**Tracking changes in glenohumeral joint position in acute post-stroke hemiparetic patients:
an observational study**

Praline Priscilla Choolun

B.Sc. (Physiotherapy)

Grad Cert Physiotherapy (Geriatrics)

School of Allied Health Sciences
Griffith University

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Statement of originality

This work has not previously been submitted for a degree or diploma in any university. To the best of my knowledge and belief, the thesis contains no material previously published or written by another person except where due reference is made in the thesis itself.

(Signed) _____

Praline Choolun

Published work by the author relevant to the thesis but not forming part of it

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“It is good to have an end to journey toward; but it is the journey that matters, in the end.”

— Ernest Hemingway

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Abstract

Up to 70% of people develop disabling hemiparetic shoulder pain (HSP) post-stroke, leading to the evasion of functional use of the hemiparetic arm, poor rehabilitation outcomes and reduced quality of life. Despite HSP being a significant issue for people with stroke, there has been limited success in preventing or managing this condition, perhaps because the aetiology of this common condition is unclear.

Although the causes of HSP remain inconclusive, positional change of the humeral head relative to the glenoid fossa (glenohumeral joint centre), particularly in the acute stage, may be a factor. Alterations in the location of the glenohumeral joint centre (GHJC) have not previously been investigated in people during the acute post-stroke phase. The primary focus of this research was to longitudinally investigate GHJC during the post-stroke phase in people who had experienced a hemiparetic stroke. The reliability of an electromagnetic tracking device to measure GHJC position in people following stroke and in a healthy population was determined. GHJC position was examined in people within two weeks of stroke onset and in a group of age, gender and body mass index matched healthy controls. Changes in the GHJC position over time (i.e. baseline to six weeks post-stroke) and the impact of demographic and clinical characteristics such as shoulder pain, hemiparetic side, and muscle tone, on GHJC position were also determined.

The GHJC position was defined relative to a scapular coordinate system with its origin at the acromion process using a functional sphere-fitting approach. Briefly, participants underwent a functional calibration trial while seated in a standardised posture in which the passive upper arm was moved by the investigator through a specified movement sequence. A 6-dimensional electromagnetic tracking device was used to track the linear and angular displacements of sensors attached to the distal humerus and the acromion process of the scapular. The humeral sensor was then transformed to the scapular coordinate system and an estimate of the GHJC location relative to the scapular coordinate system was derived using an iterative unconstrained optimisation approach in which the GHJC was estimated by minimising the cumulative sum-of-squares difference in the distance between the humeral sensor and the estimate of the GHJC over the entire length of the calibration trial. The anteroposterior and superoinferior positions of the GHJC relative to the scapular coordinate system are variables of interest for this aspect of the study.

Intra-tester reliability of anteroposterior and superoinferior position GHJC position was very good (ICCs > 0.8) in both people with an acute stroke and a healthy population. In addition, the maximum minimal detectable change for stroke participants was 26.0 mm. Thirty patients within two weeks of stroke onset (age 65 SD19 years, 60% female, 40% right side affected) were recruited to this study. At baseline, the stroke participants' GHJC was positioned posteriorly to the acromion on the hemiparetic side, and anteriorly on the non-hemiparetic side (mean difference -4.0, 95% CI -7.7 to -3.0 mm). GHJC position for the healthy control participants was positioned anterior to the acromion on both sides with no significant difference between limbs (mean difference -0.3 mm, 95% CI -10.0 to 9.5). There were no significant differences in superoinferior GHJC position within or between the stroke and control groups for the hemiparetic ($p = 0.317$) or non-hemiparetic ($p = 0.141$) limbs, with all participants exhibiting a GHJC inferior to the origin of the scapular coordinate system (i.e., the acromion process).

At six weeks follow-up the GHJC on the stroke participants' hemiparetic side had displaced anteriorly (3.1 SD 7.6 mm) ($p = 0.156$) and superiorly (-12.2 SD 16.4 mm) ($p = 0.169$) with respect to the acromion and on the non-hemiparetic side posteriorly (1.3 SD 9.1 mm) ($p = 0.308$) and superiorly (-13.3 SD 9.5 mm) ($p = 0.256$). There was no difference in the position of the GHJC in the anteroposterior direction, between the healthy controls and stroke participants' hemiparetic ($p = 0.670$) and non-hemiparetic sides ($p = 0.819$). In the superoinferior direction, the GHJC was positioned closer to the acromion in the stroke participants (mean difference 9.3, 95% CI -1.1 to 19.7 mm on hemiparetic side, 12.3, 95% CI 2.5 to 22.1 mm on the non-hemiparetic side) compared to the healthy controls.

Demographic and clinical characteristics appeared to have little association with GHJC position except for the side affected by the stroke (right or left). At baseline, in those patients with right hemiparesis, the GHJC was positioned posteriorly (-5.8 SD 8.1 mm) and more inferiorly (-24.4 SD 12.9 mm) to the acromion. In contrast, GHJC in those with left hemiparesis was positioned anteriorly (2.9 SD 3.9 mm) and less inferiorly (-11.8 SD 11.4 mm) to the acromion. At week six, GHJC in all stroke participants, regardless of side affected by stroke, had moved anteriorly in the anteroposterior direction. In the superoinferior direction, GHJC in those with right hemiparesis was positioned further from the acromion (-22.9 SD 15.4 mm) compared to those with left hemiparesis (-5.4 SD 13.4 mm).

In summary, changes in glenohumeral joint positioning occur very early post-stroke. Having the GHJC positioned closer to the acromion in participants with stroke

compared to healthy control participants suggests that glenohumeral joint impingement may occur early post-stroke. Clinicians need to be aware of this positional change and its potential role in humeral anterior subluxation and impingement, and consequently the development of hemiparetic shoulder pain. Glenohumeral joint changes on the non-hemiparetic side should also be considered during rehabilitation.

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List of abbreviations

ABS	Australian Bureau of Statistics
AGT	Acromion to greater tuberosity
AIHW	Australian Institute of Health and Welfare
AP	Anteroposterior
CRPS	Chronic regional pain syndrome
EMG	Electromyography
ER	External rotation
ETD	Electromagnetic tracking device
FIM	Functional independence measure
GHJC	Glenohumeral joint centre
HA	Helical axis
HSP	Hemiparetic shoulder pain
ICC	Intraclass correlation coefficient
IR	Internal rotation
MDC	Minimal detectable change
NEMESIS	North East Melbourne Stroke Incidence Study
NSF	National Stroke Foundation
PROM	Passive range of movement
SAIS	Subacromial impingement
SCoRE	Symmetrical centre of rotation estimation
SD	Standard deviation
SEM	Standard error of the measure
SI	Superoinferior

Chapter 1

Introduction

Stroke is a primary cause of disability in Australia (Australian Institute of Health and Welfare, 2012; Australian Institute of Health and Welfare: Senes, 2006). New and recurrent episodes of strokes suffered by Australians each year are estimated to be approximately 60,000 (Australian Bureau of Statistics, 2007; Dewey et al., 2003; Thrift, Dewey, Macdonell, McNeil, & Donnan, 2001); and this is predicted to increase each year due to our ageing population (Dewey et al., 2003; Thrift et al., 2001; Thrift, Dewey, Macdonell, McNeill, & Donnan, 2000; Thrift et al., 2006). There are significant physical, financial, and emotional impacts of stroke on the individual, their family and the community (Feigin, Barker-Collo, McNaughton, Brown, & Kerse, 2008). In Australia, the overall annual direct and indirect financial costs associated with the treatment of stroke, productivity loss and lifetime costs related to stroke, are estimated at \$2.14 billion per annum (Cadilhac, Carter, Thrift, & Dewey, 2009).

The impact of disability as a result of stroke is significant. Approximately 420,000 Australians live with the effects of stroke at a total cost of around \$5 billion (National Stroke Foundation, 2013). Sixty-five percent of this group live with the continuing impact that limits their ability to perform everyday activities independently, contributing to the presence of depressive mood symptoms and reduced quality of life (Graven, Brock, Hill, & Joubert, 2011; White et al., 2012). The annual carer costs associated with providing care for those rendered dependent by stroke is estimated at \$222 million. In summary, the majority (88%) of people who have experienced a stroke live at home and are disabled (Australian Institute of Health and Welfare, 2006).

Stroke most frequently manifests as hemiparesis of one side of the body, which is associated with weakness or loss of active movement, changes in muscle tone, and spasticity (Turner-Stokes & Jackson, 2002). Upper limb recovery following stroke is generally poor (Blennerhassett, Gyngell, & Crean, 2010; Lum et al., 2009; Williams, Galea, & Winter, 2001) and may be complicated by the development of hemiparetic shoulder pain (Kalichman & Ratmanský, 2011; Pong et al., 2012; Rajaratnam et al., 2008). Hemiparetic shoulder pain (HSP) is a common problem after stroke with up to 70% of patients reporting moderate to severe shoulder pain often within the first few days (Lindgren, et al., 2007; Ward, 2007). HSP is disabling, linked to poor rehabilitation outcomes (Turner-Stokes & Jackson, 2002) and leads to patients evading functional use of the hemiparetic arm (Vasudevan & Vasudevan, 2008). HSP limits patients' rehabilitation participation (Faria-Fortini, Michaelsen, Cassiano, & Teixeira-Salmela, 2011) and results in increased length of hospital stay (Barlak, Unsal, Kaya, Sahin-Onat, & Ozel, 2009; Wanklyn, Forster, & Young, 1996). HSP is also a predictor of reduced

quality of life (Chae & Jedlicka, 2009) and an increased incidence of depression post stroke (Lindgren, Jonsson, Norrving, & Lindgren, 2007).

Despite HSP being a significant issue for people with stroke, there has been limited success in preventing or managing this condition. Despite there being four systematic reviews on the effectiveness of interventions for HSP (Kalichman & Ratmansky, 2011; Koog, Jin, Yoon, & Min, 2010; Page & Lockwood, 2003; Rajaratnam et al., 2008), the National Stroke Foundation Clinical Guidelines for Stroke Management (National Stroke Foundation, 2010) could not recommend effective treatment methods reflecting our lack of understanding of the underlying aetiology of this condition.

The lack of clarity regarding the causes of HSP signals the multifactorial nature of the phenomenon borne out by recent systematic reviews (Kalichman & Ratmansky, 2011; Rajaratnam et al., 2008; Roosink, Renzenbrink, Geurts, & Ijzerman, 2012b). All reviews propose the possibility of glenohumeral subluxation being a potential contributor to HSP. Subluxation has primarily been investigated in the superior-inferior direction (Huang et al., 2012; Stolzenberg, Siu, & Cruz, 2012); however subluxation in the anterior-posterior direction (Huang et al., 2012) is possible and also requires investigation.

The occurrence and magnitude of glenohumeral subluxation has been measured using a range of clinical, imaging and three-dimensional motion analysis techniques. The main clinical measure used to estimate glenohumeral subluxation is palpation of the space between the inferior aspect of the acromion and the head of the humerus (Bohannon & Andrews, 1990; Paci, Nannetti, Taiti, Baccini, & Rinaldi, 2007). However, palpation is considered to be a screening tool only and is not sensitive for measuring small changes in subluxation (Kumar & Swinkels, 2009). Medical imaging techniques such as radiography and ultrasound have quantified the degree of subluxation (Barlak et al., 2009; Huang et al., 2012). Three-dimensional motion analysis techniques, such as electromagnetic tracking devices, have been used to measure changes in the centre of the glenohumeral joint. However, these have only been used in the healthy (Campbell, Lloyd, Alderson, & Elliott, 2009; Monnet, Desailly, Begon, Vallee, & Lacouture, 2007) or non-stroke (Rettig et al., 2013) population. Electromagnetic tracking devices have been used in the stroke population to measure scapular position during humeral movement in the subacute phase (Beebe & Lang, 2009; Meskers, Koppe, Konijnenbelt, Veeger, & Janssen, 2005; Niessen et al., 2008; Price, Rodgers, Franklin, Curless, & Johnson, 2001) or chronic phase (Hardwick & Lang, 2011a; Massie, Malcolm, Greene, & Browning, 2012; Rundquist, Dumit, Hartley, & Kendall,

2012) but have not yet been used to measure changes within the glenohumeral joint centre during the acute post-stroke phase. Furthermore, changes in the position of the glenohumeral joint centre may be influenced by demographic and clinical characteristics such as age, gender, whether the left or right side has been affected by the stroke, hand dominance, motor recovery and muscle tone, but have not been explored in an acute stroke population.

Therefore, the primary aims of this program of research were to investigate positional changes of the glenohumeral joint in the early post-stroke phase, compare these changes to a healthy control group and assess the impact of demographic and clinical characteristics on glenohumeral joint position.

This thesis encompasses five chapters. Chapter two provides a literature review of stroke, the changes at the shoulder that occur post-stroke, hemiparetic shoulder pain, its implications, possible causes and consequences, the role of the scapula and measurement of glenohumeral joint centre position. The third chapter describes the general methods used across the research program. Chapter four presents the results of this research program. This chapter describes the reliability of an electromagnetic tracking device measure to determine the glenohumeral joint centre position in a stroke and healthy population, outlines the results from the investigation of the glenohumeral joint centre position in both the hemiparetic and non-hemiparetic sides within two weeks of stroke onset and in a healthy population matched for age, gender and body mass index and the changes that occur over the first six weeks. The influence of demographic and clinical characteristics on the glenohumeral joint centre position in the first six weeks following stroke is also reported. Chapter five summarises the study's findings and discusses the generalisability, clinical implications and limitations of this study. Directions for future research and a summary of the project concludes the thesis.

Chapter 2

Literature review

2.1 Introduction

Stroke is a manifestation of cerebrovascular disease, where the arterial supply to a particular part of the brain suddenly becomes blocked or starts to bleed (Australian Institute of Health and Welfare, 2011; National Stroke Foundation, 2014b). This disruption to the blood supply may result in death, or brain damage, presenting as sudden impairment in one or more functional areas including vision, speaking, swallowing, cognition, movement, sensation and balance (National Stroke Foundation, 2014a). The Australian Institute of Health and Welfare estimates that 375,800 people in Australia had had a stroke at some time during their lives and that 70% were aged 65 and over (Australian Institute of Health and Welfare, 2013). Over one third of these people with stroke were disabled as a result of their stroke; they were more likely to always require assistance with core activities of daily living than other people with disabilities (Australian Institute of Health and Welfare, 2013).

The incidence of stroke in Australia in 2010 was an estimated 62,000 (Australian Institute of Health and Welfare, 2011), based on data from the NEMESIS study (Thrift et al., 2001), with 420,000 people living with the effects of stroke (National Stroke Foundation, 2013). The risk of stroke increases with age as the number of risk factors increases. Main risk factors include hypertension, smoking, diabetes, high blood cholesterol, high alcohol intake, atrial fibrillation, carotid stenosis and other heart disease (Australian Institute of Health and Welfare, 2011). Improvement in the management of these risk factors has decreased the stroke mortality rate (Australian Institute of Health and Welfare, 2011), however the increased survival levels and an ageing population has led to escalating levels of activity limitation (Moser & Ward, 2000; Sterr & Conforto, 2012).

2.2 Stroke associated activity limitation

Stroke is a significant contributor to activity limitation among adults representing approximately 7% of all people with an activity limitation (Australian Institute of Health and Welfare: Senes, 2006). The cost of stroke to Australia in 2008-09 was \$606 billion (Australian Institute of Health and Welfare, 2013), but there are also significant financial, emotional and social impacts at the individual and community level (Australian Institute of Health and Welfare: Senes, 2006). Of the 420,000 people living with the effects of stroke (National Stroke Foundation, 2013), an estimated 285,300 Australians report activity limitation, leading to difficulties with and/or requiring

assistance with mobility, self-care or communication (Australian Institute of Health and Welfare, 2013).

One of the main reasons for the ongoing high level of activity limitation associated with stroke is the frequent presence of multiple comorbidities commonly experienced by this group. The most dominant comorbidities in people with stroke which also require medical care include hypertension, diabetes, lipid disorders, depression, atrial fibrillation and ischaemic heart disease (Australian Institute of Health and Welfare: Senes, 2006). For example, people with diabetes are 2.2 times more likely to suffer a stroke than those without diabetes, a great concern with this disease on the rise (Australian Bureau of Statistics, 2011). In addition to these comorbidities, a primary feature of stroke is the sensorimotor deficits that result directly from the central neurological injury that occurs following a stroke. Sensorimotor deficits that result directly from the central neurological injury and are a primary feature of stroke, also impact activity limitation.

2.3 Sensorimotor deficits and recovery

Hemiplegia or paresis and hemianaesthesia on the side contralateral (opposite) to the side of the brain lesion are common sensorimotor deficits associated with stroke. Motor deficits are accepted to be the most common post-stroke impairment (Bohannon, 2007) occurring on the side contralateral to the stroke as well as on the side ipsilateral (same side as) to the stroke, resulting in generalised weakness (Bohannon & Andrews, 1995). This weakness results in lack of movement and/or poor control of body segments, which reduces the stroke survivor's ability to participate in functional activities such as standing up, getting dressed and walking; tasks that are essential to daily living (Bohannon, 2007). Associated with this weakness or loss of active movement are additional sensorimotor effects that include changes in muscle tone, the development of shoulder pain, subluxation of the glenohumeral joint, and spasticity (Turner-Stokes & Jackson, 2002).

Somatosensory changes occur in up to 85% of people with stroke (Connell, Lincoln, & Radford, 2008a; Kim & Choi-Kwon, 1996; Roosink, Van Dongen, et al., 2012) and contribute to functional impairment (Coupar, Pollock, Rowe, Weir, & Langhorne, 2012; Nudo, Friel, & Delia, 2000; Rose, Bakal, Fung, Farn, & Weaver, 1994). Aspects of the somatosensory system affected by stroke include light touch, pressure, temperature, pain, and proprioception (Connell et al., 2008a; Roosink, Van Dongen, et al., 2012). Common presentations of impaired somatosensory function include loss of

proprioception (sense of the position of body parts in relation to each other and the degree of effort undertaken to modulate movement) (Connell et al., 2008a), and loss of vibration sense (Kim & Choi-Kwon, 1996). Somatosensory deficits in the post-stroke upper limb are often chronic and have a direct correlation with motor impairment (Scalha, Miyasaki, Lima, & Borges, 2011). Functional impairment due to somatosensory changes are impacted by stroke related perceptual disorders (Jutai et al., 2003), such as astereognosis (inability to identify items by handling without vision) (Connell et al., 2008b), limb apraxia (inability to perform purposeful movement), agnosia (inability to recognise objects using intact visual acuity), inability to gauge depths and distances (Jutai et al., 2003), hemispatial neglect (inattention or awareness) (Roosink, Renzenbrink, et al., 2011), altered thermoception (perception of the rate of temperature change) (Kim & Choi-Kwon, 1996) and prolonged nociception (pain response to noxious stimuli) (Roosink, Renzenbrink, et al., 2012b). Combined perceptual and somatosensory impairments are associated with reduced recovery of mobility and activities of daily living (Jutai et al., 2003).

Changes in muscle tone, which is common following stroke, also impact on functional activities (Marciniak, 2011; Wissel et al., 2010). Decreased muscle tone, or flaccidity is usually present in the initial period post-stroke and may last from 24 hours to 18 months at which time the tone may increase and spasticity develop (Formisano et al., 2005; Turner-Stokes & Jackson, 2002). Conflicting evidence exists as regards the association of flaccidity and spasticity with HSP, as evidenced by recent systematic reviews (Kalichman & Ratmansky, 2011; Murie-Fernández et al., 2012). Flaccidity appears to be associated with more severe motor deficits (Formisano et al., 2005; Kwakkel, Kollen, van der Grond, & Prevo, 2003). Flaccidity is also associated with the presence of subluxation (Kalichman & Ratmansky, 2011; Murie-Fernández et al., 2012) potentially due to the biomechanical changes in the shoulder, shoulder joint malalignment and loss of shoulder mobility (Allen, Shanahan, & Crotty, 2010; Jackson et al., 2002). However, although shoulder subluxation has been associated with pain, its role as a cause of pain remains unclear (Murie-Fernández et al., 2012).

Spasticity, a velocity dependent increase in the tonic stretch reflex (Lance, 1980), can have a variable onset from days to months post stroke. Spasticity is associated with decreased function due to abnormal patterns of movement, co-contraction of muscles, associated movements and persistent limb posturing, and the development of contractures (Marciniak, 2011). Spasticity has also been associated with HSP (Kumar, Saunders, Ellis, & Whitlam, 2013; Lindgren et al., 2007; Rajaratnam et al., 2008) and glenohumeral joint subluxation (Ada & Foongchomcheay, 2002; Kumar, Kassam,

Denton, Taylor, & Chatterley, 2010; Stolzenberg et al., 2012). In summary, evidence points to both flaccidity and spasticity being associated with HSP and subluxation. It is feasible that both play a role during post-stroke temporal stages and may vary depending on the level of motor control (Huang, Liang, Pong, Leong, & Tseng, 2010) and stroke severity (Formisano et al., 2005; Ward, 2012).

Generally, the rate of hemiparetic recovery has been shown to be fastest in the first three months post-stroke (Jørgensen et al., 1995; Kwakkel, Kollen, & Twisk, 2006), with progress slowing over the next three months (Kwakkel et al., 2003) and plateauing from six to 12 months post-stroke (Formisano et al., 2005; Jørgensen et al., 1995). Motor and functional recovery in the chronic stages (beyond 12 months) post-stroke are doubtful (Page, Gater, & Bach-y-Rita, 2004), though a recent review revealed evidence of improved outcomes in chronic stroke (Teasell, Mehta, et al., 2012).

Deficits in upper limb recovery are experienced by up to 75% of people with stroke at three to six months post-stroke (Lai, Studenski, Duncan, & Perera, 2002) and even those considered to have recovered well have residual functional deficits (Lai et al., 2002). Although motor recovery on the hemiparetic side is similar in both the upper and lower limbs (Higgins, Mayo, Desrosiers, Salbach, & Ahmed, 2005; Kwakkel et al., 2006; Verheyden et al., 2008), the upper limb suffers greater activity limitation as a result of a higher level of motor and sensory control required to complete fine motor tasks (Duncan et al., 1994; Kwakkel et al., 2006). Upper limb function is frequently minimal in the first few weeks post-stroke, even in the presence of some motor recovery (Akinwuntan et al., 2005). The severity of functional and motor impairments in the initial stage following stroke predict recovery in the upper limb (Coupar et al., 2012).

Upper limb recovery is dependent in part on recovery proximally at the shoulder complex (Teasell & Heitzner, 1998). In the context of upper limb deficits being common following stroke (Lai et al., 2002), the glenohumeral joint appears to be particularly vulnerable. Loss of muscle strength (Canning, Ada, Adams, & O'Dwyer, 2004; Kumar et al., 2010), somatosensory (Connell et al., 2008a; Scalha et al., 2011) and perceptual issues (Jutai et al., 2003; Staines, Black, Graham, & McIlroy, 2002) result in maladaptive changes due to muscles resting in shortened or lengthened positions (Ada, Canning, & Low, 2003). In the shoulder girdle, these changes in muscle control and tone may affect humeral head and scapula positioning and lead to loss of active range and may subsequently result in contractures (Ada et al., 2003). Shoulder pain, appears to be linked to this loss of active movement. Both loss of range and presence

of HSP have been shown to be related to poor functional recovery and dependent arm function (Beebe & Lang, 2009; Rajaratnam et al., 2008).

2.4 Hemiparetic shoulder pain

Shoulder pain is a common post-stroke complication, which typically occurs within the first few days post-stroke (Dromerick, Edwards, & Kumar, 2008; Lindgren et al., 2007). Studies reporting HSP prevalence in stroke survivors are summarised in Table 1. Prevalence ranged from 5% (Davenport, Dennis, Wellwood, & Warlow, 1996) to 72% (Bohannon, Larkin, Smith, & Horton, 1986), however studies varied in the timing of the HSP assessment, measurement methods used, and the professional affiliation of the researchers conducting the shoulder pain assessment. Potential risk of bias in these identified studies was determined using a previously validated checklist for assessing methodological quality (Downs & Black, 1998). This checklist has been nominated as one of six assessment tools deemed suitable for systematic reviews (Deeks et al., 2003). The checklist consists of 27 items which rate randomised and non-randomised studies on reporting, external validity, bias, confounding and power. Each component of the 27-item checklist is conferred a score of 1 (yes) or 0 (no or unable to determine), except for question 5, that deals with principal confounders. This question is scored with a maximum of 2 points (0 = no, 1 = partially reported, 2 = yes). Scoring of question 27, dealing with statistical power to detect a clinical effect, was simplified to a choice of 1 (when evidence of sufficient statistical power was portrayed), or 0 (when no evidence of sufficient statistical was present) (Benjamin, van de Water, & Peiris, 2014; Tan et al., 2014). The maximum score possible is 28 with quality levels allocated to a range of scores: excellent (26-28), good (20-25), fair (15-19) and poor (≤ 14) (Benjamin et al., 2014; Downs & Black, 1998; Hooper, Jutai, Strong, & Russell-Minda, 2008; Tan et al., 2014). Only one study scored in the excellent quality range (Griffin & Bernhardt, 2006), with three scoring good quality (Adey-Wakeling et al., 2014; Huang et al., 2010; Lindgren, Lexell, Jonsson, & Brogardh, 2012), while the remaining studies scored in the fair quality range.

Table 2 summarises the quality ratings for each of the subscales (reporting, external validity, bias, confounding and power) from the Downs and Black checklist (Downs & Black, 1998). Overall, studies scored well with regard to reporting, external validity and power, but scored poorly on bias and confounding. Of the four high quality studies (scoring excellent or good), two studies estimated HSP prevalence to be between 42% and 56% in the first three weeks post-stroke (Griffin & Bernhardt, 2006; Huang et al., 2010).

Table 1. Summary of research studies regarding prevalence of hemiparetic shoulder pain

Study Lead author, year	Stroke participants' characteristics	Time post-stroke	Methods of measuring HSP and other measures	Prevalence and intensity of HSP and other outcomes
Prospective studies (in chronological order)				
Parker, 1986	N = 187 Age, mean (SD): 71.8 yrs (10.8) Men: 50%	3 months No mean and SD disclosed.	Assessor not disclosed. Participants identified in population register at 2 weeks post-stroke, but HSP not assessed at this time. 3 and 6 month assessments. Neurological information included: <ul style="list-style-type: none"> PROM shoulder Motor control for shoulder via Motricity index (0-100) Sensation test using standardised equipment 	HSP prevalence: <ul style="list-style-type: none"> At 3 months: No HSP, n = 140 (74%) HSP at rest, n = 5 (3%); >90 ° shoulder abduction, n = 39 (21%); <90 ° shoulder abduction, n = 3(1%) At 6 months: 80% had no HSP. PROM shoulder: <ul style="list-style-type: none"> At 3 months, Full PROM, n = 173 (92%) Motor control: <ul style="list-style-type: none"> At 3 months: Severe paralysis, n = 31 (17%); Moderate paralysis, n = 13 (7%); Mild paralysis, n = 94 (50%); No weakness, n = 49 (26%) At 6 months: Similar to percentages at 3 months Sensation: Abnormal sensation, n = 36 (19%)
Wanklyn, 1996	N = 108 Age, mean (range): 71 yrs (60-89) Men: 52.8%	Not disclosed, but first assessed at discharge from hospital.	Interviews and assessments via standard protocol conducted by one of the investigators on discharge, at 8 weeks and 6 months post-discharge. Interview: Presence and severity of shoulder pain. Assessment: Motor power <ul style="list-style-type: none"> Upper limb proximal (shoulder shrug) and distal (pinch grip) PROM, pain-free shoulder internal and external rotation via goniometer. Glenohumeral subluxation measured via finger-width palpation. 	HSP prevalence: At hospital discharge 38.6%; At 8 weeks 59.0%; At 6 months 36% Severity and pattern of HSP: <ul style="list-style-type: none"> At discharge: <ul style="list-style-type: none"> Severe HSP, n = 4; Moderate HSP, n = 18; Mild HSP, n = 17 HSP Constant, n = 9; HSP at night, n = 5; HSP on movement, n = 14 At 8 weeks post-discharge: <ul style="list-style-type: none"> Severe HSP, n = 12 HSP Constant, n = 6; HSP on movement, n = 25 At 6 months post-discharge: Severe HSP, n = 7 Normal shoulder control: <ul style="list-style-type: none"> At discharge = 42.6%; At 8 weeks = 54%;

Study Lead author, year	Stroke participants' characteristics	Time post-stroke	Methods of measuring HSP and other measures	Prevalence and intensity of HSP and other outcomes
Gamble, 2002	N = 123 Age, mean (range): 70.6 (29-93) yrs Men : 48%	Interview and examination at 14 days 2, 4 and 6 months. Means and SD not recorded.	All participants underwent interviews and examination, though by whom is not specified. Interview via standard questionnaire: Shoulder pain history, pain characteristics and site. Examination: <ul style="list-style-type: none"> VAS (0-100mm) for HSP intensity. Shoulder PROM. Standardised Rheumatology definition for classification of significant shoulder pain classified Neurology examination. NIHSS for weakness. Shoulder X-ray for persistent HSP. 	<ul style="list-style-type: none"> At 6 months = 59% <p>PROM reduced at discharge in 67%; at 8 weeks in 76%, at 6 months in 81%. Decreased PROM associated with HSP</p> <p>Glenohumeral subluxation prevalence at discharge 29%, at 8 weeks 23%, at 6 months 26%. No statistically significant relationship between subluxation and HSP except at 6 months.</p> <p>HSP prevalence: n = 52 (40%) developed HSP during 6 months.</p> <p>Progress of HSP:</p> <ul style="list-style-type: none"> By 2 weeks, n = 28 (55%) had developed HSP By 2 months, n = 44 (87%) had developed HSP <p>In last 4 months of study, n = 8 (16%) developed HSP</p> <p>VAS: HSP had good prognosis. n = 41 (80%) of participants with HSP improved or resolved HSP during 6 months.</p> <p>Shoulder Pain Classification: Regional causes, n = 33 (65%); Central pain, n = 9 (20%), Mixed, n = 8 (16%); Chronic widespread, n = 2 (4%).</p> <p>Cold temperature sensibility, Light touch, arm weakness and abnormal joint examinations were statistically significantly different between HSP and non-HSP groups.</p> <p>Shoulder X-rays (n = 39)</p> <ul style="list-style-type: none"> n = 26 (67%) no/minimal joint degeneration n = 5 (13%) minimal/moderate degeneration in keeping with age. n = 6 (15%) significant changes. <p>HSP prevalence:</p> <ul style="list-style-type: none"> 1 week, n = 256 (17%) 1 month, n = 261 (20%) 6 months, n = 284 (23%) <p>HSP associated with presence and severity of sensorimotor deficits.</p> <p>At 1 week, those at home were more likely to report</p>
Ratnasabapathy, 2003	N = 1,761 Age, mean (no SD) – data from earlier publication: Women 73.4 yrs Men 67.3 yrs Percentage of	Not disclosed, but all questionnaire administered within 2 weeks post-stroke.	Self-report via questionnaire (not standardised) administered by nurse at 1 week, 1 month and 6 months. Information collected included: <ul style="list-style-type: none"> Presence of HSP (relationship to stroke affected side, timing of onset, history of shoulder pain) 	

Study Lead author, year	Stroke participants' characteristics	Time post-stroke	Methods of measuring HSP and other measures	Prevalence and intensity of HSP and other outcomes
	men not disclosed.		<ul style="list-style-type: none"> Demographic information Sensory loss (lasting >24 hrs) was described and rated (nil, mild, moderate, severe and not assessable), Motor loss severity rated from not affected to severe. At 1 month, self-reported opinion for cause of HSP recorded. 	<p>HSP than those in care.</p> <p>14% with HSP had pre-stroke shoulder pain.</p> <p>11% reported HSP for the first time at 6 months.</p> <p>Perceived causes of HSP: Stroke 21%; Physiotherapy and exercise 15%; Methods of handling 12%; Non-stroke causes 28%</p> <p>Risk of HSP increased with severity of upper limb motor deficit and with increasing time post-stroke.</p> <p>Risk of developing HSP was greater for left hemiparetic shoulders.</p>
Aras, 2004	<p>N = 85</p> <p>Age, mean (SD): 59.5 (11.6) yrs</p> <p>Men: 69.4%</p>	<p>Two groups:</p> <p>With HSP:</p> <p>n = 54, mean (SD): 70.2 (45.6) days post-stroke</p> <p>Without HSP:</p> <p>n = 31, mean (SD): 59.5 (49.9) days post stroke</p>	<p>Assessor not disclosed</p> <p>Presence of HSP assessed during PROM.</p> <p>Comparisons made between those with and without HSP regarding:</p> <ul style="list-style-type: none"> Shoulder PROM Subluxation via radiographic assessment Shoulder soft tissues evaluation via ultrasound Motor recovery assessed via Brunnstrom recovery stages (1-6). Spasticity via Ashworth scale (0-4) 	<p>HSP prevalence:</p> <p>n = 54 (63.5%) (Women, n = 16, men, n = 38)</p> <p>Shoulder PROM: HSP group had statistically significantly more severe ROM restriction of flexion and external rotation.</p> <p>Subluxation: HSP group vs no HSP group: n = 27 (50%) vs n = 5 (16.1%)</p> <p>Soft tissues: No statistically significant difference between groups.</p> <p>Motor recovery: Statistically significant difference between HSP and no HSP groups.</p> <p>Spasticity: No significant difference between groups.</p>
McLean 2004	<p>N = 133</p> <p>Age, mean (SD): 68.6 (13.1) yrs.</p> <p>Men: 52%</p>	<p>Not specified, but participants recruited from in-patient, tertiary-level stroke rehabilitation unit.</p>	<p>Participants assessed on admission; outcomes determined by one of two physicians with input from the multi-disciplinary team, at weekly case conference meetings during the week of participants' discharge.</p> <p>HSP determined via self-report, mainly during passive movement.</p>	<p>HSP prevalence:</p> <p>n = 32 (24%)</p>
Griffin, 2006	<p>N = 32</p> <p>Three groups:</p> <p>Therapeutic strapping</p> <p>(n = 10).</p> <p>Age, mean (SD),</p>	<p>Therapeutic strapping group, mean (SD): 9 (5) days</p> <p>Placebo strapping group, mean (SD): 10</p>	<p>Outcome measures completed by physiotherapy assistants blinded to group allocation.</p> <p>Daily Ritchie Articular Index to assess HSP.</p> <p>Measurements at 4 weeks:</p> <ul style="list-style-type: none"> PROM shoulder with direction specified 	<p>HSP prevalence:</p> <ul style="list-style-type: none"> Therapeutic strapping group, n (%): 1 (10) Placebo strapping group n (%): 5 (50) No strapping group n (%): 5 (42) <p>PROM shoulder: No statistically significant differences between groups</p>

Study Lead author, year	Stroke participants' characteristics	Time post-stroke	Methods of measuring HSP and other measures	Prevalence and intensity of HSP and other outcomes
	<p>65.0 (10.7) yrs Men: 30%</p> <p>Placebo strapping (n = 10). Age, mean (SD), 62.1 (11.0) yrs Men: 30%</p> <p>No strapping (n = 12) Age, mean (SD), 58.8 (9.9) yrs Men: 33%</p>	<p>(4) days No strapping group, mean (SD): 12(6) days</p>	<ul style="list-style-type: none"> • Muscle tone via Modified Ashworth Scale • Upper arm item of Motor Assessment Scale 	<p>Muscle tone: No statistically significant differences between groups</p> <p>Motor Assessment Scale: No statistically significant differences between groups. The median Motor Assessment Scale score remained low for all groups.</p>
Lindgren, 2007	<p>At 4 months post stroke: N = 327, age, mean (range): 73.1 (17 – 102) yrs Men: 60%; NIHSS score, mean 5.4</p> <p>16 month follow up: N = 305, age, mean (range): 72.5 (17 – 96) yrs Men: 60% NIHSS score, mean: 5.1</p>	<p>Initial assessment: 4 months Follow-up assessment: 16 months</p>	<p>Measured by nurse and physiotherapist in collaboration.</p> <p>Shoulder pain questionnaire – not standardised. Questions included time of onset, frequency, use of analgesia, relationship to movement, rest, ADL and mobility.</p> <p>VAS (0-100mm) for self-perceived HSP intensity during previous 48 hours.</p>	<p>HSP prevalence:</p> <ul style="list-style-type: none"> • At 4 months: n = 71 (22%) • Follow-up at 16 months: n = 74 (24%) • Total number of participants with HSP during study: n = 99 (30%). <p>VAS > 40 mm:</p> <ul style="list-style-type: none"> • At 4 months: n = 48 (79%) • At 16 months: n = 41 (59%)
Dromerick, 2008	<p>N = 40 Age, mean (SD): 57.3 (25.2) yrs Men: 52%</p>	<p>Mean (SD): 18.9 (14.1) days</p>	<p>Assessment by physician via:</p> <p>Interview/self-report re presence and nature of HSP</p> <p>HSP at rest and end of passive range</p> <p>Palpation of soft tissues</p> <p>Pain intensity scale and pain diagram (McGill pain questionnaire).</p> <p>Diagnostic manoeuvres including Neer test for impingement.</p>	<p>HSP prevalence:</p> <ul style="list-style-type: none"> • Self-report: n = 17 (37%) • HSP with palpation: n = 25 (54%) <p>Pain intensity: not disclosed.</p> <p>Pain diagram: most frequently located area was anterior aspect of shoulder, n = 14 (30%).</p> <p>HSP with impingement test: n = 22 (48%).</p>
Hadianfard,	N = 152	Time post-stroke	Interview and assessment by physician at:	HSP prevalence:

Study Lead author, year	Stroke participants' characteristics	Time post-stroke	Methods of measuring HSP and other measures	Prevalence and intensity of HSP and other outcomes
2008	Age, mean (SD): 61.1 (12.3) yrs Men: 47%	not specified. First assessment at < 2 months post-stroke.	<ul style="list-style-type: none"> < 2 months post-stroke 2-4 months post-stroke 2-6 months post-stroke 6-8 months post-stroke 8-10 months post-stroke 10-12 months post-stroke <p>HSP presence via interview and measurement of intensity via VAS > 50mm (0-100mm) and reduced shoulder PROM.</p> <p>Sensation and vibration threshold via standardised assessments.</p>	<p>n = 49 (32%) during the study period with peak during 2 – 6 months post-stroke (36% of those with HSP in 2-4 months and 34% in 4-6 months)</p> <p>Sensation and vibration threshold:</p> <p>Statistically significant association with HSP.</p>
Huang, 2010	Two groups based on Brunnström motor recovery stage PMF group (Brunnström stages 1-3): n = 35, age, mean (SD): 60.2 (13.3) yrs, men: 55.9% GMF group (Brunnström stages 4-6): n = 23, age, mean (SD): 64.7 (13.3) yrs, men: 65.2%	PMF group: 20.6 (11.5) days GMF group: 18.4 (11.8) days	<p>Assessments completed by same therapist at admission and before discharge.</p> <p>VAS for HSP completed at rest and during passive ROM.</p> <p>Soft tissue lesions via sonography with diagnosis by physician.</p>	<p>HSP prevalence:</p> <ul style="list-style-type: none"> At admission: PMF group: n =19 (55.9%), GMF group: n = 10 (43.5%) Before discharge: PMF group: n =23 (67.6%) GMF group: n = 8 (34.8%) <p>VAS score (0-100mm):</p> <ul style="list-style-type: none"> At admission: PMF group: 2.47 (2.80), GMF group: 1.26 (1.74) Before discharge: PMF group: 3.62 (3.18), GMF group: 1.39 (2.37) <p>Soft tissue lesions: Before discharge, there was a statistically significant difference in frequency of total soft tissue lesions via sonography between PMF and GMF groups.</p>
Tavora, 2010	N = 337 Two groups: HSP group: (n = 45), age, mean (SD), 61.3 (10.2) yrs, men: 55.6% Control group: (n = 23), recruited from participants without HSP (N = 337)), age, mean	Mean:17.3 months (both groups) No SD specified	<p>Presence of HSP determined by PROM, but specific movements and ranges not detailed. Person conducting PROM not specified.</p> <p>MRI and X-rays both groups, but person conducting not specified.</p> <p>MRI review by two Radiologists</p>	<p>HSP prevalence:</p> <p>N = 45 (13.4%)</p> <p>MRI and X-rays:</p> <ul style="list-style-type: none"> Glenohumeral subluxation, n = 2 in HSP group Statistically significantly greater incidence of synovial capsule thickening, synovitis and inflammation of the rotator cuff in HSP group in comparison to the control.

