.40	0.97 (0.91-0.99)	1.25	3.46	
	At 45° humeral FF (°)	0.88 (0.65-0.96)	1.58	4.38	0.96 (0.68-0.99)	1.07	2.96	
	At 90° humeral FF (°)	0.95 (0.85-0.98)	1.47	4.07	0.98 (0.90-0.99)	1.67	4.62	
	At 135° humeral FF (°)	0.83 (0.48-0.96)	3.27	9.05	0.96 (0.67-0.99)	1.72	4.77	
Test 4: Max humeral elevation		0.99 (0.99-1.00)	2.44	6.76	0.99 (0.99-0.99)	1.76	4.89	
Test 5: Medial rotation test								0.73 (0.44-1.00)

Max: Maximal; FF: Forward flexion; ICC: Intraclass correlation coefficient; CI: Confidence interval; SEM: Standard error of the measurement; MDC: Minimal detectable change; K: Weighted kappa

Table 2. Descriptive statistics for the Fugl-Meyer and the different tests of the ClinScaP in IwS with different levels of proximal arm function

		Low prox arm function		Moderate prox arm function		High prox arm function		Controls	
		Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)
Fugl-Meyer	Proximal[a]	9.3	(4.1)	23.8	(1.6)	31.8	(3.2)		
	Upper limb[b]	26.8	(9.9)	49.2	(5.5)	57.4	(4.1)		
Test 2: Shoulder girdle position	Pectoralis minor index	5.9	(1.0)	6.0	(0.7)	5.7	(1.0)	5.5	(0.5)
	Scapular distance	1.6	(0.1)	1.6	(0.4)	1.6	(0.2)	1.5	(0.1)
	Acromial index	4.6	(1.0)	4.0	(0.5)	4.5	(0.9)	4.4	(0.7)
Test 3: Lateral rotation, inclinometry	Start (°)	1.9[c]	(9.1)	2.5[c]	(6.0)	4.0[c]	(5.2)	2.3[c]	(4.6)
	At 45° of FF (°)	7.9	(6.1)	8.2[d]	(5.1)	2.7	(4.9)	2.4[e]	(3.5)
	At 90° of FF (°)	26.5[d]	(7.6)	25.5[d]	(6.1)	18.3	(6.9)	12.6[e]	(8.9)
	At 135° of FF (°)			48.1[d]	(7.9)	38.2	(5.8)	36.7[e]	(5.6)
Test 4: Maximal humeral elevation		61.8[d,f]	(21.3)	118.8[d]	(23.2)	152.5	(13.3)	164.1[e]	(9.6)

		Low prox arm function			Moderate prox arm function			High prox arm function			Controls		
	Score (%)	**0**	**1**	**2**	**0**	**1**	**2**	**0**	**1**	**2**	**0**	**1**	**2**
Test 1: Observation	at rest	29.4	23.5	47.1	26.3	15.8[d]	57.9	61.9	23.8	14.3	66.6[e]	33.3	0
	during movement	54.5	0	55.5	21.1	21.1	57.8	42.9	33.3	23.8	80.0[e]	13.3	6.7
Test 5: Medial rotation test		29.4	11.8	58.8	31.6	21	47.4	52.4	23.8	23.8	73.3	0	26.7

FF: Forward flexion; prox: Proximal; [a]: Shoulder and elbow parts of the Fugl-Meyer upper limb motor scale; [b]: Fugl-Meyer upper limb motor scale; [c]: Medial rotation; [d]: Significantly different from high proximal arm function group; [e]: Significantly different from total group of patients with stroke; [f]: Significantly different from moderate proximal arm function group.

An overview of the results for the different tests of ClinScaP can be found in appendix 1.

5. Discussion

A prerequisite for the application of a clinical measure is its potential to differentiate between a pathological or non-pathological situation or between various degrees of dysfunction. Moreover, before any assessment is of value in a clinical decision-making process or to evaluate treatment efficacy, its reliability needs to be confirmed. In this study, we proposed a specific measurement protocol for scapulohumeral control (ClinScaP) for IwS, which is easily available and directly applicable in rehabilitation centers or in private practices and assessed intra-observer reliability. We furthermore determined the protocol's ability to differentiate between controls and IwS in general, and between IwS with different levels of PAF. In this way, it enables therapists to clinically identify scapular characteristics or dysfunctions, which could be related to various levels of PAF. The included tests in the ClinScaP were chosen based on their acceptable psychometric properties in musculoskeletal rehabilitation and on our assumption that these tests were related to a specific scapular rotation.[7-9,13,15,16,26] We furthermore opted to add static as well as dynamic tests, deemed feasible for IwS, even with a low PAF. Lateral rotation was therefore assessed during passive forward flexion. Additionally, this allowed maximal standardization of the test, i.e. joint angles were obtained at exactly 45°, 90° and 135° of humerothoracic forward flexion. Observation at rest, and the different tests for shoulder girdle position (PMI, AI, SDT) were chosen as passive measurements of scapular positional alterations, linked with e.g. inflexibilities or shortening of soft tissue structures around the shoulder joint, contributing to shoulder disorders (e.g. pectoralis minor).[14] However, dynamic measures are considered more functional than static measures, and thus observation of tilting and winging during movement was also included in the protocol. This test is assumed to provide information on e.g. delayed lower trapezius activation or decreased serratus anterior activity. Lastly, the medial rotation test and maximal active humeral elevation were added to dynamically assess scapular and scapulohumeral control, respectively.

Current study results could not confirm an acceptable reliability for the PMI in IwS with different levels of PAF. Measurement inaccuracies due to the difficult palpation areas and the dependence of the participant's respiration for the assessment of pectoralis minor length, could explain the lower reliability for the PMI.

The clinical value of the ClinScaP also relies on its ability to differentiate between IwS and controls, or between IwS with different levels of PAF. Results suggested that both passive, i.e. lateral rotation by inclinometry and observation at rest, as active measures, i.e. observation during movement and maximal active humeral elevation, are relevant measures for therapists to use in clinical practice in IwS.

Humeral motion can create early scapular motion by placing tension on a shortened glenohumeral capsule or stiff posterior-inferior glenohumeral muscles.[27-29] Hence, the increase in scapular lateral rotation and presence of anterior tilting and winging seen in IwS with moderate PAF compared to high PAF can be caused by restrictions in posterior glenohumeral structures. The reported significant differences in lateral rotation are moreover larger than the magnitude of the minimal detectable change, and can thus be interpreted as real differences. Together, results suggest that the inclusion of glenohumeral capsular or muscular stretching techniques in the rehabilitation of IwS with a moderate PAF might be beneficial to improve scapulohumeral control and hence arm function.

The PMI was considered less reliable in this study and could, together with the AI, SDT and medial rotation test, not differentiate between groups. The lack of differentiation of the shoulder girdle position tests (PMI, AI, SDT) might be due to the fact that these were measured with the participant's arm alongside of the body instead of an arm elevated position. Although not significantly different between IwS and controls, results for the medial rotation test, executed in an elevated arm position, did show a trend (p.056) toward reduced scapular control in IwS. The aforementioned tests are thus considered less relevant in clinical practice to differentiate between IwS and controls or between IwS with different levels of PAF. However, this selection of tests should only be applied to IwS similar to our included study sample, i.e. IwS without shoulder pain.

5.1. Limitations

In the current study, information about stroke location was extracted from the medical records. However, we did not account for the side and type of stroke or the amount of brain damage. Furthermore, participants were grouped based on the shoulder and elbow motor items of the Fugl-Meyer upper limb motor scale. As such, other upper limb impairments such as spasticity or sensory deficits were not taken into account.

5.2. Future perspectives

A first step would be to assess the feasibility and reliability of the entire ClinScaP in IwS with shoulder pain. This will allow gaining a deeper understanding of the development of shoulder pain in IwS. This is especially of interest since the alterations found in IwS with moderate compared to high PAF, i.e. more often presence of tilting and winging at rest and increased lateral rotation, are known to be related to shoulder symptoms and pathology.[30-31] Future studies should also assess the relation between objective scapular measures based on kinematic movement analysis and the different ClinScaP tests.

Finally, inter-observer reliability of the ClinScaP remains to be confirmed in IwS.

6. Conclusion

Observation of tilting and winging, inclinometry measures and maximal humeral elevation showed good reliability results, and these tests revealed distinct alterations in scapular characteristic in IwS. From a clinical perspective, the availability of the assessment of static scapular position (observation at rest, inclinometry during passive arm forward flexion) and dynamic movement control (observation during movement, maximal active humeral elevation), facilitates the understanding of the relation between scapulohumeral control and arm function in IwS. The knowledge gained from these tests will thus contribute to the further delineation of a treatment plan to target specific scapulohumeral dysfunctions, i.e decreasing scapular lateral rotation, optimizing scapular position on the thorax and enhancing scapular motor control, and eventually improve arm function.

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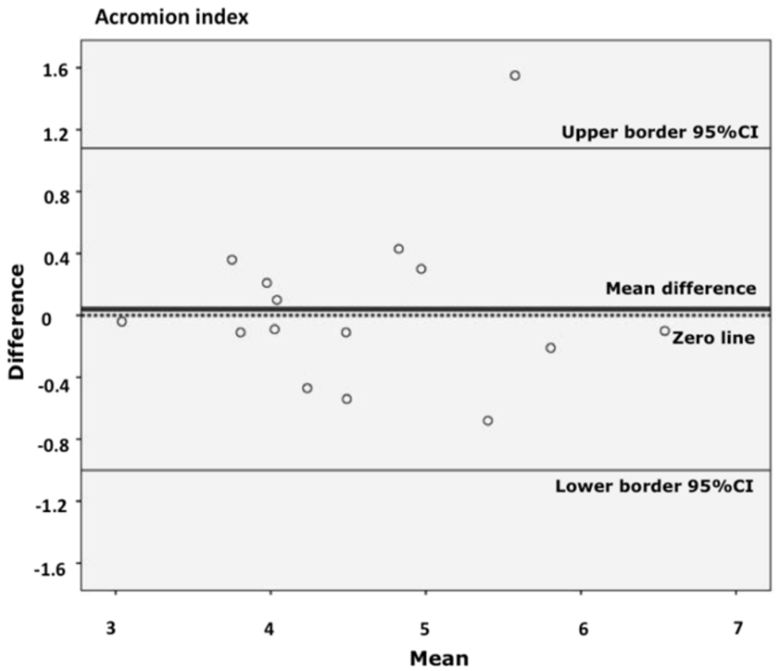
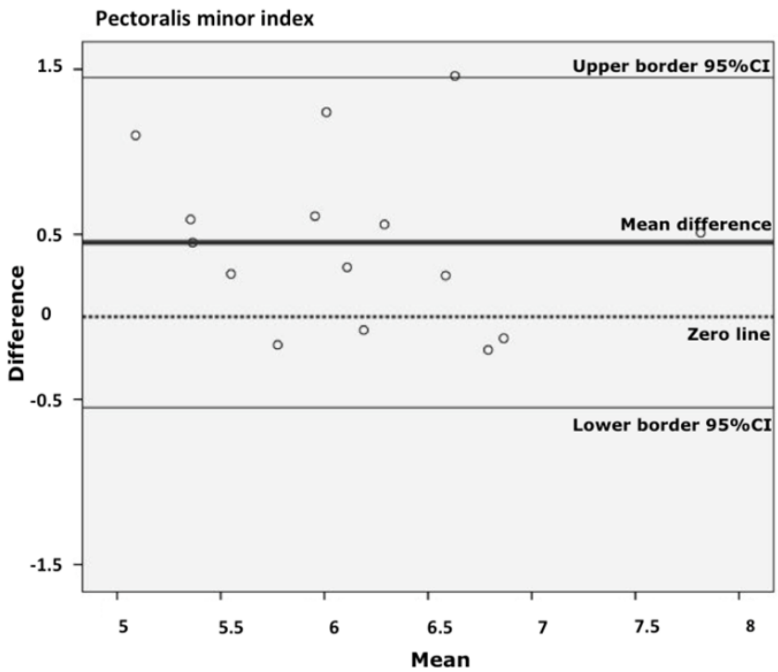
Appendix 1. Characteristics of the different tests of the ClinScaP