Study Lead author, year	Stroke participants' characteristics	Time post-stroke	Methods of measuring HSP and other measures	Prevalence and intensity of HSP and other outcomes
Roosink, 2011	(SD), 64.4 (12.6) yrs, men: 52% N = 31 Two groups: Persistent HSP group: (n = 9), age, mean (SD): 72 (10) yrs Men, n (%): 6 (67) No persistent HSP group (n = 22), age, mean (SD): 65 (13) yrs Men, n (%): 8 (36)	Persistent HSP group, mean (SD): 8 (3) days No persistent HSP group, mean (SD): 7(3) days	HSP measured at 3 time points by Biomedical scientist within 2 weeks of stroke, and 3 and 6 months post-stroke HSP frequency and intensity (Numeric rating 0, no pain; 10, maximum pain) recorded via interview. Motor function via Motricity index (0-100). PROM shoulder via pain-free abduction and external rotation. Spasticity via Modified Ashworth scale (0-4) Subluxation via palpation. Somatosensory function.	Persistent HSP group: <ul style="list-style-type: none"> Within 2 weeks, n = 8 (22%) At 3 months, n = 11 (35%) At 6 months n = 9 (29%) Persistent HSP intensity: <4 Motor function: Persistent HSP group scored mean 36-38 points less than No persistent HSP group at all time points. PROM shoulder: <ul style="list-style-type: none"> Within 2 weeks of stroke = 10% lower in persistent HSP group At 3 months = not disclosed At 6 months – 42% lower in persistent HSP group Spasticity: Persistent HSP group vs no HSP: <ul style="list-style-type: none"> At 3 months = 44% vs 9% At 6 months = 56% vs 18% Subluxation: At 6 months only = 22% vs 0% Somatosensory function: Persistent HSP group had statistically significantly more frequent abnormal and increased tactile, cold sensation and reduced proprioception; also tactile and visual extinction and visual inattention.
Lindgren, 2012	Participants with HSP at 4 months post-stroke. N = 58 Age, mean: 71 yrs Men: 63.8%	Initial assessment: 4 months Follow-up assessment: 16 months	Measured by physiotherapist Self-perceived HSP frequency and intensity via VAS (0-10 cm). PROM shoulder abduction and external rotation.	HSP prevalence: <ul style="list-style-type: none"> At 4 months, n (%): 58 (100%) At 16 months, n (%): 42 (72%) HSP intensity: No effect noted. PROM shoulder: Statistically significant differences in PROM abduction between participants with and without HSP at 4 months. HSP at 16 months was associated with left side hemiparesis, pain frequency and decreased PROM shoulder abduction at 4 months.
Adey-Wakeling,	Two groups: HSP group (n =	Mean: 8.7 days (both groups)	Assessments undertaken by nurse. Rehabilitation physician trained all assessors in	HSP prevalence: HSP prevalence varied depending on whether

Study Lead author, year	Stroke participants' characteristics	Time post-stroke	Methods of measuring HSP and other measures	Prevalence and intensity of HSP and other outcomes
2014	65) Men: 54% No HSP group (n = 161) Men: 55%	No SD provided	standard approach to objective tests with video package for ongoing reference. Assessments at baseline, 4 and 12 months. PROM tests: Modified Neer test, Passive hand-behind-neck test, Shoulder external rotation. Other assessments: VAS (0-100mm); Motor function item from NHISS (1-4 scale)	participants completed any of the assessments (due to loss-to-follow-up), or all 3 assessments: Participants who completed any assessment: At baseline 10%, At 4 months 21%, At 12 months 29% Participants assessed at all 3 time points: At baseline 8%, At 4 months 18%, At 12 months 21% VAS median pain score highest at 4 months (40mm).
Retrospective studies (in chronological order)				
Bohannon, 1986	N = 50 Age, mean (SD): Men: 40%	No pain group: 25 days (19.9) Some pain group: 28.4 days (15.6) Pronounced pain: 81.1 days (73.8)	Auditor not specified. Fugl-Meyer pain grading (no pain, some pain, pronounced pain) measured during external rotation up to 90° ROM Shoulder external rotation measured with goniometer. Spasticity of shoulder internal rotators via Ashworth scale . Mean static strength deficit of hemiparetic shoulder abductors and external rotators compared to non-hemiparetic shoulder, using a hand held dynamometer. Relationship between HSP , external rotation ROM, time post-stroke, age, spasticity shoulder internal rotators and strength of hemiparetic shoulder abductors and external rotators.	HSP prevalence: <ul style="list-style-type: none"> • Total HSP, n = 36 (72%) • Some pain, n = 20 (55.6%), • Severe pain, n = 16 (44.4%) Significant relationships identified between: HSP and external ROM; HSP and time post-stroke; Time since onset of HSP and external rotation ROM No significant relationship between: HSP and spasticity; HSP and shoulder strength; HSP and age; HSP and time post-stroke Statistically significant differences between HSP subgroups: Time post-stroke and external rotation ROM in no HSP group different from that of severe HSP group. Time post-stroke and external rotation ROM in some HSP group different from that of severe HSP group.
Davenport, 1996	N = 607 Age, median: 73 yrs Men: 46%	Complications documented from hospital admission and through in-patient stay. Mean length of stay: 37 days	Chart audit completed by one medical practitioner. Inter-rater (blinded paramedical) reliability (k = 0.74 for shoulder pain) before study commencement. HSP identified by reviewing chart entries. Diagnostic criteria not identified.	HSP prevalence: n = 9 (5%)
Demerci, 2007	N = 1,000 Age, mean (SD): 61.1 (12.3) yrs	Mean (SD): 104.1 (152) days	Chart auditor not specified. Quality control regarding data extraction not evident. Presence of HSP determined from chart entries.	HSP prevalence: n = 548 (54.8%) PROM shoulder: Statistically significant association between HSP and limitation of PROM.

Study Lead author, year	Stroke participants' characteristics	Time post-stroke	Methods of measuring HSP and other measures	Prevalence and intensity of HSP and other outcomes
	Men, (%): (47)		No diagnostic criteria specified. PROM shoulder: full or limitation of range noted, but only external rotation specified. Subluxation via X-ray Spasticity via Ashworth scale. Motor recovery via Brunnström recovery stages (1-6). ADL assessment via Barthel index Ambulation via Functional Ambulation category	Subluxation: HSP group had 52% subluxation. Spasticity: Statistically significant association between spasticity and HSP. Motor recovery: statistically negative correlation between Brunnström recovery stages and HSP. ADL: Scores statistically significantly lower in group without HSP. Ambulation: Scores statistically significantly lower in group without HSP.
Blennerhas sett, 2010	N = 94 Age, mean (range): 59 (17-80) yrs Men: 61%	Median (range): 12 days (3-181)	Chart auditor not specified. Randomised selection of medical charts, excluding LOS ≤ 6 days. Quality control regarding data extraction not evident. Information extracted from standardised PT and OT assessments , but also from ward rounds and case conferences: HSP identified if documented in the weekly therapy reports, ward round, or case conference notes. PROM shoulder via goniometer Motor Assessment scale , upper arm item Shoulder subluxation via palpation	HSP prevalence: <ul style="list-style-type: none"> On admission to rehabilitation, n = 22 (23%) During rehabilitation, n = 33 (35%) PROM shoulder: Statistically significantly limited shoulder range in participants with HSP vs those without HSP (median range flexion 130 degrees vs 180 degrees; median range external rotation 30 degrees vs 40 degrees). Motor Assessment scale , upper arm item: Statistically significantly lower scores in those participants with HSP, compared to those without HSP (median = 1 vs median = 5). Shoulder subluxation: Statistically significantly greater prevalence of subluxation in those with HSP compared to without HSP (58% vs 7%).

Abbreviations: ADL, Activities of Daily Living; GMF, Good Motor Function; HSP, Hemiparetic Shoulder Pain; MRI, Magnetic Resonance Imaging; NIH, National Institute of Health; NIHSS, National Institute of Health Stroke Scale; PROM, Passive Range of Movement, PMF, Poor Motor Function; ROM, Range of Movement; SD, Standard Deviation; yrs, years; VAS, Visual Analogue scale.

Table 2. Results of bias assessment using the Downs and Black assessment tool

Lead author, year	Reporting (11)*	External validity (3)*	Bias (7)*	Confounding (6)*	Power (1) *	Total (28) *
Prospective studies (in chronological order)						
Parker 1986	8	3	2	3	1	17
Wanklyn 1996	7	3	2	3	1	16
Gamble 2002	8	3	2	3	1	17
Ratnasabapathy 2003	7	3	3	4	1	18
Aras 2004	8	3	3	2	1	17
McLean 2004	8	3	1	2	1	15
Griffin 2006	11	3	7	6	1	28
Lindgren 2007	8	3	4	3	1	19
Dromerick 2008	8	3	3	3	1	18
Hadianfard 2008	6	3	4	3	1	17
Huang 2010	8	3	4	4	1	20
Tavora 2010	6	3	3	4	1	17
Roosink 2011	8	3	4	3	0	18
Lindgren 2012	10	3	4	3	1	21
Adey-Wakeling 2014	9	3	4	3	1	20
Retrospective studies (in chronological order)						
Bohannon 1986	6	3	3	2	1	15
Davenport 1996	7	3	2	3	1	16
Demerci 2007	6	3	3	2	1	15
Blennerhassett 2010	8	3	3	3	1	18

* **Maximum number can be scored in that criterion.**

Downs and Black score ranges: excellent (26–28); good (20–25); fair (15–19); poor (≤14)

One study only recruited participants with HSP (Lindgren et al., 2012) and the remaining study (Adey-Wakeling et al., 2014) had an unexplained low prevalence rate of 10% within four weeks post-stroke. Similar prevalence rates were reported by studies in the fair quality range (Dromerick et al., 2008; Gamble et al., 2002). HSP prevalence appears to increase during the subacute period (up to three months) to 64% (Aras, Gokkaya, Comert, Kaya, & Cakci, 2004) and then stabilise to approximately 30% (Hadianfard & Hadianfard, 2008; Roosink, Renzenbrink, et al., 2011) at three months and later post-stroke. It is possible that these relatively consistent prevalence rates are due to the measurement method used; that is via passive shoulder range. Self-reported HSP prevalence rates appear to be lower (17% to 22%) (Lindgren et al., 2007; Ratnasabapathy et al., 2003), possibly due to the subjective nature of the measurement method. No consistency in HSP prevalence rates were found with the retrospective chart audits (Blennerhassett et al., 2010; Bohannon, Larkin, Smith, & Horton, 1986; Davenport, Dennis, Wellwood, & Warlow, 1996; Demirci, Öcek, & Köseoğlu, 2007).

2.4.1 Changes in the shoulder complex associated with hemiparetic shoulder pain

HSP aetiology remains uncertain, but appears multifactorial (Kalichman & Ratmansky, 2011; Vasudevan & Browne, 2014). Various pathologies, altered central somatosensory sensitivity and biomechanical changes within the glenohumeral joint complex are among some of the proposed aetiological mechanisms. Pathologies thought to be associated with the development of HSP such as adhesive capsulitis, chronic regional pain syndrome type one (Barlak et al., 2009; Pompa et al., 2011), rotator cuff tendinopathy and bursitis are acknowledged as potential contributors. It is possible that these shoulder pathologies may be pre-existing or subclinical (Kalichman & Ratmansky, 2011; Rah et al., 2012). Knowledge about stroke survivors' prior shoulder anatomy and pathology may not always be available (Rah et al., 2012) so its link to HSP aetiology cannot be confirmed. With the prevalence of shoulder pain in the general community highest between the fifth and seventh decades (Kalichman & Ratmansky, 2011), it is feasible that stroke survivors may have had shoulder pain or pathological changes prior to the stroke. However, the relationship between pre-existing shoulder pathologies and HSP is unclear, with conflicting results reported most likely due to the confounder of motor recovery (Aras et al., 2004; Huang et al., 2010). Similarly, altered shoulder proprioception and kinematics (Niessen et al., 2009) and altered peripheral and central nervous system pain mechanisms have also been suggested to underlie hemiparetic shoulder pain (Roosink, Renzenbrink, et al., 2012b;

Zeilig, Rivel, Weingarden, Gaidoukov, & Defrin, 2013) due to the positive association with central somatosensory sensitivity (Roosink, Renzenbrink, et al., 2012b; Roosink, Van Dongen, et al., 2012).

Changes in the position of glenohumeral joint centre (GHJC) and the scapula that contribute to common post-stroke complications of subluxation and impingement (Kalichman & Ratmansky, 2011) are thought to occur as a result of motor impairments including changes in muscle tone and strength following stroke. In a healthy population, significant shoulder pain inhibits shoulder movement (Tobola, Cassas, & Sease, 2009). In the stroke population, the relationship between HSP, shoulder movement and motor recovery appear interrelated. HSP on movement is a predictor of poor functional outcome (Pong et al., 2012; Roy, Sands, Hills, Harrison, & Marshall, 1995) while poor motor recovery has been associated with presence of HSP (Pong et al., 2012; Smith, 2012). Immobility of the upper limb due to paralysis or weakness (Ada et al., 2003; Tyson, Chillala, Hanley, Selley, & Tallis, 2006) and changes in muscle control and adaptive changes in muscle length post-stroke (Ada et al., 2003; Kalichman & Ratmansky, 2011) may affect positioning of the humeral head and scapula, affecting positioning of GHJC, all leading to possible biomechanical changes in the shoulder complex. Minimal bony restraint at the glenohumeral joint enables it to be the human body's most mobile joint, allowing the humeral to translate in multiple directions within the glenohumeral joint itself (Lam, Bhatia, Mostofi, van Rooyen, & de Beer, 2007) – forwards/backwards (anteroposterior: AP), upwards/downwards (superoinferior: SI), and inwards/outwards (medio-laterally). The mobility of the glenohumeral joint together with joint instability puts it at risk of musculoskeletal complications post-stroke (Shepherd & Carr, 1998). Musculoskeletal complications at the shoulder complex lead to biomechanical changes, which are the focus of this research project.

2.4.1.1 Glenohumeral subluxation

Reduction in glenohumeral joint elevation and scapulohumeral rhythm during active movement has been demonstrated in people post-stroke using three-dimensional electromagnetic device (ETD) based protocols (Niessen et al., 2008; Rundquist et al., 2012). Excessive anterior and inferior movement of the glenoid fossa occurs at rest and during post-stroke shoulder movement and is associated with glenohumeral joint subluxation (Vidal & Pascoal, 2011). Glenohumeral subluxation has been defined as a “non-traumatic, partial or total change of relationship between the scapula and the humerus in all directions and in all planes, as compared with the non-affected shoulder, that appeared after stroke” (Paci, Nannetti, & Rinaldi, 2005, p. 558). Inferior subluxation

is one of the most common post-stroke complications (Ada & Foongchomcheay, 2002) and refers to an increase in the normal gap between the acromion and the superior aspect of the humeral head (Ikai, Tei, Yoshida, Miyano, & Yonemoto, 1998). Flaccidity and poor motor control of rotator cuff and the deltoid muscles are thought to allow the force of gravity to inferiorly translate the humerus post-stroke (Turner-Stokes & Jackson, 2002; Umphred, 2007, p. 886). Patients with severe inferior glenohumeral joint subluxation are found to also have anterior subluxation (Ikai et al., 1998; Park, Kim, Sohn, Shin, & Lee, 2007). Anterior subluxation has received little attention in the stroke population (Park et al., 2007).

Despite this, the aetiology and risk factors associated with glenohumeral joint subluxation remain unclear (Paci, 2010). Two systematic reviews demonstrated that glenohumeral joint subluxation prevalence varies from 7% to 81%, dependent on the extent of upper limb paralysis (Ada & Foongchomcheay, 2002), pain and muscle tonus at the shoulder, the population studied, time post-stroke and assessment method (Kumar et al., 2010). At the high end of subluxation prevalence (up to 81%), participants with stroke had no or poor shoulder motor recovery (Moskowitz, Goodman, Smith, Balthazar, & Mellins, 1969; Najenson, Yacubovich, & Pikielni, 1971; Van Langenberghe, Partridge, Edwards, & Mee, 1988) while at the lower end (7% to 18%) subluxation prevalence was associated with more shoulder motor control (Faghri et al., 1994; Fil, Armutlu, Atay, Kerimoglu, & Elibol, 2011; Suethanapornkul et al., 2008).

The association between shoulder pain, muscle tonus and subluxation has been investigated within several reviews (Kumar et al., 2013; Murie-Fernández et al., 2012; Paci et al., 2005), though the reviews remain equivocal about these associations. This lack of consensus could be attributable to whether recruitment and data collection occurred in the acute, subacute or chronic phase or a combination of these. Glenohumeral joint subluxation prevalence rates were lowest during the acute phase in those participants that had good motor recovery (4.2%) and higher in those with poor motor recovery (27% to 32%) (Daviet et al., 2002; Huang et al., 2010), or those whose shoulder muscles remained flaccid within the first eight weeks post-stroke (15%) (Chaco & Wolf, 1971). In the subacute phase post-stroke, the prevalence of glenohumeral joint subluxation appears to increase as time progresses. Prevalence of up to 62% has been reported in subacute stroke populations (Aras et al., 2004; Barlak et al., 2009; Demirci et al., 2007; Suethanapornkul et al., 2008). There are few studies that have investigated the prevalence of subluxation in the chronic phase post-stroke; a small study found a prevalence rate of 44% subluxation up to 12 months post-stroke, though subluxation was measured via palpation only (Lo et al., 2003).

Another reason for the disparity of reported subluxation prevalence rates is whether a clinical or more quantitative assessment such as a radiological or ultrasonographic technique was used (Park et al., 2007). Clinical assessment methods use finger-breadths, callipers or plexiglass jig to measure the gap between the inferior acromial surface and the superior humeral head (Boyd et al., 1993a; Boyd & Torrance, 1992). These are a useful screening tools, but are not sufficiently sensitive to detect small changes in glenohumeral joint displacement (Hall, Dudgeon, & Guthrie, 1995; Park et al., 2007), which may contribute to lower reported prevalence rates. Discriminatory quantification of subluxation is available via radiographic and ultrasonographic assessment methods (Boyd et al., 1993a; Hall et al., 1995; Huang et al., 2012; Park et al., 2007). These enable superior measurement precision (Hall et al., 1995) and also allow measurement of anterior subluxation (Hall et al., 1995; Lee & Han, 2000; Park et al., 2007). However, clinical utility of such methods is limited. For more precise determination of glenohumeral joint subluxation in both the inferior and anterior direction, in particular longitudinally, more precise measurement tools are needed (Kumar & Swinkels, 2009).

2.4.1.2 Scapula position

Some controversy exists regarding the scapular resting position following a stroke. A recent literature review (Struyf, Nijs, Baeyens, Mottram, & Meeusen, 2011) revealed inconsistencies with regard to the scapular resting position in both shoulders in asymptomatic healthy subjects. Any change in scapular resting position may adversely affect the congruency of the humeral head with the glenoid cavity and hence the integrity of the glenohumeral joint at rest and during movement (Constant, 1989; Hardwick, 2010). Upward rotation of the scapula during movement lifts the coracoacromial arch, thereby avoiding impingement of the humeral head against the acromion and maintains shoulder stability (Hanchard, Lenza, Handoll, & Takwoingi, 2013; Lucas, 1973). Scapular rotation occurs via actions of the trapezius and serratus anterior muscles; external rotation of the humerus, via the action of the infraspinatus muscle (Turner-Stokes & Jackson, 2002), also allows the humeral tubercles to clear the arch, further avoiding impingement (Hanchard et al., 2013; Lucas, 1973).

Scapular and humeral rotation are affected following a stroke when muscles are weakened, failing to protect the glenohumeral joint from impingement (Poppen & Walker, 1976; Turner-Stokes & Jackson, 2002) and enhancing the risk of damage to

the rotator cuff musculature during passive arm movements (Najenson et al., 1971). Glenohumeral joint impingement may result in HSP (Hardwick, 2010).

2.4.1.3 Subacromial impingement

Impingement is a common cause of shoulder pain in the non-stroke population (Garofalo et al., 2010; Hanchard et al., 2013; Heyworth & Williams, 2009; Ludewig & Braman, 2011) and has been identified in people with stroke (Barlak et al., 2009; Demirci et al., 2007; Joynt, 1992; Shah et al., 2008). Impingement may occur inside the glenohumeral joint (internal impingement) or in the space beneath the coracoacromial arch (subacromial impingement syndrome) (Hanchard et al., 2013). Internal impingement occurs when the glenoid rim, the joint capsule or glenoid labrum is encroached on by the rotator cuff or the humerus, causing shoulder pain (Garofalo et al., 2010). There are two types of internal impingement: posterior-superior impingement and anterior-superior impingement (Garofalo et al., 2010; Hanchard et al., 2013). Subacromial impingement syndrome (SAIS) is caused by a reduction in the space under the coracoacromial arch, impacting the soft tissues beneath it. In individuals who have SAIS, these tissues are pinched between the humeral tuberosities and the inferior surface of the coracoacromial arch during arm movement, causing shoulder pain (Hanchard, Cummins, & Jeffries, 2004; Hanchard et al., 2013). Secondary pathologies are often associated with impingement and include tendinopathies or tears of the rotator cuff or long head of biceps and acromial or deltoid bursitis (Hanchard et al., 2013).

It has been proposed that impingement may occur post-stroke and contribute to the development of hemiparetic shoulder pain (Barlak et al., 2009; Demirci et al., 2007; Shah et al., 2008). Impingement is common in older adults (Garofalo et al., 2010) so maybe pre-exist in a stroke population. Coordinated contraction of the rotator cuff, deltoid and scapular muscles allows accurate coupling of the humerus and scapula and pain-free gliding of the soft tissues around the glenohumeral joint and acromion. Accurate humeral and scapula coupling ensures that GHJC is maintained. Disruption of humeral and scapula coupling results in GHJC position change and adversely affects the subacromial space, resulting in soft tissues being impinged (Hardwick & Lang, 2011b). This occurs during the post-stroke period when scapular and humeral movement patterns are altered (compared to a healthy population), particularly reduced active humeral external rotation, and impingement results (Hardwick & Lang, 2011b). Spasticity or flaccidity in any of the glenohumeral joint or scapular muscles can also disrupt the scapulohumeral coupling, causing impingement of the soft tissues beneath

the coracoacromial arch (Jaraczewska & Long, 2006). Impingement gives rise to shoulder pain, which as highlighted earlier, adversely impacts participation in rehabilitation (Barlak et al., 2009) and upper limb function (Vasudevan & Vasudevan, 2008).

Despite HSP being a common post-stroke complication with consequences of upper limb dysfunction and dependency, there is a lack of agreement regarding identification of the underlying cause(s) and soft tissue lesions associated with it, such as SAIS (Dromerick, Kumar, Volshteyn, & Edwards, 2006). Reliability of a number of physical tests for soft tissue lesions have been reported in an effort to standardise this assessment of patients with shoulder pain (Dromerick et al., 2006), but to date a dearth of literature exists regarding this topic in the post-stroke population. Much of the literature involving stroke participants relate to the trial of corticosteroid injections to ease HSP in those diagnosed with SAIS (Chae & Jedlicka, 2009; Lakse, Gunduz, Erhan, & Celik, 2009; Rah et al., 2012; Viana, Pereira, Mehta, Miller, & Teasell, 2012). Comprehensive and accurate assessment of the hemiparetic shoulder girdle complex will enable pain free function of the hemiparetic upper limb to be achieved during rehabilitation (De Baets, Jaspers, Desloovere, & Van Deun, 2013).

In summary, HSP has been associated with various factors including shoulder pathologies, altered peripheral and central nervous system pain mechanisms and biomechanical changes within the glenohumeral joint complex. The relative contribution of these factors is difficult to determine, though it is likely that these factors, alone or in combination, play differing roles in the context of the individual involved, any comorbidities, time post-stroke, severity of the stroke, and management of the sequela following stroke. HSP prevalence is variable and appears to be influenced by the assessment timing, measurement methods used and the professional affiliation of the researchers conducting the study, but appears to increase over time.

2.5 Measurement of glenohumeral joint centre

The position of the head of the humerus relative to the glenoid fossa can be defined as the glenohumeral joint centre. The glenohumeral joint centre (GHJC) is defined by both the geometric centre of the head of the humerus, as well as the rotation centre of the glenohumeral joint as a whole (physiological centre), both of which coincide and experience little deviation during dynamic movement of the shoulder (Veeger, 2000). Changes in GHJC have been associated with stroke as well as with musculoskeletal pain.

Changes in GHJC position in the stroke population are inferred in conditions such as glenohumeral joint subluxation and impingement. Glenohumeral joint subluxation and impingement are common post-stroke complications in which the humeral head position translates from being centred within the glenoid fossa to being positioned inferiorly and/or anteriorly (Boyd et al., 1993b; Park et al., 2007) or superiorly (Dromerick et al., 2006; Turner-Stokes & Jackson, 2002) to the geometric and rotation (physiological) centre of the glenohumeral joint (GHJC). However, to date, changes in GHJC position in the acute stroke population have not been investigated. The current study measured GHJC in order to investigate and track its position over time in post-stroke participants, some of whom may have begun to develop these glenohumeral joint complications in the acute phase.

Evidence of displacement such as inferior subluxation (Kumar et al., 2010) or impingement (Barlak et al., 2009) within the glenohumeral joint in the subacute and chronic phases post-stroke lends weight to the necessity of determining GHJC in the acute post-stroke population. Electromagnetic tracking devices have been used to determine the location of GHJC in healthy populations; its validity and reliability have been established (Meskers, Vermeulen, Groot, van der Helm, & Rozing, 1998; Stokdijk, Nagels, & Rozing, 2000; Veeger, 2000), but its use to determine GHJC position in a stroke population has received little investigation.

Several studies have measured positional changes within the glenohumeral joint following stroke (Arsenault, Bilodeau, Dutil, & Riley, 1991; Hardwick & Lang, 2011a; Kumar, Bradley, Gray, & Swinkels, 2011; Rundquist et al., 2012). Different equipment (ETDs, motion analysis, radiography, ultrasonography), measurement and calculation methods (for locating GHJC such as predictive and sphere-fitting) and healthy and stroke populations (subacute, chronic, with HSP, without HSP) have been used. Table 3 summarises the participant group/s, methods of measurement and calculation of glenohumeral joint position for studies which have investigated the kinematics of the scapula, humerus, glenohumeral joint and upper limb using electromagnetic tracking devices. Table 4 provides a similar summary of studies which have investigated shoulder kinematics using other measurement methods including motion analysis, radiography and ultrasonography. No prospective studies were identified that investigated GHJC in an acute post-stroke population. However, glenohumeral joint position (GHJC) has been reported in a healthy population.

One study calculated GHJC in a healthy population of ten young men (Stokdijk et al., 2000) and the other in cadavers (Veeger, 2000), but both studies used an electromagnetic tracking device (Stokdijk et al., 2000; Veeger, 2000). Stokdijk and colleagues' investigations calculated the GHJC to be similarly located anterior and inferior to the acromion using three different methods (Algorithm, Helical axis and sphere-fitting) (Stokdijk et al., 2000). The cadaveric study, in comparing the geometric GHJC with the kinematic GHJC, using the helical axis and sphere-fitting methods, found that their measurements of GHJC coincided (Veeger, 2000). GHJC was again found to be located anterior and inferior to the acromion, but measurements varied from those of the first study. Comparison of the results from the two ETD studies appear to indicate that GHJC location is variable (Table 3). Evaluation of GHJC location determined using different equipment and similar methods may yield different results. Studies calculating GHJC using motion analysis systems with infrared cameras have also been undertaken (Table 4) (Lempereur, Brochard, & Remy-Neris, 2013; Lempereur et al., 2010; Monnet et al., 2007). The most recent study (Lempereur et al., 2013) compared three different sphere-fitting (Algorithm, Bias Compensation and Normalisation) and two predictive (Symmetrical Centre of Rotation Estimation and Helical Axis) methods. These same sphere-fitting (Lempereur et al., 2010) and predictive (Lempereur et al., 2010; Monnet et al., 2007) methods have been shown to be accurate and reliable for measuring GHJC location. Differences were found between the studies in the reported position of the GHJC across all measurement and calculation methods. However, it appears that sphere-fitting methods are more accurate with good within-session reliability (repeatability error mean 6.3 mm, accuracy mean [SD] 11.4 mm [8.0] and GHJC equal to half the radius of the sphere (Lempereur et al., 2010). As a result, sphere-fitting methods were chosen for the current study. ETDs have also been used to investigate other aspects of shoulder complex kinematics, but for the purposes of this study, the ETD was chosen to measure GHJC.

In addition to investigating GHJC, ETDs have been used in the healthy population to determine its reliability (Meskers et al., 1998), explore shoulder impingement (Hamming, Braman, Phadke, LaPrade, & Ludewig, 2012; Ludewig & Cook, 2000) and gauge the plane of humeral movement relative to the thorax as well as scapulothoracic rhythm during shoulder elevation (Braman, Engel, LaPrade, & Ludewig, 2009) (Table 3).

ETDs have been used in participants in the subacute to chronic phase post-stroke, to determine the position of both scapulae and humerus on the hemiparetic (Culham, Noce, & Bagg, 1995; Hardwick & Lang, 2011b; Niessen et al., 2008; Rundquist et al.,

2012) and non-hemiparetic sides (Meskers et al., 2005), as well as the extent of active range of movement of the shoulder (Beebe & Lang, 2009). The reliability of determining GHJC with an ETD using a sphere-fitting method, has not been determined in the acute phase post-stroke. Neither have ETDs been used to determine GHJC in acute patients with stroke.

Glenohumeral joint kinematic research in people with stroke and healthy people has also involved imaging techniques. Glenohumeral joint kinematic exploration has involved radiography and ultrasound in healthy participants or participants with stroke in the subacute or chronic phase or where the time post-stroke has not been specified (Table 4). Kinematic GHJC was reported to be within 6 mm of geometric GHJC in a healthy population and more than 10 mm in participants with shoulder pathology in a radiography study (Poppen & Walker, 1976). Radiographic studies involving participants with stroke have investigated the position of the humeral head. A difference between hemiparetic and non-hemiparetic sides in the V-shaped space between the humeral head and the glenoid fossa was found (Arsenault et al., 1991) as well as glenohumeral joint subluxation, impingement and soft tissue lesions in participants with HSP (Barlak et al., 2009). Humeral head centre and the scapula were displaced more in a group of participants with impingement than in control participants during Radiostereometric analysis (Hallstrom & Karrholm, 2009).

An ultrasound study reported a significantly greater distance between the acromion and the greater tuberosity on the hemiparetic than on the non-hemiparetic side (Kumar et al., 2011); this indicated that the humeral head had moved inferiorly compared to its position on the non-hemiparetic side (Table 4).

Gaps in the stroke literature have been identified. The reliability of determining GHJC position using a sphere-fitting method via an ETD has not been determined in participants with acute stroke. Furthermore, GHJC has not been measured in participants with acute stroke using any method via ETD, radiography or ultrasound as outlined in Table 3 and Table 4.

Table 3. Summary of research studies that have used electromagnetic tracking devices (ETD) to measure shoulder kinematics

Study Reference	Participants	Areas measured	Equipment	Methods of Measurement	Outcomes
Culham et al. 1995	Stroke participants N = 34 Age, range: 41 to 89 yrs 2 groups: • 17 low muscle tone • 17 high muscle tone	Scapula and humerus bilaterally and trunk	ETD (3-Space Isotrak) to measure scapular and humeral orientation Radiography to measure subluxation	Scapular, humeral, thoracic and pelvic landmarks to determine x, y and z axes for angular and linear measures of the scapula, humerus, thorax and trunk. Modified Ashworth scale used to measure muscle tone.	Low tone group: Scapula further from midline and lower on hemiparetic and non-hemiparetic side. Greater glenohumeral joint subluxation. High tone group: No differences between sides. No correlation between subluxation and scapular or humeral orientation.
Meskers et al. 1998	Healthy participants N = 15 Age mean (SD): 24.3 (8.4) yrs Mean weight (SD): 67.2 (11.1) kg Mean height (SD): 1.79 (0.24) m	Scapula and glenohumeral joint and trunk. Side not specified but unilateral results presented	ETD (Ascension Technology, Burlington, VT, USA)	Three sensors attached to thorax, scapula (via scapula locator) and humerus. Fourth sensor attached to mobile sensor (stylus). Participant performed maximal elevation in three planes with measurements recorded at 10° increments x 5 reps: a) Forward flexion b) Scapular plane c) Frontal plane	Inter- and intra-rater variability reduced with coordinate system located on bony landmarks
Stokdijk et al. 2000	Healthy men N = 10 Age, mean (SD): 21.9 (3.6) yrs	Glenohumeral joint centre – GHJC Side not specified but unilateral results presented	ETD (Ascension Technology Inc. Burlington, VT, USA)	GHJC measured using 3 methods (regression, sphere-fitting and HA) by observers Movements completed: (a) Combine forward flexion, horizontal abduction, adduction, scapular abduction and circumduction (b) circumduction x 3 forward flexion, scapular abduction and shoulder abduction	<ul style="list-style-type: none"> • All methods reliable, but GHJC location differed for each method • Inter- and intra-tester reliability higher for sphere-fitting and HA methods • HA <ul style="list-style-type: none"> AP 48.4 (2.6) mm anterior to acromion SI 39.4 (4.2) mm inferior to acromion • Algorithm (Sphere-fitting): <ul style="list-style-type: none"> AP 47.6 (3.2) mm anterior to scapula SI 38.4 (4.1) mm inferior to acromion
Veeger 2000	Cadavers (n = 4) ; no visible degeneration both shoulders	Glenohumeral joint centre – GHJC	ETD (3 Space Isotrak System)	Anatomical and kinematic GHJC measured using HA and	<ul style="list-style-type: none"> • Geometric and kinematic GHJC identical and no difference between methods.

Study Reference	Participants	Areas measured	Equipment	Methods of Measurement	Outcomes
		Side not specified but unilateral results presented		<p>sphere-fitting methods.</p> <p>Shoulder moved passively 3 times in each direction twice in approx. 15 sec</p> <p>(a) abduction-adduction (b) flexion-extension (c) internal-external rotation</p>	<ul style="list-style-type: none"> Geometric: <ul style="list-style-type: none"> AP 32.0 (0.5) mm anterior to acromion SI 25.9 mm inferior to acromion Kinematic: <ul style="list-style-type: none"> AP 31.1 (11.9) mm anterior to acromion SI 25.8 mm inferior to acromion
Ludewig and Cook 2000	<p>Healthy construction workers (men) N = 52</p> <p>2 groups (n = 26): Participants without impingement Age mean (SD): 39.9 (12.3) yrs</p> <p>Participants with impingement Age mean (SD): 39.7 (12.0) yrs</p>	Thorax, scapula and humerus on dominant side only	<p>Motion capture system (Polhemus FASTRAK) (40-Hz sampling rate)</p> <p>EMG unit</p>	<p>Sensors attached to sternum, acromion and distal humerus to track orientation of thorax, scapula and humerus.</p> <p>Digitisation of thorax, scapula and humerus. Participants completed humeral elevation in scapular plane once every 4 sec.; with and without hand held load</p> <p>EMG activity of upper and lower trapezius muscles and lower serratus anterior muscle.</p>	<ul style="list-style-type: none"> Group with impingement showed 4.1° less upward scapular rotation and 5.8° more anterior scapular tilting than group without impingement. Under load conditions group with impingement had 5.2° more scapular medial rotation than group without impingement Upper and lower trapezius muscles EMG activity increased more in impingement group. Decreased activity in serratus anterior in impingement group when loaded
Meskers et al. 2005	<p>Participants with stroke and healthy age matched controls</p> <p>Strokes: N = 10 Age, mean (SD): 53.4 (10.3) yrs Time post-stroke: 7.7 months</p> <p>Controls: N = 10 Age mean (SD): 60.8 (12.4) yrs</p>	Non-hemiparetic shoulder only	ETD (Innovative Sports Training, Chicago, IL, USA)	<p>Sensors on thorax, upper arm and scapular locator. Also mobile sensor to digitise landmarks on scapula, humeral and thorax.</p> <p>Active elevation as high as possible, stopping incrementally at marked 10° elevations; maximal passive internal and external rotation x 4 reps.</p> <p>Shoulder pain: measured via VAS Muscle tone: modified Ashworth scale.</p>	<ul style="list-style-type: none"> Shoulder elevation 12° less, scapular protraction 13° less during elevation in sagittal plane in stroke group than in control group. Humeral external rotation during elevation in frontal plane 18° less and in sagittal plane 29° less in stroke group than in control group. Max passive IR 30° less and ER 34° less in frontal plane in stroke than in control group Scapular posterior tilt 7° less during ER in sagittal plane and 11° less during ER in the frontal plane in stroke than in control

Study Reference	Participants	Areas measured	Equipment	Methods of Measurement	Outcomes
				Arm function: Fugl-Meyer score.	<ul style="list-style-type: none"> group Scapular posterior tilt was 15° more during IR in frontal plane in stroke than in the control group Non-hemiparetic shoulder should not be considered to have normal function
Niessen et al. 2008	<p>Participants with stroke and healthy age matched control participants</p> <p>Strokes: N = 27</p> <p>Two groups:</p> <ul style="list-style-type: none"> • With HSP, n = 13 Age, mean (SD): 59.3 (11.1) yrs Time post-stroke, mean (SD): 14.4 (9.3) weeks • Without HSP, n = 14 Age, mean (SD): 57.0 (9.5) yrs Time post-stroke, mean (SD): 13.0 (7.6) weeks <p>Age matched healthy controls. N = 27 Age, mean (SD): 49.3 (7.2) yrs</p>	<p>Hemiparetic and non-hemiparetic scapula and humerus</p> <p>Bilateral control upper limbs</p>	ETD (Innovative Sports Training, Chicago, IL, USA)	<p>Sensors on thorax, upper and lower arm and acromion. Also mobile sensor to digitise landmarks.</p> <p>Passive and active (if able) arm elevation to 120° with both arms x 3 reps. Also maximum humeral external and internal rotation.</p> <p>Degree of paralysis determined via Brunnstrom stage.</p>	<ul style="list-style-type: none"> At rest, 7° more scapular lateral rotation in hemiparetic side in HSP group than in hemiparetic side in no HSP group and 9° more than in same side in control group At rest, 6° more scapular lateral rotation on non-hemiparetic side in HSP group than in non-hemiparetic side in no HSP group and 10° more than on same side in control group Scapular lateral rotation similarly increased during passive and active shoulder flexion and abduction in strokes with HSP when compared to strokes without HSP and controls Humeral elevation during passive abduction decreased in strokes with HSP
Beebe and Lang 2009	<p>Participants with stroke N = 33 Age, mean (SD): 57 (10) yrs Time post-stroke, mean (SD): 18.6 (5.6) days</p> <p>3 month follow-up: N = 28 (n = 1 died, n = medically unwell, n = 2 lost contact)</p>	Hemiparetic upper limb, including shoulder, elbow, wrist and hand	ETD (Motion Monitor; Innovative Sports Training Inc, Chicago IL, USA)	<p>9 sensors attached to trunk, arm, forearm and fingers.</p> <p>Recorded 9 anti-gravity segmental movements as far as able, at self-selected pace x 2 reps, at 1 month and 3 months post-stroke. ETD calculated AROM for each of the 9 segments.</p> <p>Upper limb function, bilateral</p>	<ul style="list-style-type: none"> Shoulder and middle finger AROM measured at 1 month predicted 71% of the variance in upper limb function at 3 months Similar recovery rates occurred at all segments with no evidence of a proximal to distal recovery pattern over time

Study Reference	Participants	Areas measured	Equipment	Methods of Measurement	Outcomes
				limb strength using dynamometer, HSP, index finger joint position sense and muscle tone using Modified Ashworth scale recorded.	
Braman et al. 2009	Healthy participants N = 12 Age, mean (SD): 29.3 (6.8) yrs Mean height 1.7 (SD 0.1) metres Mean weight 77.5 (SD 13.8) kg.	Scapula and humerus on non-dominant side	ETD (Ascension Technology, Burlington, VT) Fluoroscopy (Mini View 6800 Mobile Imaging System, Milwaukee)	Under fluoroscopy guide, stainless steel pins surgically inserted into scapula spine, distal clavicle, and deltoid insertion. To record active movement, ETD sensors fixed to bone pins. Surface sensor taped to the sternum. Used mobile sensor to digitise anatomical landmarks. Participants reached upwards as far as possible, no control of the plane, height or speed of the movement, in 3 sec x 2 reps.	<ul style="list-style-type: none"> Average plane of humero-thoracic movement during reaching: 63.3° (SD 7.0) forward of the coronal plane. Mean peak elevation range: 132.9° (SD 9.9) Scapulo-thoracic rhythms different during arm raising and lowering. For each 1° of glenohumeral elevation, 0.43° upward scapula rotation. For each 1° of humeral lowering, 0.37° downward scapula rotation During overhead reaching, glenohumeral component to scapulothoracic rhythm greatest during 0° to 30° increment and decreased as angle of humerus to the trunk increased
Hardwick and Lang 2011	Participants with stroke with HSP and age and gender matched healthy control participants Participants with stroke: N = 9 Age, mean (SD): 56 (8) yrs Time post-stroke, mean (SD): 7.2 (6.7) months Healthy participants: N = 12 Age, mean (SD): 54 (9) yrs	Scapula and humerus on hemiparetic side	ETD (Innovative Sports Training, Chicago, IL, USA)	4 sensors attached to sternum, proximal to lateral epicondyle, dorsal aspect of forearm and acromion. Bony landmarks digitised on thorax, scapula and humerus at rest. 3 trials of scapular plane elevation to a target at self-selected speed with elbow extended Shoulder pain at rest measured via VAS. Pain severity measured using Shoulder Pain and Disability Index pain subscale.	<ul style="list-style-type: none"> HSP at rest mean VAS 22 mm, 48 mm on movement Mean scapular upward rotation across all humeral elevation angles = 14.7° in acute subgroup and 2.4° in chronic subgroup. No significant differences in scapular tilt between strokes and controls Stroke participants had reduced humeral external rotation compared to controls. Pain with movement was negatively correlated with scapular upward rotation. Shoulder pain disrupted daily life with score of 44.9 on this scale. 6/9 had positive Neer impingement test and positive

Study Reference	Participants	Areas measured	Equipment	Methods of Measurement	Outcomes
				Neer's impingement test and palpation long head biceps and supraspinatus	palpation of long head of biceps and supraspinatus
Hamming et al. 2012	Adults with and without shoulder impingement N = 19 Age, mean (range): 34.9 (21 to 59) yrs. Height mean (SD): 170.4 (8.9) m Weight mean (SD): 76.2 (12.3) kg 2 groups: Shoulder impingement: N = 10 Participants with no shoulder pathology: N = 9	Scapula and humerus bilaterally	ETD (Ascension Technology, Burlington, VT)	Sensors surgically attached to transcortical pins in clavicle, acromion, and deltoid insertion. Two skin sensors attached to sternum and to elbow, proximal to epicondyles. Digitisation of anatomical landmarks. Participants raised and lowered arm over 6 s x 2 reps in 3 planes (sagittal, scapular, coronal) Shoulder ER and IR with elbow at side and abducted 90°. Data recorded for bone and skin sensors simultaneously.	<ul style="list-style-type: none"> • Skin sensors accurate method of measuring humeral elevation in different planes of movement • Skin sensors not accurate when measuring axial rotation during shoulder ER and IR • Increased average error rate in participants with BMI > 25, particularly during ER and IR
Rundquist et al. 2012	Participants with stroke: N = 16 Age, mean (SD): 60.6 (11.1) yrs Time post-stroke, mean (SD): 66.2 (40) months	Scapula and humerus on hemiparetic side	ETD (Innovative Sports Training, Chicago, IL, USA)	Sensors attached to acromion process, sternal notch and superior to the epicondyles. Active shoulder elevation performed in frontal, sagittal and self-selected plane of movement x 3 reps at steady, self-selected pace	<ul style="list-style-type: none"> • Maximum humeral elevation median 106.7°. • Scapulohumeral rhythm was 4:1 when humeral elevation ranged from 45° to 50°, 1.5:1 from 80° to 95° and 2:1 from 105° to 130°. • Humeral elevation, scapular upward rotation and scapular internal rotation predicted 65.4% of motor function score variability

Abbreviations: AP, anteroposterior; AROM, active range of movement; ETD, electromagnetic tracking device; Kg, kilogram; EMG, electromyography; ER, external rotation; GHJC, glenohumeral joint centre; HA, helical axis; HSP, hemiparetic shoulder pain; IR, internal rotation; reps, repetitions; s, seconds; SD, standard deviation; SI, superoinferior; VAS, visual analogue scale; yrs, years.