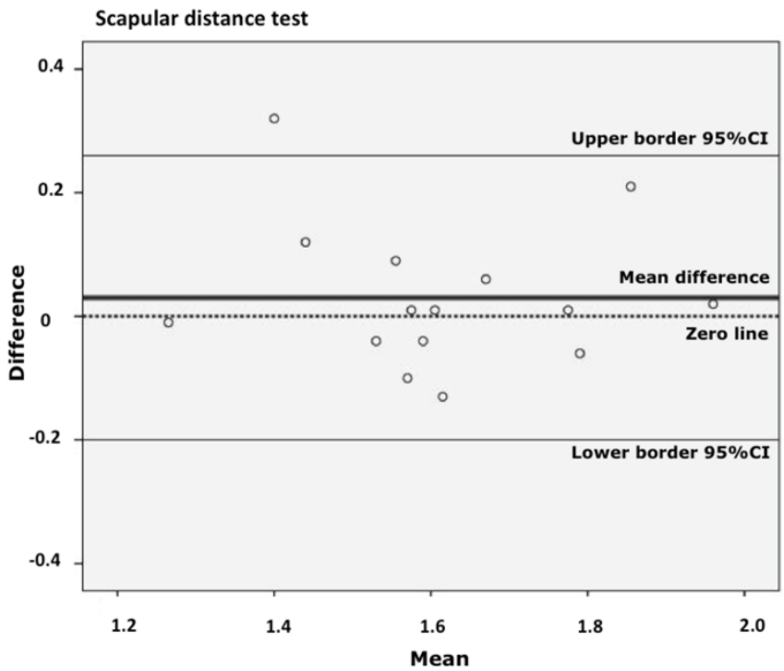
		Test characteristics				Psychometric properties		
		Active	**Passive**	**Sit**	**Supine**	**Reliable**[a]	**IwS vs controls**[b]	**IwS with different levels of PAF**[c]
Test 1: Observation of tilting and winging	At rest		X	X		X	X	X
	During movement	X		X		X	X	
Test 2: Shoulder girdle position	Pectoralis minor index		X	X				
	Scapular distance		X	X		X		
	Acromial index		X		X	X		
Test 3: Lateral rotation, inclinometry	Start (°)		X	X		X		
	At 45° FF (°)		X	X		X	X	X
	At 90° FF (°)		X	X		X	X	X
	At 135° FF (°)		X	X		X	X	X
Test 4: Maximal humeral elevation		X		X		X	X	X
Test 5: Medial rotation test		X			X	X		

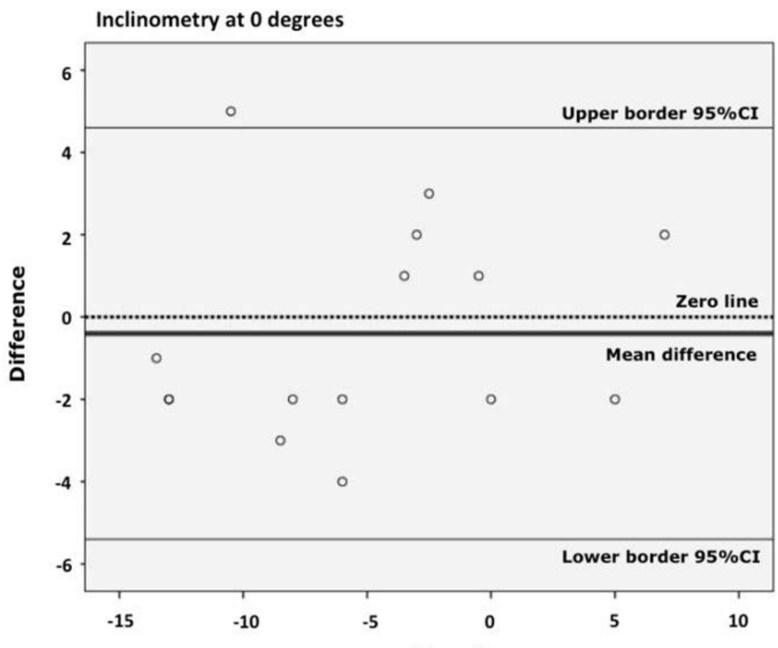
FF: forward flexion; [a] intra-observer reliability; [b] discriminative between IwS and controls; [c] discriminative between IwS with different levels of proximal arm function; PAF proximal arm function

Appendix 2. Bland-Altman plots: difference between two measurement sessions versus the mean of both

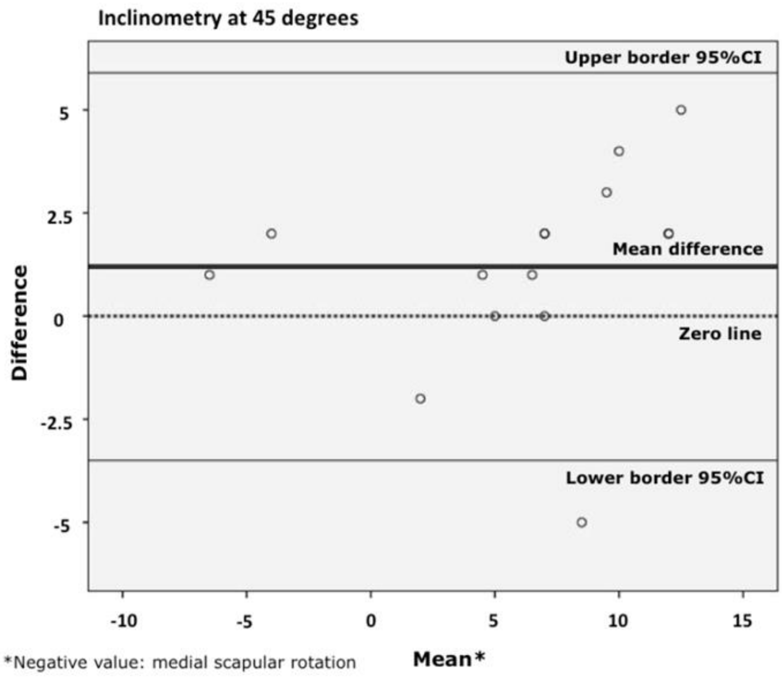


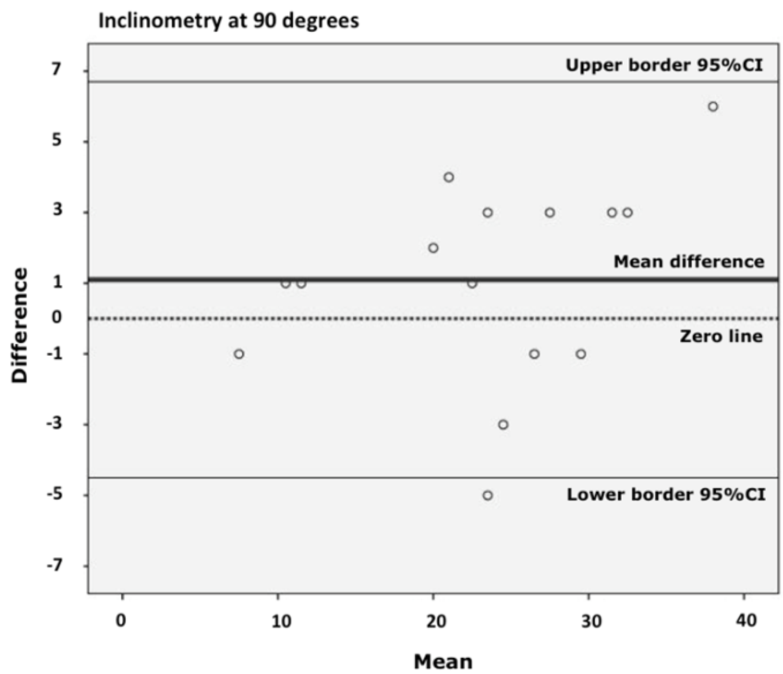


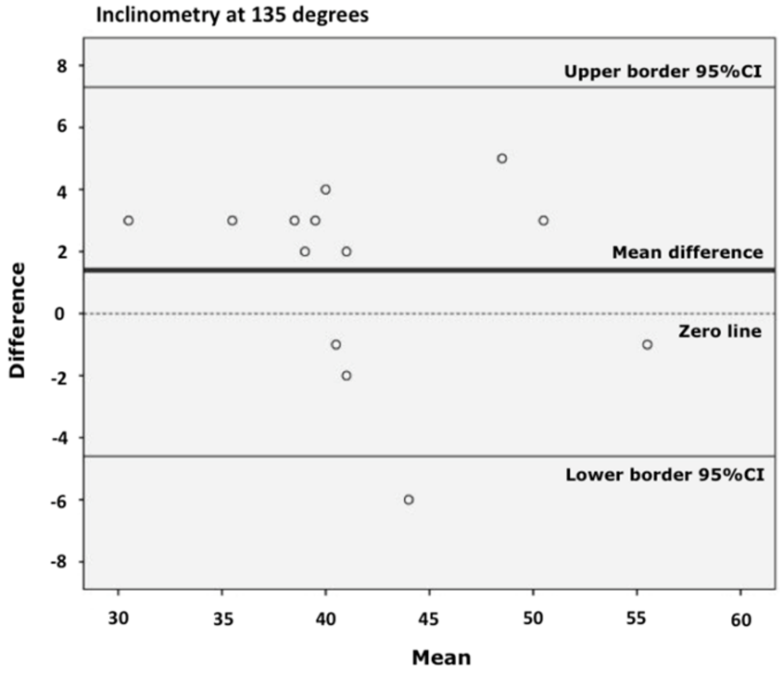




*Negative value: medial scapular rotation


Inclinometry at 45 degrees
Upper border 95%CI
Mean difference
Zero line
Lower border 95%CI
Difference
5
2.5
0
-2.5
-5
-10
-5
0
5
10
15
*Negative value: medial scapular rotation
Mean*


Inclinometry at 90 degrees
Upper border 95%CI
Mean difference
Zero line
Lower border 95%CI
Difference
7
5
3
1
0
-1
-3
-5
-7
0
10
20
30
40
Mean


Inclinometry at 135 degrees
Upper border 95%CI
Mean difference
Zero line
Lower border 95%CI
Difference
Mean
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6
4
2
0
-2
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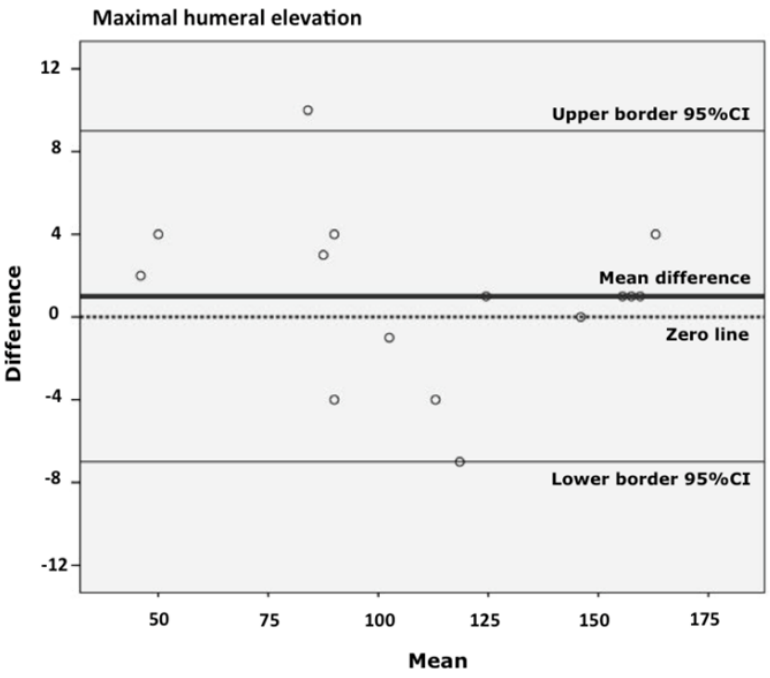
35
40
45
50
55
60


Maximal humeral elevation
Upper border 95%CI
Mean difference
Zero line
Lower border 95%CI
Difference
Mean
12
8
4
0
-4
-8
-12
50
75
100
125
150
175

Chapter 6

Disability and Rehabilitation; under review

Associations between three-dimensional scapulothoracic kinematics and the outcome of clinical scapular measures in individuals with stroke

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1. Abstract

Purpose: This study investigated the relationship between clinical scapular measures and three-dimensional (3D) scapulothoracic kinematics post-stroke.

Method: 3D scapulothoracic kinematics of 18 individuals with stroke (IwS) were calculated during a unilateral forward flexion task (active/passive, low/high). Secondly, scapular control was assessed using various clinical tests: (1) observation of tilting and winging at rest and during movement; (2) shoulder girdle position while upright and in supine; (3) scapular lateral rotation at different forward flexion heights (inclinometry); (4) maximal humeral elevation; and (5) the medial rotation test. Correlations between kinematics (at start position, at 45° (low forward flexion) and 90° (high forward flexion) and the scores for each of the clinical tests were calculated.

Results: An increase in scapular protraction at start position and at 45° of forward flexion was mainly correlated with higher values on the scapular distance test and the acromial index, indicating increased shoulder girdle protraction posture. Increased scapular protraction at 90 degrees of forward flexion was correlated with increased lateral rotation measured with inclinometry at higher forward flexion. Increased lateral rotation during low and high forward flexion was generally related with clinically measured scapular lateral rotation at different forward flexion heights. No other ClinScaP test was significantly correlated with scapulothoracic protraction/retraction or medial/lateral rotation. Anterior/posterior scapular tilting was not correlated with any of the clinical scapular measures.

Conclusions: Clinical scoring of shoulder girdle position (upright and in supine), as well as inclinometry are deemed most appropriate to clinically assess scapular control post-stroke, in terms of protraction and lateral rotation. The clinical assessment of scapular tilting remains a challenge for future research.

2. Introduction

Many individuals with stroke (IwS) suffer from upper limb impairments and shoulder pain. The reported prevalence of acute post-stroke upper limb dysfunctions varies between 48% and 77%,[1,2] and the general incidence of post-stroke shoulder pain is reported to be around 33%.[3] Bicipital and supraspinatus tenderness, and a positive Neer impingement sign are furthermore described in 54%, 48% and 30% of IwS, respectively.[4] These problems negatively affect functional arm recovery, and thus decrease autonomy in daily living.[5-7]
Given the fact that adequate scapulothoracic behavior is a prerequisite for proper and pain free shoulder movement, knowledge of scapular position and movement on the thorax seems essential to understand post-stroke shoulder dysfunctions and pain. Current evidence already suggests an altered scapulohumeral rhythm and altered scapulothoracic kinematics post-stroke,[8-13] and some authors have pointed towards the role of these alterations in the development of shoulder pain.[9,10,12]
Three-dimensional (3D) movement analysis is considered the golden standard for objective movement analysis of the scapulothoracic joint, as it allows describing scapular kinematics with respect to the thorax in three rotations (protraction/retraction, medial/lateral rotation, anterior/posterior tilt). Moreover, the reliability of 3D scapulothoracic movement analysis has been previously confirmed in IwS.[14] Although of great value in terms of detailed kinematic information, these motion-tracking systems are expensive due to the many requirements regarding materials and accommodation, and the fact that 3D assessment requires specific expertise and is time-consuming. Hence, 3D scapulothoracic movement analysis is less suited to be used by physiotherapists in a private practice or in rehabilitation centers without specific laboratory settings.
Very recently, a more clinical approach for assessing scapulohumeral control in IwS was developed,[15] i.e. the clinical scapular protocol (ClinScaP). This assessment covers different aspects of static and dynamic scapular control. Good intra-observer reliability was observed for most tests, except for the pectoralis minor index.[15] The observation of tilting and winging, inclinometry measures for lateral rotation and measures of maximal humeral elevation were found to differentiate between controls and IwS, and between IwS with different levels of proximal arm function.
In this study, we aimed to investigate how the different reliable tests of ClinScaP are related to the joint angles provided by 3D scapulothoracic movement analysis in IwS.

3. Methods

3.1. Participants

IwS were recruited from different rehabilitation centers in Flanders (Belgium). They were eligible for inclusion when they (1) were at least 1 month after a first time stroke (cortical or subcortical); (2) were able to perform 45° of

humerothoracic forward flexion (FF); and (3) had a mild to moderate upper limb motor impairment according to the upper limb motor part of the Fugl-Meyer (score ≥ 30).[16] Participants were not considered for inclusion in case they had (1) reduced communicative or cognitive abilities; (2) a known history of shoulder and/or neck pain or discomfort in the last six months prior to stroke; (4) an event of shoulder dislocation, fracture or surgery during life time; or (5) other systemic and/or neurologic diseases. All IwS received standard care and physiotherapy, attuned to their specific needs.
Prior to study participation, all participants gave informed consent, as approved by the Ethical Committee of the University Hospital Leuven (Belgium).

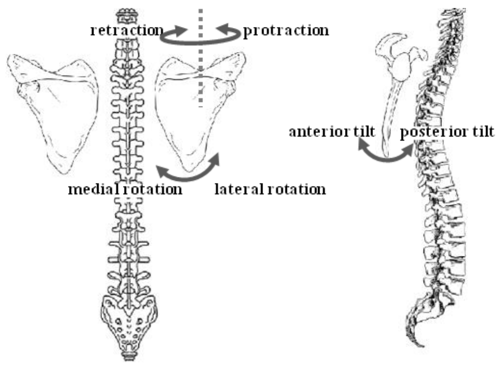
3.2. Kinematic data acquisition

3D kinematic data were captured with 15 infrared cameras sampling at 100 Hz (Vicon, Oxford Metrics, UK) and filtered with a spline-interpolation.[17] Clusters of three or four markers, mounted on tripods or cuffs, were placed on the sternum, scapula (flat part of the acromion) and upper arm (proximal, lateral) (Figure 1A). Anatomical landmarks were digitized during static trials, using a pointer with four linear markers. Anatomical landmarks were defined within their respective segmental marker cluster (CAST-procedure),[18] and subsequently used to construct anatomical coordinate systems and to calculate joint kinematics. All kinematic calculations were done according to the ISB-guidelines.[19] Scapulothoracic kinematics were described in following three rotations: protraction/retraction, medial/lateral rotation, anterior/posterior tilting. These rotations are visualized in Figure 1B.

Figure 1. Marker cluster placement (A) and (B) 3D scapulothoracic rotations

A.

B.



3.3. Measurement procedure

All measurements took place at the Clinical Motion Analysis Laboratory of the University Hospital Pellenberg. While seated on a chair with low back support, marker clusters were mounted on the participant's upper body. Static calibration trials were first collected to digitize the anatomical landmarks. IwS were subsequently instructed to perform a low and high unilateral humerothoracic elevation in the sagittal plane (FF). These active FF tasks were then repeated passively, with the participant's arm supported by the researcher during task execution. A bar was placed in front of the participants in order to indicate the appropriate FF height for the low (±60°) and high (±120°) FF tasks. After some practice trials, eight repetitions per task (active low FF, active high FF, passive low FF, passive high FF) were recorded.
After the 3D kinematic analysis, all markers were removed and the clinical scapular protocol (ClinScaP) was conducted in all participants. All measures of the ClinScaP were evaluated by one skilled physiotherapist, who was assisted by a second physiotherapist during test 3.

Test 1: Tilting and winging

While seated upright in a chair with low back support, the presence (score 1) or absence (score 0) of scapular tilting and winging was scored by observing the participant's scapular position on the thorax. This scoring was done during rest (both arms alongside the body, thumbs pointing forward) and during active unloaded FF. Participants were instructed to move bilaterally at a rate of 3 seconds up toward their maximal FF, and 3 seconds down toward the rest position. Observation was done from a dorsal and lateral position. Presence of tilting or winging indicated a prominence of the inferior tip of the scapula dorsally or prominence of the medial scapular border, respectively.[20,21] A total score was calculated for observation at rest and for observation during movement. Score '0' indicated no presence of tilting or winging, score '1' the presence of tilting or winging and score '2' the presence of both tilting and winging.

Test 2: Shoulder girdle position

Two different measures were used to evaluate the participant's shoulder girdle position.
Acromial index (AI): this index was assessed with the participant lying supine, the arms relaxed alongside the body with the palm placed on the table. The participant was instructed to stay relaxed during the measurement. In this position, the acromial angle was palpated and the vertical distance between this angle and the table was measured with a sliding carpenter. This distance was normalized to subject height and defined as the AI.[22]
Scapular distance test (SDT): this test assesses the position of the scapula on the trunk in an upright-seated position with low back support with the arms relaxed alongside of the body. The SDT was calculated by dividing the distance

between the acromial angle and the spinous process of T3 by the distance between the acromial angle and the scapular trigonum. Anatomical landmarks were first palpated and marked using a pen, and distances were subsequently measured with flexible measurement tape.[23]

Test 3: Scapular lateral rotation

Scapular lateral rotation was assessed with an inclinometer (Plurimeter -V gravity inclinometer, Dr Rippstein, Switserland), while participants were seated upright on a chair with low back support. The inclinometer was held manually on the scapular spine by the skilled physiotherapist, while the assisting physiotherapist passively elevated the participant's arm in the sagittal plane (FF). The amount of lateral rotation was read from the inclinometer at rest (arm alongside the body), and at 45°, 90° and 135° of passive FF (determined by goniometry).[24] The elbow was extended and the thumb pointed upward during task performance.

Test 4: Maximal active humeral elevation

While seated upright with low back support, the maximal range of active humerothoracic elevation in the sagittal plane (FF) was read from a goniometer. Participants were instructed to extend the elbow and to keep the thumb pointing upward during movement.