Table 4. Shoulder kinematic studies measured using motion analysis, radiography and ultrasound

Study Reference	Study design and participants	Areas measured	Equipment	Methods of Measurement	Outcomes
Motion analysis systems					
Monnet et al. 2007	Reliability study Healthy men N = 9 Age, mean (SD): 27.9 (1.1) yrs	Glenohumeral joint – GHJC Side not specified but unilateral results presented	Motion analysis system with infrared cameras (50Hz) (Biogesta, Valenciennes, France)	Three shoulder movements not exceeding shoulder height x 10 cycles each x 2 speeds (slow and medium): a) flexion-extension b) abduction-adduction circumduction	<ul style="list-style-type: none"> • SCoRE and HA methods determined same GHJC location. • SCoRE more precise than HA particularly with slow movements • GHJC position (as determined by SCoRE): <ul style="list-style-type: none"> ○ AP – 13.5 (6.7) mm anterior to acromion ○ SI - 34.7 (9.8) mm inferior to acromion SEM: SCoRE 3.0 mm, HA 4.6 mm
Lempereur et al. 2010	Reliability study Healthy participants (men) N = 4 Age mean (SD): 26.5 (1.89) yrs Height mean (SD): 1.74 (0.04) m. Weight mean (SD): 73.8 (10.4) kg.	GHJC Side not specified but unilateral results presented	Motion analysis system (Vicon MX, Oxford Metrics Ltd., Oxford, UK), 9 infrared cameras (120 Hz)	Participants completed 3 movements x 3 cycles: (a) Flexion-extension; (b) Abduction-adduction; (c) Circumduction GHJC estimated via 5 functional methods: (a) Algorithm using Gamage and Lazenby; (b) Bias Compensation; (c) SCoRE; (d) Normalisation; (e) HA	<ul style="list-style-type: none"> • SEM < 8.3 mm for all methods • Significant differences between the 5 methods. Most accurate estimation of GHJC via Gamage and Lazenby method. • SCoRE method GHJC position: AP – 35.1 mm SI - 23.8 mm
Lempereur et al. 2013	Reliability study Healthy participants N = 10 Age mean (SD): 25.8 (3.3) yrs, range 21 to 31. Height mean (SD): 1.77 (0.09) m. Weight mean (SD): 68.3 (8.0) kg.	GHJC via scapula and acromion Side not specified but unilateral results presented	Motion analysis system (Vicon MX, Oxford Metrics Ltd., Oxford, UK) with 9 infrared cameras (120 Hz).	Three sensors attached to acromion and one on scapula locator. Participants performed 3 movements x 3 reps x 3 ranges using 5 functional methods Movements: (a) Flexion-extension; (b) Abduction-adduction; (c) Circumduction Ranges: (a) Small ($\leq 30^\circ$); (b)	<ul style="list-style-type: none"> • Location of GHJC differed between methods • Reliability <ul style="list-style-type: none"> ○ Good within session reliability regardless of method; lowest error with high amplitude (ICC=0.9) ○ Moderate inter-session reliability; lowest error with high range regardless of method Functional (e.g. sphere-fitting)

Study Reference	Study design and participants	Areas measured	Equipment	Methods of Measurement	Outcomes
				Medium (0° to ≤ 60°); (c) High (≤ 90°) Methods: (a) Algorithm using Gamage and Lazenby; (b) Bias Compensation; (c) SCoRE; (d) Normalisation; (e) HA No instructions given regarding speed.	methods using high range are most reliable in locating GHJC
Radiography					
Poppen and Walker 1976.	Case-control study Participants with shoulder pathology N = 15 Age, range: 17 to 72 yrs N = 12 healthy participants Age, range: 22 to 63 yrs	Scapula, glenohumeral joint and humerus bilaterally, including GHJC	Radiography	X-ray while standing with arm at side, shoulder abducted with maximal scapula abduction. Three sets of axes for each of the torso, scapula and humerus drawn on each X-ray.	<ul style="list-style-type: none"> Glenohumeral to scapulothoracic ratio was 5:4 after 30° of abduction Abnormal glenohumeral to scapulothoracic ratio associated with painful shoulder GHJC during scapular abduction within 6mm of anatomical centre for healthy participants; for participants with shoulder pathology, GHJC was ≥ 10mm superior to the anatomical centre. Humeral excursion within glenoid cavity was within 1.5 mm in healthy participants; abnormal in those with shoulder pathology
Arsenault et al. 1991	Longitudinal cohort study Participants with stroke N = 40 Age, mean (SD): 46.5 (13.8) yrs Time post-stroke not specified 2 groups: <ul style="list-style-type: none"> n = 19 with subluxation n = 21 without 	Hemiparetic and non-hemiparetic glenohumeral joint and scapulae bilaterally	Radiography	Radiographs of hemiparetic and non-hemiparetic shoulders. Measurements of V-shaped space between humeral head and glenoid fossa, abducted arm and scapula. Shoulder pain intensity via McGill Pain questionnaire. Evaluated before, during and	<ul style="list-style-type: none"> Significant 5° difference in V-shaped angle with shoulder abducted 45°: between hemiparetic and non-hemiparetic shoulder in subluxation group Significant difference in angle of humeral abduction of 3° in subluxation group compared to those without subluxation Humeral head subluxation inferiorly relative to the scapula No change in scapula position

Study Reference	Study design and participants	Areas measured	Equipment	Methods of Measurement	Outcomes
	subluxation			after treatment (treatment details not specified) over three months.	<ul style="list-style-type: none"> No relationship of pain with subluxation
Hallström and Kärrholm 2009	<p>Participants with impingement N = 30 Age, mean (range): 50 (29 – 63) yrs</p> <p>Control group N = 11 Age, mean (range): 38 (22 – 58) yrs</p>	Scapula and humerus	Radiostereometric analysis (RSA Biomedical, Umeå, Sweden)	<p>4-6 spherical tantalum markers inserted under local anaesthetic into acromion and humeral head.</p> <p>Passive and active shoulder abduction at constant speed.</p> <p>Measured centre of humeral head translations using two images. Radiographic films scanned and measured using software and 3-D coordinates computed.</p>	<ul style="list-style-type: none"> Proximal displacement of centre of humeral head with increasing passive and active abduction of glenohumeral joint was: <ul style="list-style-type: none"> 30 mm impingement group 20 mm control group Scapula displacement was: <ul style="list-style-type: none"> 2.0 mm impingement group 0.5 mm control group Glenohumeral to scapulothoracic ratio during abduction was $\leq 1:1$ in both groups
Barlak et al. 2009	<p>Cohort study</p> <p>Participants with stroke N = 187</p> <p>Two groups: With HSP, n = 114 Age mean (SD): 62.4 (10.7) yrs Time post-stroke mean (SD): 124.3 (55.1) days</p> <p>Without HSP n = 73 Age mean (SD): 59.6 (12.0) yrs Time post-stroke, mean (SD): 89.9 (42.5) days</p>	Hemiparetic glenohumeral joint	<p>Radiography</p> <p>Ultrasound 5-12 MHz</p>	<p>AP Radiographic examination of V-shaped distance in the intra-articular space between humeral head and glenoid fossa, to determine presence and grade of subluxation.</p> <p>Linear ultrasound transducer examination of shoulder soft tissues.</p> <p>Diagnosis of CRPS recorded</p> <p>HSP via VAS during preceding 7 days.</p> <p>FIM on admission and discharge.</p> <p>PROM hemiparetic shoulder with goniometer.</p> <p>Hemiparetic upper limb motor function assessed via</p>	<ul style="list-style-type: none"> In HSP participants. <ul style="list-style-type: none"> 71 (62.3%) had glenohumeral joint subluxation Significant relationship between HSP and adhesive capsulitis and CPRS Significantly greater functional outcomes and shorter length of hospital stay in participants without HSP compared to those with HSP No correlation of HSP with gender, hemiparetic side, dominance, etiological cause or comorbidities

Study Reference	Study design and participants	Areas measured	Equipment	Methods of Measurement	Outcomes
Brunnstrom recovery stages.					
Diagnostic ultrasound					
Kumar et al. 2011	Longitudinal reliability and validity study Participants with stroke older than 50 yrs N = 26 Age mean (SD): 71 (10) yrs Time post-stroke, mean (range): 24 (7 to 54) days Follow-up: N = 21 Time between initial and follow-up, mean (range): 6 (2 to 21) days.	Acromion and humerus Hemiparetic and non-hemiparetic sides	Portable diagnostic ultrasound (TITAN model, L38/10-5MHz broadband)	Shoulder scanned with ultrasound x 3 images x 2 sets of measurements. Repeat measurement on other shoulder. Acromion to greater tuberosity (AGT) distance recorded on a still image Measurement protocol repeated within 2 weeks. Motor strength (Oxford scale) bilaterally. Muscle tone assessed bilaterally (high/low).	<ul style="list-style-type: none"> • Significant difference between hemiparetic and non-hemiparetic AGT distance; hemiparetic shoulder mean (SD) 2.3 (0.6) cm, non-hemiparetic shoulder, mean (SD) 1.9 (0.3) cm • Intra-rater reliability hemiparetic shoulder, ICC 0.98, non-hemiparetic shoulder ICC 0.95 • Between-day reliability hemiparetic shoulder, ICC 0.94, non-hemiparetic shoulder 0.76 • SEM for both shoulders < 0.2cm • MDC(90), hemiparetic shoulder 0.2, non-hemiparetic shoulder 0.1 cm • Motor power score: <ul style="list-style-type: none"> ○ n = 10, score ≤2 ○ n = 8, score =3 • n = 8, score = 4

Abbreviations: AGT, acromion to greater tuberosity; AP, anteroposterior; CRPS, chronic regional pain syndrome; ETD, electromagnetic tracking device; FIM, functional independence measure; GHJC, glenohumeral joint centre HA, helical axis; HSP, hemiparetic shoulder pain; ICC, intraclass correlation coefficient; kg, kilogram; MDC, minimal detectable change; PROM, passive range of movement; reps, repetition; SCoRE, symmetrical centre of rotation estimation; SD, standard deviation; SEM, standard error of the measure; SI, superoinferior; VAS, visual analogue scale; yrs, years.

2.6 Demographic and clinical characteristics

There is level 1 evidence (National Health and Medical Research Council, 2009) of an association between glenohumeral joint subluxation, HSP and impaired motor recovery and muscle tone (flaccidity, spasticity) in the acute phase post-stroke (Ada & Foongchomcheay, 2002; Kumar et al., 2010). Severity of upper limb paralysis has been identified as a risk factor for subluxation (Kumar et al., 2010) but the evidence is conflicting. In addition, demographic characteristics such as age, gender, side affected by the stroke (left, right) and limb dominance (left, right) (Patrick, Savage and Drača, 2012) have been associated with stroke severity, recovery and the presence of HSP.

2.6.1 Age

As we age, the risk of stroke increases. In 2009, 70% of people who had had strokes were 65 years old or more (Australian Institute of Health and Welfare, 2013). As a result, older people with stroke will also have age related musculoskeletal changes in their glenohumeral joint (Tobola et al., 2009; Turner-Stokes & Jackson, 2002). These age-related changes include degenerative alterations in the joint articular surfaces and soft tissues in and around the glenohumeral joint (Tobola et al., 2009; Turner-Stokes & Jackson, 2002). A reduction in shoulder pain-free range of movement occurs along with weakening of the rotator cuff and surrounding shoulder musculature (Degens & Korhonen, 2012). Rotator cuff thinning, fraying and tears (Yamamoto et al., 2010) as well as calcific deposits in the rotator cuff are common (Turner-Stokes & Jackson, 2002). Inflammation of the subacromial and subdeltoid bursae further exacerbates the narrowing of the subacromial space, increasing the older person's vulnerability to SAIS (Tobola et al., 2009). These changes potentially complicate and contribute to changes in the GHJC, development of HSP and recovery following stroke. As a result, age has been reported to play a significant role in the development of HSP (Aras et al., 2004), possibly due to the age-related structural and morphological changes within and surrounding the glenohumeral joint..

2.6.2 Gender

Stroke is more common in men than women (Islam et al., 2008). An Australian population-based study reported a greater incidence of ischaemic and haemorrhagic stroke in men (approximately 60%) than women (Thrift et al., 2009) and this has been borne out by a recent report by the Australian Institute of Health and Welfare

(Australian Institute of Health and Welfare, 2012). The role of gender on HSP has received some investigation with HSP appearing to be a more frequent occurrence in women than men (Demirci et al., 2007). Interestingly, men are likely to have better upper limb recovery than women (Coupar et al., 2012; Patrick, Savage, & Drača, 2012), though the reason for this is unclear. One possibility may be due to healthy women having less motor control around the glenohumeral joint complex (Ditroilo, Forte, Benelli, Gambarra, & De Vito 2010). This reduced glenohumeral joint complex motor control may be impacted further by stroke impairments, perhaps making the joint more vulnerable to pain, though this has not yet been investigated. Contrary to this, gender has also been found to have no association with functional improvement and motor recovery (Kwakkel et al., 2006).

There is also evidence to suggest interactions between gender and side affected by the stroke (Patrick et al., 2012). Women were more likely to have a stroke affecting the dominant left hemisphere than the right hemisphere and have better rehabilitation outcomes if the left hemisphere was affected; on the other hand, men with strokes affecting the non-dominant right hemisphere were more likely to have better rehabilitation outcomes (Patrick et al., 2012).

2.6.3 Side affected by the stroke

The side affected by the stroke has been found to influence stroke outcomes. HSP is more frequent in people with right hemisphere strokes, that is, those with left hemiparesis, (Demirci et al., 2007; Poulin de Courval et al., 1990). Those with right hemisphere strokes are also more likely to have persistent HSP one year later (Lindgren et al., 2012). Pain may be related to the injured area of the brain (Klit, Finnerup, & Jensen, 2009). People with right hemisphere strokes experience more pain due to altered pain perception following lesions on this side of the brain (Beschin, Cazzani, Cubelli, Della Sala, & Spinazola, 1996; Sterzi et al., 1993). As right hemisphere lesions impact on the non-dominant left upper limb, the other reason that right hemisphere strokes may have more pain in the left shoulder may be due to secondary joint changes resulting from inactivity (Harris & Eng, 2006).

Other differences between people with right and left hemisphere strokes have been found in upper limb motor performance; those with left hemisphere strokes (right hemiparesis) have upper limb movement coordination problems during the movement, while those with right hemisphere strokes have difficulty controlling upper limb

movement endpoints, exhibiting overshooting of the target (Mani et al., 2013). These differences could be in part due to specialisation (a process called lateralisation) of various movement aspects by each cerebral hemisphere (Schaefer, Haaland, & Sainburg, 2009). For example, the dominant left hemisphere controls inter-segmental movement, while the adjusting to changing task dynamics (Duff & Sainburg, 2007; Schaefer et al., 2009) and if damaged, would result in movement trajectory deficits (Mani et al., 2013). This occurs as a result of impaired coordination of the direction of the movement whilst moving toward a target (Duff & Sainburg, 2007; Mani et al., 2013; Schaefer et al., 2009). In comparison with left hemisphere specialisation, the non-dominant right hemisphere controls final limb positioning relative to the target (Duff & Sainburg, 2007; Mani et al., 2013; Schaefer et al., 2009) and if damaged, would result in difficulties gauging movement distances and problems with accurately stabilising the limb at the termination of movement, frequently overshooting the target (Mani et al., 2013).

People with right hemisphere strokes may also have left sided visuospatial deficits (Di Monaco et al., 2011; Vossel, Weiss, Eschenbeck, & Fink, 2013) and impaired awareness of performance (Kottorp, Ekstam, & Petersson Lie, 2013), which limit functional improvement. Impaired reaction to stimulation presented on the left side of the body results in slower functional gains, longer lengths of hospital stays (Cherney, Halper, Kwasnica, Harvey, & Zhang, 2001), while impaired awareness of limitations in activities of daily living limits goal setting and treatment interventions (Kottorp et al., 2013).

2.6.4 Dominant side affected

The hemisphere affected by the stroke also determines arm preference during post-stroke activities (Mani, Przybyla, Good, Haaland, & Sainburg, 2014). During activities involving reaching towards a target, people with dominant left hemisphere strokes (right hemiparesis) used their right arm significantly more than those with non-dominant right hemisphere strokes (left hemiparesis) (Mani et al., 2014). People with strokes in the dominant left hemisphere also had better rehabilitation outcomes than those with strokes in the non-dominant right hemisphere, possibly due to their showing preference in using their dominant right hemiparetic limb during functional tasks (Harris & Eng, 2006; Patrick et al., 2012). These limb choices were displayed by women, with the reverse being true for men i.e. men with left hemiparesis had better rehabilitation outcomes than men with right hemiparesis (Patrick et al., 2012). These findings have been endorsed by another study, which also reported that people with left hemisphere

strokes (right hemiparesis) engaged in bimanual tasks more than people with right hemisphere strokes, but less frequently than healthy controls (Rinehart, Singleton, Adair, Sadek, & Haaland, 2009). It is possible that this finding is due to effect of hand preference; those with left (dominant) hemispheric strokes may need to use both arms in the absence of being able to use their dominant right arm in everyday tasks. In contrast, people with right hemispheric strokes are likely to be more able to use their right unaffected dominant arm (Rinehart et al., 2009).

2.6.5 Motor recovery

Poor motor recovery following stroke is associated with poor shoulder muscle control, increased muscle tone changes and the development of HSP (Adey-Wakeling et al., 2014; Blennerhassett et al., 2010; Kalichman & Ratmansky, 2011). Glenohumeral joint subluxation is associated with impaired shoulder muscle control which has been shown to be a significant risk factor for many post-stroke complications including HSP (Kalichman & Ratmansky, 2011; Smith, 2012), subluxation (Kumar et al., 2010; Paci et al., 2007; Park et al., 2007), changes in muscle tone (Formisano et al., 1993; Formisano et al., 2005; Marciniak, 2011), tendinopathies and rotator cuff tears (Pong et al., 2009; Shah et al., 2008). Severity of initial impairment of motor function was predictive of upper limb recovery (Coupar et al., 2012) and has been correlated with impaired upper limb function within the first month post-stroke (Beebe, 2008). Motor recovery took longer or occurred more slowly in participants with flaccidity than with spasticity (Formisano et al., 2005); flaccidity was significantly more frequent a phenomenon than spasticity (Formisano et al., 1993). Poor motor recovery has also been documented as a risk factor for the development of spasticity (de Jong, Hoonhorst, Stuive, & Dijkstra, 2011); those with poor motor recovery at 48 hours post-stroke had increased odds of 13 times that spasticity would develop at six months post-stroke than those with moderate or good motor recovery (de Jong et al., 2011). It is likely that those with good motor recovery will have fewer complications post-stroke, while poor motor recovery is associated with worse shoulder pain, increased levels of subluxation, impingement, spasticity and functional dependency (Kalichman & Ratmansky, 2011).

2.6.6 Muscle tone

In a clinical setting, changes in muscle tone are described in terms of spasticity and flaccidity. However, spasticity is used to describe two main clinical signs, namely hyperreflexia, defined as “a velocity dependent increase in the tonic stretch reflexes

(muscle tone) with exaggerated tendon jerks, resulting from the hyperexcitability of the stretch reflex” (Lance, 1980, p. 487-489), and hypertonia, defined as “perceived resistance to lengthening of muscles by passive movement” (Carr & Shepherd, 2003, p. 215). Hypotonia (flaccidity) is defined as the lack of resistance to passive movement (van der Meche & van Gijn, 1986). Changes in muscle tone during this research project will refer to the range of muscle tone changes from ‘hypertonia’ or spasticity, to ‘hypotonia’ or flaccidity. Resistance to passive movement at fast and slow speeds will be measured using the Tardieu scale during this research project (Haugh, Pandyan, & Johnson, 2006; Morris, 2002).

A recent systematic review summarised the timing of spasticity onset as being extremely variable (within a very short time post-stroke or more than 12 months later). Our understanding of spasticity is affected by the development of contractures which may be thought to result from spasticity, but may be involved in its cause (Ward, 2012). Potential predictors for spasticity post-stroke include limb weakness, particularly of the left side (Ward, 2012). In addition, people with increased spasticity post-stroke are more likely to have reduced function (Marciniak, 2011; Ward, 2012) and quality of life (Marciniak, 2011). The prevalence of spasticity is up to 43% in the acute phase post-stroke (Dvorak, Ketchum, & McGuire, 2011; Urban et al., 2010), though only 16% are severely affected and spasticity is more severe in the upper limbs (Urban et al., 2010). These rates drop to 33% (Kong, Lee, & Chua, 2012) and 19% (Sommerfeld, Eek, Svensson, Holmqvist, & von Arbin, 2004) in the subacute phase following stroke. Spasticity has been linked to HSP and changes in glenohumeral joint position (Kalichman & Ratmansky, 2011). Changes in scapulohumeral rhythm caused by spasticity may cause soft tissue damage and hence potentially contribute to HSP development (Singh & Fitzgerald, 2011), though to date, no studies have investigated its link with changes in GHJC. Flaccidity has also been linked with more severe motor deficits (Formisano et al., 2005) and biomechanical changes at the shoulder (Jackson et al., 2002). This has been previously discussed in Section 2.3 (Adey-Wakeling et al., 2014; Blennerhassett et al., 2010).

2.7 Summary

In summary, hemiparetic shoulder pain is common in people who have suffered a stroke. Aetiology is uncertain, and clearly multifactorial. Many changes in the post-stroke shoulder are evident, some due to the stroke itself and some due to the demographic and clinical characteristics of the person experiencing the stroke. Factors including age, presence of glenohumeral joint subluxation, changes in neuromotor

control of the shoulder complex, musculoskeletal impairments and positional changes within the glenohumeral joint have been identified in people with hemiparetic shoulder pain.

Much of the evidence underpinning our knowledge of HSP has come from investigations conducted in people in the sub-acute and chronic phases following stroke. Muscle tone changes, motor weakness and other sensorimotor changes are evident in the acute phase following stroke. These changes are likely to impact on the position of the GHJC even in the acute phase but this has not been explored. Changes in the glenohumeral joint centre position must be explored in context with respect to age, gender and side affected by stroke; therefore comparisons should be made with healthy adults with similar demographics. Possible links between biomechanical changes, motor recovery and shoulder pain in the acute phase have also not been established.

2.8 Thesis aims and objectives

The aims of this study were to measure positional changes of the glenohumeral joint in the early post-stroke phase, compare the position of the glenohumeral joint in people with an acute stroke to a healthy matched control group, and assess the impact of demographic and clinical characteristics on glenohumeral joint position in people with acute stroke.

Specific aims of this project are to:

1. Determine the reliability of an electromagnetic tracking device to measure the position of the glenohumeral joint centre in people within two weeks of stroke onset and in a healthy population.

Objective: To perform an intra-tester reliability study on people within two weeks of stroke onset and on an age, gender and BMI matched healthy group of people.

2. Compare the glenohumeral joint centre position between the hemiparetic and non-hemiparetic sides in people at two weeks of stroke onset.

Objective: To determine the position of the glenohumeral joint centre on the hemiparetic and non-hemiparetic sides in people with an acute stroke.

3. Assess changes in the glenohumeral joint position over time (i.e. within the first six weeks) in people with an acute stroke.

Objective: To determine the change in glenohumeral joint centre position within the first six weeks in people with an acute stroke.

4. Compare the glenohumeral joint centre position in people with an acute stroke and a healthy population matched for age, gender and body mass index.

Objective: To compare the position of the glenohumeral joint centre position in people with an acute stroke to healthy people matched for age, gender and body mass index.

5. Examine the influence of demographic and clinical characteristics of age, gender, side affected by the stroke, dominance, shoulder pain, motor recovery and muscle tone on the glenohumeral joint centre position in the first six weeks following stroke.

Objective: To determine the influence of demographic and clinical characteristics on age, gender, side affected by the stroke, dominance, shoulder pain, motor recovery and muscle tone on the position of glenohumeral joint centre within the first six weeks following stroke.

Chapter 3

General methods

3.1 Overview

This chapter describes the methods used during the research program presented in this thesis. The longitudinal observational study design investigated the position of GHJC using an ETD and explanatory variables which included pain, motor recovery and muscle tone. Acute stroke unit participants were recruited within two weeks of stroke onset and age, gender and BMI matched with healthy control participants. Nine acute post-stroke participants and age-, gender- and BMI-matched healthy controls were involved in a reliability study to ensure that the protocol used to measure GHJC throughout the study had intra-tester reliability. The position of GHJC was assessed within two weeks of stroke onset in 30 acute post-stroke participants and their matched healthy participants. Stroke participants had the protocol repeated six weeks later while the healthy control participants' data were carried forwards for comparison. Assessment procedures and protocol will be detailed and sample size calculations and ethical considerations outlined. Methods used to process and analyse GHJC data are presented. Demographic and clinical characteristics including pain, motor recovery and muscle tone were explored relative to GHJC position.

3.2 Participants

Two groups of participants were recruited for this program of research; acute post-stroke participants and age-, gender- and BMI-matched healthy controls (mean age paired differences 65.6 years SD 1.1; mean BMI paired differences 24.8 kg/m² SD 0.7). Stroke participants for this program of research were recruited from patients admitted consecutively to the Royal Brisbane and Women's Hospital and Brighton Health Service for eleven months from May 2011. The Royal Brisbane and Women's Hospital is the largest general tertiary referral teaching hospital in Queensland with 987 beds. It boasts a full multi-disciplinary health team and offers a range of specialties. Services extend to the entire State, northern New South Wales, the Northern Territory and countries in the South West Pacific region. The hospital has a throughput in excess of seven hundred and eighty strokes per annum. One participant was recruited from the Brighton Health Service, which is a north side metropolitan rehabilitation facility in Queensland.

Potential stroke participants were identified by physiotherapists working on the acute stroke unit and medical wards of the Royal Brisbane and Women's Hospital, the Prince Charles Hospital and the subacute ward of the Brighton Health Service and subsequently referred to the study investigator (PC). Medical records of potential participants were screened to ascertain suitability for the study. An information sheet

and a consent form were provided and those individuals, who demonstrated interest in volunteering, were verbally screened for inclusion in the study via a standardised questionnaire (Appendix 2). Potential participants also had the opportunity to ask questions regarding their involvement in the study. Eligible volunteers were then required to provide written informed consent and arrangements for the initial assessment session were made, which was no later than 14 days post-stroke.

Control participants were recruited via advertisements to staff at the Royal Brisbane and Women's Hospital and Griffith University and to the general community via external advertising. This included advertisements via email, on community notice boards and in person to community groups such as Senior Citizens and Bowls Clubs. Those interested in participating who contacted the primary investigator (PC) were verbally screened over the phone for inclusion in the study via the same standardised questionnaire. A mutually convenient appointment was made where additional screening was undertaken, the opportunity to ask questions regarding involvement in the study and an information sheet and consent form were provided. Written informed consent was gained from all control participants (Appendix 2).

Inclusion and exclusion criteria

To be eligible for inclusion in these studies, stroke patients:

- experienced their first stroke within the previous 14 days,
- were medically stable, that is vital signs such as body temperature and blood pressure have remained stable for 24 hours (Duncan et al., 2005)
- were able to give written consent regarding their participation in the study and understand instructions,
- had initial upper limb impairment as a result of the stroke and
- were able to sit continuously for a minimum of 30 minutes with minimal support and for a total of 90minutes within a 120 minute session, and

Potential stroke participants were excluded if they:

- reported any pain either at rest or movement which would impact on measurements being recorded,
- a history of previous shoulder pain, shoulder surgery, any neuromusculoskeletal disorder involving the upper limb,
- a clinically apparent scoliosis which would affect the measures of scapular orientation,
- any metallic implants,

- a delicate or reactive skin which would be at risk of damage by use of adhesive tape

Inclusion and exclusion criteria for control participants were identical except for criteria relating to the stroke.

3.3 Study protocol

For stroke participants demographic and clinical information was obtained from the hospital's medical record. This included date of birth, gender, date, type and side of stroke, upper limb dominance, height and weight. Demographic information obtained from control participants included age, gender, dominance, height and weight. BMI for each participant was calculated from the weight and height data ($BMI = \text{weight}/\text{height}^2$).

Stroke participants underwent assessment during two sessions; baseline, and six weeks follow-up. Control participants underwent one assessment session. During each session, the following measures were recorded in this order: GHJC during rest and passive shoulder movement, pain, motor function of the affected shoulder and upper limb muscle tone. This order ensured that the primary measure of GHJC was completed for all participants. Participants were provided with rest breaks as required. For each assessment, measurements were completed within one session. Prior to commencing testing, participants were required to remove all metal items of clothing and jewellery, including, watches, coinage and metal in clothing (bra clip, jeans buttons). The trunk, shoulders and arms were exposed for the testing procedure and draped appropriately to maintain modesty/ keep warm while preparing equipment. All measures were recorded by the primary investigator (PC). A second investigator was present when required to assist with transfers of stroke participants onto and off the customised seat, the maintenance of the participant's position during measurement and to save data files on the computer. The following outcome measures were recorded.

3.3.1 Shoulder pain

Participants were questioned regarding the presence of pain on both the hemiparetic and non-hemiparetic sides for the shoulder and neck. A horizontal visual analogue scale was used to measure pain, with anchors of 0 mm representing no pain and 100 mm representing the worst pain imaginable (Carlsson, 1983) (Appendix 3). Participants were asked to rate their level of shoulder pain:

- I. at rest
- II. with passive shoulder movement
- III. with active shoulder movement

In addition, the severity of neck pain experienced during neck movement was also scored using the visual analogue scale. Neck pain was determined due to its close anatomical relationship with the shoulder and the frequency of neck pain referring to the shoulder (Fish, Gerstman, & Lin, 2011). Any reported upper limb or neck pain was plotted on a pain map (Appendix 4). Participants were asked to report any pain experienced in the stroke affected shoulder and the date of onset and any precipitating events were documented.

3.3.2 Motor recovery

Functional motor recovery of the stroke affected shoulder was assessed using the upper arm item (item 6) of the Motor Assessment scale (MAS) (Carr, Shepherd, Nordholm, & Lynne, 1985; Loewen & Anderson, 1988). The MAS is a battery test developed for stroke patients, assessing 8 motor items, each scored on a 0-6 scale. Higher scores indicate a greater level of independence, quality of movement pattern and complexity of tasks completed. The items include: rolling, lie to sit, sitting balance, sitting to standing, gait, upper arm function, hand movements and advanced hand activities. The MAS has established intra- ($r = 0.98$) and inter-tester ($r > 0.95$) and test-retest ($r = 0.98$) reliability (Carr et al., 1985; Kjendahl, Jahnsen, & Aamodt, 2005; Loewen & Anderson, 1990). Please refer to Appendix 5 for details of item 6 of the MAS. Participants were categorised according to the level of motor recovery by MAS score. Those who scored less than 6, were classified as having poor motor recovery and those who scored 6 were classified as having achieved good motor recovery.

3.3.3 Muscle tone

Muscle tone in the upper limbs was measured using the Tardieu Scale (Haugh et al., 2006). This tool assesses and compares the response of a muscle to passive movement at both slow and fast speeds. The stretch reflex within the muscle being tested is elicited during the fast speed passive movement. During this study, the assessor moved each major upper limb muscle group passively, with the limb in a position which lengthens the muscle fully through its maximum available range at slow speed. Maximum passive range was recorded and each muscle group moved in the

same direction as previously as fast as possible. The resistance detected during the movement or the joint angle at which a catch in the muscle was first felt was recorded (quality of the muscle reaction).

Quality of the muscle reaction was recorded on a scale of 0-4, with 4 being maximum resistance of the muscle and 0 being nil. This scale has previously been shown to be sensitive to change (Mehrholtz et al., 2005; Parker, Wade, & Langton Hower, 1986), and valid (kappa for elbow flexors = 1.0) (Patrick & Ada, 2006) and reliable (intra-tester reliability, ICC for elbow flexors = 0.72, inter-tester reliability, $Z > 1.96$, $p < 0.05$) (Mehrholtz et al., 2005). Although good quality evidence of validity and reliability for any clinical measure of spasticity is lacking (Malhotra, Pandyan, Day, Jones, & Hermens, 2009) (Appendix 6).

3.3.4 Glenohumeral joint centre (GHJC)

3.3.4.1 Instrumentation

Glenohumeral joint centre (GHJC) was measured using an electromagnetic tracking device (ETD) (Meskers et al., 1998; Mills, Morrison, Lloyd, & Barrett, 2007). A Polhemus Liberty HST 2000 3D electromagnetic tracking device (ETD) (Figure 1) was used to measure the 3-D position and orientation of sensors attached to the upper arm and trunk as the upper arm was moved passively through a movement sequence. This study used the standardised measurement protocol established by the International Society of Biomechanics (Wu et al., 2005). The protocol has been previously validated in a younger population (Meskers et al., 1998; Mills et al., 2007) as well as in a subacute stroke population (Niessen et al., 2008).

The device has three components: a control box which senses the magnetic field, processes the signals sent by the sensors and communicates with the computer (Figure 1a); an emitter (source) which emits low frequency magnetic field signals and is mounted on a stand/tripod (Figure 1b); and sensors which detect the electromagnetic signals emitted by the emitter. The outputs from the sensors provide their 3D position and orientation relative to the emitter. Sensors can be attached to body segments (Figure 1c) or embedded into a stylus to be used in accurate identification of anatomical landmarks (Figure 1d).

The electronics unit controls the intensity of a magnetic field and automatically adjusts it when distances between the emitter and the sensors change, so that the strength of

the field reaching the sensor remains at a constant level. The electromagnetic tracking system is affected by metal conductors, so the manufacturer recommends that metal objects be kept at least one metre from the source and sensors. PI Manager (Version 2.3) (Polhemus Incorporated, Colchester, USA) software was used to collect all ETD data.

Figure 1. Electromagnetic tracking device parts and sensors, (a) Electronics unit and sensors of Polhemus Liberty HST 2000 3D ETD, (b) ETD mounted on its stand, (c) Four ETD sensors attached to the upper sternum, inferior to the deltoid muscle insertion on the humerus, the lateral epicondyle and the acromion process, (d) The stylus sensor.

3.3.4.2 Participant set up for GHJC

Participants were required to have all upper-body clothing and metallic jewellery removed. They were then seated in a custom-built, height-adjustable, non-metal chair with a low back rest (Figure 2a), in a posture with their back straight, hips and knees flexed to 90° and shoulder and scapula exposed (Figure 2b).

Figure 2. Custom-built seat and participant position during the measurement of GHJC, (a) height adjustable, non-metal chair with low back rest, (b) participant seated with back straight, hips and knees flexed to 90 degrees and scapulae exposed.

Anatomical landmarks (Table 5) for ETD sensor attachment (four) were identified and marked with a pen (Figure 3). A fifth sensor was embedded in a stylus and used to calibrate the anatomical landmarks on the upper arm and trunk (Table 6). All sensors were attached using hypoallergenic tape (Fixomull) to marked landmarks (Figure 4).

Table 5. Anatomical landmarks for the electromagnetic tracking device (ETD) sensor attachment positions, sensor names and abbreviations

Description	Name	Abbreviation
Ventral surface of the upper sternum	Trunk sensor	St
Upper arm just below the Deltoid muscle insertion	Humeral sensor	Sh1
As close as possible to the lateral epicondyle	Humeral sensor	Sh2
Flat aspect of the acromion	Acromial sensor	Sas

Figure 3. Anatomical landmarks, (a) on the right posterior aspect of the trunk: acromial angle (AA), 7th cervical vertebra (C7), 8th thoracic vertebra (T8), (b) on the anterior aspect of the trunk: suprasternal notch (IJ) and the xiphoid process (PX), (c) on the right upper limb: the deltoid insertion, the lateral epicondyle (EL) and the medial epicondyle (EM).

Table 6. Description, names and abbreviations of anatomical landmarks for electromagnetic tracking device calibration using the stylus (fifth sensor).

Description	Name	Abbreviation
Deepest point of the Incisura Jugularis	Supra-sternal notch	IJ
Most caudal point on the sternum	Xiphoid process	PX
Spinous process of the 7th cervical vertebra	C7 spinous process	C7
Spinous process of the 8th thoracic vertebra	T8 spinous process	T8
Most caudal point on the lateral epicondyle	Lateral epicondyle	EL
Most caudal point on the medial epicondyle	Medial epicondyle	EM

Figure 4. Attachment of four electromagnetic sensors, (a) Trunk sensor (St), attached to the upper sternum, (b) Acromial sensor (Sas), attached to the flat aspect of the acromion, the humeral sensor Sh1 attached inferior to the deltoid insertion and the humeral sensor Sh2, attached to the lateral epicondyle.

The height of the chair was adjusted to ensure that the acromion process of the scapula was at least 1.0 m above the floor to avoid potential interference from metal reinforcement within the concrete. The ETD transmitter was positioned approximately 300 mm posterior to the sagittal mid-line of the participant at the height of the sternal notch (Figure 2b). A hemisphere was marked on the floor, with the origin directly inferior to the acromion process of the scapula, and the hemisphere extending to the side of the shoulder being tested. The radius of the hemisphere was approximate equal to the anterior distance projected from the acromion to the fingertips when the

participants arm was in pure flexion of 45 degrees. Targets were placed at 45 degree increments along the circumference of the hemisphere to ensure consistency of arm movement across all the measures. A mirror was placed anterior to the participants to enable them to provide a view of the arm and targets during movements towards the posterior targets.

3.3.4.3 Measuring process for GHJC

The glenohumeral joint centre (GHJC) was estimated using a functional sphere-fitting approach similar to that described by Meskers and colleagues (Meskers et al., 1998; Mills et al., 2007) using the joint coordinate system for the glenohumeral joint recommended by the International Society of Biomechanics . Data were collected at 240 Hz using an eight-channel Polhemus Liberty ETD system in conjunction with PI Manager software (Version 2.3) (Polhemus Incorporated, Colchester, USA). All participants underwent anatomical and passive calibration trials. During the anatomical calibration trial, the stylus was sequentially placed on each of the landmarks listed in Table 6 for 5 seconds. For the passive calibration trial, the upper arm was moved passively by the investigator in the following pattern of movements starting with the arm resting by the side (Figure 5a). The arm was passively elevated to 45 degrees and returned to the resting position through the following planes of movement:

- Anterior sagittal plane (flexion, Figure 5b)
- Mid-way between sagittal and frontal plane anteriorly (Figure 5c)
- Frontal plane (Figure 5d)
- Mid-way between frontal plane and sagittal plane posteriorly (Figure 5e)
- Posterior sagittal plane (extension, Figure 5f).

Figure 5. Recording shoulder passive calibration trial; (a) start position, (b) elevation in the anterior sagittal plane, (c) mid-way between sagittal and frontal planes anteriorly (d) frontal plane (e) mid-way between frontal and sagittal planes posteriorly (f) posterior sagittal plane.

Finally the participant's upper arm was moved through an arc of circumduction in 45 degrees elevation commencing in the resting position (arm resting by the side), progressing to sagittal plane anterior (flexion to 45 degrees) then sagittal plane

posterior (extension to 45 degrees) and finishing in the resting position. The process was then repeated for the contralateral limb.

3.4 Data processing for GHJC

Data processing was performed using custom software written in Matlab programming language (Mathworks Inc.; Natick, MA). The software was used to extract and calculate position and angle data. Sensor data were filtered using a zero-lag second order Butterworth low-pass filter with a cut off frequency of 2 Hz. The locations of anatomical landmarks within their respective segmental coordinate systems were defined by firstly transforming anatomical landmarks positions from the anatomical calibration trials (i.e., the stylus tip) from the stylus coordinate system to the emitter coordinate system, and then transforming the anatomical landmarks from the emitter coordinate system to the segmental coordinate system, defined by the sensor attached to the respective segment. The GHJC was then estimated using a sphere-fitting approach (Leardini et al., 1999; Lempereur et al., 2013) in which the variance in distance between the humeral sensors and the GHJC throughout the passive calibration trial was minimized using unconstrained optimization. The initial guess for the location of GHJC was 0.02 m inferior, 0.02 m anterior and 0.02 m lateral to the acromion.

The optimization routine was iterated five times with the x, y and z components of the initial guess randomly perturbed by a constant between 0 and 0.1 m. The best estimate of the GHJC was then transformed to correspond with its position relative to the acromion coordinate system in the seated anatomical calibration trial; the outcome variables being the anteroposterior (AP) (y-coordinate) and superoinferior (SI) (z-coordinate) position of the GHJC relative to the acromion.

The three dimensional position of the sensors were used to calculate GHJC. Glenohumeral joint centre has been reported in this study via the AP (y-coordinate) and SI (z-coordinate) positions. The AP and SI positional data were transformed to a right handed coordinate system so that the GHJC relative to the acromion could be reported in a consistent manner. Data for the right side anteroposterior position (y-coordinate) were multiplied by negative one, while data for the left side superoinferior position (z-coordinate) were multiplied by negative one. Positive-y was designated as being positioned anterior to the acromion, y-negative as being positioned posterior to the acromion, z-positive as being superior to the acromion and z-negative as being inferior to the acromion.

Control participants were assigned the same hemiparetic side as the matched stroke participant. If the stroke participant was right hemiparetic, the control participant's right side was assigned as the designated hemiparetic side for purposes of comparison.

3.5 Data analysis

All data were entered into an Excel spreadsheet (Microsoft, USA), and then analysed using a statistical software package (SPSS Statistics software V21.0, IBM, Chicago USA). Alpha levels were set at $p < 0.05$.

3.5.1 Reliability analysis

The reliability of the ETD to measure GHJC in a stroke population has not previously been investigated. This measure has established intra- and inter-tester reliability in young healthy adults (Meskers et al., 1998; Stokdijk et al., 2000). Intra-tester reliability of measuring GHJC during passive movement using an ETD was determined in acute stroke participants within 14 days post stroke and age, gender and body mass index (BMI) matched control participants. Participants able to complete three valid repeated measures of GHJC position at baseline for reliability of GHJC measures were identified and used in the reliability analysis.

Reliability of the GHJC measures for each group (Stroke, Control) were calculated for the AP and SI axes from the three baseline trials and for each side (hemiparetic, non-hemiparetic). To calculate reliability, intraclass correlation coefficients (ICC)_(3,k) (Portney & Watkins, 2009, p. 590; Shrout & Fleiss, 1979) and 95% confidence intervals were used. Intraclass correlation coefficients of 0.81 to 1.0 were considered to be good correlation, 0.61 to 0.80 were considered to be moderate and those below 0.60 were considered to be poor correlation (Shrout, 1998). Secondly, the standard error of the measure (SEM = standard deviation* $\sqrt{1-ICC}$) was calculated in millimetres in order to quantify measurement error which could be used to distinguish error from clinically significant change in GHJC (Bialocerkowski, Klupp, & Bragge, 2010). Minimal detectable change ($MDC_{95} = 1.96*\sqrt{2*SEM}$) was calculated for each GHJC direction (Bialocerkowski et al., 2010), to determine with 95% confidence whether the differences in GHJC measures between sides and groups identified in subsequent analyses were greater than the measurement error (Portney & Watkins, 2009, p. 646).

3.5.2 Baseline GHJC analysis

To assess GHJC positions in each direction (AP and SI) on the hemiparetic and non-hemiparetic sides in the stroke group and on matched hemiparetic and non-hemiparetic sides in the healthy control participants at baseline, descriptive statistics were used to calculate the means and standard deviations for each GHJC position. To assess between-limb comparisons of GHJC positions in each direction on hemiparetic and non-hemiparetic sides in stroke participants and right and left sides in the healthy control participants, general linear measures of mean GHJC positions were used. To assess and compare GHJC positions in both directions between strokes and controls at baseline, general linear measures were used.

3.5.3 Week six GHJC analysis

Descriptive analysis was completed at week six to ensure that the stroke and control groups remained statistically comparable following any loss-to-follow-up of stroke participants. To assess the stroke group's between-limb comparison of GHJC position at week six general linear measures using means of three trials of GHJC measures for each side (hemiparetic and non-hemiparetic) at week six were conducted. To assess and compare GHJC position in both directions (AP and SI) between strokes and controls (carried forwards from baseline) at week six, general linear measures were used. Repeated measures ANOVA of mean GHJC measures at each session were used to compare baseline measures with those at week six on the hemiparetic and non-hemiparetic sides.

3.5.4 Demographic and clinical measures

All variables were checked for normality. For non-normally distributed variable, data were analysed using non-parametric analyses. Normally distributed data were analysed using parametric analyses. To ensure that demographic characteristics for age, gender and BMI for the matched stroke and control groups did not differ statistically, descriptive statistics (means, standard deviations or mediate range) were completed. Univariate analyses were used at baseline and week six to compare subgroups based on median age ($\geq$ or $<$ 68 years), gender (men, women), side affected by the stroke (right, left), whether the dominant side was affected by the stroke (yes, no), whether pain was present on the hemiparetic side (yes, no), motor recovery scores ($= 6$, < 6) and the presence of muscle tone changes (no, yes).

3.6 Sample size

Apriori sample size was calculated based on data output from GPower 3.1 (Faul, Erdfelder, Buchner, & Lang, 2009). No studies were found that reported GHJC in stroke survivors or healthy older adults. Kumar and colleagues (Kumar et al., 2011) reported a minimum detectable change (MDC) in the distance between the lateral acromial border and the apex of the greater tuberosity of the humerus of 2.0 mm, measured using portable ultrasound. Sample size calculations, based on an MDC of 2.0 mm (no SD available) (Kumar et al., 2011), effect size 0.3 to account for minimal detectable change in the GHJC between stroke and controls and stroke survivors over time, power of 0.80 and alpha set at 0.05, gave a sample size of 24 participants. Allowing for 25% attrition in this sample of acute stroke participants who, with their extensive co-morbidities and risk of subsequent stroke, could potentially be lost to follow-up, 30 participants were recruited. Thirty age, gender and BMI matched healthy participants were required to make up the control group.

3.7 Ethical considerations

This research study aimed to improve our understanding of biomechanical changes which may occur in the glenohumeral joint following stroke and the association these changes may play in the development of hemiparetic shoulder pain. An improved understanding may assist in the development of effective interventions to prevent and treat this debilitating condition, thereby potentially improving functional upper limb ability and independence for the individual. Improved upper limb function and independence may enable individuals to interact with and contribute more substantially to their community. Furthermore, the development of efficacious interventions, decrease reliance on health care services for support and equipment will significantly reduce the cost of stroke to carers and the broader community.

The potential significance of this research was established during the ethical clearance process undertaken at the Royal Brisbane and Women's Hospital, Prince Charles Hospital and Brighton Subacute Service from which participants were either recruited or followed up. Ethical clearance was also gained from Griffith University as the overseeing institution. Please refer to Appendix 1 for copies of Ethical Clearances from all institutions. All participants were required to provide written informed consent prior to participation.

Chapter 4

Study results

4.1 Reliability of glenohumeral joint centre measurement

4.1.1 Participants

Thirteen participants with acute stroke were eligible for inclusion in this study. Two participants did not have a complete dataset (i.e. no data for the non-hemiparetic side) and were excluded from the reliability analyses. In addition, two other participants had missing data points, due to errors during data collection and/or processing (one trial on the hemiparetic side and two trials on the non-hemiparetic side), leaving nine participants with three valid trials of GHJC measures on both sides, for inclusion in reliability analyses. Stroke and control participant characteristics for the reliability analyses are available in Table 7. There were no significant differences in age ($p = 0.96$) or BMI ($p = 0.71$) between the groups.

4.1.2 Within-session intra-tester reliability

GHJC means, Intraclass correlation coefficients (ICC) with 95% confidence intervals (CI) for average measures, SEM and MDC for the AP and SI directions of GHJC position in the hemiparetic and non-hemiparetic shoulders are presented in Table 8. There was excellent intra-tester reliability (ICCs > 0.8) for all measures for both stroke and control participants, except for the assigned non-hemiparetic side in the control participants (ICC 0.78). Maximum SEM and MDC measures for stroke participants were 9.4 mm and 26.0 mm respectively. Greater variability was recorded for the control group with wide 95% confidence intervals, maximum SEM 15.3 mm and MDC 42.4 mm (Table 8).

Table 7. Participant characteristics for the study assessing the reliability of the GHJC using an ETD-based functional sphere-fitting technique

Characteristic	Stroke N = 9	Control N = 9
Age, years, mean (SD)	65.1 (15.2)	66.2 (16.0)
Gender, n (%) females	2 (22.2)	2 (22.2)
Side affected by stroke		
Right, n (%)	6 (66.7)	N/A
Dominant side affected, yes, n (%)	8 (88.9)	N/A
Dominance		
Right, n (%)	7 (77.8)	8 (88.9)
Body mass index, kg/m ² , mean (SD)	27.1 (4.1)	26.8 (4.1)
Time post stroke at baseline, days, mean (SD)	4.0 (2.6)	N/A
Type of stroke, Ischaemic n (%)	9 (100)	N/A

Table 8. Means, intraclass correlation coefficients (ICC), 95% confidence intervals (CI), standard error of measurement (SEM) and minimal detectable change (MDC) for glenohumeral joint centre position for stroke and control groups at baseline.

		Strokes		Controls	
GHJC position	Measure	Hemi N = 9	Non-hemi N = 9	Hemi* N = 9	Non-hemi* N = 9
Anteroposterior	Mean (SD) (mm)†	-0.9 (9.4)	4.0 (5.7)	5.1 (13.3)	-0.8 (25.4)
	ICC (95% CI)	0.97 (0.90 to 0.99)	0.99 (0.78 to 0.98)	0.80 (0.36 to 0.95)	0.99 (0.97 to 1.00)
	SEM (mm)	3.3	1.1	11.8	5.0
	MDC (mm)	9.2	3.1	32.7	13.8
Superoinferior	Mean (SD) (mm)‡	-22.0 (15.6)	-5.4 (13.8)	-26.3 (23.7)	-27.9 (16.7)
	ICC (95% CI)	0.99 (0.96 to 1.00)	0.88 (0.62 to 0.97)	0.91 (0.73 to 0.98)	0.78 (0.31 to 0.95)
	SEM (mm)	3.6	9.4	13.7	15.3
	MDC (mm)	10.0	26.0	38.0	42.5

*Control sides matched to hemi- and non-hemi side from the stroke group

†Negative scores indicate centre of rotation axis sits posterior to the acromion

‡Negative scores indicate centre of rotation axis sits inferior to the acromion

Hemi, Hemiparetic; Non-hemi, Non-hemiparetic.

4.2 Case-control and longitudinal results

4.2.1 Participants

One hundred and ninety-three people admitted with a confirmed diagnosis of an acute stroke were screened for inclusion into this study. One hundred and forty-nine people were excluded (Figure 6), including one participant whose BMI was 37.1, due to the possibility of the skin-mounted sensors being susceptible to skin movement artefact (Hamming et al., 2012). Reasons for exclusion are detailed in Figure 6. Forty-four people were eligible for inclusion in the study. Nine declined consent resulting in 35 people with stroke who were recruited. Of these, 30 were age, gender and BMI-matched with healthy control participants and enrolled in the study (Figure 6).

Participant characteristics (n = 30) are reported in Table 9. Twelve participants were male (40%) aged between 35 and 82 years, with a mean age of 62.4 years (SD 15.9). Participant comorbidities included the following diagnostic categories: hypertension 66.6%, cardiovascular 43.4%, medical 53.3%, orthopaedic 30%, pulmonary 20%, surgical 23.3% and non-insulin dependent diabetes 16.7%.

Twenty-three participants were followed up at week 6, with 23.3% lost to follow-up. Reasons for the loss to follow-up included one death due to medical complications, two participants being medically unwell and four declining further participation (Figure 6).

Forty-five control participants were recruited, 30 of whom were age-, gender and BMI-matched with stroke participants (Figure 6) and were included in this program of research (Table 9). Twelve were males (40%) aged between 34 and 82 (mean 63.4 years, SD 16.1).

Figure 6. Flow of participants through the case-control and longitudinal studies

Table 9. Participant characteristics for the case-control and longitudinal studies*.

Characteristic	Stroke N = 30	Control N = 30
Age, years	65.1 (19.0)	65.3 (18.2)
Women	66.9 (21.0)	66.5 (19.9)
Men	62.4 (15.9)	63.4 (16.1)
Gender, n (%) females	18 (60)	18 (60)
Side affected by stroke		
Right, n (%)	12 (40)	
Dominance		
Right, n (%)	25 (83.3)	29 (96.7)
Dominant side affected by stroke		
Yes, n (%)	15 (50)	
Body mass index, kg/m²	25.1 (4.0)	24.8 (4.0)
Time post stroke, days	6.9 (4.2)	
Type of stroke, Ischaemic, n (%)	28 (93.3)	
Hemiparetic shoulder pain on passive movement, n (%)	10 (33.3)	
Women, n (%)	8 (26.7)	
Hemiparetic shoulder pain severity, visual analogue scale (0-100 mm)		
At rest	2.6 (8.8)	
During passive shoulder movement	18.6 (31.0)	
During active shoulder movement (unable to move actively n=4)	7.2 (16.6)	
During neck movement	0.9 (3.8)	
Motor assessment scale Item 6, /6	4.3 (2.2)	
Score = 6, n (%)	14 (46.7)	30 (100)
Score < 6, n (%)	16 (53.3)	
Muscle tone (Tardieu), n (%)		
No tonal changes	24 (80.0)	
Tonal changes present	5 (16.7)	
Unable to assess (fatigue)	1 (3.3)	

* Data is reported as mean (standard deviation) unless otherwise indicated

4.2.2 GHJC position in matched strokes and controls at baseline

All baseline measurements were completed within one session, with the exception of two participants who were measured over two consecutive days due to fatigue or the interruption of medical investigations. Data collection and/or data processing errors resulted in 27 stroke participants' data being available for analysis of GHJC position in the anteroposterior (AP) and superoinferior (SI) directions on the hemiparetic and non-hemiparetic sides. Glenohumeral joint centre position in the AP and SI directions for the matched strokes and controls at baseline are presented in Table 10.

4.2.2.1 Comparison between hemiparetic and non-hemiparetic sides

The GHJC in the AP direction was positioned 4.0 mm (95%CI 0.3 to 7.7) more anteriorly on the non-hemiparetic side compared to the hemiparetic in the stroke group ($p = 0.036$; $F_{(1, 26)} = 0.654$) at baseline. There was no significant difference in GHJC position in the SI direction between sides in the stroke group ($p = 0.56$; $F_{(1, 26)} = 1.205$). Regardless of side, the average position of the GHJC in the SI direction was 17.0 mm (95%CI 19.9 to 14.1) inferior to the acromion in the stroke group. In the control group there were no significant differences in GHJC position between sides, in either the AP ($p = 0.95$; $F_{(1, 29)} = 0.003$) or the SI ($p = 0.34$; $F_{(1, 29)} = 0.95$) directions (Table 10). Regardless of side, the average position of the GHJC was 1.6 mm (95%CI -2.0 to 5.2) anterior to, and 23.8 mm (95%CI -30 to -17.6) inferior to the acromion in the control group.

4.2.2.2 Comparison between stroke and control groups

The between-group differences for hemi and non-hemi sides in each direction of GHJC are reported in Table 10. There were no significant interaction ($p=0.485$; $F_{(1, 55)} = 0.494$) or main effects for Group (Control versus Stroke; $p = 0.938$; $F_{(1, 55)} = 0.006$) or Side (hemi versus non-hemi; $p=0.422$; $F_{(1, 55)} = 0.654$) in the AP direction at baseline. Similarly, there was no significant interaction ($p = 0.798$; $F_{(1, 55)} = 0.66$) or main effect for Side ($p = 0.277$, $F_{(1, 55)} = 1.205$) in the SI direction at baseline. However, the main effect for Group ($p = 0.056$, $F_{(1, 55)} = 3.799$) approached significance. Regardless of side, the stroke group GHJC was positioned an average of 6.8 mm (95%CI -0.2 to 13.7) closer to the acromion (SI direction) than the control group.

Table 10. Means and standard deviations for GHJC anteroposterior and superoinferior positions at week 0 and week 6 and mean differences with 95% confidence intervals (CI) between matched stroke and control participants between week 0 and week 6 and changes within stroke group from week 0 to week 6.

GHJC position	Stroke group Mean (SD) (n = 27)			Control group Mean (SD) (n = 30)			Difference in GHJC position between control and stroke groups Mean difference (95% CI)	
	Hemi	Non-hemi	Mean difference (95%CI)	Hemi [†]	Non-hemi [†]	Mean difference (95%CI)	Hemi	Non-hemi
Week 0								
AP mm	-0.2 (7.4)	3.8 (7.4)	-4.0* (-7.7 to -0.3)	1.5 (16.8)	1.7 (15.5)	-0.3 (-10.0 to 9.5)	2.2 (-9.0 to 4.7)	2.2 (-4.2 to 8.7)
SI mm	-17.1 (16.6)	-17.5 (16.3)	0.1 (-12.1 to 12.2)	-21.5 (20.3)	-26.0 (21.4)	4.5 (-4.9 to 13.8)	4.5 (-4.5 to 13.6)	8.6 (-2.4 to 19.5)
Week 6								
AP mm	3.1 (7.6)	1.3 (9.1)	2.2 (-3.6 to 8.0)	1.5 (16.8)	1.7 (15.5)	-0.3 (-10.0 to 9.5)	1.6 (-6.0 to 9.2)	-0.8 (-8.1 to 6.5)
SI mm	-12.2 (16.4)	-13.3 (9.5)	-1.1 (-8.8 to 11.1)	-21.5 (20.3)	-26.0 (21.4)	4.5 (-4.9 to 13.8)	9.3 (-1.1 to 19.7)	12.3* (2.5 to 22.1)
Difference Week 0 to 6								
AP mm, mean (95%CI)	3.1 (-1.3 to 7.5)	-2.2 (-6.5 to 2.2)						
SI mm, mean (95%CI)	-6.2 (-15.2 to 2.9)	-3.8 (-10.5 to 2.9)						

*p<0.05

† Control sides matched to hemi- and non-hemi side from the stroke group
GHJC Glenohumeral joint centre, AP anteroposterior, SI superoinferior
Hemi, Hemiparetic; Non-hemi, Non-hemiparetic.

4.2.2.3 GHJC position in matched strokes and controls at week six

4.2.2.3.1 Participants at week six

The seven participants lost to follow up had a mean age of 57.7 years (SD 26.5), mean BMI 23.0 kg/m² (SD 3.6) and mean MAS score 5.0 (1.8). Participants who remained in the study were older with a mean age of 67.3 years (SD 16.1), had a mean BMI of 25.7 kg/m² (SD 4.0) and mean MAS score of 4.0 (SD 2.3). There was no significant difference between the two groups for age ($p = 0.246$), BMI ($p = 0.112$) and mean MAS scores ($p = 0.325$). Glenohumeral joint centre AP and SI positions for the matched strokes and controls at week six are presented in Table 10.

4.2.2.3.2 Position of GHJC at week six - comparison between hemiparetic and non-hemiparetic sides in the Stroke group

There were no significant difference between sides in GHJC position for either the AP (mean difference 2.2 mm, 95%CI -3.6 to 8.0) or SI (mean difference -1.1 mm, 95%CI -8.8 to 11.1) directions, in the stroke group at 6 weeks (Table 10). Regardless of side, the average position of the GHJC at week six in the stroke group was 2.0 mm (95% CI -0.2 to 4.2) anterior to the acromion and 13 mm (96% CI -15.8 to -9.8) inferior to the acromion.

4.2.2.3.3 Position of GHJC at week six - comparison between stroke and control groups

Data for the control group were carried forward from baseline and as previously reported. At week 6, the GHJC was positioned anteriorly and inferiorly to the acromion in both the stroke and control groups. There were no significant differences between the groups for either side at week 6, except for SI direction on the non-hemiparetic side (Table 10). The non-hemiparetic GHJC was positioned 12.3 mm (95%CI 2.5 to 22.1) closer to the acromion in the stroke group compared with the control group ($p = 0.005$, $F_{(1, 51)} = 8.829$).

4.2.2.3.4 Change in GHJC position in stroke group from baseline to week six

There were no statistically significant changes in GHJC position from baseline to week six on the hemiparetic or non-hemiparetic sides, in either the AP ($p > 0.156$; $F_{(1, 21)} < 2.164$) or SI directions ($p > 0.169$; $F_{(1, 21)} < 2.029$), in the stroke group. Overall, the

GHJC moved superiorly and anteriorly on the hemiparetic side, and posterior-superiorly on the non-hemiparetic side, over the 6-week follow up (Table 10).

4.2.2.4 Influence of clinical characteristics on GHJC: A subgroup analysis

The clinical and demographic characteristics included in the subgroup analyses were median age ($\geq$ or $\leq$ 68 years), gender (men, women), side affected by the stroke (right, left), dominant side affected by the stroke (yes, no), pain present on the hemiparetic side (yes, no), motor recovery scores (≥ 6 , <6) and the presence of muscle tone changes (no, yes). Means and standard deviations of GHJC position in AP and SI directions for these demographic and clinical characteristics, found following the subgroups analyses, are reported in Table 11.

4.2.2.4.1 Age and gender

There were no significant differences in GHJC position in AP or SI directions on the hemiparetic and non-hemiparetic sides between younger and older stroke participants at baseline or week six (Table 11). There were no significant differences between men and women in the stroke group for GHJC position at baseline in AP or SI directions on the hemiparetic and non-hemiparetic sides. There were no significant differences between men and women in the stroke group at week 6 in AP or SI directions on the hemiparetic side and in the SI direction on the non-hemiparetic side for gender. However, there was a significant difference ($F_{(1,21)} = 7.908$, $p = 0.010$) in GHJC position in AP direction on the non-hemiparetic side in stroke participants at week six, with the men positioned 6.2 mm (SD 6.0) anteriorly to the acromion, while the women were positioned 3.2 mm (SD 9.2) posteriorly to the acromion (Table 11). There was no difference in GHJC in either direction, for either side, between age or gender subgroups in the control group.

4.2.2.4.2 Right versus left hemiparesis

Baseline

Subgroup analysis of the side affected by the stroke revealed (i.e. *right* versus *left*) a significant difference in GHJC position in the AP and SI directions at baseline. In participants with *right* hemiparesis the GHJC was positioned 5.8 mm (SD 8.1) posterior to the acromion, while in participants with *left* hemiparesis the GHJC was positioned 2.9 mm (SD 3.9) anterior to the acromion ($F_{(1, 27)} = 14.704$, $p = 0.001$) on the

hemiparetic side (Table 11). In addition, the GHJC in the SI direction, was positioned 24.4 mm (SD 12.9) inferior to the acromion in participants with *right* hemiparesis compared to 11.8 mm (SD 11.4) inferior to the acromion in participants with *left* hemiparesis ($F_{(1, 27)} = 7.756$, $p = 0.010$). In participants with *right* hemiparesis, the non-hemiparetic GHJC was positioned 6.9 mm (SD 12.7) inferior to the acromion compared with 26.2 mm (SD 12.5) inferior to the acromion on the non-hemiparetic side in participants with *left* hemiparesis (Table 11).

Week 6

At week 6, a significant difference between right and left hemiparetic sides were evident in the SI direction. In participants with right hemiparesis, the GHJC on the hemiparetic side in the SI direction was positioned 22.9 mm (SD 15.4) inferior to the acromion compared to 5.4 mm (SD 13.4) inferior to the acromion in participants with left hemiparesis ($F_{(1, 21)} = 8.392$, $p = 0.009$) (Table 11). No other significant differences were found for either the hemiparetic or non-hemiparetic side between right and left hemiparetic sides in the AP direction.

4.2.2.4.3 Hand dominance

Dominant side affected subgroup analysis revealed no significant differences between the sides in either the AP or SI direction for either baseline or at week 6 with one exception. At baseline, for those participants who had their dominant side affected by the stroke, GHJC position in the SI direction on the non-hemiparetic side was 10.9 mm (SD 14.8) inferior to the acromion compared to 26.3 mm (SD 12.6) inferior to the acromion in stroke participants who had their non-dominant side affected ($F_{(1, 26)} = 8.827$, $p = 0.006$) (Table 11).

4.2.2.4.4 Pain, motor recovery and muscle tone

There was no statistically significant relationship between pain, motor recovery and muscle tone changes on GHJC position in the AP or the SI direction for the hemiparetic or non-hemiparetic sides at baseline or week 6 with one exception. At week 6 those participants who had MAS scores less than 6 (indicating a poor motor recovery) the GHJC was positioned in the AP direction on the non-hemiparetic side 5.7 mm (SD 10.4) posterior to the acromion, in contrast to the group with scores equal to six (good motor recovery) which were positioned 3.3 mm (SD 7.7) anterior to the acromion ($F_{1, 21} = 4.971$, $p = 0.037$) (Table 11).

Table 11. Means and standard deviations for anteroposterior and superoinferior GHJC positions for hemiparetic (hemi) and non-hemiparetic (non-hemi) sides by demographic and clinical subgroups for anteroposterior and superoinferior GHJC positions at baseline and week six for stroke group.

GHJC position	Week 0 AP Means (SD) mm		Week 6 AP Means (SD) mm		Week 0 SI Means (SD) mm		Week 6 SI Means (SD) mm	
	Hemi	Non-hemi	Hemi	Non-hemi	Hemi	Non-hemi	Hemi	Non-hemi
Age[†]								
< 68 years, n = 14	-2.1 (8.0)	4.9 (8.6)	4.4 (8.3)	1.4 (9.5)	-16.1 (13.3)	-21.7 (14.8)	-9.3 (18.9)	-15.8 (10.8)
≥ 68 years, n = 15	0.1 (6.6)	3.1 (6.2)	2.0 ((7.2)	0.5 (9.2)	-17.8 (13.9)	-15.9 (16.3)	-14.5 (14.5)	-11.4 (8.3)
Gender								
Men, n =12	-0.8 (8.8)	6.2 (7.4)	0.7 (6.7)	6.2* (6.0)	-21.6 (16.6)	-17.0 (22.7)	-12.0 (11.5)	-11.2 (3.8)
Women, n = 18	-0.6 (6.3)	2.6 (7.1)	4.9 (8.0)	-3.2* (9.2)	-13.7 (9.9)	-19.7 (9.4)	-12.4 (19.8)	-15.0 (12.1)
Stroke side								
Right, n = 12	-5.8* (8.1)	3.6 (3.4)	-0.5 (8.6)	5.3 (3.1)	-24.4* (12.9)	-6.9* (12.7)	-22.9* (15.4)	-8.6 (3.3)
Left, n = 18	2.9* (3.9)	4.2 (9.1)	5.4 (6.2)	-1.9 (10.7)	-11.8* (11.4)	-26.2* (12.5)	-5.4* (13.4)	-16.4 (11.0)
Dominant side affected								
Yes, n = 15	-3.1 (8.6)	4.3 (5.2)	1.6 (9.4)	3.6 (8.0)	-21.5 (13.8)	-10.9* (14.8)	-17.0 (21.3)	-11.7 (9.4)
No, n = 15	1.9 (4.6)	3.7 (9.2)	4.4 (5.6)	-1.6 (9.7)	-12.2 (11.5)	-26.3* (12.6)	-7.8 (8.9)	-14.9 (9.7)
MAS[‡] baseline								
<6, n = 16	-0.8 (6.4)	4.1 (5.8)	2.2 (9.2)	-1.1 (10.4)	-18.2 (14.1)	-17.5 (16.0)	-11.3 (18.9)	-15.0 (9.7)
=6, n = 14	-0.6 (8.4)	3.8 (9.0)	4.2 (5.2)	3.5 (6.8)	-15.7 (13.0)	-19.9 (15.8)	-13.4 (13.3)	-11.2 (9.2)

MAS* week 6

<6, n = 6			4.5 (11.2)	-5.7*(10.4)			-9.1 (27.9)	-14.8 (13.4)
=6, n = 17			2.6 (6.3)	3.3* (7.7)			-13.3 (11.0)	-12.8 (8.2)

Pain§ baseline

Yes, n = 13	-2.3 (7.1)	4.3 (7.8)	4.4 (7.3)	1.3 (9.4)	-19.8 (15.1)	-24.6 (14.7)	-13.9 (13.6)	-15.1 (8.1)
No, n = 17	0.4 (7.5)	3.7 (7.1)	2.1 (8.0)	0.6 (9.3)	-13.0 (9.9)	-13.5 (15.0)	-10.9 (18.7)	-12.0 (10.6)

Pain§ week 6

Yes, n = 13			3.2 (8.1)	1.4 (9.2)			-16.0 (14.5)	-14.9 (8.4)
No, n = 10			3.0 (7.6)	0.5 (9.4)			-9.4 (17.7)	-12.2 (10.4)

Muscle tone|| baseline

Tonal changes, n = 24	-3.1 (5.7)	5.7 (5.1)	1.6 (12.9)	-2.6 (13.0)	-15.8 (19.6)	-19.3 (5.7)	-2.9 (28.0)	-16.5 (14.8)
No tonal changes, n = 5	-0.2 (7.6)	3.4 (7.7)	2.9 (6.3)	2.1 (8.4)	-17.2 (12.3)	-18.2 (17.1)	-14.2 (13.6)	-12.1 (8.3)

Muscle tone|| week 6

Yes, n = 16			0.9 (8.5)	-2.5 (8.6)			-16.4 (18.7)	-12.2(8.0)
No, n = 7			4.0 (7.3)	2.4 (9.2)			-10.4 (15.6)	-13.8 (10.3)

*p < 0.05;

† equal to or greater than and less than 68 years;

‡ motor assessment scale scores (equal to or less than six);

§ visual analogue scale 0=no pain, 100=worst pain imaginable;

|| participants with and without tonal muscle changes

GHJC, glenohumeral joint centre; MAS, Motor Assessment scale.

Hemi, Hemiparetic; Non-hemi, Non-hemiparetic.

Chapter 5

Discussion

5.1 Summary of study outcomes

The aims of this prospective longitudinal observational study were to investigate changes in the glenohumeral joint centre of rotation over time in an acute stroke population (within and between sides), and compare GHJC between a stroke and healthy control cohort.

This study demonstrated that using a sphere-fitting approach of an ETD to measure GHJC in an acute stroke and healthy population was reliable. At baseline, in participants with stroke, the hemiparetic GHJC in the AP direction was positioned slightly posteriorly to the acromion. This was significantly different to the GHJC position in the AP direction on the non-hemiparetic side, which was positioned anterior to the acromion. Similarly, the GHJC for the age, gender and BMI matched healthy controls was positioned anterior to the acromion on both sides with no significant difference between them. There were no differences within or between strokes and controls for either the hemiparetic or non-hemiparetic GHJC position in the SI direction.

At six weeks follow-up the stroke group displayed changes in GHJC position on the hemiparetic side with GHJC moving anteriorly and more superiorly with respect to the acromion. The GHJC on the non-hemiparetic side moved posteriorly and superiorly compared to baseline. Neither of these changes were statistically significant. Compared to the healthy controls, the stroke participants' GHJC in both the hemiparetic and non-hemiparetic sides were similarly positioned in the AP direction. In the SI direction, the GHJC was positioned closer to the acromion in the stroke participants compared to the healthy controls. This reached statistical significance in the non-hemiparetic side only.

Overall, clinical characteristics of this cohort of people with stroke appeared to have little influence on GHJC position. At baseline, age, gender, degree of motor recovery, pain and tonal changes did not influence GHJC position in either arm in either direction. The GHJC was influenced by which side was affected by the stroke (right or left). At baseline, in those with right hemiparesis the GHJC was positioned posteriorly to the acromion. In contrast, in those with left hemiparesis the GHJC was positioned anteriorly to the acromion. At week six, the GHJC centre in all stroke participants, regardless of side affected by stroke, had moved anteriorly in the AP direction. In the SI direction, those with right hemiparesis had the GHJC positioned significantly more inferiorly relative to the acromion compared to those with left hemiparesis, both at baseline and week six. Gender may have influenced GHJC but the findings were

inconclusive. Other clinical characteristics investigated such as age, dominance, pain, motor recovery and muscle tone did not influence GJHC.

5.2 Within session intra-tester reliability

This is the first study that has investigated intra-tester reliability of GHJC position measurement using an ETD in healthy older adults and those following stroke. Single assessor within session intra-tester reliability of measurement of GHJC position using the sphere-fitting method approach of an ETD was excellent in this group of acute stroke and age, gender and BMI matched healthy control participants. Overall, ICCs were greater than 0.80 (except for control group non-hemiparetic GHJC in the SI direction = 0.78). Measurement error was consistently less for the stroke participants compared to the healthy control participants, regardless of side tested. The maximum SEM reached in the stroke group was 9.4 mm with 15.3 mm in the healthy control group and the MDC reached was 26.0 mm in the stroke group and 42.5 mm in the healthy control group. The measurement error or variability in the measures appears to be greater in the healthy control group compared to the stroke participants in both directions (AP, SI).

The measurement error of GHJC has been reported in a few studies. The magnitude of measurement error of GHJC centre in this healthy control group is consistent with that reported in a previous *in vitro* cadaveric study, which reported SEMs ranging from 8 to 16.5 mm (Veeger, 2000). Two other studies (Lempereur et al., 2013; Monnet et al., 2007) measured GHJC but in younger populations and reported SEMs similar or larger to the SEMs calculated for healthy control participants in the current study (SEM 30.0 to 76.0 mm (Monnet et al., 2007); 12.9 to 44.1 mm (Lempereur et al., 2013)), with similar ICC values (>0.7) (Lempereur et al., 2013).

It appears that differences in participant demographics, equipment, and method of calculating the GHJC, have little influence on the measurement error in a healthy population. Compared to previous studies, the healthy control group in the current study was older (mean age 65.3 compared to 25.8 years) (Lempereur et al., 2013). In addition, previous studies used camera-based three-dimensional motion analysis systems such as the Vicon MX (Oxford Metrics Ltd., Oxford, UK and Biogesta, Valenciennes, France) (Lempereur et al., 2013; Monnet et al., 2007). GHJC has also been measured previously during active movement within three planes of movement and at varying speeds (Lempereur et al., 2013; Monnet et al., 2007). In contrast, the current study measured error during passive movement in five planes of movement, and at a constant medium speed.

Measurement error in the stroke group was consistently less than that of the healthy control group in the current study, regardless of side (Table 8). Greater measurement error was also found on the non-hemiparetic side compared to the hemiparetic side in the stroke participants. Several possible factors could have contributed to the variability of measurement error reported in the current study, including differences in participant characteristics and method of measurement. Stroke participants were recruited on average a few days post stroke. It is possible that even in this short time frame some muscle wasting and muscle tone changes may have already occurred, particularly on the hemiparetic side (Canning et al., 2004; Wissel et al., 2010). These changes in muscle wasting and muscle tone may potentially have made it easier to palpate anatomical landmarks for sensor placement on the hemiparetic side in the stroke participants and hence greater accuracy when compared to the healthy controls as well as on the non-hemiparetic side of stroke participants. The possibility of skin movement evoking errors during manual palpation of bony landmarks has been suggested (Harlick, Milosavljevic, & Milburn, 2007); this may also have contributed to some inaccuracy of sensor placement.

The current study involved the assessor performing passive movement on the measured arm for both the stroke and healthy control groups. Passive movement was chosen as it was anticipated that at least some of the stroke participants would not have sufficient control of their hemiparetic arm to perform active movement. Participants' ability to allow true passive movement of the limb may have been different for hemiparetic and non-hemiparetic sides in the stroke group and between stroke and healthy control groups. The stroke group had an acute condition, were generally unwell, and had reduced muscle activity, as evidenced by the scores on the Motor Assessment Scale (Table 9). In contrast, our healthy control group were well, community dwelling older adults with no history of shoulder pain or current upper limb musculoskeletal injury. The measurement method during passive calibration involved the assessor passively elevating the arm to 45 degrees through a standardised pattern of movement (see section 3.3.4.3). Despite standardised instructions being given to both groups, it is possible that healthy control participants as well as stroke participants during movement of their non-hemiparetic side, did not fully relax and therefore the movement may not have been truly passive. This may have led to a slightly different glenohumeral joint position due to active muscle contraction in the healthy control group, as opposed to passive movement (Chopp, Fischer, & Dickerson, 2011; Chopp, O'Neill, Hurley, & Dickerson, 2010; James & Parker, 1989).

5.3 Baseline position of GHJC in acute strokes and matched control group

This is the first study to investigate GHJC position in healthy older adults and in people with acute hemiparetic stroke. It is also the first study to investigate GHJC position within two weeks of stroke onset. There were no differences in GHJC position between sides or between stroke and control groups at baseline with one exception: there was a significant difference between hemiparetic and non-hemiparetic GHJC in the AP direction in the stroke group. Regardless of side, the GHJC was positioned 23.8 mm inferiorly in the healthy controls, compared to 17 mm inferiorly in the stroke group. The GHJC in the stroke group was positioned 0.2 mm posteriorly to the acromion on the hemiparetic side, compared with 3.8 mm anteriorly on the non-hemiparetic side. In comparison, the GHJC was positioned anteriorly on both sides by an average of 1.6 mm in the control group. Given that the minimal detectable change is 9.2 mm for the stroke group (see Table 8), a between-side difference of 4 mm may be the result of measurement error rather than a true side difference. Therefore, regardless of side or group, the position of the GHJC in this study sits 17 to 24 mm inferiorly, and 0 to 4 mm anteriorly to the acromion.

5.3.1 Control participants

Two previous studies have investigated GHJC position measured using a similar electromagnetic tracking device and methodology in healthy controls. Stokdijk's investigations calculated the GHJC to be 38.2 mm (SD 8.3) posterior and 44.8 mm (SD 7.1) inferior to the acromion (Stokdijk et al., 2000). While Monnet and colleagues calculated the centre to be 14.5 mm (SD 6.2) posterior and 31.5 mm (11.8) inferior to the acromion, using a motion analysis system with infrared cameras (Monnet et al., 2007). In contrast, the current study found the GHJC was positioned anteriorly and less inferiorly to the acromion in the healthy control group. These differences in GHJC between the current study and previous work may be due to differences in population characteristics. Both Stokdijk et al. and Monnet et al. recruited young healthy adults (i.e. less than 28 years of age), whereas participants in the current study were older (average 65 years). Age impacts the shoulder joint in older healthy people and people with stroke alike (McBeth & Jones, 2007; Tobola et al., 2009). Older adults may experience shoulder pathologies such as impingement (Garofalo et al., 2010; Ludewig & Braman, 2011), tendinopathies (Yamamoto et al., 2010) and capsulitis (Dundar, Toktas, Cakir, Evcik, & Kavuncu, 2009) that may influence GHJC position.

5.3.2 GHJC position in strokes

Abnormal excursion of the humerus within the glenohumeral joint typically is referred to as subluxation and has been mainly measured in the SI direction (inferior subluxation) in stroke participants (Paci et al., 2005). The current study is one of the few that have investigated excursion of the humerus in the AP direction (Boyd et al., 1993b; Hall et al., 1995; Park et al., 2007). We found that the hemiparetic GHJC was positioned posterior to the acromion while the non-hemiparetic GHJC was anterior to the acromion (Table 10).

One reason for this difference between the hemiparetic and non-hemiparetic sides may be due to post-stroke participants spending more time lying in bed compared to prior to their stroke (Kroeders, Bernhardt, & Cumming, 2013). In the supine position, the gravitational forces acting on the head of the humerus, even in the mildly impaired group such as ours, may lead to posterior glide of the head of humerus within the glenohumeral joint. The anatomical alignment of the glenohumeral joint suggests that there is less constraint of the humeral head in the AP direction (Howell & Galinat, 1989), making it more vulnerable to changes in humeral head position in this direction. Add to this the impaired rotator cuff control: the compressive forces which usually ensure accurate coupling of the humeral head and the glenoid fossa could allow excessive humeral movement in the AP direction (Halder, Kuhl, Zobitz, Larson, & An, 2001; Lam et al., 2007).

It has previously been identified that subluxation does not occur uni-dimensionally, and that further research into the measurement of the AP component is needed (Boyd et al., 1993b). Anterior subluxation combined with inferior subluxation in 35% of stroke participants has previously been reported during a combined radiological and palpatory examination of 20 people up to ten years post stroke (Hall et al., 1995). Issues surrounding radiological tests limit their clinical utility. A more recent study has found that anterior subluxation determined via ultrasonography was superior to radiological measures while correlating with clinical measurements of muscle tone and strength in a group of subacute post-stroke participants (Park et al., 2007).

Though ETDs have not previously been used to measure GHJC in stroke participants, they have been used to measure humeral and scapular position in people with stroke (Culham et al., 1995; Niessen et al., 2008) (Beebe & Lang, 2009). In people with stroke with low muscle tone, greater glenohumeral subluxation and positioning of the scapula lower and further from the midline, have been shown at rest compared to stroke

participants with high tone (Culham et al., 1995). Similarly, the inferior angle of the scapula has been shown to be positioned away from the midline at rest (rotation laterally) and during shoulder abduction and flexion in stroke participants with HSP compared to those without HSP (Niessen et al., 2008). Only one other study was found that used an ETD in people with stroke; however, scapular protraction and humeral rotation were measured only on the non-hemiparetic arm during active elevation (Meskers et al., 2005). Reduced scapula protraction and humeral external rotation on the non-hemiparetic side was reported in comparison to controls (Meskers et al., 2005). Although resting scapular position was not measured, it is reasonable to assume that the resting position of the non-hemiparetic shoulder was altered.

The size of the subacromial space, that is, the distance between the glenohumeral head and acromion, may also provide information on glenohumeral joint position on the hemiparetic and non-hemiparetic sides in people with stroke (Arsenault et al., 1991; Barlak et al., 2009; Demirci et al., 2007; Kumar et al., 2011). Glenohumeral joint examination with radiographs or ultrasound have reported conflicting results. Several studies report an increase in the subacromial space on the hemiparetic side, which infers loss of the passive restraints and subsequent inferior subluxation of the glenohumeral joint at rest (Arsenault et al., 1991; Barlak et al., 2009; Demirci et al., 2007; Kumar et al., 2011). All these studies investigated humeral and scapular position in subacute (Arsenault et al., 1991; Barlak et al., 2009; Demirci et al., 2007; Kumar et al., 2011; Meskers et al., 2005) or chronic stroke populations (Culham et al., 1995; Niessen et al., 2008).

A decrease in the subacromial space in the hemiparetic side following stroke has previously been suggested as contributing to HSP (Chae & Jedlicka, 2009; Dromerick et al., 2006; Lakse et al., 2009; Shah et al., 2008). However, these suggestions have been based on clinical assessments of soft tissue impingement (Chae & Jedlicka, 2009; Dromerick et al., 2006) or diagnoses of soft tissue lesions via radiographical measures (Lakse et al., 2009) or magnetic resonance imaging (Shah et al., 2008) rather than specific measurement of the subacromial space or humeral head position as undertaken in the current study.

5.3.3 GHJC position – comparison between strokes and controls

The current study found no difference in position of the GHJC in the AP and SI direction between stroke and control participants at baseline, suggesting that the glenohumeral joint position may not be affected immediately following a stroke. This is

in contrast to other literature (Huang et al., 2010; Paci et al., 2007). One reason potentially contributing to this lack of difference in GHJC position at baseline between strokes and control participants is the timing of the baseline measurement. GHJC was measured within the first two weeks post-stroke, when any complications such as changes in shoulder joint and therefore GHJC position may not yet have taken place (Kuptniratsaikul, Kovindha, Suethanapornkul, Manimmanakorn, & Archongka, 2009). Although subluxation rates (indicating movement in the GHJC position inferiorly to the acromion) of up to 50% have been reported in the early to subacute phase (Huang et al., 2010; Paci et al., 2007), the subacute phase (Aras et al., 2004; Barlak et al., 2009; Demirci et al., 2007; Kuptniratsaikul et al., 2009; Lindgren et al., 2012; Pompa et al., 2011) and the chronic post-stroke phase (Boyd et al., 1993b; Lo et al., 2003). However, the mild deficits of the recruited sample may have also contributed to the lack of the difference in GHJC position between strokes and controls.