Test 5: Medial rotation test

This test assessed scapular dynamic control while participants laid supine with the upper arm passively supported by a wedge in 90° of humerothoracic scapular plane elevation (30° anterior to the frontal plane), and the elbow flexed. While actively performing a movement towards glenohumeral internal rotation, i.e. moving the forearm towards the table, the participant was instructed to keep the scapula still. Meanwhile, the assessor palpated the anterior humeral head and the coracoid process and judged the amount of anterior humeral translation and scapular movement. Aberrant dynamic control indicated excessive anterior humeral translation (more than 4 mm, judged by palpation) or scapular movement (more than 6 mm, judged by palpation in the direction of anterior tilt, downward rotation or scapular elevation) before 60° of internal glenohumeral rotation. A total score of '0' indicated correct humeral translation and scapular movement. A score of '1' meant aberrant humeral translation or aberrant scapular movement, and a score of '2' indicated aberrant humeral translation and aberrant scapular movement. Every participant received some practice trials to get familiarized with the test before the formal test was executed.[25,26]

3.4. Kinematic Data Analysis

From the eight recorded kinematic trials, only the middle six repetitions per FF task were selected for data analysis (as these were considered free from initiation/completion strategies). Movement cycles were visually defined from start to highest arm position. Data was further processed with Matlab®, using U.L.E.M.A. (https://github.com/u0078867/ulema-ul-analyzer). Each movement cycle was time-normalized and joint angles were visualized as a function of time to check for erroneous signals. Discrete parameters of interest from the kinematic analysis were 3D joint angles of the scapulothoracic joint (1) at start position; (2) at 45° of FF in the low FF tasks; and (3) at 90° of FF in the high FF tasks (45° and 90° of humerothoracic FF were chosen as these heights were reached by all participants).

3.5. Statistical analysis

Normal distribution of the continuous data was verified with the Kolmogorov-Smirnov test. Pearson correlations (r) were calculated to investigate the associations between the discrete 3D scapulothoracic joint angles (at start, 45°, 90° of both active and passive FF) and the outcomes on test 2, 3 and 4 of the ClinScaP. A biserial correlation (r_b) was calculated between the discrete 3D scapulothoracic joint angles and the outcome on test 1 and 5 of the ClinScaP. Correlations >0.90 were considered very high, 0.70-0.89 high, 0.50-0.69 moderate, 0.30-0.49 low and <0.29 very low.[27] All analyses were done with IBM SPSS statistics 22.0.

4. Results

4.1. Participants

Eighteen participants were included in the current study. An overview of participants' characteristics and their results on the several tests of ClinScaP is given in Table 1 and 2, respectively. Average values of 3D scapulothoracic joint angles are provided in Table 3. There were no missing values.

Table 1. Participants' characteristics

Participants,	N, male/female	18, 12/6
Age,	mean, SD (years)	51.8 ± 13.3
Dominant side,	right/left	18/0
Hemiplegic side,	right/left	10/8
Time since stroke,	weeks	15.6 ± 10.0
Lesion location,	cortical/subcortical	14/4
Fugl-Meyer proximal*	range	24-35
Fugl-Meyer upper limb¥	range	45-63

*: Shoulder and elbow parts of the Fugl-Meyer upper limb motor scale, maximum score 36; ¥: Fugl-Meyer upper limb motor scale, maximum score 66

Table 2. Descriptive statistics for the different tests of the ClinScaP

		Mean (SD)	Score 0	Score 1	Score 2
Test 1: Observation	At rest		61.1%	27.8%	11.1%
	During movement		22.2%	50%	27.8%
Test 2: Shoulder girdle position	Scapular distance test	4.3 (0.8)			
	Acromial index	1.6 (0.2)			
Test 3: Lateral rotation	Start	2.7 (6.2)			
	At 45° humeral elevation	4.2 (6.3)			
	At 90° humeral elevation	20.8 (7.8)			
	At 135° humeral elevation	40.1 (6.3)			
Test 4: Max humeral elevation		154.7 (12.5)			
Test 5: Medial rotation test			44.4%	22.2%	33.3%

SD: standard deviation; Max: Maximum; Mean and SD are expressed in degrees for test 3 and 4. Test 2 has no unit (normalized distances).

Table 3. 3D scapulothoracic joint angles (degrees) at start, 45° and 90° of forward flexion

	Protraction Mean	Protraction (SD)	Lateral rotation Mean	Lateral rotation (SD)	Anterior tilting Mean	Anterior tilting (SD)
0°, start FF	29.4	(7.2)	1.1	(7.0)	9.0	(6.4)
45°, active FF	37.9	(6.3)	8.8	(7.8)	2.8	(6.8)
45°, passive FF	37.2	(5.4)	10.6	(7.9)	3.3	(7.3)
90°, active FF	46.3	(9.8)	36.2	(12.7)	0.9	(9.4)
90°, passive FF	45.7	(9.6)	35.0	(11.7)	2.1	(9.8)

FF: forward flexion; SD: standard deviation; Mean and SD are expressed in degrees

4.2. Relation between 3D scapulothoracic measures and the ClinScaP

Correlation coefficients between 3D joint angles of the scapulothoracic joint and the outcomes on the different ClinScaP tests are given in Table 4. At start position (0° FF), low to moderate correlations were found between 3D *protraction/retraction* and inclinometry at 135° of FF (0.52), the acromial index (0.47) and the scapular distance test (0.65). The latter two ClinScaP tests were also moderately correlated with 3D protraction/retraction at 45° FF (active and passive)(0.5-0.65). At 90° of active and passive FF, 3D protraction/retraction showed moderate correlations only with inclinometry at 135° of FF (0.5-0.57). Higher scores for the acromial index, the scapular distance test and inclinometry at 135° of FF were associated with an increase in 3D protraction for all FF tasks.

Moderate to high correlations were found between 3D *medial/lateral rotation* at start position (0° FF) and inclinometry at start (0.72) and at 45° of FF (0.64). At 45° of FF, medial/lateral rotation correlated low (passive; 0.49) to moderately

(active; 0.52) with inclinometry at start; moderately (active; 0.68) to high (passive; 0.8) with inclinometry at 45° of FF; and moderately (passive; 0.0.54-0.62) with inclinometry at 90° and 135° of FF. At 90° of FF, low correlations were found between medial/lateral rotation and inclinometry at 135° (passive FF; 0.49); and moderate correlations were found between for inclinometry at 45° of FF (active and passive FF; 0.52-0.57). For all FF tasks, an increase in lateral rotation measured with inclinometry was associated with an increase in 3D lateral rotation values.
No significant correlations between 3D *anterior/posterior tilt* and any of the ClinScaP tests were found.

5. Discussion

From a clinical perspective, it is an opportunity to gain knowledge on 3D scapulothoracic kinematics through the use of clinical scapular measures. Therefore, this study assessed the association between the ClinScaP and the 3D scapulothoracic assessment in a group of IwS with mild to moderate upper limb impairments.

5.1. Relation between 3D scapulothoracic and ClinScaP measures

Correlation coefficients were calculated to investigate which 3D scapular rotations at different FF heights (0°, 45° or 90° of FF) were associated with which clinical scapular tests. Inclinometry measures at 0° and at 45° of FF were related to 3D scapular lateral rotation (at start, 45° and 90° of FF), i.e. an increase in scapular lateral rotation measured with inclinometry was also found in the kinematic lateral rotation data. Inclinometry measures at 90° and at 135° of FF were generally not associated with 3D scapular lateral rotation. In contrast, higher inclinometry measures at 135° of FF, as well as the increased scapular distance measures were moderately related to increased 3D scapular protraction at start position. Increased inclinometry at 135° was also moderately correlated to increased 3D scapular protraction at 90° of FF. 3D scapular protraction at 45° was mainly associated with the scapular distance test (active FF) and the acromial index (passive FF), whereby higher scores for both ClinScaP tests corresponded to an increase in 3D scapular protraction.
The possibility to assess scapular protraction and lateral rotation in IwS based on clinical tests has high clinical relevance. Alterations in scapular protraction have been previously related to posterior-inferior glenohumeral capsular or muscular stiffness. Glenohumeral posterior stiffness in turn, is linked to the development of glenohumeral pathologies.[28] Our study results showed that an increased 3D scapular protraction at rest might be reflected in higher values on the scapular distance test. During movement, increased 3D protraction was also related to higher values on the scapular distance test, the acromial index, and inclinometry at 135°. Alterations in scapular protraction have been described in IwS during reaching tasks, which confirms the clinical relevance of the

Table 4. Correlation coefficients between discrete 3D scapulothoracic joint angles and the outcomes for the different tests of the ClinScaP

3D scapulothoracic joint angles		Observation (r_b)		Shoulder girdle position (r)		Lateral rotation (r)				Maximal humeral elevation (r)	Medial rotatio test (r_b
		Rest	Movement	SDT	AI	start	45°	90°	135°		
Protraction	Start FF	0.29	0.30	**0.65****	0.47*	0.07	0.10	0.34	**0.52***	0.34	0.25
	45°, active FF	0.14	0.17	**0.65****	**0.55***	0.21	0.21	0.34	0.40	0.03	0.24
	45°, passive FF	0.12	0.20	**0.50***	**0.64****	0.23	0.36	0.31	0.43	0.35	0.24
	90°, active FF	0.09	-0.27	0.36	0.36	0.16	0.37	0.45	**0.57***	0.30	-0.16
	90°, passive FF	0.20	-0.23	0.32	0.32	0.11	0.41	0.41	**0.50***	0.29	-0.13
Lateral rotation	Start FF	0.14	-0.12	0.03	0.02	**0.72****	**0.64****	0.33	0.27	-0.20	0.04
	45°, active FF	-0.03	-0.24	-0.01	-0.25	**0.52***	**0.68****	0.39	-0.39	0.04	-0.10
	45°, passive FF	0.02	-0.28	0.04	-0.09	0.49*	**0.80****	**0.54***	**0.62****	0.22	-0.06
	90°, active FF	0.13	-0.12	-0.19	-0.16	0.26	**0.57***	0.35	0.42	0.28	-0.17
	90°, passive FF	0.07	-0.23	-0.12	-0.23	0.14	**0.52***	0.43	0.49*	0.17	-0.37
Tilting	Start FF	-0.12	-0.23	0.03	0.41	-0.26	-0.19	-0.10	-0.09	0.25	-0.19
	45°, active FF	-0.10	0.01	0.14	0.26	-0.21	-0.05	-0.13	-0.08	-0.02	-0.16
	45°, passive FF	-0.19	-0.18	0.01	0.24	-0.22	-0.02	-0.04	-0.04	0.02	-0.14
	90°, active FF	-0.04	0.26	0.06	0.15	-0.07	0.01	-0.06	-0.05	0.03	0.13
	90°, passive FF	-0.24	0.01	0.03	0.27	0.22	0.24	0.13	0.08	-0.10	0.18

FF: Forward flexion; r_b: biserial correlation coefficients; r: pearson correlation coefficients

*: significance level ≤.05; **: significance level ≤.01; Moderate and high correlations are marked in bold

availability of tests related to scapular protraction.[29] The availability of clinical measurements associated with scapular lateral rotation is also valuable in the clinical setting, since alterations in scapular lateral rotation are known to contribute to the development of shoulder pathologies such as subacromial impingement.[30-33] Previous research has already shown alterations in 3D scapular lateral rotation in IwS compared to controls.[30,31] More specifically, IwS with shoulder pain use more scapular lateral rotation during active and passive abduction and FF of the hemiplegic arm.[30] In contrast, others have reported that those IwS with more shoulder pain had less 3D scapulothoracic lateral rotation during scapular plane arm elevation.[31]

Maximal humeral elevation, as measured with goniometry, and the medial rotation test were not significantly correlated to any 3D scapulothoracic angle. Moreover, the clinical observation of tilting and winging was, contrary to our expectations, not correlated to the 3D measures of scapular anterior/posterior tilt or protraction/retraction. It appears that visually rating abnormalities in scapular position on the thorax might be better described with the general term "scapular dyskinesis". This term refers to a clearly apparent abnormality in scapular positioning, rather than an alteration in a specific rotation.[32,33] Although not associated to one specific 3D scapular rotation, we have previously shown that both the observation of tilting and winging and the assessment of maximal humeral elevation were valuable measures to discriminate between IwS and healthy controls and between IwS with different levels of proximal arm function.[15] It thus appears that the observed dyskinesis and decreased maximal humeral elevation in IwS compared to controls, and in IwS with reduced proximal arm function encompasses alterations in a combination of the three scapular rotations.