The lack of anteroinferior subluxation found in the current study may also reflect the effectiveness of current best practice guidelines which promote safe positioning and handling of the upper limb to minimise subluxation forces and potential trauma to the passive restraints of the shoulder in the acute phase (National Stroke Foundation, 2010). Additionally, in Australia in recent years, improved access to stroke units, increased awareness of and implementation of post-stroke management programs, including thrombolysis (Tai & Yan, 2013) may have also contributed to improved recovery and reducing post-stroke disability (Nazir, Petre, & Dewey, 2009; Quain et al., 2008).

The link in stroke participants, between poor motor recovery and subluxation has been confirmed in a meta-analysis (Ada & Foongchomcheay, 2002), systematic review (Kumar et al., 2010), and medical history audit (Blennerhassett et al., 2010). The relatively mild nature of the motor deficits experienced in our stroke group may have limited any change in GHJC position immediately following the stroke. Fourteen (47%) participants had good shoulder motor recovery at baseline (MAS score =6) and an additional 6 (20%) participants were able to lift their arm to 90 degrees shoulder elevation. As 67% of the stroke group in this current study could elevate their arm to at least 90 degrees, it may be reasonable to assume that the motor control of their shoulder was minimally, or unaffected by the stroke. Coordinated activity of the rotator cuff muscles is responsible for accurate coupling of the humeral head and glenoid cavity at rest and during dynamic activity (Chopp et al., 2010; Lam et al., 2007). With the majority of our stroke participants having antigravity shoulder control, this might explain the similarities in positioning of the glenohumeral joint centre to that of the

healthy controls. The remaining nine (30%) stroke participants who had a MAS score at baseline of equal or less than three (not able to move shoulder against gravity), also had no significant difference in baseline GHJC positions in the AP or SI direction at baseline or 6-week follow-up on either side compared to stroke participants with good motor recovery. This finding though is most likely due to our overall small sample size; in particular, our small numbers of participants following stroke with poor motor recovery and HSP. This is further discussed in Section 5.9.

In conclusion, the current study results differ from studies involving stroke participants in all phases post-stroke, in not finding any difference between stroke and control participants. The main reasons are the timing of the measurement (within two weeks post-stroke) and the stroke group's relatively mild motor deficits. These features differentiate the position of the glenohumeral joint post-stroke from that of other studies and align them with that of the controls within the current study.

5.4 Changes in GHJC position over time

5.4.1 GHJC position in strokes

Twenty-three participants were followed up at week six. Participant loss to follow-up involved one death, two participants being medically unwell and four declining further involvement in the study. Although, not statistically different, clinical observation indicated that the majority of participants who declined further involvement were younger, physically mildly affected by the stroke, had shorter in-patient stays and were unprepared for the stroke diagnosis. Anecdotally, these participants were not keen to be followed up for any reason other than for essential medical appointments, seeking to distance themselves from the hospital environment and staff (they also declined the offer of follow-up at home). As a result the remaining participants were older, had slightly higher BMI and had less motor recovery than the loss-to-follow-up group.

Few studies have been conducted with acute post stroke participants. The current study's 23.3% loss-to-follow-up appears to be consistent with a two month shoulder taping randomized control trial that commenced in the acute phase, which reported a 25.5% loss (Hanger et al., 2000). Greater losses of up to 36% at six months have also been reported in a longitudinal study of acute stroke participants being investigated for hemiparetic shoulder pain (Pong et al., 2012). Loss to follow-up rates for trials involving sub-acute and chronic stroke participants have been similar (22.7%) (Desrosiers et al., 2003) or lower for studies involving upper limb positioning (12%) (Ada, Goddard,

McCully, Stavrinou, & Bampton, 2005) or exercise (14%) (English, Hillier, & Stiller, 2008) in sub-acute post-stroke participants and in a study investigating the effect of electrical stimulation on shoulder pain (4%) follow-up after 24 months (Chantraine, Baribeault, Uebelhart, & Gremion, 1999).

There were no significant differences in GHJC in the stroke group, either between sides or over time. At week 6, the GHJC on the hemiparetic side had moved anteriorly and superiorly compared to the baseline position. In contrast, the non-hemiparetic side moved posteriorly and superiorly compared to the baseline position (Table 10). The GHJC position change in the SI direction at week 6, closer to the acromion, contradicts the general assumption that the glenohumeral joint undergoes inferior subluxation following an acute stroke (Huang et al., 2010; Kumar et al., 2013; Kuptniratsaikul et al., 2009; Lo et al., 2003). Instead, the current study found the position change analogous with a superior shift in the position of the humeral head. The humeral head position change could lead to the greater tuberosity contacting the superior aspect of the glenoid cavity or coracoacromial arch and impinge the soft tissues (including the rotator cuff) in the subacromial space (subacromial impingement syndrome) (SAIS) (Garofalo et al., 2010; Heyworth & Williams, 2009). This GHJC position change combined with the move anteriorly on the hemiparetic side, fits the criteria for SAIS (Garofalo et al., 2010).

5.4.2 GHJC position - comparison between strokes and controls

Comparison of stroke GHJC position in the AP direction on both the hemiparetic and non-hemiparetic sides with that of controls on the same side (carried forwards from baseline), revealed that there was no difference between the two groups. However, the GHJC was positioned more superiorly on both sides in the stroke group compared to the controls, and this was significant for the non-hemiparetic side. These findings infer that the strokes tended to have a reduced subacromial space on both sides compared to the controls, but more so on the non-hemiparetic side. The current study's significant finding of the stroke group's non-hemiparetic side being positioned closer to the acromion than in the control group infers the possibility of some impingement (Garofalo et al., 2010). This is the first study to identify changes over time in the position of the non-hemiparetic glenohumeral joint.

Changes in glenohumeral joint positioning on the non-hemiparetic side post-stroke have been found previously in only two studies (Kumar et al., 2011; Park et al., 2007). These studies used radiographic and/or ultrasonographic (Kumar et al., 2011; Park et

al., 2007) measurements of the distance between the greater tuberosity and the lateral acromion to determine the extent of subluxation. Contrary to our findings, these reliability studies reported greater humeral to acromial distances on the hemiparetic than the non-hemiparetic sides (Kumar et al., 2011; Park et al., 2007). One of the reasons for these contrasting findings may be due to difference in the stroke populations between studies. In the study by Kumar et al almost 40% of participants had less than antigravity strength (grade ≤ 2) as measured via the Medical Research Council's Oxford scale (Compston, 2010), compared to 30% in our study (Kumar et al., 2011). Similarly, the median Motricity Index score for shoulder abductors in the second study (Park et al., 2007) was 14, which indicates that participants did not have anti-gravity control (Demeurisse, Demol, & Robaye, 1980). Only one other study reported differences between hemiparetic and non-hemiparetic stroke upper limbs, but the differences related to scapular rather than glenohumeral joint positioning (Yoon & Lee, 2007).

It is perhaps not unexpected to find changes on the non-hemiparetic GHJC over the six week period of the study. At a simplistic level, motor deficits following a single unilateral cortical event such as stroke is most common on the contralateral (opposite side to the side of the cerebral hemisphere damage) upper and/or lower limb (Barrett & Meschia, 2013, p. 2), through the brainstem cross-over of the ascending and descending corticospinal tracts. Efferent signals to a limb travel from the cerebral cortex via the corticospinal tracts, cross to the opposite side at the brainstem and descend in the spinal cord where they synapse in the dorsal horns with peripheral nerves (Tymianski, 2013). The process is reversed for afferent signals from the limbs to the cerebral cortex. However, there is evidence to indicate that there is inter-hemispheric cortical control of each side of the body in a healthy population, hence a lesion in one cerebral hemisphere may produce bilateral changes in the periphery (Desrosiers, Bourbonnais, Bravo, Roy, & Guay, 1996; Mani et al., 2013). A more likely scenario, however, might be the reliance on the functional non-hemiparetic arm to perform activities of daily living resulting in increased use of this arm which may have also contributed to the lack of changes (Michielsen, Selles, Stam, Ribbers, & Bussmann, 2012; Nakayama, Jorgensen, Raaschou, & Olsen, 1994).

Several studies indicate that in unilateral stroke, the ipsilateral (same side as the side of the cerebral hemisphere damage) upper and/or lower limb can also be affected (Bohannon & Andrews, 1995; Jung, Yoon, & Park, 2002; Meskers et al., 2005; Schaefer et al., 2009). Upper limb muscle weakness was found during dynamometer tests (Bohannon & Andrews, 1995) and manual muscle testing (Jung et al., 2002);

positional errors and multi-joint coordination impairments were detected during multiple targeted reaching tasks (Schaefer et al., 2009); and impaired scapular and humeral range of movement were observed during investigation via an ETD of upper limb elevation (Meskers et al., 2005). Functional consequences as a result of ipsilateral upper limb impaired speed and coordination have also been suggested (Nakamura, Abreu, Patterson, Buford, & Ottenbacher, 2008; Pohl, Luchies, Stoker-Yates, & Duncan, 2000). The raft of ipsilateral upper limb impairments relating to muscle strength, control and function is evident. As a result, changes in both GHJC are not unexpected.

No longitudinal studies of GHJC position in the healthy or post-stroke population or studies comparing GHJC with healthy controls were found. As a consequence, the results of the current study cannot be directly compared with other studies. Few longitudinal studies involving post-stroke participants have investigated changes in glenohumeral joint position. Most investigated the effectiveness post-stroke of electrical stimulation of the rotator cuff and other shoulder muscles to reduce subluxation or the relationship between subluxation and HSP, though this relationship remains controversial (Kumar et al., 2013). Reduction of subluxation was reported during a meta-analysis of several studies using electrical stimulation post-stroke (Ada & Foongchomcheay, 2002). Radiographic evidence demonstrated prevention of 6.5 mm of subluxation but a reduction of only 1.9 mm in those who were already subluxed. One study not included in the meta-analysis demonstrated significant differences in reduction of subluxation to normal in the intervention group (80%) compared with the control group, adding further evidence of glenohumeral joint position changes over time (Chantraine et al., 1999).

More recent investigation of the effect of galvanic stimulation on shoulder subluxation in the acute stage post-stroke, reported significant improvements in subluxation at discharge (Fil et al., 2011), compared to stroke participants who did not receive the galvanic stimulation. This result is similar to an earlier longitudinal study investigating the effect of electrical stimulation on subluxation (Koyuncu, Nakipoğlu-Yüzer, Doğan, & Özgirgin, 2010). Other studies have explored subluxation's relationship with HSP. Subluxation measurements were taken at baseline only (Barlak et al., 2009; Paci et al., 2007), with no glenohumeral joint positioning information data collected at discharge (Barlak et al., 2009; Paci et al., 2007) or several weeks post-discharge (Paci et al., 2007). In another study, subluxation measurements were taken on admission, discharge and six months post-discharge; but because no correlation was demonstrated between degree of subluxation and HSP, the data were not reported

(Pong et al., 2012). As a result, it is not possible to ascertain whether subluxation had changed over time. It appears that sufficient or improvement in motor control prevents or reduces the impact of subluxation at the glenohumeral joint. However, caution should be observed with regards to interpreting the findings from cross sectional observational study designs, as they do not infer a causal effect (Portney & Watkins, 2009, p. 280).

5.5 Subacromial impingement, stroke and hemiparetic shoulder pain

The current study found that the GHJC was positioned closer to the acromion in the stroke participants than control participants in both arms in both sides at both time points (though not statistically significant). This equates to a reduction in the subacromial space, which may suggest the potential for subacromial impingement (Barlak et al., 2009; Demirci et al., 2007) is a possibility.

Both subluxation (Arsenault et al., 1991; Barlak et al., 2009; Kumar et al., 2011) and subacromial impingement (Barlak et al., 2009; Demirci et al., 2007) are common post-stroke and may even co-exist (Barlak et al., 2009). With approximately 60% of people with stroke with HSP demonstrating subacromial impingement and subluxation (Barlak et al., 2009) a better understanding of the potential impact of these conditions on the GHJC is required.

The presence of subluxation or impingement of the glenohumeral joint infers a change in location of the glenohumeral joint centre. To date, no studies have been found that have measured GHJC in a population with subacromial impingement syndrome (SAIS), despite this condition being the most common cause of shoulder pain in older adults (Garofalo et al., 2010; Hanchard et al., 2013; Tekavec et al., 2012; Yamaguchi et al., 2006). Impingement occurs in the space under the coracoacromial arch when this space is reduced by bony or soft tissue structures or joint instability (Hanchard et al., 2004; Mostaed, 2004). Secondary pathologies which may be associated with SAIS include subacromial bursitis, subdeltoid bursitis, rotator cuff or long head of biceps tendinopathies or tears and glenoid labrum damage (Hanchard et al., 2013; Mostaed, 2004). As a result of these pathologies or shoulder muscle weakness or imbalance, the humeral greater tuberosity has insufficient space to clear the coracoacromial arch during arm movement. Soft tissues are pinched, causing pain and dysfunction (Mostaed, 2004). Based on the current study's results, some of the same pathologies and mechanisms may have occurred in the stroke participants. However, differences in

methods, equipment, and calculation of glenohumeral joint position, limits our ability to compare and contrast our findings with previous work.

Studies investigating the causes of HSP in the post-stroke population have found a varying prevalence of SAIS. Barlak and colleagues reported that 61.4 % of participants with HSP had SAIS identified via radiographical examination (Barlak et al., 2009), while other researchers reported as little as 14.8% prevalence (Demirci et al., 2007). Another imaging study that attempted to identify the causes of HSP, reported that almost 51% of stroke participants had effusion of acromial or deltoid bursae on ultrasonographic examination (Lee et al., 2009). Magnetic resonance imaging revealed extensive soft tissue pathology in a chronic post-stroke population with HSP (Shah et al., 2008). Thirty-five percent of participants had tears of at least one rotator cuff, deltoid or biceps muscle and 53% had tendinopathies of at least one rotator cuff, deltoid or biceps muscle (Shah et al., 2008). SAIS is usually one of many pathologies found in strokes participants with HSP and more than one pathology may occur in any given stroke patient (Yu, 2004). Additionally, the extent or severity of pathological changes may not always correlate with symptoms in people reporting HSP following stroke (Shah et al., 2008) or in the non-stroke population (Harrison & Flatow, 2011).

People with stroke and HSP may have reduction in the subacromial space associated with a high incidence of rotator cuff tendinopathies and tears (Kalichman & Ratmansky, 2011). Since the rotator cuff muscles are vital to glenohumeral joint stability in healthy people and people with stroke alike, impingement of the subacromial tissues may contribute to instability (Heyworth & Williams, 2009) and HSP (Chae & Jedlicka, 2009). The rotator cuff usually stabilises the humeral head centrally in the glenoid cavity during shoulder movement by moving it slightly downwards as the humerus moves upwards (Rah et al., 2012). The ability of the rotator cuff to control positioning of the humeral head has been highlighted in studies of healthy people (Chopp et al., 2010; Teyhen, Miller, Middag, & Kane, 2008). When the rotator cuff muscles were deliberately fatigued, the upward translation of the humeral head during shoulder elevation was unable to be prevented (Chopp et al., 2010; Teyhen et al., 2008). Further evidence of the rotator cuff playing a vital role in humeral positioning in the glenoid cavity and thereby influencing GHJC, is available in a recent review of shoulder movement dysfunction (Ludewig & Braman, 2011). The review reports that impaired rotator cuff activation results in increased humeral head translation superiorly or anteriorly, reducing the subacromial space and potentially leading to an increased risk of glenohumeral joint impingement (Ludewig & Braman, 2011).

In participants with stroke who have weakened or degenerated rotator cuff muscles (Rah et al., 2012), or who have suffered impaired motor control or changes in muscle tone (Huang et al., 2010), the humeral head tends to move upwards, leading to impingement and potential soft tissue damage (Rah et al., 2012; Shah et al., 2008). Given that older people have a higher prevalence of rotator cuff pathology (Tobola et al., 2009), prevalence is likely to be similar post-stroke and may not necessarily cause or be associated with HSP (Távora et al., 2010). This has implications for the current study: even though the stroke group in the current study had little or no pain, the movement of the head of humerus superiorly may predispose this population to subacromial impingement, and subsequently the development of shoulder pain post-stroke.

5.6 Relationship between GHJC position and demographic and clinical characteristics.

Various factors have the potential to influence clinical presentations and outcomes in stroke. Some of these include age, gender, side affected by the stroke, dominance, pain, motor recovery and muscle tone. The current study is the first to explore the impact of these factors on the position of GHJC. More details on these factors are available in chapter 2 (section 2.6). Having pain (Barlak et al., 2009; Klit, Finnerup, & Jensen, 2009), being older (Australian Institute of Health and Welfare, 2013), male (Islam et al., 2008; Thrift et al., 2009), having hemiparesis on the left side (Demirci et al., 2007), non-dominant side affected by stroke (Harris & Eng, 2006), poor motor recovery (Coupar et al., 2012; Hendricks, van Limbeek, Geurts, & Zwarts, 2002) and increased muscle tone (Wissel et al., 2010) have been associated with a poorer prognosis following stroke.

The stroke participants in the current study presented with a severity level distinguishable from other studies measuring the shoulder joint in this population. Participants with mainly minor severity strokes were recruited as a result of participants with more severe strokes not meeting eligibility criteria within the first two weeks of stroke onset (unable to give consent due to communication difficulties, medically unstable, insufficient sitting balance or endurance). Minor severity levels of shoulder pain, shoulder motor control and tonal muscle changes resulted: only 13.3% of participants had shoulder pain at rest and 33.3% during passive shoulder movement; severity of shoulder pain was low, with mean VAS at rest and during passive movement 2.6 mm (SD 8.8) and 18.6 mm (SD 31.0) respectively; 47% had full motor

recovery as measured via MAS and only 30% scored three or less, indicating inability to move the hemiparetic shoulder against gravity; only 16.7% had tonal muscle changes.

5.6.1 Pain

Pain did not influence GHJC within the first six weeks following stroke. Perhaps the main reason for this finding is our cohort of stroke participants did not have shoulder pain prior to their stroke (this was a specific exclusion criterion) and only 10 participants developed pain over the short investigation period. It is possible that more of our participants may have developed HSP or experienced increased severity of their HSP had a longer follow-up period been used and this may have influenced the GHJC position. Interestingly, more women experienced HSP compared to men in the current study at baseline (26% vs 6.7%) and week six (26% vs 8.7%). The relationship between gender and HSP has received little investigation; with studies reporting little (Demirci et al., 2007) or no relationship between HSP and gender (Aras et al., 2004; Barlak et al., 2009; Suethanapornkul et al., 2008).

There is conflicting evidence for the association of glenohumeral subluxation and HSP. Barlak et al. (2009) found that 71/108 (66%) patients with radiographic evidence of GH subluxation reported HSP compared to 37 (34%) of patients who had no pain despite evidence of GH subluxation. In contrast, Aras et al. (2004), Demirci et al. (2007), and Paci et al. (2007) found only 50% of patients with HSP had evidence of GH subluxation, suggesting that patients with GH subluxation have equal odds of experiencing, or not experiencing, HSP following a stroke. Similarly, while some studies report an association between soft tissue changes in the shoulder (e.g. rotator cuff tendinopathy), as identified using ultrasound assessment, and poor motor recovery and HSP following stroke (Aras et al., 2004; Demirci et al., 2007; Huang et al., 2010), the diagnosis of shoulder pathology such as subacromial impingement or capsulitis cannot be made on the basis of ultrasound or radiographic evidence alone (Carter et al., 2012; Hanchard et al., 2013). Therefore, there is insufficient evidence to confirm an association between local tissue pathology or biomechanical subluxation in the shoulder and HSP.

5.6.2 Age and gender

Age did not influence GHJC position in either direction at baseline or week 6. The influence of age on the head of humerus position post-stroke has not previously been

explored, although it is reasonable to suggest that age-related changes in the shoulder may affect GHJC position post-stroke. Some studies have reported age-related changes in scapula position during shoulder elevation in both healthy (Cutti et al., 2014) and shoulder pathology populations (Fayad et al., 2008; Hébert, Moffet, McFadyen, & Dionne, 2002; McClure, Michener, & Karduna, 2006). These changes include increased scapular retraction, lateral rotation and posterior tilting during shoulder elevation. While we did not measure scapular position relative to the thorax in the current study, scapular kinematics are considered an important factor in the development of shoulder pain in non-stroke populations (Fayad et al., 2008; Ludewig & Cook, 2000), and may therefore be important in the development of HSP in people with stroke. Further research is needed to investigate this relationship.

Gender did not significantly influence GHJC position at baseline or week 6 with one exception. GHJC in the AP direction on the non-hemiparetic side at week 6 in men was positioned anteriorly to the acromion in contrast to being positioned posteriorly in the women. Only general reference has been made previously to different upper limb outcomes following stroke for men and women. Men who survived right hemisphere strokes reported better upper limb recovery and functional outcomes compared to women (Coupar et al., 2012; Patrick et al., 2012). This may also be attributed to hemiparetic shoulder pain being more prevalent in women (Demirci et al., 2007). Improved upper limb recovery and function with a lower frequency of shoulder pain in men post-stroke may be factors associated with improved ability to centre the humeral head in the glenoid cavity during upper limb movement (Abreu, Buford, Nakamura, Ottenbacher, & Patterson, 2008; Nakamura et al., 2008). If this is the case, our finding of the anteriorly positioned GHJC in men relative to women is consistent with previous research, which have found the GHJC lies anterior to the acromion in healthy populations (Labriola, Lee, Debski, & McMahon, 2005; Meskers et al., 2005).

5.6.3 Stroke affected side

This is the first study to identify a potential relationship between the side affected by stroke (right vs left) and GHJC. In participants with left-sided hemiparetic, the GHJC was positioned closer to the acromion than participants with right-sided hemiparesis. The implication is that the GHJC in participants with right-sided hemiparesis more closely approximates to a 'normal position' compared to healthy control data, and may therefore be at less risk of experiencing subacromial impingement compared to participants with left-sided hemiparesis.

A reduced subacromial space, as indicated by the smaller SI value, suggests that participants with left sided hemiparesis may have an increased risk of shoulder pain, consistent with other studies which cite an association between left hemiparesis and HSP (Demirci et al., 2007; Ratnasabapathy et al., 2003). Perceptual deficits are more common in patients with right cerebral hemisphere strokes (i.e. left hemiparesis) (Vossel et al., 2013). Perceptual deficits result in loss or altered somatosensory input which may lead to changes in supraspinal nociceptive processing associated with pain perception (Klit et al., 2009). Pain perception and nociceptive processing changes may occur following stroke, consequent to prolonged upper limb immobility and tissue trauma due to poor motor control (Roosink, Renzenbrink, et al., 2012b). Altered pain perception and nociception may therefore play a role in the development of HSP (Demirci et al., 2007; Roosink, Renzenbrink, et al., 2012b).

Another factor associated with the side affected by the stroke is shoulder range of movement. Reduced passive range of shoulder movement has been reported in left-side affected strokes compared to right-side affected strokes and an association drawn with increased HSP when the group was followed up at four months post-stroke (Lindgren et al., 2012). The current study did not measure hemiparetic shoulder range of passive movement, so cannot be compared with the current literature. However, it is likely that the better positioning of GHJC in participants with left-sided hemiparesis in the current study would result in their having greater shoulder passive range than participants with right-sided hemiparesis. Another aspect which may influence the stroke affected side is whether it is the dominant hand or not.

5.6.4 Dominant side affected

Hand dominance appeared to have little effect on GHJC position in either the hemiparetic or non-hemiparetic sides at all stages during the measurement period. The only significant finding was that at baseline, only participants with their dominant side affected by the stroke had their GHJC on the non-hemiparetic side positioned significantly closer to the acromion in the SI direction than those who did not. Given that reduced subacromial space has been implicated in SAIS and shoulder pain, a reduced SI distance from the acromion on the non-hemiparetic side may be interpreted as a risk factor for development of shoulder pain. Interestingly, this difference between sides was no longer apparent at 6 weeks follow up.

Changes in the non-hemiparetic upper limb has previously been identified, but only during the subacute rehabilitation phase (Meskers et al., 2005; Nakamura et al., 2008;

Schaefer et al., 2009). To date, no studies have investigated changes in the non-hemiparetic limb in the acute phase post-stroke. The differences between sides in people whose dominant side was affected may be due to either a real change, or a measurement error, particularly as the side-to-side difference lies within the MDC (26.0 mm). Immediate changes on the non-hemiparetic side at baseline may occur through changes in inter-hemispheric communication and motor control following injury to the central nervous system (Lee & Jang, 2011; Robertson, Roche, & Roby-Brami, 2012). The lack of differences between sides at 6 weeks follow up may be interpreted as an improvement or normalisation in motor control and therefore positioning of the GHJC on the non-hemiparetic side. This is feasible, given the early therapeutic intervention participants received, consistent with national clinical guidelines for rehabilitation (National Stroke Foundation, 2010).

Interestingly, the GHJC on the hemiparetic dominant side was positioned more optimally than the non-hemiparetic side. Previous studies have reported less impairment and superior motor recovery and function following stroke, when the dominant side is affected (Harris & Eng, 2006; Mani et al., 2014; Rinehart et al., 2009). This is thought to be due to more frequent use of their hemiparetic arm during functional tasks, which are commonly performed with the dominant (in this case, affected) side. A future study with a larger sample size is required to confirm these findings and investigate the predictive capacity of affected dominant side on GHJC changes over time and the subsequent development of HSP.

5.6.5 Motor recovery

At baseline, the degree of motor recovery appeared to have no influence on GHJC position. However, at week 6 on the non-hemiparetic side, stroke participants who scored less than six on the MAS had GHJC positioned significantly posterior to the acromion in the AP direction, compared to those in the MAS group who scored six that had GHJC positioned anterior to the acromion. There were no other significant relationships pertaining to motor recovery on the hemiparetic or non-hemiparetic sides at baseline or week 6.

The current study's participants' motor control on the hemiparetic side was only mildly affected by stroke which may have limited any changes occurring in GHJC position. Fourteen (47%) participants had good shoulder motor recovery at baseline (MAS score = 6) and an additional 6 (20%) participants were able to lift their arm to 90 degrees shoulder elevation. By week six 73.9% scored the maximum on the MAS. Coordinated

activity of the rotator cuff muscles is responsible for accurate coupling of the humeral head and glenoid cavity at rest and during dynamic activity (Chopp et al., 2010; Lam et al., 2007). With this high level of motor control, it would be anticipated that the majority of participants would have some ability to centre the humeral head in the glenoid cavity and so maintain a reasonable GHJC position, similar to that of the healthy controls.

This study was not powered for subgroup analysis; and with the low numbers of stroke participants (n = 9, 30%) with poor motor recovery (MAS score ≤ 3 , not able to move the shoulder against gravity), no significant difference was found in GHJC positions in the AP or SI direction at baseline or 6-week follow-up on either side compared to stroke participants with good motor recovery.

The possibility exists in the current study that stroke had a more adverse impact on the non-hemiparetic side in the poor motor recovery group than in the good motor recovery group at week six. Changes on the non-hemiparetic side and the implications of these have previously been discussed (section 5.4.2); with the current study lending support to such changes occurring at least in those with poor motor recovery following stroke.

Clinical implications arise from this changes GHJC position on the non-hemiparetic side. The non-hemiparetic shoulder can be impacted by the stroke, and as a result, post-stroke assessments should include detailed evaluation of the non-hemiparetic limb rather than assuming it to be unaffected by the stroke. Evaluation should include tasks involving coordination of the unilateral non-hemiparetic upper limb as well as bilateral upper limbs, designed to detect more subtle movement disorders. Therapeutic interventions should be directed towards addressing the more subtle deficits on the non-hemiparetic side in addition to interventions (such as constraint induced movement) directed towards maximising outcomes on the hemiparetic side.

5.6.6 Muscle tone changes

Muscle tone changes did not influence GHJC position in either direction at baseline or week 6. Although not powered for subgroup analysis, this may also have been as a result of our mildly affected group of participants with stroke, of whom less than 17% had tonal changes at baseline and fewer again at week six. Studies which have reported the impact of tonal changes have generally had participants with stroke who have been more severely affected by the stroke. The influence of muscle tone on GHJC post-stroke has not previously been explored. However, the effect of tonal changes on the hemiparetic shoulder and upper limb has been investigated.

A significant relationship between increased muscle tone (spasticity) and subluxation was found in a subacute, poor motor functioning group of stroke participants together with an increased incidence of soft tissue lesions (Huang et al., 2010). Spasticity, more specifically in the shoulder adductors, has been related to shoulder subluxation in a group of acute post-stroke participants (Daviet et al., 2002). Contrary to this finding, spasticity is also thought to reduce subluxation by enabling the supraspinatus muscle to respond to loading of the glenohumeral joint (Chaco & Wolf, 1971). Spasticity together with subluxation have been associated with complex regional pain syndrome (also known as shoulder hand syndrome) though the mechanism is uncertain (Kocabas, Levendoglu, Ozerbil, & Yuruten, 2007; Pertoldi & Di Benedetto, 2005).

Spasticity has been linked to poor upper limb function post-stroke. Moderate to severe spasticity has been reported to be associated with markedly impaired upper limb activity, reduced function (Kong et al., 2012; Marciniak, 2011; Rosales, Kanovsky, & Fernandez, 2011) and reduced passive shoulder range of movement (Lo et al., 2003; Teasell, Foley, Pereira, Sequeira, & Miller, 2012). Reduced upper limb activity and function as a result of spasticity may lead to contractures (Ada, O'Dwyer, & O'Neill, 2006; Kwah, Harvey, Diong, & Herbert, 2012; Rosales & Chua-Yap, 2008) which further impact function. Prolonged spasticity may result in poorly synchronised muscle activity around the shoulder or unbalanced scapulothoracic rhythm that may predispose a person to shoulder soft tissue damage (Kalichman & Ratmansky, 2011).

Clinicians need to be aware of the need to assess changes in muscle tone over the course of the rehabilitation period. Clinicians need to be cognisant of the potential impact altered muscle tone may have on changes in positioning within the glenohumeral joint, the effect on surrounding muscles and soft tissues, as well as its adverse consequence on hemiparetic upper limb range of movement, activity and function.

5.7 Generalisability

The average age of both the acute stroke and age-matched control participants was 65.1 years. Ninety-three percent of stroke participants had an ischaemic stroke and 60% had their left sides affected by the stroke. These characteristics are consistent with other studies involving measurement of humeral and scapular orientation post-stroke (Culham et al., 1995; Meskers et al., 2005; Niessen et al., 2008). Medical imaging studies involving the glenohumeral joint post-stroke also have cohort characteristics comparable to our study (Barlak et al., 2009; Beebe & Lang, 2009;

Demirci et al., 2007). Time post-stroke distinguishes the current study from those involving measurement of the shoulder joint using ETD or medical imaging in stroke populations (Barlak et al., 2009; Demirci et al., 2007; Kumar et al., 2011; Niessen et al., 2008). The majority of participants in these previous studies were in the subacute or chronic stages post-stroke (range 3 months to several years), with the exception of one study which had recruited participants with a mean time of 18.6 (SD 5.6) days post-stroke to investigate shoulder active range using an ETD (Beebe & Lang, 2009).

Some differences are apparent between participants in the current study and those of previously reported literature with respect to gender, presence of hemiparetic shoulder pain, degree of motor recovery and muscle tone changes. Australian population-based studies report stroke patients to be approximately 60% male (Islam et al., 2008; Thrift et al., 2009) which is different to the 40% of males recruited to the current study. Reasons underlying the gender difference in the current study are not clear; men may have had more severe strokes and experienced a greater mortality rate than women. Hospital data on mortality rate following acute stroke was not collected during the current study period. A reduced representation of men in this stroke cohort may have been due to men being less inclined to participate in research. However, of the nine eligible volunteers who declined to participate, four ((44%) were men, indicating that men were not more likely to decline than women. On the other hand, women have a greater risk of stroke due to their longer life expectancy (Seshadri et al., 2006), and given that women in the current study were older than men, this may have increased the representation of women.

Differences in other demographic and clinical characteristics with previous literature should be regarded with caution due to the small numbers of participants with hemiparetic shoulder pain, poor motor recovery and muscle tone changes in the current study. HSP rates in two recent literature reviews were cited as varying from 5% to 84% (Kalichman & Ratmansky, 2011; Kumar et al., 2013). One-third of the current study's participants reported HSP which is comparable to studies involving more acute stroke participants (Gamble, Barberan, Bowsher, Tyrrell, & Jones, 2000; Hadianfard & Hadianfard, 2008; Lindgren et al., 2007; Roosink, Renzenbrink, et al., 2011). Severity of HSP (mean VAS 18.6 mm SD 31.0) was also comparable to the current study with only a single study investigating pain severity in acute to subacute stroke patients (mean VAS 24.5 mm SD 28.0) (Huang et al., 2010).

HSP has previously been linked to poor motor recovery (Blennerhassett et al., 2010; Huang et al., 2010; Kalichman & Ratmansky, 2011; Pong et al., 2012) and muscle tone

changes (Kalichman & Ratmansky, 2011). Overall, few participants in the current study had poor motor control or altered muscle tone at the time of recruitment. Selection criteria for the current study limited the inclusion of people with stroke to those who had sufficient sitting balance and endurance to be able to sit for 30 minutes at a time for a total of two hours. These criteria were necessary for data collection techniques to be performed, but resulted in those with poor motor recovery being excluded. Therefore, we cannot rule out that poor motor recovery and muscle tone changes may impact GHJC position.

A systematic review of studies reporting recovery of motor control post-stroke found that most participants had moderate to severe paresis at study admission (Hendricks et al., 2002). In a recent review of motor recovery prediction tools, report was made of motor arm scores of the National Institutes of Health Stroke scale in a study of 360 participants two weeks after a stroke (Stinear, 2010). The majority of participants had no movement and were unable to maintain or achieve hemiparetic upper limb movement against gravity (Stinear, 2010). By contrast, overall motor recovery in the current study was relatively good with almost half of the cohort making a full motor recovery by six weeks. Likewise, stroke participants' tonal changes in the current study were minimal in comparison with other studies. At baseline, less than 17% of stroke participants had tonal changes. Moderate tonal changes are frequently reported in stroke participant studies with up to 100% of stroke participants with HSP in the subacute phase having muscle tone changes (Pompa et al., 2011). The rate of spasticity, for example, has been reported to be as high as 25% within two weeks of stroke (Wissel et al., 2010). The reason for the low rate of tonal changes in the current study is likely due to the inclusion criteria. People with acute stroke who met the inclusion criteria of sufficient sitting balance, endurance and communication skills were more likely to be less severe and therefore have no tonal changes.

As the current study's stroke cohort appears to differ in severity from other studies, the GHJC changes found may be more applicable to patients with mild physical deficits and minimal HSP. It is possible that the position of the GHJC in both the AP and SI direction in those people with a more severe stroke (i.e. less motor recovery, more muscle tone changes) and HSP may be different from the current study's findings. Given that subluxation of the hemiparetic shoulder is a common feature in people with a severe stroke (Blennerhassett et al., 2010; Kumar et al., 2010; Paci et al., 2007; Stolzenberg et al., 2012), the GHJC position in those people may demonstrate features such as more significant inferior and anterior movement of the humeral head within the

glenohumeral joint (anteroinferior subluxation) over time. Greater changes in GHJC may result in increased levels of HSP in people more severely affected.

5.8 Clinical implications

The clinical relevance of GHJC position in stroke participants compared to control participants at baseline and the change in GHJC position in stroke participants at week six as well as the difference between stroke and control participants are manifold.

Significant difference between the stroke hemiparetic and non-hemiparetic GHJC position in the AP direction at baseline indicate that changes in GHJC position occur very early post-stroke. As a result, clinicians need to be aware of these early positional changes and the potential for this to influence the development of HSP, anterior humeral subluxation and glenohumeral impingement.

The GHJC was positioned closer to the acromion in the stroke group compared to the control group at baseline and week six, which bears similarity with musculoskeletal changes in the glenohumeral joint in non-stroke populations. Similarities can be drawn with SAIS in older populations (Jia, Ji, Pannirselvam, Petersen, & McFarland, 2011; Kanatli et al., 2013; Tobola et al., 2009). In older populations with SAIS, shoulder pain has been associated with soft tissue impingement within the subacromial space during shoulder movement (Yamaguchi et al., 2006) and rotator cuff muscle weakness or imbalance when initiating a change in the position of the humeral head relative to the glenoid fossa (Chopp et al., 2010; Hanchard et al., 2004). The change in humeral head position leads to disruption of the scapulohumeral rhythm (Chopp et al., 2010; Ludewig & F., 2009) with potential for damage of other soft tissues such as tendons, bursae, joint capsule and glenoid labrum (Tobola et al., 2009; Yamaguchi et al., 2006).

Changes in humeral head position such as those that occur in SAIS and subluxation have been cited as two of the possible contributors to HSP (Aras et al., 2004; Barlak et al., 2009; Chae & Jedlicka, 2009; Huang et al., 2010; Kumar et al., 2013). However, the aetiology of HSP is thought to be multifactorial with no one dominant factor (Kalichman & Ratmanský, 2011; Roosink, Renzenbrink, Geurts, & Ijzerman, 2012a; Roosink, Renzenbrink, et al., 2012b). HSP incidence is frequently low in the acute phase post-stroke (Ratnasabapathy et al., 2003; Roosink, Renzenbrink, et al., 2011), increasing during the subacute and chronic phases (Aras et al., 2004; Demirci et al., 2007; Lindgren et al., 2012). Few stroke participants in the current study developed HSP. Several possible reasons might account for these findings. Participants in the current

study were recruited early, within 2 weeks post stroke when research indicates that the incidence of HSP is low. Furthermore, participants were only followed up for six weeks from baseline, which might still be considered part of the acute phase following stroke. It is possible that there may have been a greater incidence of HSP if participants had been followed up for a longer time period.

Clinicians managing acute stroke patients are aware that prevention of HSP is of paramount importance. The National Stroke Foundation Clinical Guidelines for Stroke Management 2010 (National Stroke Foundation, 2010) inform us that positioning and handling are important to prevent shoulder subluxation and onset of shoulder pain. It is possible that staff adherence to and awareness of, best practice principles post-stroke have contributed to minimising the development of early onset shoulder pain in the current study. Clinicians need to employ best practice prophylactic guidelines in order to avoid the onset of HSP, monitor and assess the glenohumeral joint for signs of the development of HSP and seek appropriate interventions in the event of its development. Particular care needs to be taken during passive or active shoulder ranging, ensuring adequate humeral external rotation so as to avoid aggravating subacromial impingement.

People who had a right-sided stroke had better recovery towards a normal GHJC position compared to those with a left-sided stroke, perhaps because they use their dominant hemiparetic arm more (Mani et al., 2014; Rinehart et al., 2009) than patients with a non-dominant hemiparetic arm. This suggests that close attention needs to be given to patients with left (non-dominant) hemiparesis, to encourage functional use of their affected left upper limb. Increased functional use of their upper limb may be best achieved via constraint induced movement therapy (Khan, Oesch, Gamper, Kool, & Beer, 2011; Stevenson, Thalman, Christie, & Poluha, 2012; Wang, Zhao, Zhu, Li, & Meng, 2011) for patients with non-dominant upper limb hemiparesis.

The GHJC position was closer to the acromion on the non-hemiparetic side in our stroke group compared to the control group at six weeks, suggesting that the non-hemiparetic side is at risk of subacromial impingement following stroke. While the focus of rehabilitation is primarily on the hemiparetic upper limb, bilateral training, is increasingly recognised as being a vital element of rehabilitation (Haaland et al., 2012; National Stroke Foundation, 2010), due to inter-hemispheric cortical control of each side of the body (Robertson et al., 2012; Schwerin et al., 2008). National Stroke Foundation Clinical Guidelines for Stroke Management (National Stroke Foundation, 2010) recommend that interventions inclusive of bilateral upper limb activities be one of the future research priorities (Stoykov, Lewis, & Corcos, 2009). Changes seen in GHJC

position on the non-hemiparetic side in this study support the need to include the non-hemiparetic side in the rehabilitation program (Akinwuntan et al., 2005; Desrosiers et al., 1996; Nakamura et al., 2008). Long-term consequences of changes in GHJC position in the non-hemiparetic upper limb are unknown; however, if the non-hemiparetic arm is used more following stroke, then the risk of impingement (Harrison & Flatlow, 2011) is important for clinicians to consider as part of the rehabilitation program (Michaleff & Kamper, 2013).

Current physiotherapy neurological practices within stroke rehabilitation, such as functional task practice (Corti, McGuirk, Wu, & Patten, 2012; Winstein et al., 2004), task repetition (Blennerhassett & Dite, 2004), specificity and feedback (O'Dell, Lin, & Harrison, 2009; Van Peppen et al., 2004), focus heavily on interventions which maximise recovery via changes driven by neuroplasticity (Arya, Pandian, Verma, & Garg, 2011; Sterr & Conforto, 2012). Musculoskeletal conditions, that may be the underlying cause of HSP, and co-exist with neurological deficits, may often not receive appropriate assessment and treatment following stroke. Conventional musculoskeletal physiotherapy focuses on movement impairment without necessarily considering the potential of neuroplasticity to maximise neuromotor control (Snodgrass et al., 2014). Improved integration of neurological and musculoskeletal intelligence needs to occur so that patients post-stroke who present with a combination of these conditions are optimally managed (Snodgrass et al., 2014). National Stroke Foundation Clinical Guidelines for Stroke Management (National Stroke Foundation, 2010) recommend evidence-based musculoskeletal interventions for those patients who develop shoulder pain post-stroke.