5.2. Future directions

The ClinScaP includes specific clinical scapular measures that are commonly used in musculoskeletal rehabilitation.[22] None of the included clinical tests correlated with 3D scapulothoracic tilting, which points towards the importance of including other measures to improve the clinical assessment of these angles. For example, Scibek and Carcia (2014) recently introduced a clinical measure for scapular tilting movement by means of a modified inclinometer.[34] One might moreover consider to go beyond specific measures on body function level (International Classification of Functioning, ICF),[35] and additionally investigate the relation between 3D scapulothoracic measures and measurement scales at the level of activity (e.g. ARAT).

We only included IwS with a moderate to mild upper limb dysfunction, based on the Fugl-Meyer upper limb motor scale. However, further generalization of results would benefit from the inclusion of IwS with lower upper limb motor function.

Lastly, the clinical implementation of the ClinScaP also requires the availability of reference data to distinguish between e.g. different levels of motor impairments or to identify IwS at risk to develop shoulder pain, based on respectively 'functional' or 'dysfunctional' scores, or 'painful' and 'pain free' scores for the different tests. To accomplish this, larger study samples per subgroup of IwS (low, moderate or high arm motor function; with or without shoulder pain) should be measured, and cut-off values per test should be determined for inclusion in a specific group.

6. Conclusion

The use of 3D scapulothoracic kinematics in clinical settings is not feasible given the high cost and specific requirements of a movement analysis lab. Hence, the quantification of scapular position and movement characteristics by means of clinical measures becomes more important. Given the kinematic alterations in scapular protraction and lateral rotation already described in IwS and their relation with the development of shoulder pathologies,[36-38] early detection of alterations in scapular position in terms of protraction and lateral rotation are crucial. The availability of clinical tests associated with these kinematic rotations is thus highly relevant. This study revealed that (1) inclinometry at 45° of FF is related to 3D lateral rotation at start, 45° and 90° of active and passive FF; (2) inclinometry at 90° and 135° of FF is related to 3D lateral rotation at 45° of passive FF; (3) inclinometry at 135° of FF is linked to 3D lateral rotation at 45° of passive FF as well as to 3D protraction at start and at 90° of active and passive FF; and (4) the scapular distance test and acromial index are related to scapular protraction at start and at 45° of FF. Scapulothoracic tilting was not significantly related to any test of the ClinScaP, prompting a search for alternative clinical measures for this specific scapular rotation.

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General discussion

The general discussion will first address the different aims and research questions of this doctoral thesis, followed by a focus toward several specific methodological issues. Subsequently, we discuss the implications of our results for upper limb assessment post-stroke and present a hypothetical assessment and treatment-planning framework in IwS, including the scapulothoracic joint.

1. Reflection on aims and research questions

The scope of this doctoral project was to contribute to the understanding of impaired scapular control in IwS, based on following research questions:

1. How can we assess the scapulothoracic joint and its function in a laboratory and clinical setting (**Chapter 1, 2** and **5**)?
2. Can measures of scapular control identify deficits in scapular positioning, movement and muscle activation patterns in IwS (**Chapter 3, 4,** and **5**)?
3. What is the relationship between three-dimensional (3D) and clinical measures of scapular position and movement control (**Chapter 6**)?

1.1. Reflection on research question 1: Introduction of 3D scapular kinematic measurement and the ClinScaP in IwS

1.1.1. 3D scapular kinematics

The review of literature showed a clear lack of scapular kinematic measurements and scapular muscle timing assessments in IwS (**Chapter 1**). As such, a protocol for the assessment of 3D scapular movement analysis, and combined assessment of scapular muscle timing parameters was developed. However, clinical implementation of a newly developed protocol firstly requires the establishment of its reliability. Given the challenging and demanding protocol we developed for IwS, feasibility of the assessment was also tested (**Chapter 2**). We evaluated if IwS were able to adhere to the elevation protocol as requested, as well as the amount of training necessary for the assessor to be able to perform accurate placement of the marker clusters and palpation of anatomical landmarks.
The reliability of scapular start position, total range of motion (ROM) and the scapular joint movement patterns was assessed using ICCs (discrete data) and CMCs (continuous data). However, the magnitude of the ICC is dependent on the variability of between-subject data, i.e. a high ICC can hide poor trial-to-trial consistency in case of high between-subject variability.[1] Conversely, limited between-subject variability could result in poor ICCs even when trial-to-trial consistency is high. Similarly, the CMC is influenced by the total ROM covered during the movement, i.e. movement patterns with higher ROM result in higher CMCs, despite high trial-to-trial variability.[2] Therefore, ICCs and CMCs were always considered in conjunction with measurement errors (**Chapter 2**), i.e. standard error of measurement (SEM) for discrete data and sigma for continuous data.[1,3-5] Measurement

errors have the same unit as the measurement of interest (in this case degrees), and are thus an indicator of precision of the measurement protocol. Lastly, measurement errors allow distinguishing changes due to natural variability or inaccuracy from a true change or difference in performance. Knowledge of the stroke specific measurement errors was crucial when comparing IwS versus healthy controls in **Chapter 4**. Based on our reliability results of **Chapter 2**, i.e. higher reliability for forward flexion tasks executed with the hemiplegic arm (IwS) or the dominant arm (controls), a sound reasoning for parameter and task selection for kinematic studies in IwS was also provided. We adhered to these recommendations in our further kinematic studies (**Chapter 4** and **6**).

1.1.2. ClinScaP

Notwithstanding the value of 3D measurement systems in terms of objectivity and detailed kinematic information, motion-tracking systems are only available in a specific laboratory environment. As such, these systems have limited potential to measure IwS in a clinical setting. By introducing the ClinScaP, we wanted to offer an accessible and low-cost assessment tool to identify abnormal scapular characteristics in IwS. Scapular mal-positioning and dyskinesis are linked to e.g. delayed lower trapezius activation or decreased serratus anterior activity. This might induce scapular winging (protraction), increased anterior tilting, or reduced lateral rotation. The 3D character of scapulothoracic movement challenges the clinical scapular assessment. Based on knowledge from musculoskeletal rehabilitation, those tests that were expected to be associated with a specific scapular rotation were retained (**Chapter 5**).

To test the amount of scapular lateral rotation, we specifically included inclinometry measures at different forward flexion (FF) heights. This test was performed during passive static FF, as opposed to active abduction in standard musculoskeletal testing. Furthermore, clinical observation of the medial (winging) and the inferior (anterior tilting) scapular border prominence during arm elevation was added as a dynamic test for scapular dyskinesis. Uhl et al. (2009) suggested that arm FF is the optimal task for evaluating such scapular dyskinesis dynamically.[6] Dynamic scapular control was further assessed using the medial rotation test, and maximal humeral elevation was also added to the protocol.[7]

To ensure IwS with various levels of arm motor function could be assessed using the ClinScaP, we additionally included static measures (acromial distance, scapular distance, pectoralis minor index, observation at rest) to assess shoulder girdle position and scapular positioning at rest. Inflexibility or shortening of soft tissue structures has been suggested as a possible contributor to shoulder disorders. For example, stiffness of pectoralis minor can induce increased scapular anterior tilt and protraction due to its pull on the coracoid.[8] Many authors have suggested that such an altered scapular position is associated with a reduction of the subacromial space[9,10] and that

it leads to muscle imbalances and adaptive shortening of postural muscles and other soft tissue, e.g. posterior shoulder structures.[11] Inflexibility of posterior shoulder structures in return is associated with glenohumeral internal rotation deficits, which is related to a more anteriorly tilted scapular position.[12] As such, a vicious circle of inflexibility, leading to scapular malposition and further inflexibility is created.
We found sufficient reliability for most ClinScaP tests in IwS, except for the pectoralis minor index, which was therefore excluded in the subsequent study using ClinScaP (**Chapter 6**).

1.2. Reflection on research question 2: Observed differences in scapular characteristics

We provided evidence that 3D kinematic scapular analysis and scapular muscle timing assessment can identify differences between IwS and healthy controls (**Chapter 3** and **4**). We furthermore showed that inclinometry measures for lateral rotation, observation of tilting and winging, and maximal humeral elevation, are useful tests to distinguish between IwS and controls and between IwS with different levels of proximal arm function (**Chapter 5**).
In IwS with a *low proximal arm function without shoulder pain*, increased lateral rotation at high elevation angles and less active humeral elevation were generally observed in comparison to IwS with a moderate to high proximal arm function and healthy controls (**Chapter 5**). IwS with a *moderate proximal arm function without shoulder pain* also showed increased lateral rotation, as well as scapular mal-positioning and dyskinesis, and less active humeral elevation in comparison to high functioning IwS and healthy controls (**Chapter 5**). IwS with a *high proximal arm function without shoulder* pain did not perform differently on the ClinScaP compared to controls. However, muscle recruitment patterns indicated early and prolonged infraspinatus activity in high FF compared to controls and compared to IwS with a high proximal arm function with shoulder pain (**Chapter 3** and **5**). IwS with a *high proximal arm function with shoulder pain* showed later activity of lower trapezius in comparison to high functioning IwS without shoulder pain. Serratus anterior was furthermore earlier inactive compared to controls and high functioning IwS without shoulder pain (**Chapter 3**). Finally, in IwS with *a moderate to high proximal arm function without shoulder pain*, delayed activation and early inactivation of serratus anterior, early lower trapezius activation and late infraspinatus inactivation were reported during low FF compared to healthy controls. In high FF, less posterior tilting and more lateral rotation were observed (**Chapter 4**).

1.3. Reflection on research question 3: Association between ClinScaP and 3D scapulothoracic kinematics

Lastly, we investigated which clinical measures were associated with altered 3D scapulothoracic kinematics for specific degrees of FF (**Chapter 6**). This increased our understanding of which 3D scapulothoracic angles are assessed with the various ClinScaP tests. As expected, 3D lateral rotation was related to inclinometry measures. Scapular tilting angles, however, could not be associated with any clinical test. Protraction of the scapulothoracic joint was not only related to the scapular distance test and the acromial index, but also to inclinometry measures at 135 degrees of passive FF. IwS with low proximal arm function showed increased lateral rotation at 135 degrees (inclinometry) compared to those with high proximal arm function. Lower proximal arm function thus seems related to increased 3D scapulothoracic lateral rotation, as well as protraction.