5.9 Limitations

While we attempted to minimise bias and maximise the internal validity of this study, there are limitations that must be acknowledged. Patients with more severe stroke presentation were largely excluded in this study due to their physical incapacity to cope with the time required for data collection (approximately two hours), which impacted on participant eligibility. Additionally, patients were excluded if they had shoulder pain pre-existing or present during recruitment and screening. It was possible that the physical requirements for measuring GHJC used in this study may have placed undue burden on these patients. Further, for patients who were in the acute post-stroke phase, the time required for data collection makes this measurement tool suitable for research purposes only.

Inclusion of a global stroke scale such as the modified Rankin scale (Koudstaal, Visser, Schouten, & van Gijn, 1988) or the National Institute of Health Stroke scale (Brott et al., 1989; Dewey et al., 1999) would have assisted in quantifying the severity of stroke symptoms in this study. The current study relied only on specific physical measures such as the MAS for motor recovery and the Tardieu scale for muscle tone. Employment of a global stroke scale in the inclusion criteria may possibly have ensured that the study recruited participants with a range of stroke severities while also meeting other inclusion criteria.

The time required for data collection was determined by the instrumentation and methods employed. Instrumentation and methods required that participants be seated for at least 30 minutes at a time for a total time of two hours. Exploration of other instrumentation and methods, such as having participants in a reclined position during measurement, may have better suited an acute post-stroke population and enabled recruitment of participants with a wide range of stroke severity.

High measurement error associated with use of the ETD was not apparent during the data collection phase, but became evident during the data processing phase, when individual participant GHJC positional data points were missing. This reduced the availability of three trials for each participant for each session and hence the number of participants' data for analysis. As a result, the final number of participants with sufficient valid data available for analysis at baseline (N=27 per group) and week six (N=23 per group, including seven participants who were lost to follow-up for other reasons) was less than the *apriori* sample size calculations (N=30 per group). This reduced the power of the study. The original sample size was calculated on a minimal clinical difference in humeral head position change (Kumar et al., 2011). No data was available *apriori* regarding GHJC position change in normal or stroke populations. It is also possible that we underestimated the sample required as the variability and differences between hemiparetic and non-hemiparetic GHJC, and stroke and control groups, were not known. Additionally, our study was not powered to enable subgroup analyses (age, gender, side affected by the stroke, hand dominance, pain, motor recovery and muscle tone). The subgroup analysis undertaken within the current study was exploratory only. This study now provides more specific data to inform future sample size calculations. Sample size calculations may also need to account for approximately 25% loss of participant data when employing this measurement method.

In contrast to previous work (Monnet et al., 2007; Veeger, 2000), this study did not calculate the mediolateral position of GHJC relative to the acromion. Rather, this study

focussed on the AP and SI directions, as these are more commonly reported in both the stroke (Atalar et al., 2009; Kumar & Swinkels, 2009; Murie-Fernández et al., 2012) and musculoskeletal shoulder pain literature (Garofalo et al., 2010; Heyworth & Williams, 2009). Determining the mediolateral position may be relevant, particularly as it may complete the three dimensional representation of the glenohumeral joint position in the post-stroke population. This added information may shed more light on post-stroke glenohumeral joint complications such as HSP, impingement and subluxation.

The glenohumeral joint is formed by the scapula's glenoid fossa and the humeral head. The coordinated movement of the scapula and humerus, also known as scapulohumeral rhythm is vital to the integrity of the glenohumeral joint (Braman et al., 2009; Sang, Dae Suk, Ha Yong, & Won Sik, 2013). Disruption of the scapulohumeral rhythm may impact on GHJC position and result in development of shoulder pain (Fayad et al., 2008; Hallstrom & Karrholm, 2009; Rundquist et al., 2012). Though the current study determined GHJC relative to the acromion, measurement of the scapular position was not included. Measurement of changes in scapular position relative to the thorax, and with respect to changes in GHJC and the development of HSP in people following acute stroke, are needed to fill this gap in our knowledge base.

The low incidence of stroke participants experiencing shoulder pain in this study did not allow subgroup analysis at week six. Research indicates that shoulder pain, subluxation and impingement worsen in the subacute and chronic phases (Kalichman & Ratmansky, 2011; Kumar et al., 2013; Lindgren et al., 2007). This study was not powered to inform subgroup analyses regarding the relationship between age, gender, side affected by the stroke, hand dominance, pain, motor recovery or muscle tone. Therefore, future studies that include this in sample size calculations as well as include longer term follow up of the stroke group, may reveal more about the course of their shoulder pain and the relationship with position of GHJC.

Study time constraints and consideration of the time commitments involved for control participants resulted in the control group being measured only once. Control participants were recruited from various suburbs around Brisbane as well as the Gold and Sunshine Coasts. Each participant data collection session took four hours, which included equipment set-up and dismantling, preparation of materials and measurement. As a result, it was assumed that GHJC position in the healthy controls would remain stable and their baseline data was carried forwards for comparison with the matched stroke group at week six. It would have been ideal to include a second measurement session for control participants at week six.

5.10 Future directions

A number of indicators for future research became evident during the current research program. Some were as a result of the current study's limitations, such as measurement of the scapular position and the mediolateral position of the GHJC. Investigation of scapular position relative to GHJC position will complement the knowledge about GHJC gained during the current study. Post-stroke changes in scapular position have been investigated and reported in the literature (Hardwick & Lang, 2011b; Niessen et al., 2008; Rundquist et al., 2012), but have not been linked to GHJC position (Hardwick & Lang, 2011b; Niessen et al., 2008; Rundquist et al., 2012).

Aspects of HSP which the current study was unable to explore, require investigation, particularly with regards to the temporal aspect of the development of HSP (Kalichman & Ratmansky, 2011; Lindgren et al., 2012; Roosink, Renzenbrink, et al., 2011) and the sensory changes associated with HSP (Connell et al., 2008a; Roosink, Renzenbrink, et al., 2011; Roosink, Van Dongen, et al., 2012; Zeilig et al., 2013). Sensory changes post-stroke may include hyperalgaesia to a variety of sensory stimuli, including mechanical. This has been considered in non-stroke populations (O'Leary, Falla, Hodges, Jull, & Vicenzino, 2007; Teys, Bisset, & Vicenzino, 2008; Vicenzino, Collins, Benson, & Wright, 1998) and subacute (Motti, Ruth, & Nachum, 2012) and chronic (Soo Hoo, Paul, Chae, & Wilson, 2013) stroke populations, but in only one acute post-stroke population (Roosink, Van Dongen, et al., 2012). One method used to explore mechanical hyperalgaesia is pressure pain algometry (Chesterton, Sim, Wright, & Foster, 2007; Teys et al., 2008), which is quick and simple to administer in a clinical setting (Chesterton et al., 2007). Furthermore, widespread mechanical hyperalgaesia may be indicative of central sensitisation, which is considered a dysfunction in central pain processing (Arendt-Nielsen & Yarnitsky, 2009). Information gained about mechanical hyperalgaesia post-stroke may throw light on its role in the development of HSP, and help define the pain processes underlying HSP (Roosink, Buitenweg, Renzenbrink, Geurts, & Ijzerman, 2011; Roosink, Van Dongen, et al., 2012; Zeilig et al., 2013).

Changes in GHJC position post-stroke related to SAIS requires further investigation. This musculoskeletal condition is linked to shoulder pain (Demirci et al., 2007; Dromerick et al., 2006; Hardwick & Lang, 2011a), and even though the incidence and intensity of HSP had retreated by week six in the current study, our findings showed that GHJC was positioned closer to the acromion, which is consistent with signs of

subacromial impingement as a loss of subacromial space. Investigation of a larger and more severely affected group of patients post-stroke may confirm our finding and enable generalisation to all patients in the acute post-stroke phase. Longitudinal follow-up of GHJC position with regards to changes in glenohumeral joint impingement and its association with HSP over time, would add vital, pertinent information to the clinical and research knowledge base.

The current study found differences in the GHJC position of the stroke group with right versus left hemiparesis. GHJC position in those participants with right hemiparesis resembled that of the healthy controls more than those with left hemiparesis. Further research is required to confirm this finding within a larger post-stroke group and establish whether there are any differences in the way in which left and right hemiparetic post-stroke participants centre the humeral head in the glenoid cavity during active functional tasks such as forward reaching.

5.11 Summary

The current study is the first to investigate positional changes of GHJC in the acute post-stroke phase and to compare these changes to a healthy age, gender and BMI matched control group. Intra-tester reliability of a sphere-fitting method, using an ETD to measure the position of GHJC within two weeks of stroke onset and in a healthy age and gender matched population was established. The GHJC was positioned closer to the acromion in the stroke group compared to healthy controls, at baseline and week six. This is suggestive of a reduced subacromial joint space, which may predispose patients to the development of HSP, although HSP was not a significant factor in this stroke cohort. Furthermore, this difference in SI position was apparent in both the hemi- and non-hemiparetic shoulders in the stroke group.

Several gaps were apparent in the stroke literature, particularly with regard to acute post-stroke shoulder complex and specifically the glenohumeral joint. Glenohumeral joint pain (HSP) has been researched extensively, but uncertainty remains regarding its aetiology. Further research is required to investigate GHJC in a more severely affected acute stroke cohort, track GHJC changes over a longer time frame, and explore the association between GHJC and HSP. A larger acute post-stroke cohort is required to enable greater power and more significant findings. Post-stroke pain mechanisms such as central sensitisation and neuropathic pain characteristics using quantitative sensory testing such as pressure pain threshold, heat and cold pain thresholds need to be

investigated in the acute phase. The correlation of pressure pain threshold with the onset of HSP in particular will inform its aetiology.

Scapular position needs to be measured in the acute post-stroke phase in order to inform GHJC position more fully. Changes in scapular position over time (into subacute phase) need pursuing as well as its impact on GHJC changes and functional recovery of the upper limb. An improved understanding of GHJC, its course over time and its association with HSP and upper limb function will enable the development of therapeutic interventions that specifically target the glenohumeral joint and scapula position.

It is important for clinicians to assess and manage the position of the glenohumeral joint in both the hemi- and non-hemiparetic arms in patients following an acute stroke. Knowledge of both neuromotor and musculoskeletal factors associated with hemiparetic impairments is essential in developing and implementing effective management plans for these complex patients. Advancements in early therapeutic interventions to address shoulder complex problems post-stroke will give rise to improved upper limb functional outcomes and independence, greater community participation, reduced carer burden and enhanced quality of life.

Appendix 1. Ethical clearances

Royal Brisbane and Women's Hospital
Metro North Health Service District

Queensland
Government

Office of the Human Research Ethics Committees

Queensland Health

Enquiries to: Ann Marie Gordon
Admin Coordinator
Phone: 07 3636 5493
Fax: 07 3636 5649
Our Ref: HREC/10/QRBW/175
E-mail: BJRWHEC@health.qld.gov.au

Dr Leanne Bisset
School of Physiotherapy & Exercise Science
Griffith University
Gold Coast Campus
Gold Coast Qld 4222

Dear Dr Bisset,

Re: Ref N°: HREC/10/QRBW/175: Tracking changes in scapular position in acute post-stroke hemiplegia patients: an observational study

Thank you for submitting the above project for ethical and scientific review. This project was considered at the Royal Brisbane & Women's Hospital Human Research Ethics Committee (HREC) meeting held on 17 May, 2010.

I am pleased to advise that the Human Research Ethics Committee has granted approval of this research project on 14 July, 2010. HREC approval is valid for three (3) years from the date of this letter.

This HREC is constituted and operates in accordance with the National Health and Medical Research Council's (NHMRC) *National Statement on Ethical Conduct in Human Research (2007)*, *NHMRC and Universities Australia Australian Code for the Responsible Conduct of Research (2007)* and the *CPMP/ICH Note for Guidance on Good Clinical Practice*. Attached is the HREC Composition with specialty and affiliation with the Hospital (*Attachment 1*).

You are reminded that this letter constitutes ethical approval only. You must not commence this research project at a site until separate authorisation from the District CEO or Delegate of that site has been obtained.

A copy of this approval will also be sent to the District Research Governance Office (RGO). Please ensure you submit a completed Site Specific Assessment (SSA) Form to the RGO for authorisation from the CEO or Delegate to conduct this research at the Royal Brisbane & Women's Hospital Metro North District.

The documents reviewed and approved include:

The Royal Brisbane & Women's Hospital Human Research Ethics Committee is constituted and operates according to the NHMRC's National Statement on Ethical Conduct in Human Research (2007).

Office	Postal	Phone	Fax
Bethfield Street Herston Q 4029	Post Office Herston Queensland 4029 Australia	07 3636 5450 ISD +61 7 3636 5450	07 3636 2849

<i>Document</i>	<i>Version</i>	<i>Date</i>
Covering Letter		25 April 2010
Application: NEAF	Version 2.0 (2008)	26 April 2010
CTN Form (<i>requiring District approval</i>)		
Curriculum Vitae of Suzanne Kuys		
Curriculum Vitae of Peter Mills		
Curriculum Vitae of Natalie Collins		
Participation Information Sheet & Consent Form for Healthy Participants	1	23 April 2010
Grant approval letter from RBWH Foundation		06 December 2008
Screening Assessment Questionnaire for Stroke Group	1	23 April 2010
Screening Assessment Questionnaire for Healthy Group	1	23 April 2010
Research Funding Schedule (<i>reviewed in accordance with Section 3.3.18 of the National Statement</i>)		
Email recruitment advertisement to Griffith University staff and students		
Email recruitment advertisement to RBWH staff		
Mailout and newspaper recruitment advertisement for local community		
Noticeboard recruitment advertisement for community		
Noticeboard recruitment advertisement for hospital staff		
Noticeboard recruitment advertisement for University staff and students		
Tardieu Scale	1	23 April 2010
Pain Visual Analogue Scale	1	23 April 2010
Pain Map	1	23 April 2010
Motor Assessment Scale	1	23 April 2010
Response to Request for Further Information		12 July 2010
Protocol	2	05 July 2010
Response to Request for Further Information		13 July 2010
Participant Information Sheet & Consent Form (Stroke Group)	2	05 July 2010

Please note the following conditions of approval:

1. The Principal Investigator will immediately report anything which might warrant review of ethical approval of the project in the specified format, including:
 - Unforeseen events that might affect continued ethical acceptability of the project.

Serious Adverse Events must be notified to the Committee as soon as possible. In addition, the Investigator must provide a summary of the adverse events, in the specified format, including a comment as to suspected causality and whether changes are required to the Patient Information and Consent Form. In the case of Serious Adverse Events occurring at the local site, a full report is required from the Principal Investigator, including duration of treatment and outcome of event.

2. Amendments which do not affect either the ethical acceptability or site acceptability of the project (e.g. typographical errors) should be submitted in hard copy to the HREC Coordinator. These should include a covering letter from the Principal Investigator providing a brief description of the changes and the rationale for the changes, and accompanied by all relevant updated documents with tracked changes.
3. Proposed amendments to the research project which may affect both the ethical acceptability and site suitability of the project must be submitted firstly to the HREC for review and, once HREC approval has been granted, then submitted to the Research Governance Office.
4. Amendments to the research project which only affect the ongoing site acceptability of the project are not required to be submitted to the HREC for review. These amendment requests should be submitted directly to the Research Governance Office (by-passing the HREC).
5. Amendments to the research project which may affect the ongoing ethical acceptability of a project must be submitted to the HREC for review. Major amendments should be reflected in a revised online NEAF (accompanied by all relevant updated documentation and a covering letter from the Principal Investigator, providing a brief description of the changes, the rationale for the changes, and their implications for the ongoing conduct of the study). Hard copies of the revised NEAF, the cover letter and all relevant updated documents with tracked changes must also be submitted to the HREC Coordinator as per standard HREC SOP. Further advice on submitting amendments is available from http://www.health.qld.gov.au/ohmr/documents/researcher_userguide.pdf
6. The HREC will be notified, giving reasons, if the project is discontinued at a site before the expected date of completion.
7. The HREC will be notified, giving reasons, on any sponsor reports or other information which might affect the ongoing ethical acceptability in line with the requirements of the ICH GCP guidelines as annotated by the TGA: <http://www.tga.gov.au/docs/pdf/euguide/ich/ich13595.pdf>
8. The Principal Investigator will provide an Annual Report to the HREC and at completion of the study in the specified format.
9. The District Administration and the Human Research Ethics Committee may inquire into the conduct of any research or purported research, whether approved or not and regardless of the source of funding, being conducted on Hospital premises or claiming

any association with the Hospital, or which the Committee has approved if conducted outside Royal Brisbane & Women's Hospital Metro North Health Service District.

Should you have any queries about the HREC's consideration of your project please contact the HREC Coordinator on 07 3636 5490. The HREC terms of Reference, Standard Operating Procedures, membership and standard forms are available from http://www.health.qld.gov.au/ohmr/html/regu/regu_home.asp

Once authorisation to conduct the research has been granted, please complete the Commencement Form (*Attachment II*) and return to the office of the Human Research Ethics Committee.

The HREC wishes you every success in your research.

Yours sincerely,

Dr Conor Brophy
Chairperson RBWH Human Research Ethics Committee
Metro North **District**
14.07.2010

Dr Leanne Bisset
Physiotherapy Department
Royal Brisbane and Women's Hospital
Herston Road
HERSTON QLD 4006

Human Research Ethics Committee Queensland Health
The Prince Charles Hospital
Metro North Health Service District
Rode Road,
Chermside QLD 4032

Executive Officer (07) 3139 4500
Research & Ethics Ph:

Office Ph: (07) 3139 4691

Fax: (07) 3359 5756

Our Ref: PL/JL/ Expedited Approval

27 September 2010

Dear Dr Bisset,

Re: HREC/10/QPCH/137: Tracking changes in scapular position in acute post-stroke hemiplegia patients: an observational study. L. Bisset; S. Kuys; P. Mills; J. Grayson; P. Choolun.

I am pleased to advise that The Prince Charles Hospital Human Research Ethics Committee reviewed your submission and upon recommendation, the Chair has granted expedited approval for your research project.

Approval of this project is subject to the same confidentiality and privacy requirements as apply to other research projects and research subjects are not recognisable in publications or oral presentations.

Please complete the Commencement Form before starting your study and return to the office of the Human Research Ethics Committee.
http://www.health.qld.gov.au/northside/documents/form_notification

If you intend to publish the results of your work, it is advisable to ascertain from prospective journal editor/s the actual requirements for publication e.g. some journals may require full ethical review of all studies. When results are published, appropriate acknowledgment of the hospital should be included in the article. Please forward copies of all publications resulting from the study for inclusion in the Internet website list.

On behalf of the Human Research Ethics Committee, I would like to wish you every success with your research endeavour.

Yours truly,

Philip Lee, MBA (UQ); BAppSc (QUT); FRCNA; AFAIM
Executive Officer - Research, Ethics and Governance Unit
Email: Philip_Lee@health.qld.gov.au

Office	Postal	Phone	Fax
The Prince Charles Hospital	Rode Road Chermside Q 4032	(07) 3139 4500/3139 4691	(07) 3359 5756

Nov-2010

Dear Mrs Choolun

I write further to the additional information provided in relation to the provisional approval granted to your application for ethical clearance for your project "Prior Review: Tracking changes in scapular position in acute post-stroke hemiplegia patients: an observational study" (GU Ref No: PES/32/10/HREC).

The additional information was considered by Office for Research.

This is to confirm that this response has addressed the comments and concerns of the HREC.

Please correct the URL for the Privacy Plan to <http://www.griffith.edu.au/privacy-plan> as per changes within the PVC (A) website.

Consequently, you are authorised to immediately commence this research on this basis.

The standard conditions of approval attached to our previous correspondence about this protocol continue to apply.

Regards

Karen Moorehead

Office for Research
N54 2.39 Nathan Campus
Griffith University
ph:
fax: 07 5552 9058
email: k.moorehead@griffith.edu.au
web:

Cc:

At this time all researchers are reminded that the Griffith University Code for the Responsible Conduct of Research provides guidance to researchers in areas such as conflict of interest, authorship, storage of data, & the training of research students. You can find further information, resources and a link to the University's Code by visiting

<http://www62.gu.edu.au/policylibrary.nsf/xupdatemonth/e7852d226231d2b44a25750c0062f457?opendocument>

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REPORT TO GOVERNING COMMITTEE

Topic	Research Project Title: Tracking scapular position in acute post-stroke hemiplegia patients: an observational study.
Program/Facility	Royal Brisbane and Women's Hospital
Service Area or Functional Group	Acute Stroke patients transferred for rehabilitation to Brighton Rehabilitation Unit

Risk Rating:Low

Date: 1/11/10

Accountable Officer: - Executive Director – SA&RCS

Risk Register Ref No

Report Prepared by Praline Choolun

Position Team Leader Physiotherapist

Issue(s)

- Patients who have consented to participate in the research project while inpatients at the Royal Brisbane and Women's Hospital or The Prince Charles Hospital, may be transferred to Brighton Rehabilitation Unit prior to the six week follow up period of the project.
- As a result the project is required to collect these participants' data after their transfer to Brighton Rehabilitation Unit.
- Permission is being sought to collect the required participant data (participants would already have had explanations regarding this follow up after transfer to a rehabilitation facility and would have had to consent to this).

Background

- Cardiovascular disease has been identified as one of the National Health Priority areas, with stroke second only to coronary heart disease as a leading cause of disability in Australia.[3-5] The significant financial, emotional and social impact of stroke occurs at both the individual and community level. 230 300 Australians reported one or more disabling conditions associated with the stroke, while 77% of these required assistance or had difficulties with mobility, self care or communication.[6] Furthermore, the cost of stroke to Australia is estimated at \$2.14 billion per annum. [3]

Stroke most frequently manifests as paresis of one side of the body (hemiplegia). The natural progression of hemiplegia over time is characterised by changes in muscle tone, weakness and loss of normal range of movement.[7] Immediately following a stroke, there is an initial flaccid paralysis in over 90% of individuals, which is then replaced by reduced selective activation and control of muscles, and spasticity in some cases. This may vary in onset from 24 hours to 12 months. [10] It is known that adaptive changes of muscles about the shoulder girdle rapidly occur as a consequence of muscles resting in either shortened or lengthened positions. [8] These changes in motor control and

muscle tone may affect shoulder girdle posture including humeral head and scapula positioning.

Hemiplegic shoulder pain (HSP) is a common problem in stroke, affecting up to 70% of stroke patients.[1, 2] Despite this, the underlying aetiology of HSP remains unclear [7]; specifically, the relative contribution of the glenohumeral joint (GHJ) and scapula. Several studies have demonstrated a relationship between HSP and reduced shoulder external rotation range of motion (ROM) [9-11], while an association between spasticity and HSP has also been reported. [7] Despite this, and that a number of papers propose a link between spasticity, loss of shoulder external rotation ROM and HSP, the evidence is either conflicting or the studies are of poor methodological quality.[7] Whilst inferior subluxation of the humerus is well documented [12], the presence of anteroposterior subluxation at the GHJ is unclear. It is feasible that adaptive changes and/or spasticity of muscles that directly attach to the humerus may result in mal-alignment of the humeral head in the glenoid fossa. Clinically, it is proposed that anterior displacement of the humerus in the fossa is associated with subacromial impingement in an otherwise healthy population [13], and may also contribute to HSP in the stroke population.

Authors have argued that alterations in scapular position may affect scapular stability and the generation of muscular forces, as coordinated muscle patterns are believed to be necessary for normal shoulder function and muscle force production. [14-17] Previous studies that looked at changes in scapula position either used populations that were at least six months post-stroke [18-20], or did not specify duration following stroke.[21] One observational study compared the resting position of the scapula in a small population of stroke patients to healthy controls, and determined that, six months post-stroke, GHJ subluxation was minimally associated with scapular resting position.[19] Another study that investigated the effect of muscle tone on motor recovery pattern in hemiplegia, found that motor recovery occurred earlier and plateaued earlier in patients with spasticity, compared to those with flaccidity.[22] Although these studies provide preliminary data, the impact of adaptive changes of scapular muscles due to resting position, and the effect of muscle tone on shoulder pain and subluxation, remains unclear.[22]

In summary, there remain substantial gaps in the knowledge base regarding the aetiology of HSP. The relationship between changes in muscle tone, shoulder ROM and positional changes of the scapula and GHJ is unclear. Furthermore, no study to date has assessed changes in scapula position in the acute phase post-stroke. It is therefore unknown at what point post-stroke scapula position starts to alter, or whether changes in the acute phase are associated with the onset of HSP. Finally, the relationship between changes in scapula position and GHJ axis of rotation during the acute phase post-stroke is unknown.

Scientific reference list

1. Ward, A.B., Reducing muscle tone in the subscapularis muscle with botulinum toxin can help in the treatment of shoulder pain in patients with spastic hemiplegia. *Journal of Neurology, Neurosurgery, and Psychiatry*, 2007. **78**: p. 789.
2. Lindgren, I., et al., Shoulder pain after stroke: a prospective population-based study. *Stroke*, 2007. **38**(2): p. 343-8.
3. Australian Bureau of Statistics, Cardiovascular Disease in Australia: A Snapshot, 2004-05 ABS, Editor. 2006: Canberra.
4. Najenson, T., E. Yacubovich, and S.S. Pikielni, Rotator cuff injury in shoulder joints of hemiplegic patients. *Scand J Rehabil Med*, 1971. **3**: p. 131-137.

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5. Fitzgerald-Finch, O.P. and I.I. Gibson, Subluxation of the shoulder in hemiplegia. *Age Aging*, 1975. **4**: p. 16–18.
6. Australian Bureau of Statistics, Disability, Ageing and Carers: Summary of Results, Australia, 2003, ABS, Editor. 2004: Canberra.
7. Turner-Stokes, L. and D. Jackson, Shoulder pain after stroke: a review of the evidence base to inform the development of an integrated care pathway. *Clinical Rehabilitation*, 2002. **16**: p. 276–298.
8. Ada, L., C.G. Canning, and S.-L. Low, Stroke patients have selective muscle weakness in shortened range. *Brain*, 2003. **126**(3): p. 724–31.
9. Bohannon, R.W., et al., Shoulder pain in hemiplegia: statistical relationship with five variables. *Arch Phys Med Rehabil*, 1986. **67**: p. 514–16.
10. Cheng, P.T., et al., Risk factors of hemiplegic shoulder pain in stroke patients. *Journal of Musculoskeletal Pain*, 1995. **3**: p. 59–73.
11. Wanklyn, P., A. Forster, and J. Young, Hemiplegic shoulder pain (HSP): natural history and investigation of associated features. *Disability and Rehabilitation*, 1996. **18**: p. 497–501.
12. Zorowitz, R.D., et al., Shoulder subluxation after stroke: a comparison of four supports. *Arch Phys Med Rehabil*, 1995. **76**(8): p. 763–771.
13. Hess, S.A., Functional stability of the glenohumeral joint. *Manual Therapy*, 2000. **5**(2): p. 63–71.
14. Glusman, R.E., et al., Dynamic electromyographic analysis of the throwing shoulder with glenohumeral instability. *J Bone Joint Surg Am*, 1988. **70**: p. 220–226.
15. Moseley, B.J., et al., EMG analysis of the scapular muscles during a baseball rehabilitation program. *Am J Sports Med*, 1992. **20**: p. 128–134.
16. Paine, R.M. and M. Voight, The role of the scapula. *J Orthop sports Phys Ther*, 1993. **18**: p. 386–391.
17. Popen, N.K. and P.S. Walker, Normal and abnormal motion of the shoulder. *J Bone joint Surg Am*, 1976. **58**: p. 195–201.
18. Culham, E.G., R.R. Noce, and S.D. Bagg, Shoulder complex position and glenohumeral subluxation in hemiplegia. *Arch Phys Med Rehabil*, 1995. **76**: p. 857–64.
19. Price, C.I., et al., Glenohumeral subluxation, scapula resting position, and scapula rotation after stroke: a noninvasive evaluation. *Arch Phys Med Rehabil*, 2001. **82**: p. 955–960.
20. Prevost, R., et al., Rotation of the scapula and shoulder subluxation in hemiplegia. *Arch Phys Med Rehabil*, 1987. **68**(11): p. 786–90.
21. Arsenaault, A.B., et al., Clinical Significance of the V-Shaped Space in the Subluxed Shoulder of Hemiplegics. *Stroke*, 1991. **22**(7): p. 867–871.
22. Formisano, R., et al., Late Motor Recovery Is Influenced by Muscle Tone Changes After Stroke. *Arch Phys Med Rehabil*, 2005. **86**: p. 308–311.

Implications

- Stroke has a significant financial, emotional, physical and social impact at both the individual and community level. The clinical implications of this research are twofold. An improved understanding of the role that changes in scapular and glenohumeral joint position

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have in the development of hemiplegic shoulder pain may assist in the development of effective interventions to prevent and treat this debilitating condition.

For the individual, this may improve their functional ability, independence and quality of life, and enable them to interact with and contribute more substantially to their community. Furthermore, the development of efficacious interventions, as well as decreased reliance on health care services for support and equipment will significantly reduce the financial cost of stroke to the broader community.

Actions / Recommendations / Solutions

- Human Research Ethics approval has been obtained from the Royal Brisbane and Women's and The Prince Charles Hospital. Permission is being sought from the Governing Committee at Brighton Rehabilitation Unit to follow through and complete data collection with those few research participants who are transferred to the facility before the final week of data collection (six weeks post-stroke).

Governing Committee Use Only			
Have each of the recommendations been addressed?	☐ Yes	☐ No	
Does the committee agree that this is a "Risk"?	☐ Risk	☐ Opportunity	
Does it need to go on the QH Risk Register?	☐ Yes	☐ No	
Executive Sponsor Nominated:			
Are there any other stakeholders who need feedback on this item? If yes, Who?	☐ Yes	☐ No	
Is there anything else the Governing Committee needs to do? If yes, what?	☐ Yes	☐ No	
Governing Committee Recommendation		Date	Date of meeting & Ref No

Details of recommendation made at Management Committee meeting (excerpt from meeting summary provided by secretariat)

Minute to be prepared and forwarded to:

Staff member who prepared report

Executive Sponsor

Quality Facilitator

Stakeholders/Committee identified

Executive Director Decision (if applicable)

Executive Director Comments:

Approved / Not Approved

Date 26.11.0

Signed (chair of governing committee)

Appendix 2. Participant information and consent forms

Royal Brisbane and Women's Hospital

Health Service District

Participant Information and Consent Form – participants with stroke Royal Brisbane and Women's Hospital

Full Project Title: Tracking changes in scapular position in acute post-stroke hemiplegia patients: an observational study.

Lay Project Title: Tracking the position of the shoulder blade in patients for the first six weeks after a stroke.

Principal Researcher:

Dr Leanne Bisset (Research Fellow/Senior Lecturer, Royal Brisbane and Women's Hospital, Griffith University)

Associate researchers:

Mrs Praline Choolun (Physiotherapist, Royal Brisbane and Women's Hospital)

Dr Peter Mills (Biomechanist/Senior Lecturer, Griffith University)

Dr Suzanne Kuys (Research Fellow, The Prince Charles Hospital, Griffith University)

Dr Jane Grayson (Research Fellow, Physiotherapy, Royal Brisbane and Women's Hospital, Griffith University)

1. Introduction

You are invited to take part in this research project. The research project aims to track shoulder and arm movement so that we are able to investigate the reasons for the development of pain in the shoulder after a stroke and assist with the development of effective treatments for patients with this problem.

For this project, we require participants who have had a stroke as well as healthy participants of a similar age, so that we can compare what happens to the shoulders of healthy people to what happens after a stroke.

This Participant Information and Consent Form tells you about the research project. It explains what is involved to help you decide if you want to take part.

Please read this information carefully. Ask questions about anything that you don't understand or want to know more about. Before deciding whether or not to take

part, you might want to talk about it with a relative, friend or your local health worker.

Participation in this research is voluntary. If you don't wish to take part, you don't have to.

If you decide you want to take part in the research project, you may be asked to sign the consent section. By signing it you are telling us that you:

- understand what you have read;
- consent to take part in the research project;
- consent to be involved in the procedures described;
- consent to the use of your personal and health information as described.

You will be given a copy of this Participant Information and Consent Form to keep.

2. What is the purpose of this research project?

- **Shoulder pain is a common problem after stroke**, yet the **reason** for this condition and the **evidence for the best way to manage it are not clear**.
- This project aims to investigate changes in the position of the shoulder after a stroke in order to improve our understanding of **factors which contribute** to the development of **shoulder pain**.
- For this project we will **measure the position of structures around the shoulder**, as well as the **level of pain, movement and spasticity** around the shoulder, and **track** these measures during the **first week**, and then at **three and six weeks** immediately after a stroke.
- We will recruit **26 post-stroke** participants, and another **26 healthy** participants who are matched in terms of age and gender to those who have had a stroke.
- Post-stroke participants will be recruited from the **Royal Brisbane and Women's Hospital (RBWH)** and **The Prince Charles Hospital (TPCH)** Stroke Units and general medical wards, but will be **followed up at three** and then at **six weeks after their strokes**, when they may be transferred for rehabilitation to the Rehabilitation Unit at the RBWH, TPCH, the Brighton Rehabilitation facility of TPCH, or the Day Hospital at Rosemount (RBWH) or TPCH.
- This study involves researchers from the Royal Brisbane and Women's Hospital, Griffith University and University of Queensland working in collaboration. A grant from the Royal Brisbane and Women's Hospital Foundation Research will fund this project.
- The results of this research will be used by Mrs Praline Choolun to obtain a **Masters of Research at Griffith University**.

3. What does participation in this research project involve?

- One of the researchers will **contact you in person** and **ask you a few questions** to determine if you are **suitable** to participate in the trial.
- You may be eligible to participate in this study if you are aged over 18 years and have been diagnosed with a hemiplegic stroke with arm impairment within the last seven days, are able to sit for at least 30 minutes at a time, weigh less than 150 kg, and prior to your stroke have no history of shoulder pain, shoulder

surgery, or any other condition that has affected your arm. Also, you will be excluded if you have a scoliosis (curvature in your back), are allergic to adhesive tape on your skin, or have any metal implants.

- You will be required to attend the physiotherapy gymnasium at the hospital where you will be an inpatient or outpatient, on **three occasions** in the first six weeks after your stroke.
- A **private cubicle** in the physiotherapy gymnasium will be used.
- The immediate area has to be **free of metal** so as not to affect the very sensitive measurement device being used. You will be asked to remove **all metal jewellery, glasses, hearing aids belt buckles and bras** that use metal clasps. It is best if you leave any valuable items in a locked area on the ward or with a family member.
- **Each session will take approximately 90 minutes**, during which time you will be **seated** on a chair with a low back rest.

- **The following measurements will be taken at each session:**

Position of your shoulder joint and shoulder blade:

- Specific areas will be marked with **adhesive tags** on the surface of the **skin** of your **arm, shoulder blade, spine** and **chest**.
- A small, light-weight **sensor** connected to an electronic tracking device will be attached to your **upper breast bone**, using **low-allergenic tape**, and another sensor attached to your **arm** using a **cuff**.
- A third **sensor** will be attached to a **plastic frame** which will be adjusted to fit over your shoulder blade. This will be **held over your shoulder blade** by the **researcher** while your **arm is at rest** by your side and while it is **being gently moved a small amount** in different directions **without causing you any pain**. A **series of measurements** will be recorded by the researcher, using a fourth **sensor** which **looks like a pointer**. It maps out the positions of your upper body and arm identified by the adhesive tags.
- These measurements will be repeated on your other arm.
- This information will be **recorded by a computer program** and will measure any changes in your shoulder after the stroke.

- Your **shoulder blades** and will be **measured** with a **standard tape measure**.

Pressure pain threshold:

- Using a **device** that **measures force**, the amount of pressure that **provokes the first onset of pain** will be measured over a site on your shoulder.

Severity of shoulder pain:

- Any **pain in your shoulder** will be marked **on a measurement scale**. Shoulder pain will need to be treated by your physiotherapist.
- Your **ability to move your shoulders** and any **spasticity** you may develop from the stroke will be assessed.
- The **information collected** will be **compared** with that of **healthy participants** who are of the **same age and gender** as you.
- Information regarding **your age, medical history, type of stroke** and **hand dominance** will be sourced from your medical record and collated with the measures we take.
- No part of this project will be video or audio-taped.
- You will **not be paid for your participation** in this research. We will pay the reimbursement of parking costs (up to \$10.00) for those participants attending The Prince Charles Hospital Day Hospital, on the occasions they attend for the research study only.

4. What are the possible benefits?

- Your **participation may benefit patients who have strokes in the future**, particularly those who **experience shoulder pain**. The information gathered from this project may allow them to have better function in the arm affected by the stroke and thereby enable them to better contribute to society.
5. **What are the possible risks?**
- **There are no risks associated with your participation in this research project** except for minimal shoulder pain while your arm is unsupported during measurement for up to 30 seconds. Every effort will be made to support your weak arm at all other times. The electronic tracking device emits a small amount of radiation which is well below international safety standards and is much less than that emitted by a television.
 - **If you become upset or distressed** as a result of your participation in the research, **the researcher is able to arrange for counselling or other appropriate support**. Any counselling or support will be provided by staff who are not members of the research team.
6. **Do I have to take part in this research project?**
- **Participation in any research project is voluntary**. If you do not wish to take part, you **do not have to**. If you decide to take part and later change your mind, you are **free to withdraw from the project at a later stage**.
 - If you decide to leave the project, the researchers would like to keep the personal and/or health information about you that has been collected. This is to help them make sure that the results of the research can be measured properly. If you do not want them to do this, you must tell them before you withdraw from the research project.
 - Your decision whether to take part or not, or to take part and then withdraw, will not affect your relationship with the researchers or the Royal Brisbane and Women's Hospital.
7. **How will I be informed of the final results of this research project?**
- Information obtained through completion of this project will be submitted to a peer reviewed journal for publication. You will be informed when these results are published. Please be aware that personal data collected about you will not be used to identify you in any publication arising from this research study.
8. **What will happen to information about me?**
- Data will be recorded on a printed form and transferred into an electronic database. **All data will be kept confidential and secure** in a locked cabinet of a private office for hard copy information and by restricted password for computerised information. Hard copy data will be shredded and computer files deleted after 15 years.
 - This research project does not involve the establishment of a databank.
 - **Any information obtained in connection with this research project** that can identify you **will only be used for the purpose of this research project**. It will only be disclosed with your permission, except as required by law. In any

publication and/or presentation, information will be provided in such a way that you cannot be identified, except with your permission.

- **Your consent is being sought for this project only.** Once the study is complete your name will be removed from the data to keep the information anonymous.
- 9. Can I access research information kept about me?**
- In accordance with Australian privacy and other relevant laws, **you have the right to access the information collected and stored by the researchers about you.** Please contact one of the researchers named at the end of this document if you would like to access your information.
 - In addition, in accordance with regulatory guidelines, the information collected in this research project will be kept for at least 15 years. Once the identifying factors in the information have been removed, you will no longer be able to access the information.
- 10. Is this research project approved?**
- The ethical aspects of this research project have been approved by the Human Research Ethics Committee of the Royal Brisbane and Women's Hospital.
 - This project will be carried out according to the *National Statement on Ethical Conduct in Human Research (2007)* produced by the National Health and Medical Research Council of Australia. This statement has been developed to protect the interests of people who agree to participate in human research studies.

Full Project Title: Tracking changes in scapular position in acute post-stroke hemiplegia patients: an observational study.

Lay Project Title: Tracking the position of the shoulder blade in patients for the first six weeks after a stroke.

Consent

I have read, or have had this document read to me in a language that I understand, and I understand the purposes, procedures and risks of this research project as described within it.

I have had an opportunity to ask questions and I am satisfied with the answers I have received.

I freely agree to participate in this research project, as described.

I understand that I will be given a signed copy of this document to keep.

Participant's name (printed)

Signature

Date

.....

Declaration by researcher*: I have given a verbal explanation of the research project, its procedures and risks and I believe that the participant has understood that explanation.

Researcher's name (printed)

Signature

Date

.....

.....

12. Who can I contact?

The person you may need to contact will depend on the nature of your query. Therefore, please note the following:

For further information or appointments:

If you want any further information concerning this project or if you have any problems which may be related to your involvement in the project (for example, feelings of distress), you can contact the principal researcher:

Dr Leanne Bisset

Physiotherapy Department

Royal Brisbane and Women's Hospital

Telephone: 07 36360665/ 55527717

Email: l.bisset@griffith.edu.au

or any of the following people:

Praline Choolun

Associate Researcher

Physiotherapy Department

Royal Brisbane and Women's Hospital

Telephone: 07 36241252

Email: praline_choolun@health.qld.gov.au

Dr Peter Mills

Associate Researcher, Supervisor

School of Physiotherapy and Exercise Science

Griffith University

Telephone: 07 55528917

Email: p.mills@griffith.edu.au

Dr Suzanne Kuys

Associate Researcher

The Prince Charles Hospital

Telephone: 07 31394418

Email: Suzanne_kuys@health.qld.gov.au

For complaints:

If you have any complaints about any aspect of the project, the way it is being conducted or any questions about being a research participant in general, then you may contact:

The Coordinator

Human Research Ethics Committee

Level 7 Block 7

Royal Brisbane and Women's Hospital

Butterfield Street

Herston.

Telephone: 07 36365490

Email: RBWH-Ethics@health.qld.gov.au

Royal Brisbane and Women's Hospital

Health Service District

Participant Information and Consent Form - Healthy participants Royal Brisbane and Women's Hospital

Full Project Title: Tracking changes in scapular position in acute post-stroke hemiplegia patients: an observational study.

Lay Project Title: Tracking the position of the shoulder blade in patients for the first six weeks after a stroke.

Principal Researcher: Dr Leanne Bisset

Associate researchers: Mrs Praline Choolun, Dr Peter Mills, Dr Suzanne Kuys, Dr Natalie Collins

11. Introduction

You are invited to take part in this research project, which aims to track shoulder and arm movement so that we are able to investigate the reasons for the development of pain in the shoulder after a stroke and assist with the development of effective treatments for patients with this problem.

For this project, we require participants who have had a stroke as well as healthy participants of a similar age, so that we can compare what happens to the shoulders of healthy people to what happens after a stroke.

This Participant Information and Consent Form tells you about the research project. It explains what is involved to help you decide if you want to take part.

Please read this information carefully. Ask questions about anything that you don't understand or want to know more about. Before deciding whether or not to take part, you might want to talk about it with a relative, friend or your local health worker.