Although IwS with different levels of proximal arm function performed differently on the observation of winging and tilting and for maximal active humeral elevation (**Chapter 5**), these differences were not related to one specific 3D scapular rotation (**Chapter 6**). We assume that the observed scapular dyskinesis and decreased maximal humeral elevation in IwS with lower proximal arm function (**Chapter 5**) encompasses alterations in a combination of the three scapular rotations. However, the exact role of these alterations in creating or aggravating shoulder dysfunction is not yet fully understood.

1.4. Reflection on the reported differences in this doctoral thesis

Five important scapular movement alterations were seen in IwS: (1) increased lateral rotation during passive FF (independent from muscle activity) (**Chapter 5**); (2) increased lateral rotation and decreased posterior tilting during active FF (without simultaneous EMG timing alterations) (**Chapter 4**); (3) increased lateral rotation measured by inclinometry at 135 degrees of passive FF, partially explaining an increased scapular protraction posture (**Chapter 5** and **6**); and (4) presence of scapular dyskinesis at rest (**Chapter 5**). Since these differences exist independently from altered muscle activity, they may be explained by restrictions in glenohumeral structures that pull the scapula in a more protracted, less posterior tilted and increased lateral rotated position, e.g. posterior-inferior capsular stiffness or muscular tightness/shortening (teres major, latissimus dorsi, pectoralis minor, biceps brachii). Increased anterior tilt and a protracted scapular posture has indeed previously been reported in persons with a short pectoralis minor length and posterior capsular restrictions.[8,12] Moreover, humeral motion (e.g. arm elevation) can create early scapular protraction and lateral rotation by placing tension on the glenohumeral capsule and muscles, especially in the presence of a glenohumeral internal rotation deficit.[12] However, the presence of a glenohumeral internal rotation deficit was not specifically assessed in our studies. In unpublished results on 18

IwS, we found that, apart from glenohumeral internal rotation, elbow flexion was related to scapular anterior tilting movement during arm elevation in IwS. The decreased 3D posterior tilting during active arm elevation might thus be caused by a hypertonic or short head of the biceps brachii in IwS. Conversely, an improper or sloughed spinal position during inclinometry might also explain the increased scapular lateral rotation at 135 degrees of passive FF and decreased posterior tilting.[13] However, although not formally and objectively controlled for this thoracic position, we took care of a proper upright spinal posture during the measurements.[13]
It is important to note that some alterations in scapular motion are potentially compensatory to avoid stress on tissues at risk for impingement. For instance, increased lateral rotation during active arm elevation (**Chapter 4**) could be a compensatory movement to provide sufficient subacromial space, which is believed a prerequisite for a pain free shoulder.[14,15] Unfortunately, we did not measure kinematics of IwS with shoulder pain, and cannot conclude whether this increased scapular lateral rotation during active arm elevation is also apparent in IwS with shoulder pain.
With regard to EMG measurements in IwS (with and without shoulder pain), we found specific timing characteristics for the serratus anterior that might negatively influence shoulder kinematics and lead to subacromial pathology. Indeed, late serratus anterior activity, early serratus anterior inactivity and decreased serratus anterior strength is often associated with subacromial impingement syndrome due to limited posterior tilting and lateral rotation movement during arm elevation.[16] Also delayed lower trapezius activation contributes to subacromial disorders by altering scapular posterior tilting and lateral rotation.[17] Since IwS without shoulder pain showed early lower trapezius activation and prolonged infraspinatus activity in comparison to controls, we assume that this is adaptive muscle timing to compensate for the late activity and early inactivity of serratus anterior. IwS with shoulder pain had a later lower trapezius onset and no prolonged infraspinatus activity compared to IwS without shoulder pain. This further strengthens our suggestion that early lower trapezius activity and late infraspinatus inactivity are successful muscle timing adaptations to avoid the development of shoulder pain.

1.5. Conclusion

We presented an extensive method to assess the scapula in IwS. This allowed us to identify distinct alterations in scapulothoracic position and movement patterns in IwS with proximal arm dysfunction and/or shoulder pain. However, whether these alterations are a cause or a compensation strategy remains to be determined. Gaining these insights will further expand our understanding of ideal and faulty shoulder movement patterns.

2. Methodological considerations

Proper scapulothoracic treatment can only be attained via a meaningful and comprehensive assessment. Such assessment should provide detailed information on scapular movement alterations in different directions, and on altered activity of a specific scapular muscle or combination of muscles. Specific methodological choices and considerations related to the objective measurement method are discussed below.

2.1. General methodological choices

2.1.1. Selection of participants

We chose to only include IwS who were able to perform at least 45 degrees of arm elevation. Even though we thereby excluded many IwS with shoulder dysfunctions or pain, we believe that scapulothoracic therapy becomes crucial once a patient is able to move the hemiplegic arm in a low range of arm elevation. From that point in recovery, it is essential to focus treatment on moving in the proper manner, i.e. outside of synergistic movement patterns. This can only be achieved by retraining correct patterns of muscle activity and movement. Our developed measurement protocol specifically aims at assessing scapular movement patterns in this subgroup of IwS at risk to develop shoulder pathology, i.e. those IwS who have regained the ability to lift their arm to 45 degrees and higher.

2.1.2. Movement protocol

The rationale for choosing arm elevation rather than a functional task arose from the evaluation of the shoulder in clinical practice. The elevation task is easy to perform, non-invasive, and at the same time sensitive enough to provide valuable information on scapular changes associated with shoulder pathology.[18,19] Although a vast amount of literature in healthy controls focuses on three-dimensional (3D) kinematics of the scapulothoracic joint during scapular plane elevation, we choose to include frontal (abduction, AB) and sagittal (forward flexion, FF) plane elevation in the protocol. First, these tasks are similar to the available literature on 3D scapulothoracic kinematics in IwS.[20-22] Second, it has been reported that FF simulates forward reach.[23]

When assessing the shoulder after stroke, it is essential to have objective reference data on scapular kinematics and muscle activity during this task. **Chapter 1** outlines such reference data on 3D scapular kinematics and muscle timing during arm elevation in controls with and without shoulder pain.

It was also a distinct choice to assess scapulothoracic kinematics during a low as well as a high elevation task. The inclusion of a low elevation task allowed for the early measurement of IwS who still have limited movement capacities and this task is relevant with respect to the occurrence of subacromial impingement of the supraspinatus tendon.[24] Around 30 to 60 degrees of humeral elevation, the supraspinatus tendon is in closest proximity to the undersurface of the acromion;[25] beyond 70 degrees of elevation, the rotator cuff tendons have moved medially and posteriorly and are less susceptible to mechanical impingement by the acromion. At these higher elevation angles, bursal rather than rotator cuff compression might be the source of pain. Also, internal impingement on the glenoid may occur at elevation angles beyond 90 degrees. Lastly, higher elevation tasks were included as scapulothoracic protraction is minimal prior to 100 degrees of elevation.[26,27]

2.2. Three-dimensional scapulothoracic movement analysis

3D movement analysis has found its way into the upper limb evaluation for some decades. It is an adequate assessment tool for upper limb function, to outline treatment, but also to measure treatment efficacy and to allow follow-up over time. The main advantage of such 3D analysis is that it offers a detailed and objective description of upper limb movement patterns. However, this assessment is challenging since the upper limb has a large number of degrees of freedom, a wide variety of functional capacities and typically performs non-cyclic tasks. Therefore, the International Society of Biomechanics (ISB) has proposed specific standards for measuring and reporting upper limb data.[28] Ever since, these standards have been widely adopted, also in our kinematic studies (**Chapter 2, 4** and **6**).

At the level of the scapulothoracic joint, 3D movement analysis entails several specific challenges. Tracking the movement of the scapula using motion capture technology is complex due to the fact that it is a broad, flat shaped bone, covered with substantial soft-tissue, and with significant skin motion over it. In view of this, scapular movement assessment by means of anatomical markers placed directly on bony landmarks is not recommended. The gold standard procedure for the assessment of scapular kinematics is based on the insertion of cortical bone pins, thereby removing all soft-tissue artefacts.[18] This method is however highly invasive, and thus has limited clinical applicability. Various non-invasive techniques for scapular tracking have been developed, i.e. scapular palpation,[29,30] the scapula locator,[31,32] the acromion marker cluster,[19,33-35] and scapular sensors.[36,37] The first two techniques are applied during static recordings of scapular positions, and are therefore less suited to assess scapular kinematics during arm movement. The acromion marker cluster and the scapula sensors on the other hand allow dynamic recordings. From a clinical perspective, these are of utmost importance to identify natural versus compensatory or aberrant scapular movements.

In our kinematic studies, we used the acromion marker cluster (**Chapter 2, 4** and **6**). In the set-up of our study protocols, we adhered to the guidelines and recommendations proposed by van Andel et al. (2009) and Shaheen et al. (2011) regarding its use and applicability.[33,38] We analyzed scapulothoracic angles up to 90 degrees of elevation and placed the acromion cluster at the meeting point between the scapular spine and the acromion to minimize soft tissue artifact errors. A recent systematic review

on validity and reliability of 3D marker based scapular motion analysis also strongly recommends the acromion marker cluster,[39] combined with a calibration of the scapula. This combined method has good to excellent within session reliability and moderate to excellent between session reliability across literature[39] and a reported accuracy of about 5 degrees (average of the three scapular rotation errors) during arm FF below 90-100 degrees.[33] These results are in line with our reported reliability results (**Chapter 2**). In an attempt to improve this method and to allow for scapular tracking at higher elevation angles and to correct the underestimation of motion at these higher elevation angles, multiple calibrations at 90 degrees and 120 degrees of arm elevation have also been recently proposed.[35,40]
Given that scapular movement is a combination of movement in the sternoclavicular and acromioclavicular joint, future assessment might also benefit from including these joint movements into the protocol. However, isolating and measuring the relatively small movement between the scapula and clavicula in the acromioclavicular joint is technically difficult and requires specific algorithm development to calculate the respective joint angles. Due to these methodological challenges, acromioclavicular movement analysis has not yet been performed in IwS.

2.3. Scapular muscle timing

The inability to coordinate muscle activity during a task that requires only minimal strength (e.g. arm elevation) can exist independently from other motor impairments like weakness, excessive co-contraction or spasticity in IwS.[41] It reflects a loss of the skill to generate temporal muscle activation patterns. Correcting such temporal coordinated activation between muscles is believed to improve proper synergetic actions of these muscles, and as such task performance. Deficient movement is thus assumed to be characterized by an asynchronous pattern of muscle (in)activation in IwS. Specifically for the shoulder complex, early activation of the scapular stabilizers and a correct temporal sequence of scapular musculature in relation to prime mover activity and actual arm movement at the glenohumeral joint are essential for proper scapular position and coordinated scapulohumeral motion.[42,43]

2.3.1. Muscle selection and measurement method

Scapulothoracic muscles of interest in our electromyography (EMG) studies (**Chapter 3** and **4**) were serratus anterior and lower and upper trapezius. Both muscles coordinate normal scapular motion during arm elevation.[19,43] Infraspinatus was also measured given its role as stabilizer of the glenohumeral joint. Finally, also the prime mover for FF, anterior deltoid, was examined. Our review (**Chapter 1**) revealed that nothing had been published at that time on scapular muscle recruitment patterns, which prompted the need for the studies conducted in this doctoral thesis.