Participation in this research is voluntary. If you don't wish to take part, you don't have to.

If you decide you want to take part in the research project, you may be asked to sign the consent section. By signing it you are telling us that you:

- understand what you have read;
- consent to take part in the research project;

- consent to be involved in the procedures described;
- consent to the use of your personal and health information as described.

You will be given a copy of this Participant Information and Consent Form to keep.

12. What is the purpose of this research project?

- Shoulder pain is a common problem after stroke, yet the reason for this condition and the evidence for the best way to manage it are not clear.
- This project aims to investigate changes in the position of the shoulder after a stroke in order to improve our understanding of factors which contribute to the development of shoulder pain.
- For this project we will measure the position of structures around your shoulders at rest and during small movements.
- We will recruit twenty- six post-stroke participants, and another twenty-six healthy participants who are matched in terms of age and gender to those who have had a stroke.
- Healthy participants will be recruited from the general community, hospital staff, and staff and students at Griffith University. You may be included in the study if you are aged over 18 years, weigh no more than 150 kg and have no history of shoulder pain, shoulder surgery or any other condition involving the upper limb, no clinically apparent scoliosis, no implants which contain metal, and are not sensitive to adhesive tape on your skin.
- This is not a follow-on study, but does involve researchers from the Royal Brisbane and Women's Hospital, Griffith University and University of Queensland working in collaboration. A grant from the Royal Brisbane and Women's Hospital Foundation Research will fund this project.
- The results of this research will be used by Mrs Praline Choolun to obtain a Masters of Research at Griffith University.

13. What does participation in this research project involve?

- Once you make contact with one of the researchers, they will ask you a few questions to determine if you are suitable to participate in the trial.
- You will be invited to attend the physiotherapy gymnasium at the hospital or the School of Physiotherapy and Exercise Science at Griffith University (Gold Coast Campus) for one session of 90 minutes, during which time you will be seated on a chair with a low back rest.
- A private cubicle in the physiotherapy gymnasium or in a research laboratory will be used.
- The immediate area has to be free of metal so as not to affect the very sensitive measurement device being used. You will be asked to remove all metal jewellery, glasses, hearing aids belt buckles and bras that use metal clasps. It is best if you leave any valuable items at home.

The following measurements will be taken at each session:

- Specific areas will be marked with adhesive tags on the surface of the skin of your arm, shoulder blade, spine and chest.
- One light-weight sensor of an electronic tracking device will be attached to your upper breast bone, using low-allergenic tape, and another attached to your upper arm using a cuff.
- A third sensor will be attached to a plastic frame which will be adjusted to fit over your shoulder blade. This will be held over your shoulder blade by the researcher while your arm is at rest by your side and while it is being gently moved a small amount in different directions without causing you any pain.

- A series of measurements will be recorded by the researcher, using a fourth sensor which looks like a pointer. It maps out the positions of the areas on your body identified by the adhesive tags.
- This process will be repeated on your other arm.
- This information will be recorded by a computer program.

- Your shoulder blades and will be measured with a standard tape measure.

- Your ability to move your shoulders will be assessed.
- The information collected will be compared with that of participants who have suffered a recent stroke and are of a similar age and gender to you.
 - Information regarding your age, medical history and hand dominance will be recorded.
 - No part of this project will be video or audio-taped.
 - You will not be paid for your participation in this research and you will not be reimbursed for any costs that you incur as a result of participating in this research project.
- 4. What are the possible benefits?**
- Your participation may benefit patients who have strokes in the future, particularly those who experience shoulder pain. The information gathered from this project may allow them to have better function in the arm affected by the stroke and thereby enable them to better contribute to society.

5. What are the possible risks?

- There are no risks associated with your participation in this research project
- If you become upset or distressed as a result of your participation in the research, the researcher is able to arrange for counselling or other appropriate support. Any counselling or support will be provided by staff who are not members of the research team.

6. Do I have to take part in this research project?

- Participation in any research project is voluntary. If you do not wish to take part, you do not have to. If you decide to take part and later change your mind, you are free to withdraw from the project at a later stage.
- If you decide to leave the project, the researchers would like to keep the personal and/or health information about you that has been collected. This is to help them make sure that the results of the research can be measured properly. If you do not want them to do this, you must tell them before you withdraw from the research project.
- Your decision whether to take part or not, or to take part and then withdraw, will not affect your relationship with the researchers or the Royal Brisbane and Women's Hospital.

○ **How will I be informed of the final results of this research project?**

- Information obtained through completion of this project will be submitted to a peer reviewed journal for publication. You will be informed when these results are published. Please be aware that personal data collected about you will not be used to identify you in any publication arising from this research study.

○ **What will happen to information about me?**

- Data will be recorded on a printed form and transferred into an electronic database. All data will be kept confidential and secure in a locked cabinet of a private office for hard copy information and by restricted password for computerised information. Hard copy data will be shredded and computer files deleted after 15 years.
- This research project does not involve the establishment of a databank.
- Any information obtained in connection with this research project that can identify you will only be used for the purpose of this research project. It will only be disclosed with your permission, except as required by law. In any publication and/or presentation, information will be provided in such a way that you cannot be identified, except with your permission.
- Your consent is being sought for this project only. Once the study is complete your name will be removed from the data to keep the information anonymous.

9. Can I access research information kept about me?

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- The ethical aspects of this research project have been approved by the Human Research Ethics Committee of the Royal Brisbane and Women's Hospital.
- This project will be carried out according to the *National Statement on Ethical Conduct in Human Research (2007)* produced by the National Health and Medical Research Council of Australia. This statement has been developed to protect the interests of people who agree to participate in human research studies.

11. Consent

I have read, or have had this document read to me in a language that I understand, and I understand the purposes, procedures and risks of this research project as described within it.

I have had an opportunity to ask questions and I am satisfied with the answers I have received.

I freely agree to participate in this research project, as described.

I understand that I will be given a signed copy of this document to keep.

Participant's name (printed)

Signature

Date

Declaration by researcher*: I have given a verbal explanation of the research project, its procedures and risks and I believe that the participant has understood that explanation.

Researcher's name (printed)

Signature

Date

12. Who can I contact?

The person you may need to contact will depend on the nature of your query. Therefore, please note the following:

For further information or appointments:

If you want any further information concerning this project or if you have any problems which may be related to your involvement in the project (for example, feelings of distress), you can contact the principal researcher:

Dr Leanne Bisset

Physiotherapy Department

Royal Brisbane and Women's Hospital

Telephone: 07 36360665/ 55527717

Email: l.bisset@griffith.edu.au

Or any of the following people:

Praline Choolun

Associate Researcher

Physiotherapy Department

Royal Brisbane and Women's Hospital

Telephone: 07 36241252 Mobile 0438339854

Email: praline_choolun@health.qld.gov.au

Dr Peter Mills

Associate Researcher, Supervisor

School of Physiotherapy and Exercise Science

Griffith University

Telephone: 07 55528917

Email: p.mills@griffith.edu.au

For complaints:

If you have any complaints about any aspect of the project, the way it is being conducted or any questions about being a research participant in general, then you may contact:

The Coordinator

Human Research Ethics Committee

Level 7 Block 7

Royal Brisbane and Women's Hospital

Butterfield Street

Herston.

Telephone: 07 36365490

Email: RBWH-Ethics@health.qld.gov.au

Appendix 3. Visual analogue scale

Participant Name:

Date:

Pain Visual Analogue Scale

Think about any shoulder pain you currently experience at rest.
I am now going to ask you to make a mark on a horizontal line.
One end of the line means no pain.
The other end of the line means that the pain is the worst it could be.
Where would you mark the line?

Think about any shoulder pain you experienced when your arm is passively moved.
I am now going to ask you to make a mark on a horizontal line.
One end of the line means no pain.
The other end of the line means that the pain is the worst it could be.
Where would you mark the line?

Think about any shoulder pain you experience when you move your neck.

I am now going to ask you to make a mark on a horizontal line.
One end of the line means no pain.
The other end of the line means that the pain is the worst it could be.
Where would you mark the line?

NO PAIN

Appendix 4. Pain map

Pain Map

Patient Name: _____

Date: _____

Appendix 5. Motor assessment scale

Patient Name: _____

Date: _____

Motor Assessment Scale

Upper Arm Function

1. Supine, protract shoulder girdle with arm in 90 degrees of shoulder flexion. (Therapist places arm in position and supports elbow extension).
2. Supine, hold arm in 90 degrees of shoulder flexion for 2 seconds. (Therapist places arm in position and patient must maintain position with some (45 degrees) external rotation. Elbow must be held within at least 20 degrees of full extension).
3. Supine, hold arm in 90 degrees of shoulder flexion, flex and extend elbow to take palm to forehead. (Therapist may assist supination of forearm).
4. Sitting, hold extended arm in forward flexion at 90 degrees to body for 2 seconds. (Therapist should place arm in position and patient maintains position. Patient must hold arm in mid-rotation (thumb pointing up). Do not allow excess shoulder elevation).
5. Sitting, patient lifts arm to above position, holds it there for 10 seconds and then lowers it. (Patient must maintain position with some external rotation. Do not allow pronation).
6. Standing, hand against wall. Maintain hand position, while turning body toward wall. (Arm is abducted to 90 degrees with palm flat against the wall).

Score: /6

Appendix 6. Tardieu scale

Tardieu Scale

Objective: To measure spasticity during passive movement at varying velocities

Type of scale: Ordinal rating scale, 5 points.

Instructions: Starting position for upper limb is sitting with the elbow flexed to 90 degrees and shoulder neutral.

Procedure: Take muscle through full passive range of movement (PROM) to determine full range at slow speed (V1)
Repeat test at speed (as fast as possible) (V3). The angle at which a catch (resistance) is first felt is noted.

Scoring: **Quality of muscle reaction (X)**

0=no resistance throughout course of passive movement

1=slight resistance throughout course of PROM, with no clear catch at a precise angle

2=clear catch at a precise angle, interrupting PROM, followed by release

3=fatigable clonus (<10s when maintain pressure) occurring at precise angle

4=infatigable clonus (>10s when maintain pressure) at precise angle

Angle of muscle reaction (Y)

The difference between the full PROM (during V1) and the angle of catch at V3= the spasticity angle. Normal value would be 0 difference and equal to normal range

Scores:

Upper limb muscle	R	L	Movement	Angle in degrees at which muscle reaction occurs			
				V1	V3	Y	X

References

- Abreu, B., Buford, W., Nakamura, T., Ottenbacher, K., & Patterson, R. (2008). Upper-limb kinematics of the presumed-to-be-unaffected side after brain injury. *American Journal of Occupational Therapy*, 62(1), 46.
- Ada, L., Canning, C., & Low, S. (2003). Stroke patients have selective muscle weakness in shortened range. *Brain: A Journal of Neurology*, 126(Part 3), 724-731.
- Ada, L., & Foongchomcheay, A. (2002). Efficacy of electrical stimulation in preventing or reducing subluxation of the shoulder after stroke: a meta-analysis. *Australian Journal of Physiotherapy*, 48(4), 257-267.
- Ada, L., Goddard, E., McCully, J., Stavrinou, T., & Bampton, J. (2005). Thirty minutes of positioning reduces the development of shoulder external rotation contracture after stroke: a randomized controlled trial. *Archives of Physical Medicine & Rehabilitation*, 86(2), 230-234.
- Ada, L., O'Dwyer, N., & O'Neill, E. (2006). Relation between spasticity, weakness and contracture of the elbow flexors and upper limb activity after stroke: an observational study. *Disability & Rehabilitation*, 28(13-14), 891-897.
- Adey-Wakeling, Z., Arima, H., Crotty, M., Leyden, J., Kleinig, T., Anderson, C. S., & Newbury, J. (2014). Incidence and Associations of Hemiplegic Shoulder Pain Post Stroke: A prospective population based study. *Archives of Physical Medicine & Rehabilitation*. doi:10.1016/j.apmr.2014.09.007
- Akinwuntan, A. E., De Weerd, W., Feys, H., Pauwels, J., Baten, G., et al. (2005). Effect of simulator training on driving after stroke: a randomized controlled trial. *Neurology*, 65(6), 843-850.
- Allen, Z. A., Shanahan, E. M., & Crotty, M. (2010). Does suprascapular nerve block reduce shoulder pain following stroke: a double-blind randomised controlled trial with masked outcome assessment. *BMC Neurology*, 10, 83-83.
- Aras, M. D., Gokkaya, N. K. O., Comert, D., Kaya, A., & Cakci, A. (2004). Shoulder pain in hemiplegia: results from a national rehabilitation hospital in Turkey. *American Journal of Physical Medicine and Rehabilitation*, 83(9), 713-719.
- Arendt-Nielsen, L., & Yarnitsky, D. (2009). Experimental and clinical applications of quantitative sensory testing applied to skin, muscles and viscera. *Journal of Pain*, 10(6), 556-572.
- Arsenault, A. B., Bilodeau, M., Dutil, E., & Riley, E. (1991). Clinical significance of the V-shaped space in the subluxed shoulder of hemiplegics. *Stroke*, 22(7), 867-871.

- Arya, K. N., Pandian, S., Verma, R., & Garg, R. K. (2011). Movement therapy induced neural reorganization and motor recovery in stroke: A review. *Journal of Bodywork & Movement Therapies*, 15(4), 528-537.
- Atalar, H., Yilmaz, C., Polat, O., Selek, H., Uraş, I., & Yanik, B. (2009). Restricted scapular mobility during arm abduction: implications for impingement syndrome. *Acta Orthopaedica Belgica*, 75(1), 19-24.
- Australian Bureau of Statistics. (2007). *Australian Social Trends 2007. (Catalogue no. 4102.0.)* Retrieved from [http://www.ausstats.abs.gov.au/ausstats/subscriber.nsf/0/51EE403E951E7FDA CA25732F001CAC21/\\$File/41020_Australian%20Social%20Trends_2007_ADJ USTED.pdf](http://www.ausstats.abs.gov.au/ausstats/subscriber.nsf/0/51EE403E951E7FDA CA25732F001CAC21/$File/41020_Australian%20Social%20Trends_2007_ADJ USTED.pdf).
- Australian Bureau of Statistics. (2011). *Diabetes in Australia: A Snapshot, 2007-08. (Catalogue No. 4820.0.55.001)*. Canberra: Australian Bureau of Statistics Retrieved from <http://www.abs.gov.au/ausstats/abs@.nsf/Lookup/4820.0.55.001main+features 32007-08>.
- Australian Institute of Health and Welfare. (2006). *Australian Institute of Health and Welfare 2006. Australia's health 2006*. Canberra: Retrieved from <http://www.aihw.gov.au/WorkArea/DownloadAsset.aspx?id=6442453483>.
- Australian Institute of Health and Welfare. (2011). Cardiovascular disease: Australian facts 2011. Cardiovascular disease series *Cardiovascular disease: Australian facts 2011. Cardiovascular disease series*: Australian Institute of Health and Welfare.
- Australian Institute of Health and Welfare. (2012). *Australia's Health 2012*. Canberra: Australian Institute of Health and Welfare Retrieved from <http://www.aihw.gov.au/WorkArea/DownloadAsset.aspx?id=10737422169>.
- Australian Institute of Health and Welfare. (2013). *Stroke and its management in Australia: an update*. Canberra: AIHW Retrieved from <http://www.aihw.gov.au/WorkArea/DownloadAsset.aspx?id=60129543611>.
- Australian Institute of Health and Welfare: Senes, S. (2006). *How we manage stroke in Australia. AIHW cat. no. CVD 31*. Canberra: Australian Institute of Health and Welfare.: Retrieved from <http://www.aihw.gov.au/WorkArea/DownloadAsset.aspx?id=6442454967>.
- Barlak, A., Unsal, S., Kaya, K., Sahin-Onat, S., & Ozel, S. (2009). Poststroke shoulder pain in Turkish stroke patients: relationship with clinical factors and functional outcomes. *International Journal of Rehabilitation Research*, 32(4), 309-315.
- Barrett, K. M., & Meschia, J. F. (2013). *Stroke*: Wiley-Blackwell.

- Beebe, J. A. (2008). *Recovery of upper extremity movement control and the relationships with upper extremity function in individuals with acute hemiparesis*. (Ph.D.), Washington University in St. Louis. Retrieved from <http://libraryproxy.griffith.edu.au/login?url=http://search.ebscohost.com/login.aspx?direct=true&db=rzh&AN=2010544081&site=ehost-live&scope=site> Available from EBSCOhost rzh database.
- Beebe, J. A., & Lang, C. E. (2009). Active range of motion predicts upper extremity function 3 months after stroke. *Stroke* (00392499). Retrieved from <http://libraryproxy.griffith.edu.au/login?url=http://search.ebscohost.com/login.aspx?direct=true&db=cin20&AN=2010264819&site=ehost-live&scope=site>.
- Benjamin, D. R., van de Water, A. T., & Peiris, C. L. (2014). Effects of exercise on diastasis of the rectus abdominis muscle in the antenatal and postnatal periods: a systematic review. *Physiotherapy*, 100(1), 1-8.
- Beschin, N., Cazzani, M., Cubelli, R., Della Sala, S., & Spinazzola, L. (1996). Ignoring left and far: an investigation of tactile neglect. *Neuropsychologia*, 34(1), 41-49.
- Bialocerkowski, A., Klupp, N., & Bragge, P. (2010). How to read and critically appraise a reliability article. *International Journal of Therapy & Rehabilitation*, 17(3), 114-120.
- Blennerhassett, J., & Dite, W. (2004). Additional task-related practice improves mobility and upper limb function early after stroke: a randomised controlled trial. *Australian Journal of Physiotherapy*, 50(4), 219-224.
- Blennerhassett, J., Gyngell, K., & Crean, R. (2010). Reduced active control and passive range at the shoulder increase risk of shoulder pain during inpatient rehabilitation post-stroke: an observational study. *Journal of Physiotherapy*, 56(3), 195-199.
- Bohannon, R. W. (2007). Muscle strength and muscle training after stroke. *Journal of Rehabilitation Medicine*, 39(1), 14-20.
- Bohannon, R. W., & Andrews, A. W. (1990). Shoulder subluxation and pain in stroke patients. *American Journal of Occupational Therapy*, 44(6), 507-509.
- Bohannon, R. W., & Andrews, A. W. (1995). Limb Muscle Strength is Impaired Bilaterally after Stroke. *Journal of Physical Therapy Science*, 7(1), 1-7.
- Bohannon, R. W., Larkin, P. A., Smith, M. B., & Horton, M. G. (1986). Shoulder pain in hemiplegia: statistical relationship with five variables. *Archives of Physical Medicine and Rehabilitation*, 67(8), 514-516.
- Boyd, E. A., Goudreau, L., O'Riain, M. D., Grinnell, D. M., Torrance, G. M., & Gaylard, A. (1993a). A radiological measure of shoulder subluxation in hemiplegia: its reliability and validity. *Archives of Physical Medicine & Rehabilitation*, 74(2), 188-193.

- Boyd, E. A., Goudreau, L., O'Riain, M. D., Grinnell, D. M., Torrance, G. M., & Gaylard, A. (1993b). A radiological measure of shoulder subluxation in hemiplegia: its reliability and validity. *Archives of Physical Medicine & Rehabilitation*, 74(2), 188-193.
- Boyd, E. A., & Torrance, G. M. (1992). Clinical measures of shoulder subluxation: their reliability. *Canadian Journal of Public Health*, 83, S24-28.
- Braman, J. P., Engel, S. C., LaPrade, R. F., & Ludewig, P. M. (2009). In vivo assessment of scapulohumeral rhythm during unconstrained overhead reaching in asymptomatic subjects. *Journal of Shoulder and Elbow Surgery*, 18(6), 960-967.
- Brott, T., Adams, H. P., Jr., Olinger, C. P., Marler, J. R., Barsan, W. G., et al. (1989). Measurements of acute cerebral infarction: a clinical examination scale. *Stroke*, 20(7), 864-870.
- Cadilhac, D. A., Carter, R., Thrift, A. G., & Dewey, H. M. (2009). Estimating the long-term costs of ischemic and hemorrhagic stroke for Australia: new evidence derived from the North East Melbourne Stroke Incidence Study (NEMESIS). *Stroke*, 40(3), 915-921.
- Campbell, A. C., Lloyd, D. G., Alderson, J. A., & Elliott, B. C. (2009). MRI development and validation of two new predictive methods of glenohumeral joint centre location identification and comparison with established techniques. *Journal of Biomechanics*, 42(10), 1527-1532.
- Canning, C. G., Ada, L., Adams, R., & O'Dwyer, N. J. (2004). Loss of strength contributes more to physical disability after stroke than loss of dexterity. *Clinical Rehabilitation*, 18(3), 300-308.
- Carlsson, A. M. (1983). Assessment of chronic pain. I. Aspects of the reliability and validity of the visual analogue scale. *Pain*, 16(1), 87-101.
- Carr, J., & Shepherd, R. (2003). *Stroke rehabilitation: guidelines for exercise and training to optimize motor skill*. New York; Edinburgh: Butterworth-Heinemann.
- Carr, J., Shepherd, R., Nordholm, L., & Lynne, D. (1985). Investigation of a New Motor Assessment Scale for Stroke Patients. *Physical Therapy*, 65(2), 175-180.
- Carter, T., Hall, H., McIntosh, G., Murphy, J., MacDougall, J., et al. (2012). Intertester reliability of a classification system for shoulder pain. *Physiotherapy*, 98(1), 40-46.
- Chaco, J., & Wolf, E. (1971). Subluxation of the glenohumeral joint in hemiplegia. *American Journal of Physical Medicine*, 50, 139-143.
- Chae, J., & Jedlicka, L. (2009). Subacromial corticosteroid injection for poststroke shoulder pain: an exploratory prospective case series. *Archives of Physical Medicine and Rehabilitation*, 90(3), 501-506.

- Chantraine, A., Baribeault, A., Uebelhart, D., & Gremion, G. (1999). Shoulder pain and dysfunction in hemiplegia: Effects of functional electrical stimulation. *Archives of Physical Medicine and Rehabilitation*, 80(3), 328-331.
- Cherney, L. R., Halper, A. S., Kwasnica, C. M., Harvey, R. L., & Zhang, M. (2001). Recovery of functional status after right hemisphere stroke: relationship with unilateral neglect. *Archives of Physical Medicine & Rehabilitation*, 82(3), 322-328.
- Chesterton, L. S., Sim, J., Wright, C. C., & Foster, N. E. (2007). Interrater reliability of algometry in measuring pressure pain thresholds in healthy humans, using multiple raters. *Clinical Journal of Pain*, 23(9), 760-766.
- Chopp, J. N., Fischer, S. L., & Dickerson, C. R. (2011). The specificity of fatiguing protocols affects scapular orientation: Implications for subacromial impingement. *Clinical Biomechanics*, 26(1), 40-45.
- Chopp, J. N., O'Neill, J. M., Hurley, K., & Dickerson, C. R. (2010). Superior humeral head migration occurs after a protocol designed to fatigue the rotator cuff: a radiographic analysis. *Journal of Shoulder and Elbow Surgery*, 19(8), 1137-1144.
- Compston, A. (2010). Aids to the investigation of peripheral nerve injuries. Medical Research Council: Nerve Injuries Research Committee. *Brain*, 133 (Part 10), 2838-2844.
- Connell, L. A., Lincoln, N. B., & Radford, K. A. (2008a). Somatosensory impairment after stroke: frequency of different deficits and their recovery. *Clinical Rehabilitation*, 22(8), 758-767.
- Connell, L. A., Lincoln, N. B., & Radford, K. A. (2008b). Somatosensory impairment after stroke: frequency of different deficits and their recovery. *Clinical Rehabilitation*, 22(8), 758-767.
- Constant, C. R. (1989). Historical background, anatomy and shoulder function. *Baillière's Clinical Rheumatology*, 3(3), 429-435.
- Corti, M., McGuirk, T. E., Wu, S. S., & Patten, C. (2012). Differential Effects of Power Training Versus Functional Task Practice on Compensation and Restoration of Arm Function After Stroke. *Neurorehabilitation & Neural Repair*, 26(7), 842-854.
- Coupar, F., Pollock, A., Rowe, P., Weir, C., & Langhorne, P. (2012). Predictors of upper limb recovery after stroke: a systematic review and meta-analysis. *Clinical Rehabilitation*, 26(4), 291-313.
- Culham, E., Noce, R., & Bagg, S. (1995). Shoulder complex position and glenohumeral subluxation in hemiplegia. *Archives of Physical Medicine and Rehabilitation*, 76, 857 - 864.

- Cutti, A. G., Parel, I., Raggi, M., Petracci, E., Pellegrini, A., et al. (2014). Prediction bands and intervals for the scapulo-humeral coordination based on the Bootstrap and two Gaussian methods. *Journal of Biomechanics*. doi: 10.1016/j.jbiomech.2013.12.028.
- Davenport, R. J., Dennis, M. S., Wellwood, I., & Warlow, C. P. (1996). Complications After Acute Stroke. *Stroke*, 27(3), 415-420.
- Daviet, J. C., Salle, J. Y., Borie, M. J., Munoz, M., Rebeyrotte, I., et al. (2002). Clinical factors associate with shoulder subluxation in stroke patients. *Annales De Réadaptation Et De Médecine Physique: Revue Scientifique De La Société Française De Rééducation Fonctionnelle De Réadaptation Et De Médecine Physique*, 45(9), 505-509.
- De Baets, L., Jaspers, E., Desloovere, K., & Van Deun, S. (2013). A systematic review of 3D scapular kinematics and muscle activity during elevation in stroke subjects and controls. *Journal of Electromyography and Kinesiology*, 23(1), 3-13.
- de Jong, L. D., Hoonhorst, M. H., Stuive, I., & Dijkstra, P. U. (2011). Arm motor control as predictor for hypertonia after stroke: a prospective cohort study. *Archives of Physical Medicine and Rehabilitation*, 92(9), 1411-1417.
- Deeks, J. J., Dinnes, J., D'Amico, R., Sowden, A. J., Sakarovich, C., et al. (2003). Evaluating non-randomised intervention studies. *Health Technology Assessment (Winchester, England)*, 7(27), iii-x, 1-173.
- Degens, H., & Korhonen, M. (2012). Factors contributing to the variability in muscle ageing. *Maturitas*, 73(3), 197-201.
- Demeurisse, G., Demol, O., & Robaye, E. (1980). Motor evaluation in vascular hemiplegia. *European Neurology*, 19(6), 382-389.
- Demirci, A., Öcek, B., & Köseoğlu, F. (2007). Shoulder pain in hemiplegic patients *Journal of Physical Medicine and Rehabilitation Sciences*, 10(1), 25-30.
- Desrosiers, J., Bourbonnais, D., Bravo, G., Roy, P., & Guay, M. (1996). Performance of the "unaffected" upper extremity of elderly stroke patients. *Stroke*, 27(9), 1564-1570.
- Desrosiers, J., Malouin, F., Bourbonnais, D., Richards, C., Rochette, A., et al. (2003). Upper limb and leg impairments and disabilities after stroke rehabilitation: relationship to handicap. *Clinical Rehabilitation*, 17(6), 666-673.
- Dewey, H. M., Donnan, G. A., Freeman, E. J., Sharples, C. M., Macdonell, R. A., et al. (1999). Interrater reliability of the National Institutes of Health Stroke Scale: rating by neurologists and nurses in a community-based stroke incidence study. *Cerebrovascular diseases*, 9(6), 323-327.

- Dewey, H. M., Sturm, J., Donnan, G. A., Macdonell, R. A., McNeil, J. J., & Thrift, A. G. (2003). Incidence and outcome of subtypes of ischaemic stroke: initial results from the north east Melbourne stroke incidence study (NEMESIS). *Cerebrovascular diseases*, 15(1-2), 133-139.
- Di Monaco, M., Schintu, S., Dotta, M., Barba, S., Tappero, R., et al. (2011). Severity of Unilateral Spatial Neglect Is an Independent Predictor of Functional Outcome After Acute Inpatient Rehabilitation in Individuals With Right Hemispheric Stroke. *Archives of Physical Medicine & Rehabilitation*, 92(8), 1250-1256.
- Ditroilo, M., Forte, R., Benelli, P., Gambarara, D., & De Vito, G. (2010). Effects of age and limb dominance on upper and lower limb muscle function in healthy males and females aged 40-80 years. *Journal of Sports Sciences*, 28(6), 667-677.
- Downs, S. H., & Black, N. (1998). The feasibility of creating a checklist for the assessment of the methodological quality both of randomised and non-randomised studies of health care interventions. *Journal of Epidemiology & Community Health*, 52(6), 377-384.
- Dromerick, A. W., Edwards, D. F., & Kumar, A. (2008). Hemiplegic Shoulder Pain Syndrome: Frequency and Characteristics During Inpatient Stroke Rehabilitation. *Archives of Physical Medicine & Rehabilitation*, 89(8), 1589-1593.
- Dromerick, A. W., Kumar, A., Volshteyn, O., & Edwards, D. F. (2006). Hemiplegic shoulder pain syndrome: interrater reliability of physical diagnosis signs. *Archives of Physical Medicine & Rehabilitation*, 87(2), 294-295.
- Duff, S. V., & Sainburg, R. L. (2007). Lateralization of motor adaptation reveals independence in control of trajectory and steady-state position. *Experimental Brain Research*, 179(4), 551-561.
- Duncan, P. W., Goldstein, L. B., Horner, R. D., Landsman, P. B., Samsa, G. P., et al. (1994). Similar motor recovery of upper and lower extremities after stroke. *Stroke*, 25(6), 1181-1188.
- Duncan, P. W., Zorowitz, R. D., Bates, B., Choi, J. Y., Glasberg, J. J., et al. (2005). Management of Adult Stroke Rehabilitation Care: A Clinical Practice Guideline. *Stroke*, 36(9), e100-e143.
- Dundar, U., Toktas, H., Cakir, T., Evcik, D., & Kavuncu, V. (2009). Continuous passive motion provides good pain control in patients with adhesive capsulitis. *International Journal of Rehabilitation Research*, 32(3), 193-198.
- Dvorak, E. M., Ketchum, N. C., & McGuire, J. R. (2011). The underutilization of intrathecal baclofen in poststroke spasticity. *Topics in Stroke Rehabilitation*, 18(3), 195-202.

- English, C., Hillier, S., & Stiller, K. (2008). Incidence and severity of shoulder pain does not increase with the use of circuit class therapy during inpatient stroke rehabilitation: a controlled trial. *Australian Journal of Physiotherapy*, 54(1), 41-46.
- Faghri, P. D., Rodgers, M. M., Glaser, R. M., Bors, J. G., Ho, C., et al. (1994). The effects of functional electrical stimulation on shoulder subluxation, arm function recovery, and shoulder pain in hemiplegic stroke patients. *Archives of Physical Medicine & Rehabilitation*, 75(1), 73-79.
- Faria-Fortini, I., Michaelsen, S. M., Cassiano, J. G., & Teixeira-Salmela, L. F. (2011). Upper Extremity Function in Stroke Subjects: Relationships between the International Classification of Functioning, Disability, and Health Domains. *Journal of Hand Therapy*, 24(3), 257-264.
- Faul, F., Erdfelder, E., Buchner, A., & Lang, A.-G. (2009). Statistical power analyses using G*Power 3.1: tests for correlation and regression analyses. *Behavior Research Methods*, 41(4), 1149-1160.
- Fayad, F., Roby-Brami, A., Yazbeck, C., Hanneton, S., Lefevre-Colau, M., et al. (2008). Three-dimensional scapular kinematics and scapulohumeral rhythm in patients with glenohumeral osteoarthritis or frozen shoulder. *Journal of Biomechanics*, 41(2), 326-332.
- Feigin, V. L., Barker-Collo, S., McNaughton, H., Brown, P., & Kerse, N. (2008). Long-term neuropsychological and functional outcomes in stroke survivors: current evidence and perspectives for new research. *International Journal of Stroke*, 3(1), 33-40.
- Fil, A., Armutlu, K., Atay, A. O., Kerimoglu, U., & Elibol, B. (2011). The effect of electrical stimulation in combination with Bobath techniques in the prevention of shoulder subluxation in acute stroke patients. *Clinical Rehabilitation*, 25(1), 51-59.
- Fish, D. E., Gerstman, B. A., & Lin, V. (2011). Evaluation of the patient with neck versus shoulder pain. *Physical Medicine & Rehabilitation Clinics of North America*, 22(3), 395-410.
- Formisano, R., Barbanti, P., Catarci, T., De Vuono, G., Calisse, P., et al. (1993). Prolonged muscular flaccidity: frequency and association with unilateral spatial neglect after stroke. *Acta Neurologica Scandinavica*, 88(5), 313-315.
- Formisano, R., Pantano, P., Buzzi, M. G., Vinicola, V., Penta, F., et al. (2005). Late motor recovery is influenced by muscle tone changes after stroke. *Archives of Physical Medicine and Rehabilitation*, 86(2), 308-311.

- Gamble, G. E., Barberan, E., Bowsher, D., Tyrrell, P. J., & Jones, A. K. (2000). Post stroke shoulder pain: more common than previously realized. *European Journal of Pain*, 4(3), 313-315.
- Gamble, G. E., Barberan, E., Laasch, H., Bowsher, D., Tyrrell, P. J., et al. (2002). Poststroke shoulder pain: a prospective study of the association and risk factors in 152 patients from a consecutive cohort of 205 patients presenting with stroke. *European Journal of Pain*, 6(6), 467-474.
- Garofalo, R., Karlsson, J., Nordenson, U., Cesari, E., Conti, M., et al. (2010). Anterior-superior internal impingement of the shoulder: an evidence-based review. *Knee Surgery, Sports Traumatology, Arthroscopy*, 18(12), 1688-1693.
- Graven, C., Brock, K., Hill, K., & Joubert, L. (2011). Are rehabilitation and/or care co-ordination interventions delivered in the community effective in reducing depression, facilitating participation and improving quality of life after stroke? *Disability & Rehabilitation*, 33(17/18), 1501-1520.
- Griffin, A., & Bernhardt, J. (2006). Strapping the hemiplegic shoulder prevents development of pain during rehabilitation: a randomized controlled trial. *Clinical Rehabilitation*, 20(4), 287-295.
- Haaland, K. Y., Mutha, P. K., Rinehart, J. K., Daniels, M., Cushnyr, B., et al. (2012). The Relationship between Arm Usage and Instrumental Activities of Daily Living after Unilateral Stroke. *Archives of Physical Medicine and Rehabilitation*, 93(11), 1957-1962.
- Hadianfard, H., & Hadianfard, M. J. (2008). Predictor factors of hemiplegic shoulder pain in a group of stroke patients. *Iranian Red Crescent Medical Journal*, 10(3), 215-219.
- Halder, A. M., Kuhl, S. G., Zobitz, M. E., Larson, D., & An, K. N. (2001). Effects of the glenoid labrum and glenohumeral abduction on stability of the shoulder joint through concavity-compression : an in vitro study. *The Journal Of Bone And Joint Surgery. American Volume*, 83-A(7), 1062-1069.
- Hall, J., Dudgeon, B., & Guthrie, M. (1995). Validity of clinical measures of shoulder subluxation in adults with poststroke hemiplegia. *American Journal of Occupational Therapy*, 49(6), 526-533.
- Hallstrom, E., & Karrholm, J. (2009). Shoulder rhythm in patients with impingement and in controls: dynamic RSA during active and passive abduction. *Acta Orthopaedica*, 80(4), 456-464.
- Hamming, D., Braman, J. P., Phadke, V., LaPrade, R. F., & Ludewig, P. M. (2012). The accuracy of measuring glenohumeral motion with a surface humeral cuff. *Journal of Biomechanics*, 45(7), 1161-1168.

- Hanchard, N., Cummins, J., & Jeffries, C. (2004). *Evidence-based clinical guidelines for the diagnosis, assessment and physiotherapy management of shoulder impingement syndrome*. London, UK. : Chartered Society of Physiotherapy. London, UK.
- Hanchard, N., Lenza, M., Handoll, H., & Takwoingi, Y. (2013). Physical tests for shoulder impingements and local lesions of bursa, tendon or labrum that may accompany impingement. Retrieved from <http://onlinelibrary.wiley.com/doi/10.1002/14651858.CD007427.pub2/abstract>
- Hanger, H. C., Whitewood, P., Brown, G., Ball, M. C., Harper, J., et al. (2000). A randomized controlled trial of strapping to prevent post-stroke shoulder pain. *Clinical Rehabilitation*, 14(4), 370-380.
- Hardwick, D. D. (2010). *Shoulder movement and pain after stroke*. (Doctor of Philosophy), Washington University, St. Louis. (UMI Number: 3397851)
- Hardwick, D. D., & Lang, C. E. (2011a). Scapula and humeral movement patterns and their relationship with pain: a preliminary investigation *International Journal of Therapy and Rehabilitation*, 18(4), 210-220.
- Hardwick, D. D., & Lang, C. E. (2011b). Scapular and Humeral Movement Patterns of People With Stroke During Range-of-Motion Exercises. *Journal of Neurologic Physical Therapy*, 35(1), 18-25.
- Harlick, J. C., Milosavljevic, S., & Milburn, P. D. (2007). Palpation identification of spinous processes in the lumbar spine. *Manual Therapy*, 12(1), 56-62.
- Harris, J. E., & Eng, J. J. (2006). Individuals with the Dominant Hand Affected following Stroke Demonstrate Less Impairment Than Those with the Nondominant Hand Affected. *Neurorehabilitation and Neural Repair*, 20(3), 380-389.
- Harrison, A. K., & Flatow, E. L. (2011). Subacromial impingement syndrome. *Journal of the American Academy of Orthopaedic Surgeons*, 19(11), 701-708.
- Haugh, A. B., Pandyan, A. D., & Johnson, G. R. (2006). A systematic review of the Tardieu Scale for the measurement of spasticity. *Disability and Rehabilitation* 28(15), 899-907.
- Hébert, L. J., Moffet, H., McFadyen, B. J., & Dionne, C. E. (2002). Scapular behavior in shoulder impingement syndrome. *Archives of Physical Medicine and Rehabilitation*, 83(1), 60-69.
- Hendricks, H. T., van Limbeek, J., Geurts, A. C., & Zwarts, M. J. (2002). Motor recovery after stroke: a systematic review of the literature. *Archives of Physical Medicine and Rehabilitation*, 83(11), 1629-1637.
- Heyworth, B. E., & Williams, R. J., III. (2009). Internal impingement of the shoulder. *American Journal of Sports Medicine*, 37(5), 1024-1037.

- Higgins, J., Mayo, N. E., Desrosiers, J., Salbach, N. M., & Ahmed, S. (2005). Upper-limb function and recovery in the acute phase poststroke. *Journal of Rehabilitation Research and Development*, 42(1), 65-76.
- Hooper, P., Jutai, J. W., Strong, G., & Russell-Minda, E. (2008). Age-related macular degeneration and low-vision rehabilitation: a systematic review. *Canadian Journal of Ophthalmology*, 43(2), 180-187.
- Howell, S. M., & Galinat, B. J. (1989). The glenoid-labral socket. A constrained articular surface. *Clinical Orthopaedics And Related Research*, (243), 122-125.
- Huang, H. J., & Ferris, D. P. (2009). Upper and lower limb muscle activation is bidirectionally and ipsilaterally coupled. *Medicine & Science in Sports & Exercise*, 41(9), 1778-1789.
- Huang, S., Liu, S., Tang, H., Wei, T., Wang, W., et al. (2012). Relationship between severity of shoulder subluxation and soft-tissue injury in hemiplegic stroke patients. *Journal of Rehabilitation Medicine*, 44(9), 733-739.
- Huang, Y., Liang, P., Pong, Y., Leong, C., & Tseng, C. (2010). Physical findings and sonography of hemiplegic shoulder in patients after acute stroke during rehabilitation. *Journal of Rehabilitation Medicine*, 42(1), 21-26.
- Ikai, T., Tei, K., Yoshida, K., Miyano, S., & Yonemoto, K. (1998). Evaluation and treatment of shoulder subluxation in hemiplegia: relationship between subluxation and pain. *American Journal of Physical Medicine and Rehabilitation*, 77(5), 421-426.
- Islam, M. S., Anderson, C. S., Hankey, G. J., Hardie, K., Carter, K., et al. (2008). Trends in incidence and outcome of stroke in Perth, Western Australia during 1989 to 2001: the Perth Community Stroke Study. *Stroke*, 39(3), 776-782.
- Jackson, D., Turner-Stokes, L., Khatoon, A., Stern, H., Knight, L., et al. (2002). Development of an integrated care pathway for the management of hemiplegic shoulder pain. *Disability and Rehabilitation*, 24(7), 390-398.
- James, B., & Parker, A. W. (1989). Active and passive mobility of lower limb joints in elderly men and women. *American Journal of Physical Medicine & Rehabilitation*, 68(4), 162-167.
- Jaraczewska, E., & Long, C. (2006). Kinesio taping in stroke: improving functional use of the upper extremity in hemiplegia. *Topics in Stroke Rehabilitation*, 13(3), 31-42.
- Jia, X., Ji, J. H., Pannirselvam, V., Petersen, S. A., & McFarland, E. G. (2011). Does a positive neer impingement sign reflect rotator cuff contact with the acromion? *Clinical Orthopaedics And Related Research*, 469(3), 813-818.
- Jørgensen, H. S., Nakayama, H., Raaschou, H. O., Vive-Larsen, J., Støier, M., et al. (1995). Outcome and time course of recovery in stroke. Part II: Time course of

- recovery. The Copenhagen stroke study. *Archives of Physical Medicine and Rehabilitation*, 76(5), 406-412.
- Joynt, R. L. (1992). The source of shoulder pain in hemiplegia. *Archives of Physical Medicine & Rehabilitation*, 73, 409-413.
- Jung, H. Y., Yoon, J. S., & Park, B. S. (2002). Recovery of proximal and distal arm weakness in the ipsilateral upper limb after stroke. *NeuroRehabilitation*, 17(2), 153-159.
- Jutai, J. W., Bhogal, S. K., Foley, N. C., Bayley, M., Teasell, R. W., et al. (2003). Treatment of visual perceptual disorders post stroke. *Topics in Stroke Rehabilitation*, 10(2), 77-106.
- Kalichman, L., & Ratmansky, M. (2011). Underlying pathology and associated factors of hemiplegic shoulder pain. *American Journal of Physical Medicine and Rehabilitation*, 90(9), 768-780.
- Kanatli, U., Gemalmaz, H. C., Ozturk, B. Y., Voyvoda, N. K., Tokgoz, N., et al. (2013). The role of radiological subacromial distance measurements in the subacromial impingement syndrome. *European Journal Of Orthopaedic Surgery & Traumatology: Orthopédie Traumatologie*, 23(3), 317-322.
- Khan, C. M., Oesch, P. R., Gamper, U. N., Kool, J. P., & Beer, S. (2011). Potential effectiveness of three different treatment approaches to improve minimal to moderate arm and hand function after stroke--a pilot randomized clinical trial. *Clinical Rehabilitation*, 25(11), 1032-1041.
- Kim, J. S., & Choi-Kwon, S. (1996). Discriminative sensory dysfunction after unilateral stroke. *Stroke*, 27(4), 677-682.
- Kjendahl, A., Jahnsen, R., & Aamodt, G. (2005). Motor assessment scale in Norway: translation and inter-rater reliability. *Advances in Physiotherapy*, 7(1), 7-12.
- Klit, H., Finnerup, N. B., & Jensen, T. S. (2009). Central post-stroke pain: clinical characteristics, pathophysiology, and management. *Lancet Neurology*, 8(9), 857-868.
- Kocabas, H., Levendoglu, F., Ozerbil, O. M., & Yuruten, B. (2007). Complex regional pain syndrome in stroke patients. *International Journal of Rehabilitation Research*, 30(1), 33-38.
- Kong, K. H., Lee, J., & Chua, K. S. (2012). Occurrence and temporal evolution of upper limb spasticity in stroke patients admitted to a rehabilitation unit. *Archives of Physical Medicine and Rehabilitation*, 93(1), 143-148.
- Koog, Y. H., Jin, S. S., Yoon, K., & Min, B. I. (2010). Interventions for hemiplegic shoulder pain: Systematic review of randomised controlled trials. *Disability & Rehabilitation*, 32(4), 282-291.

- Kottorp, A., Ekstam, L., & Petersson Lie, I. (2013). Differences in awareness between persons with left and right hemispheric stroke. *Scandinavian Journal of Occupational Therapy*, 20(1), 37-44. doi: 10.3109/11038128.2012.688864
- Koudstaal, P. J., Visser, M. C., Schouten, H. J., & van Gijn, J. (1988). Interobserver agreement for the assessment of handicap in stroke patients. *Stroke*, 19(5), 604-607.
- Koyuncu, E., Nakipoğlu-Yüzer, G. F., Doğan, A., & Özgirgin, N. (2010). The effectiveness of functional electrical stimulation for the treatment of shoulder subluxation and shoulder pain in hemiplegic patients: A randomized controlled trial. *Disability and Rehabilitation*, 32(7), 560-566.
- Kroeders, R., Bernhardt, J., & Cumming, T. (2013). Physical inactivity, depression and anxiety in acute stroke. *International Journal of Therapy & Rehabilitation*, 20(6), 289-293.
- Kumar, P., Bradley, M., Gray, S., & Swinkels, A. (2011). Reliability and validity of ultrasonographic measurements of acromion-greater tuberosity distance in poststroke hemiplegia. *Archives of Physical Medicine and Rehabilitation*, 92(5), 731-736.
- Kumar, P., Kassam, J., Denton, C., Taylor, E., & Chatterley, A. (2010). Risk factors for inferior shoulder subluxation in patients with stroke. *Physical Therapy Reviews*, 15(1), 3-11.
- Kumar, P., Saunders, A., Ellis, E., & Whitlam, S. (2013). Association between glenohumeral subluxation and hemiplegic shoulder pain in patients with stroke. *Physical Therapy Reviews*, 18(2), 90-100.
- Kumar, P., & Swinkels, A. (2009). A critical review of shoulder subluxation and its association with other post-stroke complications. *Physical Therapy Reviews*, 14(1), 13-25.
- Kuptniratsaikul, V., Kovindha, A., Suethanapornkul, S., Manimmanakorn, N., & Archongka, Y. (2009). Complications during the rehabilitation period in Thai patients with stroke: a multicenter prospective study. *American Journal of Physical Medicine and Rehabilitation*, 88(2), 92-99.
- Kwah, L. K., Harvey, L. A., Diong, J. H. L., & Herbert, R. D. (2012). Half of the adults who present to hospital with stroke develop at least one contracture within six months: an observational study. *Journal of Physiotherapy*, 58(1), 41-47.
- Kwakkel, G., Kollen, B., & Twisk, J. (2006). Impact of time on improvement of outcome after stroke. *Stroke*, 37(9), 2348-2353.
- Kwakkel, G., Kollen, B. J., van der Grond, J., & Prevo, A. J. (2003). Probability of regaining dexterity in the flaccid upper limb: impact of severity of paresis and time since onset in acute stroke. *Stroke*, 34(9), 2181-2186.

- Labriola, J. E., Lee, T. Q., Debski, R. E., & McMahon, P. J. (2005). Stability and instability of the glenohumeral joint: the role of shoulder muscles. *Journal of Shoulder and Elbow Surgery*, 14(1 Suppl S), 32S-38S.
- Lai, S., Studenski, S., Duncan, P. W., & Perera, S. (2002). Persisting consequences of stroke measured by the Stroke Impact Scale. *Stroke*, 33(7), 1840-1844.
- Lakse, E., Gunduz, B., Erhan, B., & Celik, E. C. (2009). The effect of local injections in hemiplegic shoulder pain: A prospective, randomized, controlled study. *American Journal of Physical Medicine and Rehabilitation*, 88(10), 805-811.
- Lam, F., Bhatia, D. N., Mostofi, S. B., van Rooyen, K., & de Beer, J. F. (2007). Biomechanical considerations of the normal and rotator cuff deficient shoulders and the reverse shoulder prosthesis. *Current Orthopaedics*, 21(1), 40-46.
- Lance, J. W. (1980). Spasticity: Disorder of Motor Control. In R.G. Feldman, R.R. Young and W.P. Koella (Eds.), *Symposium Synopsis* (pp.487-494). Chicago, United States of America.
- Leardini, A., Cappozzo, A., Catani, F., Toksvig-Larsen, S., Petitto, A., et al. (1999). Validation of a functional method for the estimation of hip joint centre location. *Journal of Biomechanics*, 32(1), 99-103.
- Lee, C., & Han, S. (2000). Evaluations of Anterior Displacement of Humeral Head in Hemiplegic Shoulder Subluxation Patients. *Archives of Physical Medicine & Rehabilitation*, 81(9), 1300.
- Lee, I. S., Shin, Y. B., Moon, T. Y., Jeong, Y. J., Song, J. W., et al. (2009). Sonography of patients with hemiplegic shoulder pain after stroke: correlation with motor recovery stage. *American Journal of Roentgenology*, 192(2), W40-44.
- Lee, M. Y., & Jang, S. H. (2011). Ipsilateral motor cortex activation by unaffected hand movements in patients with cerebral infarct. *NeuroRehabilitation*, 29(4), 359-364.
- Lempereur, M., Brochard, S., & Remy-Neris, O. (2013). Repeatability assessment of functional methods to estimate the glenohumeral joint centre. *Computer Methods in Biomechanics and Biomedical Engineering*, 16(1), 6-11.
- Lempereur, M., Leboeuf, F., Brochard, S., Rousset, J., Burdin, V., et al. (2010). In vivo estimation of the glenohumeral joint centre by functional methods: Accuracy and repeatability assessment. *Journal of Biomechanics*, 43(2), 370-374.
- Lindgren, I., Jonsson, A., Norrving, B., & Lindgren, A. (2007). Shoulder pain after stroke: a prospective population-based study. *Stroke*, 38(2), 343-348.
- Lindgren, I., Lexell, J., Jonsson, A. C., & Brogardh, C. (2012). Left-Sided Hemiparesis, Pain Frequency, and Decreased Passive Shoulder Range of Abduction Are Predictors of Long-Lasting Poststroke Shoulder Pain. *American Academy of Physical Medicine and Rehabilitation* 4(8), 561-568.

- Lo, S. F., Chen, S. Y., Lin, H. C., Jim, Y. F., Meng, N. H., et al. (2003). Arthrographic and clinical findings in patients with hemiplegic shoulder pain. *Archives of Physical Medicine and Rehabilitation*, 84(12), 1786-1791.
- Loewen, S. C., & Anderson, B. A. (1988). Reliability of the Modified Motor Assessment Scale and the Barthel Index. *Physical Therapy*, 68(7), 1077-1081.
- Loewen, S. C., & Anderson, B. A. (1990). Predictors of stroke outcome using objective measurement scales. *Stroke*, 21(1), 78-81.
- Lucas, D. B. (1973). Biomechanics of the Shoulder Joint. *Archives of Surgery*, 107(3), 425-432.
- Ludewig, P. M., & Braman, J. P. (2011). Shoulder impingement: biomechanical considerations in rehabilitation. *Manual Therapy*, 16(1), 33-39.
- Ludewig, P. M., & Cook, T. M. (2000). Alterations in Shoulder Kinematics and Associated Muscle Activity in People With Symptoms of Shoulder Impingement. *Physical Therapy*, 80(3), 276-291.
- Ludewig, P. M., & F., R. J. (2009). The Association of Scapular Kinematics and Glenohumeral Joint Pathologies *Journal of Orthopaedic and Sports Physical Therapy*, 39(2), 90-104.
- Lum, P. S., Mulroy, S., Amdur, R. L., Requejo, P., Prilutsky, B. I., et al. (2009). Gains in upper extremity function after stroke via recovery or compensation: Potential differential effects on amount of real-world limb use. *Topics in Stroke Rehabilitation*, 16(4), 237-253.
- Malhotra, S., Pandyan, A., Day, C., Jones, P., & Hermens, H. (2009). Spasticity, an impairment that is poorly defined and poorly measured. *Clinical Rehabilitation*, 23(7), 651-658.
- Mani, S., Mutha, P. K., Przybyla, A., Haaland, K. Y., Good, D. C., et al. (2013). Contralesional motor deficits after unilateral stroke reflect hemisphere-specific control mechanisms. *Brain*, 136 (Part 4), 1288-1303.
- Mani, S., Przybyla, A., Good, D. C., Haaland, K. Y., & Sainburg, R. L. (2014). Contralesional Arm Preference Depends on Hemisphere of Damage and Target Location in Unilateral Stroke Patients. *Neurorehabilitation & Neural Repair*. doi: 10.1177/1545968314520720
- Marciniak, C. (2011). Poststroke hypertonicity: upper limb assessment and treatment. *Topics in Stroke Rehabilitation*, 18(3), 179-194.
- Massie, C. L., Malcolm, M. P., Greene, D. P., & Browning, R. C. (2012). Kinematic motion analysis and muscle activation patterns of continuous reaching in survivors of stroke. *Journal of Motor Behaviour*, 44(3), 213-222.
- McBeth, J., & Jones, K. (2007). Epidemiology of chronic musculoskeletal pain. *Best Practice and Research Clinical Rheumatology*, 21(3), 403-425.

- McClure, P. W., Michener, L. A., & Karduna, A. R. (2006). Shoulder function and 3-dimensional scapular kinematics in people with and without shoulder impingement syndrome. *Physical Therapy*, 86(8), 1075-1090.
- McLean, D. E. (2004). Medical complications experienced by a cohort of stroke survivors during inpatient, tertiary-level stroke rehabilitation. *Archives of Physical Medicine & Rehabilitation*, 85(3), 466-469.
- Mehrholz, J., Wagner, K., Meissner, D., Grundmann, K., Zange, et al. (2005). Reliability of the Modified Tardieu Scale and the Modified Ashworth Scale in adult patients with severe brain injury: a comparison study. *Clinical Rehabilitation*, 19(7), 751-759.
- Meskers, C., Koppe, P., Konijnenbelt, M., Veeger, D., & Janssen, T. (2005). Kinematic Alterations in the Ipsilateral Shoulder of Patients with Hemiplegia due to Stroke. *American Journal of Physical Medicine & Rehabilitation*, 84(2), 97-105.
- Meskers, C., Vermeulen, H., Groot, J., van der Helm, F., & Rozing, P. (1998). 3D shoulder position measurements using a six-degree-of-freedom electromagnetic tracking device. *Clinical Biomechanics*, 13, 280-292.
- Michaeleff, Z. A., & Kamper, S. J. (2013). PEDro systematic review update: the effectiveness of physiotherapy exercises in subacromial impingement syndrome. *British Journal of Sports Medicine*, 47(14), 927-928.
- Michielsen, M. E., Selles, R. W., Stam, H. J., Ribbers, G. M., & Bussmann, J. B. J. (2012). Quantifying non-use in chronic stroke patients: a study into paretic, non-paretic and bimanual upper-limb use in daily life. *Archives of Physical Medicine & Rehabilitation*, 93(11), 1975-1981.
- Mills, P., Morrison, S., Lloyd, D., & Barrett, R. (2007). Repeatability of 3D gait kinematics obtained from an electromagnetic tracking system during treadmill locomotion. *Journal of Biomechanics*, 40, 1504-1511.
- Monnet, T., Desailly, E., Begon, M., Vallee, C., & Lacouture, P. (2007). Comparison of the SCoRE and HA methods for locating in vivo the glenohumeral joint centre. *Journal of Biomechanics*, 40(15), 3487-3492.
- Morris, S. (2002). Ashworth and Tardieu Scales: their clinical relevance for measuring spasticity in adult and paediatric neurological populations. *Physical Therapy Reviews*, 7(1), 53-62.
- Moser, V. F., & Ward, A. B. (2000). Commentary on the scope for rehabilitation in severely disabled stroke patients. Gladman JRF, Sackley CM. The scope for rehabilitation in severely disabled stroke patients. *Disability and Rehabilitation* 1998; 20(10): 391-394. *Disability & Rehabilitation*, 22(4), 188.
- Moskowitz, H., Goodman, C. R., Smith, E., Balthazar, E., & Mellins, H. Z. (1969). Hemiplegic shoulder. *New York State Journal of Medicine*, 69(4), 548-550.

- Mostaedi, B. B. (2004). *Role of the shoulder muscles in controlling the glenohumeral joint and prevention of subacromial impingement in overhead activities: A meta-study*. (3122854 Ph.D.), Union Institute and University, Ann Arbor. Retrieved from ProQuest Dissertations & Theses Global database.
- Motti, R., Ruth, D., & Nachum, S. (2012). A randomized controlled study of segmental neuromyotherapy for post-stroke hemiplegic shoulder pain. *Journal of Rehabilitation Medicine*, 44(10), 830-836.
- Murie-Fernández, M., Carmona Iragui, M., Gnanakumar, V., Meyer, M., Foley, N., et al. (2012). Painful hemiplegic shoulder in stroke patients: Causes and management. *Neurologia*, 27(4), 234-244.
- Najenson, T., Yacubovich, E., & Pikielni, S. S. (1971). Rotator cuff injury in shoulder joints of hemiplegic patients. *Scandinavian Journal of Rehabilitation Medicine*, 3(3), 131-137.
- Nakamura, T., Abreu, B. C., Patterson, R. M., Buford, W. L., Jr., & Ottenbacher, K. J. (2008). Upper-limb kinematics of the presumed-to-be-unaffected side after brain injury. *American Journal of Occupational Therapy*, 62(1), 46-50.
- Nakayama, H., Jorgensen, H. S., Raaschou, H. O., & Olsen, T. S. (1994). Recovery of upper extremity function in stroke patients: the Copenhagen Stroke Study. *Archives of Physical Medicine & Rehabilitation*, 75(4), 394-398.
- National Health and Medical Research Council. (2009). *NHMRC additional levels of evidence and grades for recommendations for developers of guidelines*. National Health and Medical Research Council Retrieved from https://www.nhmrc.gov.au/_files_nhmrc/file/guidelines/developers/nhmrc_levels_grades_evidence_120423.pdf.
- National Stroke Foundation. (2010). National Stroke Foundation. Clinical Guidelines for Stroke Management 2010. Melbourne Australia. Retrieved from http://strokefoundation.com.au/site/media/clinical_guidelines_stroke_management_2010_interactive.pdf.
- National Stroke Foundation. (2013). The Economic Impact of Stroke in Australia. Retrieved from <http://strokefoundation.com.au/site/media/Final-Deloitte-Stroke-Report-14-Mar-13.pdf>.
- National Stroke Foundation. (2014a). Signs of stroke. Retrieved from <http://strokefoundation.com.au/what-is-a-stroke/signs-of-stroke>.
- National Stroke Foundation. (2014b). Types of stroke. Retrieved from <http://strokefoundation.com.au/what-is-a-stroke/types-of-stroke>.
- Nazir, F. S., Petre, I., & Dewey, H. M. (2009). Introduction of an acute stroke team: an effective approach to hasten assessment and management of stroke in the emergency department. *Journal Of Clinical Neuroscience*, 16(1), 21-25.

- Niessen, M., Janssen, T., Meskers, C., Koppe, P., Konijnenbelt, M., et al. (2008). Kinematics of the contralateral and ipsilateral shoulder: a possible relationship with post-stroke shoulder pain. *Journal of Rehabilitation Medicine*, 40(6), 482-486.
- Niessen, M. H., Veeger, D. H., Meskers, C. G., Koppe, P. A., Konijnenbelt, M. H., et al. (2009). Relationship among shoulder proprioception, kinematics, and pain after stroke. *Archives of Physical Medicine & Rehabilitation*, 90(9), 1557-1564.
- Nudo, R. J., Friel, K. M., & Delia, S. W. (2000). Role of sensory deficits in motor impairments after injury to primary motor cortex. *Neuropharmacology*, 39(5), 733-742.
- O'Dell, M. W., Lin, C. C., & Harrison, V. (2009). Stroke rehabilitation: strategies to enhance motor recovery. *Annual Review of Medicine*, 60, 55-68.
- O'Leary, S., Falla, D., Hodges, P. W., Jull, G., & Vicenzino, B. (2007). Specific Therapeutic Exercise of the Neck Induces Immediate Local Hypoalgesia. *The Journal of Pain*, 8(11), 832-839.
- Paci, M. (2010). Risk factors for shoulder subluxation after stroke. *Physical Therapy Reviews*, 15(2), 119-119.
- Paci, M., Nannetti, L., & Rinaldi, L. A. (2005). Glenohumeral subluxation in hemiplegia: an overview. *Journal of Rehabilitation Research and Development*, 42(4), 557-568.
- Paci, M., Nannetti, L., Taiti, P., Baccini, M., & Rinaldi, L. A. (2007). Shoulder subluxation after stroke: relationships with pain and motor recovery. *Physiotherapy Research International*, 12(2), 95-104.
- Page, S. J., Gater, D. R., & Bach-y-Rita, P. (2004). Reconsidering the motor recovery plateau in stroke rehabilitation. *Archives of Physical Medicine & Rehabilitation*, 85(8), 1377-1381.
- Page, T., & Lockwood, C. (2003). Prevention and management of shoulder pain in the hemiplegic patient. *Joanna Briggs Institute Reports*, 1(5), 149-165.
- Park, G., Kim, J., Sohn, S., Shin, I., & Lee, M. Y. (2007). Ultrasonographic measurement of shoulder subluxation in patients with post-stroke hemiplegia. *Journal of Rehabilitation Medicine*, 39(7), 526-530.
- Parker, V. M., Wade, D. T., & Langton Hewer, R. (1986). Loss of arm function after stroke: measurement, frequency, and recovery. *International Rehabilitation Medicine*, 8(2), 69-73.
- Patrick, E., & Ada, L. (2006). The Tardieu Scale differentiates contracture from spasticity whereas the Ashworth Scale is confounded by it. *Clinical Rehabilitation*, 20(2), 173-182.

- Patrick, P. D., Savage, R. C., & Drača, S. (2012). Gender and stroke lateralization: Factors of functional recovery after the first-ever unilateral stroke? *NeuroRehabilitation*, 30(3), 247-254.
- Pertoldi, S., & Di Benedetto, P. (2005). Shoulder-hand syndrome after stroke: a complex regional pain syndrome. *Europa Medicophysica*, 41(4), 283-292.
- Pohl, P. S., Luchies, C. W., Stoker-Yates, J., & Duncan, P. W. (2000). Upper extremity control in adults post stroke with mild residual impairment. *Neurorehabilitation & Neural Repair*, 14(1), 33-41.
- Pompa, A., Clemenzi, A., Troisi, E., Di Mario, M., Tonini, A., et al. (2011). Enhanced-MRI and ultrasound evaluation of painful shoulder in patients after stroke: a pilot study. *European Neurology*, 66(3), 175-181.
- Pong, Y. P., Wang, L. Y., Huang, Y. C., Leong, C. P., Liaw, M. Y., et al. (2012). Sonography and physical findings in stroke patients with hemiplegic shoulders: A longitudinal study. *Journal of Rehabilitation Medicine*, 44(7), 553-557.
- Pong, Y. P., Wang, L. Y., Wang, L. Y., Leong, C. P., Huang, Y. C., et al. (2009). Sonography of the shoulder in hemiplegic patients undergoing rehabilitation after a recent stroke. *Journal of Clinical Ultrasound*, 37(4), 199-205.
- Poppen, N., & Walker, P. (1976). Normal and abnormal motion of the shoulder. *Journal of Bone and Joint Surgery*, 58(2), 195-201.
- Portney, L. G., & Watkins, M. P. (2009). *Foundations of clinical research : applications to practice* (3rd ed. ed.). Upper Saddle River, New Jersey: Pearson/Prentice Hall.
- Poulin de Courval, L., Barsauskas, A., Berenbaum, B., Dehaut, F., Dussault, R., et al. (1990). Painful shoulder in the hemiplegic and unilateral neglect. *Archives of Physical Medicine & Rehabilitation*, 71(9), 673-676.
- Price, C. I., Rodgers, H., Franklin, P., Curless, R. H., & Johnson, G. R. (2001). Glenohumeral subluxation, scapula resting position, and scapula rotation after stroke: A noninvasive evaluation. *Archives of Physical Medicine and Rehabilitation*, 82(7), 955-960.
- Quain, D. A., Parsons, M. W., Loudfoot, A. R., Spratt, N. J., Evans, M. K., et al. (2008). Improving access to acute stroke therapies: a controlled trial of organised pre-hospital and emergency care. *The Medical Journal Of Australia*, 189(8), 429-433.
- Rah, U. W., Yoon, S. H., Moon, D. J., Kwack, K. S., Hong, J. Y., et al. (2012). Subacromial Corticosteroid Injection on Poststroke Hemiplegic Shoulder Pain: A Randomized, Triple-Blind, Placebo-Controlled Trial. *Archives of Physical Medicine & Rehabilitation*, 93(6), 949-956.

- Rajaratnam, B. S., Lim, M. G., Chia, H. L. C., Chua, Y. Q. S., Gan, M. C., et al. (2008). Clinical features associated with hemiplegic shoulder pain: a systematic review. *Physiotherapy Singapore*, 11(4), 11-17.
- Ratnasabapathy, Y., Broad, J., Baskett, J., Pledger, M., Marshall, J., et al. (2003). Shoulder pain in people with stroke: a population-based study. *Clinical Rehabilitation*, 17(3), 304-311.
- Rettig, O., Maier, M. W., Gantz, S., Raiss, P., Zeifang, F., et al. (2013). Does the reverse shoulder prosthesis medialize the center of rotation in the glenohumeral joint? *Gait and Posture*, 37(1), 29-31.
- Rinehart, J. K., Singleton, R. D., Adair, J. C., Sadek, J. R., & Haaland, K. Y. (2009). Arm Use After Left or Right Hemiparesis Is Influenced by Hand Preference. *Stroke*, 40(2), 545-550.
- Robertson, J. V., Roche, N., & Roby-Brami, A. (2012). Influence of the side of brain damage on postural upper-limb control including the scapula in stroke patients. *Experimental Brain Research*, 218(1), 141-155.
- Roosink, M., Buitenweg, J. R., Renzenbrink, G. J., Geurts, A. C., & Ijzerman, M. J. (2011). Altered cortical somatosensory processing in chronic stroke: A relationship with post-stroke shoulder pain. *NeuroRehabilitation*, 28(4), 331-344.
- Roosink, M., Renzenbrink, G. J., Buitenweg, J. R., Van Dongen, R. T., Geurts, A. C., et al. (2011). Persistent shoulder pain in the first 6 months after stroke: results of a prospective cohort study. *Archives of Physical Medicine and Rehabilitation*, 92(7), 1139-1145.
- Roosink, M., Renzenbrink, G. J., Geurts, A. C. H., & Ijzerman, M. J. (2012a). Re: Underlying pathology and associated factors of hemiplegic shoulder pain. *American Journal of Physical Medicine & Rehabilitation*, 91(3), 279-280.
- Roosink, M., Renzenbrink, G. J., Geurts, A. C. H., & Ijzerman, M. J. (2012b). Towards a mechanism-based view on post-stroke shoulder pain: Theoretical considerations and clinical implications. *NeuroRehabilitation*, 30(2), 153-165.
- Roosink, M., Van Dongen, R. T., Buitenweg, J. R., Renzenbrink, G. J., Geurts, A. C., et al. (2012). Multimodal and Widespread Somatosensory Abnormalities in Persistent Shoulder Pain in the First 6 Months After Stroke: An Exploratory Study. *Archives of Physical Medicine & Rehabilitation*, 93(11), 1968-1974.
- Rosales, R. L., & Chua-Yap, A. S. (2008). Evidence-based systematic review on the efficacy and safety of botulinum toxin-A therapy in post-stroke spasticity. *Journal Of Neural Transmission*, 115(4), 617-623.

- Rosales, R. L., Kanovsky, P., & Fernandez, H. H. (2011). What's the "catch" in upper-limb post-stroke spasticity: expanding the role of botulinum toxin applications. *Parkinsonism and Related Disorders*, 17 Suppl 1, S3-10.
- Rose, L., Bakal, D. A., Fung, T. S., Farn, P., & Weaver, L. E. (1994). Tactile extinction and functional status after stroke. A preliminary investigation. *Stroke*, 25(10), 1973-1976.
- Roy, C. W., Sands, M. R., Hills, L. D., Harrison, H. S., & Marshall, S. (1995). The effect of shoulder pain on outcome of hemiplegia. *Clinical Rehabilitation*, 9, 21-27.
- Rundquist, P. J., Dumit, M., Hartley, J., & S., K. (2012). Three-dimensional shoulder complex kinematics in individuals with upper extremity impairment from chronic stroke. *Disability and Rehabilitation*, 34(5), 402-407.
- Sang, K. L., Dae Suk, Y., Ha Yong, K., & Won Sik, C. (2013). A comparison of 3D scapular kinematics between dominant and nondominant shoulders during multiplanar arm motion. *Indian Journal of Orthopaedics*, 47(2), 135-142.
- Scalha, T. B., Miyasaki, E., Lima, N. M., & Borges, G. (2011). Correlations between motor and sensory functions in upper limb chronic hemiparetics after stroke. *Arquivos de Neuropsiquiatria*, 69(4), 624-629.
- Schaefer, S. Y., Haaland, K. Y., & Sainburg, R. L. (2009). Hemispheric specialization and functional impact of ipsilesional deficits in movement coordination and accuracy. *Neuropsychologia*, 47(13), 2953-2966.
- Schwerin, S., Dewald, J. P., Haztl, M., Jovanovich, S., Nickeas, M., et al. (2008). Ipsilateral versus contralateral cortical motor projections to a shoulder adductor in chronic hemiparetic stroke: implications for the expression of arm synergies. *Experimental Brain Research*, 185(3), 509-519.
- Seshadri, S., Beiser, A., Kelly-Hayes, M., Kase, C. S., Au, R., et al. (2006). The lifetime risk of stroke: estimates from the Framingham Study. *Stroke*, 37(2), 345-350.
- Shah, R. R., Haghighpanah, S., Elovic, E. P., Flanagan, S. R., Behnegar, et al. (2008). MRI findings in the painful poststroke shoulder. *Stroke*, 39(6), 1808-1813.
- Shepherd, R. B., & Carr, J. H. (1998). The Shoulder following Stroke: Preserving Musculoskeletal Integrity for Function. *Topics in Stroke Rehabilitation*, 4(4), 35-53.
- Shrout, P. E. (1998). Measurement reliability and agreement in psychiatry. *Stat Methods Med Res*, 7(3), 301-317.
- Shrout, P. E., & Fleiss, J. L. (1979). Intraclass correlations: uses in assessing rater reliability. *Psychol Bull*, 86(2), 420-428.
- Singh, J. A., & Fitzgerald, P. M. (2011). Botulinum toxin for shoulder pain: a cochrane systematic review. *Journal of Rheumatology*, 38(3), 409-418.

- Smith, M. (2012). Management of hemiplegic shoulder pain following stroke. *Nursing Standard*, 26(44), 35-44.
- Snodgrass, S. J., Heneghan, N. R., Tsao, H., Stanwell, P. T., Rivett, D. A., et al. (2014). Recognising neuroplasticity in musculoskeletal rehabilitation: A basis for greater collaboration between musculoskeletal and neurological physiotherapists. *Manual Therapy*. doi: <http://dx.doi.org/10.1016/j.math.2014.01.006>.
- Sommerfeld, D. K., Eek, E. U. B., Svensson, A. K., Holmqvist, L. W., & von Arbin, M. H. (2004). Spasticity After Stroke: Its Occurrence and Association With Motor Impairments and Activity Limitations. *Stroke*, 35(1), 134-139.
- Soo Hoo, J., Paul, T., Chae, J., & Wilson, R. D. (2013). Central Hypersensitivity in Chronic Hemiplegic Shoulder Pain. *American Journal of Physical Medicine and Rehabilitation*, 92(1), 1-9.
- Staines, W. R., Black, S. E., Graham, S. J., & McIlroy, W. E. (2002). Somatosensory Gating and Recovery From Stroke Involving the Thalamus. *Stroke*, 33(11), 2642-2651.
- Sterr, A., & Conforto, A. B. (2012). Plasticity of adult sensorimotor system in severe brain infarcts: challenges and opportunities. *Neural Plasticity*. doi:10.1155/2012/970136.
- Sterzi, R., Bottini, G., Celani, M. G., Righetti, E., Lamassa, M., Ricci, S., & Vallar, G. (1993). Hemianopia, hemianaesthesia, and hemiplegia after right and left hemisphere damage. A hemispheric difference. *Journal of Neurology, Neurosurgery & Psychiatry*, 56(3), 308-310.
- Stevenson, T., Thalman, L., Christie, H., & Poluha, W. (2012). Constraint-Induced Movement Therapy Compared to Dose-Matched Interventions for Upper-Limb Dysfunction in Adult Survivors of Stroke: A Systematic Review with Meta-analysis. *Physiotherapy Canada*, 64(4), 397-413.
- Stinear, C. (2010). Prediction of recovery of motor function after stroke. *Lancet Neurology*, 9(12), 1228-1232.
- Stokdijk, M., Nagels, J., & Rozing, P. (2000). The glenohumeral joint rotation centre in vivo. *Journal of Biomechanics*, 33, 1629-1636.
- Stolzenberg, D., Siu, G., & Cruz, E. (2012). Current and Future Interventions for Glenohumeral Subluxation in Hemiplegia Secondary to Stroke. *Topics in Stroke Rehabilitation*, 19(5), 444-456.
- Stoykov, M. E., Lewis, G. N., & Corcos, D. M. (2009). Comparison of bilateral and unilateral training for upper extremity hemiparesis in stroke. *Neurorehabilitation and Neural Repair*, 23(9), 945-953.

- Struyf, F., Nijs, J., Baeyens, J. P., Mottram, S., & Meeusen, R. (2011). Scapular positioning and movement in unimpaired shoulders, shoulder impingement syndrome, and glenohumeral instability. *Scandinavian Journal Of Medicine & Science In Sports*, 21(3), 352-358.
- Suethanapornkul, S., Srisa-an Kuptniratsaikul, P., Kuptniratsaikul, V., Uthensut, P., Dajpratha, P., et al. (2008). Post Stroke Shoulder Subluxation and Shoulder Pain: A Cohort Multicenter Study. *Journal of Medical Association of Thailand*, 91(12), 1885-1893.
- Tai, Y. J., & Yan, B. (2013). Minimising time to treatment: targeted strategies to minimise time to thrombolysis for acute ischaemic stroke. *Internal Medicine Journal*, 43(11), 1176-1182.
- Tan, L., Wang, M.-J., Modini, M., Joyce, S., Mykletun, A., Christensen, H., & Harvey, S. B. (2014). Preventing the development of depression at work: a systematic review and meta-analysis of universal interventions in the workplace. *BMC Medicine*, 12(1), 74-74. doi: 10.1186/1741-7015-12-74.
- Távora, D. G. F., Gama, R. L., Bomfim, R. C., Nakayama, M., & Silva, C. E. P. (2010). MRI findings in the painful hemiplegic shoulder. *Clinical Radiology*, 65(10), 789-794.
- Teasell, R., Foley, N., Pereira, S., Sequeira, K., & Miller, T. (2012). Evidence to Practice: Botulinum Toxin in the Treatment of Spasticity Post Stroke. *Topics in Stroke Rehabilitation*, 19(2), 115-121.
- Teasell, R., Mehta, S., Pereira, S., McIntyre, A., Janzen, S., et al. (2012). Time to rethink long-term rehabilitation management of stroke patients. *Topics in Stroke Rehabilitation*, 19(6), 457-462.
- Teasell, R. W., & Heitzner, J. D. (1998). The painful hemiplegic shoulder. *Physical Medicine and Rehabilitation*, 12(3), 489-500.
- Tekavec, E., Jöud, A., Rittner, R., Mikoczy, Z., Nordander, C., et al. (2012). Population-based consultation patterns in patients with shoulder pain diagnoses. *BMC Musculoskeletal Disorders*, 13, 238-238.
- Teyhen, D. S., Miller, J. M., Middag, T. R., & Kane, E. J. (2008). Rotator cuff fatigue and glenohumeral kinematics in participants without shoulder dysfunction. *Journal of Athletic Training*, 43(4), 352-358.
- Teys, P., Bisset, L., & Vicenzino, B. (2008). The initial effects of a Mulligan's mobilization with movement technique on range of movement and pressure pain threshold in pain-limited shoulders. *Manual Therapy*, 13(1), 37-42.
- Thrift, A. G., Dewey, H. M., Macdonell, R. A., McNeil, J. J., & Donnan, G. A. (2001). Incidence of the major stroke subtypes: initial findings from the North East Melbourne stroke incidence study (NEMESIS). *Stroke*, 32(8), 1732-1738.

- Thrift, A. G., Dewey, H. M., Macdonell, R. A., McNeill, J. J., & Donnan, G. A. (2000). Stroke Incidence on the East Coast of Australia : The North East Melbourne Stroke Incidence Study (NEMESIS). *Stroke*, 31, 2087-2092.
- Thrift, A. G., Dewey, H. M., Sturm, J. W., Paul, S. L., Gilligan, A. K., et al. (2006). Greater incidence of both fatal and nonfatal strokes in disadvantaged areas: the Northeast Melbourne Stroke Incidence Study. *Stroke*, 37(3), 877-882.
- Thrift, A. G., Dewey, H. M., Sturm, J. W., Srikanth, V. K., Gilligan, A. K., et al. (2009). Incidence of stroke subtypes in the North East Melbourne Stroke Incidence Study (NEMESIS): differences between men and women. *Neuroepidemiology*, 32(1), 11-18.
- Tobola, A., Cassas, K., & Sease, F. (2009). Shoulder pain in older patients: interpreting clinical findings. *Journal of Musculoskeletal Medicine*, 26(11), 426.
- Turner-Stokes, L., & Jackson, D. (2002). Shoulder pain after stroke: a review of the evidence to inform the development of an integrated care pathway. *Clinical Rehabilitation*, 16, 276-298.
- Tymianski, D. (2013). Neuroanatomy and physiology. *Canadian Journal of Neuroscience Nursing*, 35(3), 6-22.
- Tyson, S. F., Chillala, J., Hanley, M., Selley, A. B., & Tallis, R. C. (2006). Distribution of weakness in the upper and lower limbs post-stroke. *Disability and Rehabilitation*, 28(11), 715-719.
- Umphred, D. A. (2007). *Neurological rehabilitation*. St. Louis, Mo: Mosby Elsevier.
- Urban, P. P., Wolf, T., Uebele, M., Marx, J. J., Vogt, T., et al. (2010). Occurrence and clinical predictors of spasticity after ischemic stroke. *Stroke*, 41(9), 2016-2020.
- van der Meche, F. G., & van Gijn, J. (1986). Hypotonia: an erroneous clinical concept? *Brain*, 109(Pt 6), 1169-1178.
- Van Langenberghe, H. V. K., Partridge, C. J., Edwards, M. S., & Mee, R. (1988). Shoulder pain in hemiplegia—a literature review. *Physiotherapy Theory and Practice*, 4(3), 155-162.
- Van Peppen, R. P., Kwakkel, G., Wood-Dauphinee, S., Hendriks, H. J., Van der Wees, P. J., et al. (2004). The impact of physical therapy on functional outcomes after stroke: what's the evidence? *Clinical Rehabilitation*, 18(8), 833-862.
- Vasudevan, J. M., & Browne, B. J. (2014). Hemiplegic Shoulder Pain: An Approach to Diagnosis and Management. *Physical Medicine and Rehabilitation Clinics of North America*. Retrieved from <http://www.sciencedirect.com/science/article/pii/S1047965114000114>.
- Vasudevan, J. M., & Vasudevan, S. V. (2008). Hemiplegic shoulder pain: diagnosis and management. *Critical Reviews in Physical & Rehabilitation Medicine*, 20(3), 207-220.

- Veeger, H. E. (2000). The position of the rotation center of the glenohumeral joint. *Journal of Biomechanics*, 33(12), 1711-1715.
- Verheyden, G., Nieuwboer, A., De Wit, L., Thijs, V., Dobbelaere, J., et al. (2008). Time course of trunk, arm, leg, and functional recovery after ischemic stroke. *Neurorehabilitation & Neural Repair*, 22(2), 173-179.
- Viana, R., Pereira, S., Mehta, S., Miller, T., & Teasell, R. (2012). Evidence for Therapeutic Interventions for Hemiplegic Shoulder Pain During the Chronic Stage of Stroke: A Review. *Topics in Stroke Rehabilitation*, 19(6), 514-522.
- Vicenzino, B., Collins, D., Benson, H., & Wright, A. (1998). An investigation of the interrelationship between manipulative therapy-induced hypoalgesia and sympathoexcitation. *Journal of Manipulative and Physiological Therapeutics*, 21(7), 448-453.
- Vidal, A. C., & Pascoal, A. G. (2011). The hemiplegic's shoulder. Analysis of the potential contribution of the scapula's position on the upper limb spastic pattern and glenohumeral subluxation during three exercises for arm elevation. *Journal of Neurology*, 258(Suppl1), s133.
- Vossel, S., Weiss, P. H., Eschenbeck, P., & Fink, G. R. (2013). Anosognosia, neglect, extinction and lesion site predict impairment of daily living after right-hemispheric stroke. *Cortex*, 49(7), 1782-1789.
- Wang, Q., Zhao, J. L., Zhu, Q. X., Li, J., & Meng, P. P. (2011). Comparison of conventional therapy, intensive therapy and modified constraint-induced movement therapy to improve upper extremity function after stroke. *Journal of Rehabilitation Medicine*, 43(7), 619-625.
- Wanklyn, P., Forster, A., & Young, J. (1996). Hemiplegic shoulder pain (HSP): Natural history and investigation of associated features. *Disability and Rehabilitation*, 18(10), 497-501.
- Ward, A. B. (2012). A literature review of the pathophysiology and onset of post-stroke spasticity. *European Journal of Neurology*, 19(1), 21-27.
- White, J. H., Magin, P., Attia, J., Sturm, J., Carter, G., et al. (2012). Trajectories of psychological distress after stroke. *Annals of Family Medicine*, 10(5), 435-442.
- Williams, B. K., Galea, M. P., & Winter, A. T. (2001). What is the functional outcome for the upper limb after stroke? *Australian Journal of Physiotherapy*, 47(1), 19-27.
- Winstein, C. J., Rose, D. K., Tan, S. M., Lewthwaite, R., Chui, H. C., et al. (2004). A randomized controlled comparison of upper-extremity rehabilitation strategies in acute stroke: a pilot study of immediate and long-term outcomes. *Archives of Physical Medicine & Rehabilitation*, 85(4), 620-628.

- Wissel, J., Schelosky, L. D., Scott, J., Christe, W., Faiss, J. H., et al. (2010). Early development of spasticity following stroke: a prospective, observational trial. *Journal of Neurology*, 257(7), 1067-1072.
- Wu, G., van der Helm, F., Veeger, H., Makhsous, M., Van Roy, P., et al. (2005). ISB recommendation on definitions of joint coordinate systems of various joints for the reporting of human joint motion - Part 11: shoulder, elbow, wrist and hand. *Journal of Biomechanics*, 38(5), 981-992.
- Yamaguchi, K., Ditsios, K., Middleton, W. D., Hildebolt, C. F., Galatz, L. M., et al. (2006). The demographic and morphological features of rotator cuff disease. A comparison of asymptomatic and symptomatic shoulders. *The Journal Of Bone And Joint Surgery. American Volume*, 88(8), 1699-1704.
- Yamamoto, A., Takagishi, K., Osawa, T., Yanagawa, T., Nakajima, D., et al. (2010). Prevalence and risk factors of a rotator cuff tear in the general population. *Journal of Shoulder and Elbow Surgery*, 19(1), 116-120.
- Yoon, T. S., & Lee, S. J. (2007). Abduction motion analysis of hemiplegic shoulders with a fluoroscopic guide. *Yonsei Medical Journal*, 48(2), 247-254.
- Yu, D. (2004). Shoulder pain in hemiplegia. *Physical Medicine and Rehabilitation Clinics of North America*, 15(3), 683-697.
- Zeilig, G., Rivel, M., Weingarden, H., Gaidoukov, E., & Defrin, R. (2013). Hemiplegic shoulder pain: Evidence of a neuropathic origin. *Pain*, 154(2), 263-271.