Surface EMG is an accurate, reliable, and non-invasive method to measure muscle activity.[44] However, placement of electrodes, type of data processing, calculation of outcome parameters etc. should always be considered when interpreting EMG results. Moreover, this technique is prone to signal noise, and requires thorough standardization. The use of surface electrodes to record infraspinatus is not valid at activation levels smaller than 10% maximal voluntary contraction and one might argue the validity of our results for this muscle.[45] Since we did not measure maximal voluntary contractions in our study samples, we relied on existing literature where infraspinatus activity levels above 10% of the maximal voluntary contraction have been reported during unloaded arm elevation.[7] Sciascia and colleagues (2012) also reported an activity of 20-50% of the maximal voluntary contraction of both the rotator cuff and scapular musculature during scapular plane elevation.[46] As such, the use of surface EMG sensors for infraspinatus in our movement protocol was justified.

2.3.2. Reliability of timing parameters

Reliability results of scapular muscles timing relative to anterior deltoid and/or movement start were reported by Padke and Ludewig (2013) and Seitz and Uhl (2012) in healthy controls and persons with shoulder impingement.[47,48] They reported highest SEMs and minimal detectable change (MDC) values for lower trapezius. We performed a pilot reliability study for muscle onset and offset timing relative to anterior deltoid in five IwS, using the same protocol as described in study 3 (**Chapter 3**) and thereby confirmed the reliability of our protocol. The significant differences between IwS and healthy controls (**Chapter 3**) were larger than the MDC/SEM values reported in our pilot study and the studies of Seitz and Uhl (2012) and Phadke and Ludewig (2013).[47,48] The significant difference between IwS and healthy controls for lower trapezius (**Chapter 4**) cannot be compared to measurement errors of our EMG protocol, though were smaller than the MDC reported by Seitz and Uhl (2012).[48] However, the latter authors evaluated onset timing during scapular plane elevation in non-stroke subjects, and used a different muscle onset determination method.[48] The lack of reliability data concerning the EMG protocol as applied in **Chapter 4** is considered a limitation.

3. A hypothetical framework for future research

Besides gaining knowledge on alterations in scapular characteristics in IwS, this doctoral thesis also wants to propose valuable avenues for future research. With this work, we have demonstrated a clear need for more clinical measures to further deepen our knowledge on proximal arm dysfunctions and pain in IwS. Furthermore, the question whether the observed alterations in scapular control can be extrapolated to effective clinical interventions, needs to be answered. In this last section, we propose a hypothesis-generating framework for the assessment and treatment of the shoulder complex and trunk in IwS.

3.1. Clinical assessment of the shoulder complex

Identification of modifiable factors associated with shoulder dysfunctions and/or pain should be a key objective in the upper limb assessment post-stroke. This allows a relevant prognosis based on the expected factors underlying the development and persistence of dysfunction and pain.

We have already identified specific scapulothoracic tests (observation at rest, inclinometry) that allow distinguishing between IwS with different levels of proximal arm function. We thereby paved the road for future development of a solid, all-encompassing clinical examination of the shoulder complex in IwS, extended to the trunk. Figure 1 presents a framework that serves as the foundation to formulate new research questions on shoulder assessment in IwS. Such a thorough, in-depth examination will allow for a complete understanding of ideal and faulty movement patterns, and can be used longitudinally to expand our knowledge on the cause or compensation strategy of shoulder complex alterations in proximal arm dysfunction and pain in IwS.

3.2. Individualized treatment planning of the shoulder complex

It is well accepted that movement control depends on the contribution of active, passive and of control systems. Within this interpretation, ideal control relies on the appropriate passive support (i.e. proper flexibility of passive structures), combined with proper muscle control (i.e. correct timing characteristics of e.g. lower trapezius and serratus anterior) that is coordinated by the nervous system. Conversely, changes in any of these systems can lead to less than optimal control. This has formed the basis of a range of passive (manual therapy and muscle stretching) and active (motor control retraining) rehabilitation techniques that aim to restore control and reduce disability and/or pain. However, the implementation of this musculoskeletal model into clinical stroke rehabilitation is not straightforward given the complexity and heterogeneity in clinical presentation of the post-stroke individual. This further stresses the need for individualized therapy planning, rather than a one-size-fits-all rehabilitation model.

In the next section, we first go into motor control training in general, and its link with neuroplasticity. Then we propose a hypothetical framework for shoulder complex rehabilitation including the trunk for IwS.

3.2.1. Motor control training

Motor control has been broadly defined as the combination of neurophysiological and biomechanical mechanisms that contribute to movement control.[49] Various brain areas are involved in motor control, including cortical (primary somatosensory cortex, posterior parietal cortex, primary motor cortex, supplementary motor area, premotor cortex, prefrontal cortex) and subcortical structures (basal ganglia), the brainstem (vestibular nuclei, reticular formation), as well as the cerebellum, and spinal networks. Any of these areas might be affected due to stroke and thus impact on motor control, which causes the sensorimotor impairments that underlie the development and persistence of dysfunction and pain. Motor control alterations can be either, neither or both cause and consequence of movement dysfunctions or pain, and thereby initiate a vicious circle of pathology leading to pain, which negatively influences motor control in turn. Sensorimotor adaptations after stroke that relate to our studies are for example (1) redistribution of activity within and between muscles, both augmented or compromised (i.e. earlier activity in lower trapezius or delayed serratus anterior activity in IwS without shoulder pain, respectively); and (2) changes in mechanical features including posture and movement (i.e. increased scapular lateral rotation and decreased scapular posterior tilting in IwS without shoulder pain).

In general, motor control training aims to optimize muscle activation patterns and proprioception, and to improve posture, joint alignment and movement patterns. When translated to the shoulder complex in IwS, this involves training of not only the upper limb, but also the core of the body[50] and the trunk.[51] Postural anticipatory adaptations, core and proximal trunk stability, correct scapulothoracic setting and control of glenohumeral external rotators (infraspinatus, posterior deltoid) and triceps brachii are prerequisites to efficiently take the hand forward and perform a proper reach.[52-55]

3.2.2. Neuroplasticity and timing of motor control training in IwS

Cortical neuroplasticity can be defined as a morphological or functional change in neuronal properties, such as strength of internal connections, altered representational patterns or reorganization of neuronal territories,[56,57] and aims to complete or almost complete recovery in the long run. In IwS, neuroplastic changes underlie the patients' functional recovery. As such, therapy focused on the restorative potential of the brain and neural structures constitutes an important part of neurological rehabilitation post-stroke.[58,59]

Neuroplasticity occurs mainly in the acute and sub-acute stage after stroke[60-62] and patients will benefit most from targeted neuro-rehabilitation in these early stages.[63,64] Although it is assumed that spontaneous recovery slows down after a few weeks and reaches a plateau after several months, an exact limit for recovery potential has never been established.[65] In contrast, it has been shown that adapted neuro-rehabilitation leads to functional gains and reorganization of brain activity even in chronic stroke patients.[66] This suggests that the complete potential of neuroplasticity and the associated recovery is often not yet fully exploited.

In healthy individuals, novel motor skill training, and not repetition of general exercise, is associated with improvement in task performance and increase in representation of the trained muscle in the primary motor cortex.[67] Given the evidence that novel motor skill training is associated with rapid changes in cortical excitability and cortical reorganization, this type of training is also considered relevant for IwS. Motor control exercises for the trunk, scapulothoracic as well as glenohumeral joint can be regarded as such novel motor skill training and should thus, as soon as possible, be added to the rehabilitation protocol. As such, a novel skill stimulus is given early after stroke toward the injured side of the brain, enhancing the likelihood for neuroplastic advantageous changes.

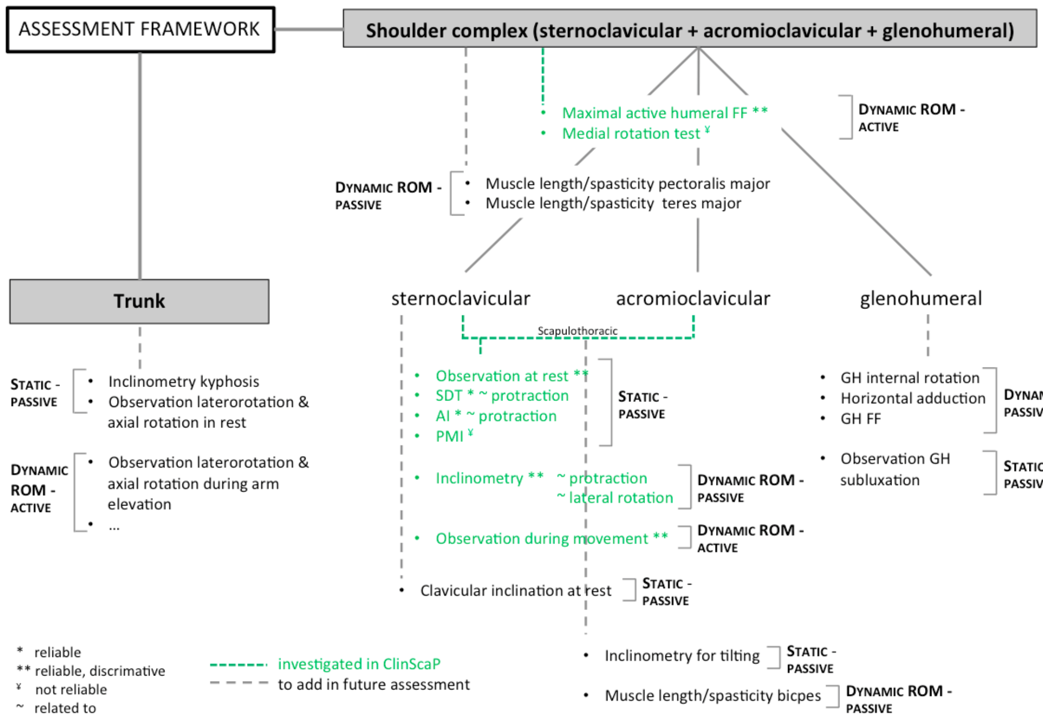
3.2.3. Hypothetical framework for shoulder complex rehabilitation

It is crucial to know whether observed alterations in trunk, scapulothoracic or glenohumeral characteristics can be translated in effective clinical interventions. In the second part of Figure 1, we present a treatment framework for each of the possible affected joints. Such therapy encompasses correct positioning, passive mobilization, stretching exercises, and active exercise therapy to create optimal position, movement and muscle recruitment. Assessing the effectiveness and efficiency of these trunk, scapulothoracic and glenohumeral focused treatments constitutes an important objective in future shoulder research in IwS.

4. Conclusion

This doctoral project made a unique contribution to the understanding of impaired scapulothoracic control in IwS by providing an extensive method to assess the scapula in IwS, including a 3D movement analysis, surface EMG measurements and a clinical test battery (ClinScaP). These assessments allowed us to identify distinct scapulothoracic deficits in relation to different levels of proximal arm function. The measurement of altered muscle timing parameters contributed to our knowledge of the development of shoulder pain post-stroke. Further elaboration of current measurement methods is recommended to gain more in-depth insights in shoulder complex deficits and their relation to trunk deficits, to functional abilities and to the development of shoulder pain. In the long run, these insights will provide a sound base for individualized therapeutic interventions aimed at optimizing upper limb function in IwS.

Figure 1. A hypothetical framework for future research



Cut-off values[a] for the different tests of TR, SC and GH to establish[b]:

1) Functional vs. dysfunctional scores
2) Pain vs. pain free scores

[a] Based on e.g. mean values ± 1 standard deviation

[b] Requires large study samples per subgroup of IwS (low, moderate or high proximal arm function; IwS with and without shoulder pain)

THERAPY FRAMEWORK

CATEGORIZATION BASED ON CUT-OFF VALUES

	I.	**II.**	**III.**	**IV.**	**V.**	**VI.**
Dysfunctional / painful	ST	ST & TR	ST, TR & GH	TR & GH	GH	-
Functional / pain free	TR & GH	GH	-	ST	ST & TR	ST, TR & GH

INDIVIDUALIZED TREATMENT PLANNING

I.	**II.**	**III.**	**IV.**	**V.**	**VI.**
ST focused	ST + TR focused	ST + TR + GH focused	TR + GH focused	GH focused	Prevention & guaranteeing function

SCAPULAR FOCUSED THERAPY:
- Positioning
- Manual therapy
  - ST (SC & AC) joint mobilization
  - Muscle stretching techniques
- Active scapular muscle recruitment re-education
  - Active control ST at rest with(out) assistance (verbal, tactile, …)
  - Control ST during active movement
  - Muscle specific exercises for serratus anterior and lower trapezius

TRUNK FOCUSED THERAPY:
- Positioning
- Manual therapy
  - Thoracic spine mobilization
  - Muscle stretching techniques (latissimus dorsi, …)
- Active muscle recruitment re-education
  - Exercises for thoracic extensors

GLENOHUMERAL FOCUSED THERAPY:
- Positioning
- Manual therapy
  - GH joint mobilization
  - Muscle stretching techniques (teres major, pectoralis major, …)
- Active muscle recruitment re-education
  - Active control of rotator cuff
  - Muscle specific exercises for infraspinatus

ST: scapulothoracic joint, TR: trunk, GH: glenohumeral joint, AC: acromioclavicular joint, SC: sternoclavicular joint

Remarks

Assessment:

- Assessment of the core (i.e. the spine, hips and pelvis, proximal lower limb and abdominal structures) and lower extremity should eventually also be integrated in the framework. Also, the shoulder complex should be further analyzed as a segment of the complete upper limb chain.
- Specific impairments following the stroke event should be assessed like sensory disorders, neglect… .
- Potential pre-existing abnormalities should also be considered (e.g. rotator cuff degeneration related to age).

Therapy:

- Impairments following the stroke-event should be taken into account. For example, a neglect or sensory disorder will have an influence on therapy.
- An optimal interaction between training on body function versus activity level should be attained. The therapist should always ensure that the specific analytical exercises are translated toward the activity level of the IwS. This will increase the patients’ motivation and will generalize the beneficial effects of scapular muscle control training to daily life.

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About the author

About the author

Liesbet De Baets was born on May 11, 1984 in Leuven (Belgium). She graduated in Greek-Mathematics at Heilig Hart College Tervuren (Belgium) in 2002. Afterwards, she started her studies in Rehabilitation Sciences and Physiotherapy at KULeuven (Leuven, Belgium) and obtained a Master degree, specialization musculoskeletal rehabilitation, in 2007. In the following three years, she worked as self-employed physical therapist, specialized in musculoskeletal rehabilitation in Leuven. The last year, she combined her work as a self-employed physiotherapist with a part-time appointment (50%) as scientific researcher at REVAL – Rehabilitation Research Center (Hasselt University). In October 2010, she started a six years PhD trajectory at REVAL (Hasselt University) under the supervision of Dr. Sara Van Deun and Dr. Ellen Jaspers. Within this PhD project, she combined her scientific work (50%) with teaching within the musculoskeletal domain of the Bachelor program of Rehabilitation Sciences and Physiotherapy (50%).

During these past years, a scientific national network with regard to movement analysis expertise centers, as well as clinical partners was established (UZ Pellenberg, Jessa Hasselt, ZOL Lanaken, Rehabilitation and MS center Overpelt).

Apart from her research work, she followed several courses to improve her scientific and transferable skills organized by the Doctoral School of Medicine and Life Sciences, such as project management, scientific writing and scientific communication. She also became a coach in scientific communication for other PhD students at Hasselt University.

Articles in international peer reviewed journals

De Baets L, Jaspers E, Van Deun S. Associations between three-dimensional scapulothoracic kinematics and the outcome of clinical scapular measures in individuals with stroke. Disability and rehabilitation, under review.

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Jaspers, Ellen and **De Baets, Liesbet**. Objective upper limb assessment in adult stroke and unilateral CP: from measurement to interpretation. 23st Annual meeting of the European Society of Movement analysis in Adults and Children (ESMAC), Rome (Italy), 29 September – 4 October 2014.
Invited lecture

De Baets, Liesbet, Jaspers, Ellen, Van Deun, Sara. Scapular muscle recruitment in stroke patients and healthy controls. 28ème Congrès de Médecine Physique et de Réadaptation (SOFMER), Reims (France), 17-19 October 2013.

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Liesbet De Baets. Kinesiologie van het schoudercomplex. Symposium 'Impingement gerelateerde schouderklachten', February 11, 2012, Hasselt (Belgium)

Poster presentations

De Baets, Liesbet, Jaspers, Ellen, Van Deun, Sara. Scapular muscle timing in stroke patients. International NeuroRehabilitation symposium (INRS), Zürich (Switzerland), 11-13 September 2013

De Baets, Liesbet, Jaspers, Ellen, Van Deun, Sara. Repeatability of a three-dimensional scapular movement analysis in persons after stroke. World Conference NeuroRehabiliation 2012, Melbourne, Australia.

De Baets, Liesbet, Jaspers, Ellen, Van Deun, Sara. Repeatability of a three-dimensional bilateral scapular movement analysis in persons after stroke. Slot-event RevalidatieRobotica II, PHL Research Centre, Hasselt (Belgium)

Participations

20th Annual Meeting of ESMAC: European society of movement analysis in Adults and Children, 12-17 September, 2011, Vienna (Austria).

Summary
Samenvatting

A proper and pain free shoulder function is essential for accurate performance of daily activities and contributes to daily life autonomy and quality of life. The brain damage underlying a stroke results in several motor impairments such as muscle weakness, increased muscle tone, pathological muscle synergies and altered temporal muscle activity. At the level of the shoulder complex, these impairments may specifically hamper scapulohumeral control, i.e. the adaptation of scapular position and movement according to the humeral position. Reduced scapulohumeral control is known to contribute to the difficulties individuals with stroke (IwS) experience when moving their paretic arm. Upper limb rehabilitation after stroke could benefit from specific training to enhance scapular positioning and movement control. However, such therapy planning firstly requires an extensive evaluation of the scapulothoracic joint.

Chapter 1 outlines a systematic search on three-dimensional (3D) movement patterns and muscle activity of the scapulothoracic joint in healthy persons, persons with primary shoulder pain and IwS. Little was found on scapulothoracic kinematics post-stroke, and no results were available on scapular muscle activity post-stroke. The results of this systematic review formed the basis for the development of a protocol for 3D movement analysis in IwS, which was presented in **Chapter 2**. Moreover, its feasibility and reliability in IwS and healthy controls was assessed and discussed. Since our systematic search revealed that there was no literature on scapular muscle timing post-stroke, a first exploration of muscular timing characteristics in IwS was performed in **Chapter 3**. We found alterations in muscle activation patterns of main scapulothoracic and glenohumeral stabilizers in IwS, i.e. lower trapezius, serratus anterior and infraspinatus. Additionally, alterations in 3D scapulothoracic movement patterns, i.e. in tilting and lateral rotation, were reported in IwS in comparison to healthy controls (**Chapter 4**).

Given that 3D movement analysis is furthermore difficult to use in clinical stroke practice, current knowledge on scapulohumeral control in individuals with stroke was further enhanced by means of clinical scapular measures. **Chapter 5** introduced a clinical scapular measurement protocol and reported good reliability results for most tests, along with significant differences between controls and IwS, and between IwS with various degrees of arm function for some tests of the protocol. **Chapter 6** additionally described the relation between 3D scapulothoracic kinematics and the outcomes of the different tests of the clinical scapular protocol.

This doctoral project provided more insights into altered scapulohumeral control in individuals with stroke. Thereby, this project paved the road for improved shoulder rehabilitation management post-stroke.

Een correcte en pijnvrije schouderfunctie is essentieel voor de accurate en autonome uitvoering van dagelijkse taken, en draagt bij aan een goede levenskwaliteit. De hersenbeschadiging die optreedt bij personen met een cerebrovasculaire accident (PmCVA) resulteert in verschillende motorische dysfuncties zoals spierzwakte, verhoogde spiertonus, pathologische spiersynergiën, en een veranderde temporele spieractiviteit. Deze dysfuncties kunnen ter hoogte van het schoudercomplex het scapulothoracale ritme verstoren. Dit wil zeggen dat ze de aanpassing van de scapulaire positie aan de humerale positie verstoren en daardoor bijdragen aan een verminderde scapulohumerale controle. Deze verminderde controle kan op zijn beurt bijdragen aan de moeilijkheden die personen na een CVA ondervinden wanneer ze hun paretische schouder willen bewegen. In de revalidatie van het bovenste lidmaat kunnen PmCVA voordeel halen uit specifieke training ter verbetering van de scapulaire bewegingscontrole. Dergelijke therapie vereist dan wel dat er eerst en vooral een goede en uitgebreide evaluatie van het scapulothoracale gewricht plaatsvindt.

In **hoofdstuk 1** werden de resultaten van een systematische review over de driedimensionale (3D) scapulaire bewegingspatronen en spiertiming bij gezonde personen, gezonde personen met schouderpijn en PmCVA besproken. Beperkte wetenschappelijke evidentie bleek beschikbaar over 3D scapulaire bewegingspatronen na een CVA. Over scapulaire spiertiming na CVA werd er zelfs geen literatuur gevonden. Op basis van de literatuur bij gezonde personen werd een bewegingsprotocol ter evaluatie van 3D scapulaire bewegingspatronen bij PmCVA ontwikkeld. Dit protocol, tezamen met zijn betrouwbaarheid bij PmCVA werd in **hoofdstuk 2** voorgesteld. Aangezien er geen literatuur met betrekking tot scapulaire spiertiming beschikbaar was bij PmCVA, werd een exploratieve studie hieromtrent opgezet (**hoofdstuk 3**). We vonden veranderingen in spieractivatiepatronen van de belangrijkste scapulothoracale en glenohumerale stabilisatoren (lower trapezius, serratus anterior en infraspinatus) bij PmCVA. Bovendien vonden we ook veranderingen in 3D scapulaire bewegingspatronen. PmCVA vertoonden een verminderde posterieure scapulothoracale tilt en een vergrote scapulothoracale laterale rotatie in vergelijking met gezonde controle personen (**hoofdstuk 4**). Aangezien dat 3D bewegingsanalyse een meetprocedure is die moeilijk te hanteren is in de klinische praktijk, werd er voorts een klinisch scapulair bewegingsprotocol ontwikkeld. In **hoofdstuk 5** werd dit klinische protocol geïntroduceerd, en werden goede betrouwbaarheidsresultaten tezamen met significante verschillen voor sommige testen van het protocol gerapporteerd bij PmCVA. **Hoofdstuk 6** ging tenslotte de relatie na tussen de uitkomsten van de verschillende testen van het klinische scapulair protocol en de 3D scapulaire bewegingen bij PmCVA.

Met dit doctoraatsproject is er inzicht verworven in de veranderde scapulohumerale controle bij PmCVA. Op deze manier draagt dit project bij tot de ontwikkeling van een betere schouderrevalidatie na een CVA.

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