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Scientific Review

Preserving transfer independence among individuals with spinal cord injury

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Study design: Literature review.
Objectives: Upper extremity (UE) joint degeneration, particularly at the shoulder, detrimentally influences functional independence, quality of life, cardiovascular disease risk, and life expectancy of individuals following spinal cord injury (SCI). This review (1) describes UE use for transfers among individuals with SCI; (2) describes contributing factors associated with UE joint degeneration and loss of transfer independence; (3) summarizes and identifies gaps in existing research; and (4) provides suggestions for future research.
Results: Investigations of wheelchair transfer related UE joint and function preservation among individuals with SCI should consider factors including age and length of time from SCI onset, interface between subject-wheelchair, pain, shoulder joint range of motion (ROM) and muscle strength deficiencies or imbalances, exercise capacity and tolerance for the physical strain of activities of daily living (ADL), body mass and composition, previous UE injury or disease history, and transfer techniques. Existing studies of transfers among individuals with SCI have relied on small groups of either asymptomatic or non-impaired subjects, with minimal integration of kinematic, kinetic and electromyographic data. Descriptions of UE joint ROM, forces, and moments produced during transfers are lacking.
Conclusions: Biomechanical measurement and computer modeling have provided increasingly accurate tools for acquiring the data needed to guide intervention planning to prevent UE joint degeneration and preserve function among individuals with SCI. The identification of stressful sub-components during transfers will enable intervening clinicians and engineers who design and modify assistive and adaptive devices to better serve individuals with SCI.
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Introduction

Historically, often due to medical complications and secondary disorders, individuals with spinal cord injury (SCI) were not expected to live long productive lives. Advances in medicine and health care since the Second World War have increased life expectancies for individuals with SCI. For the first time, a cohort of individuals with SCI is approaching their elder years. Since SCI is endemic to a younger population who are living longer lives, a critical concern is how to maintain independence with activities of daily living (ADL) and functional mobility (transfers and wheelchair propulsion) over time. The ability of individuals with SCI to safely transfer from a wheelchair depends upon preserving upper extremity (UE) joint and functional integrity.

Among non-impaired individuals the UE is used primarily for reaching and grasping. Following SCI, the UE must be conditioned to withstand the cumulative forces of life-long weight bearing during wheelchair transfers (Figure 1) and propulsion. Bruehlmeier *et al*[1] in comparing topographic maps of the primary sensorimotor brain regions of individuals with SCI reported an expansion of the cortical hand area toward the cortical leg area. They postulated that brain activation changes following SCI suggested an adaptation of UE movement patterns to substitute for wheelchair transfer and propulsion demands.[1]

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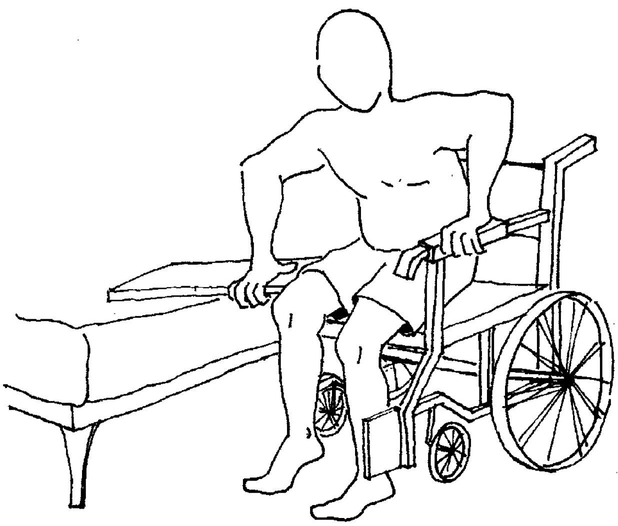

Figure 1 Sliding board transfer from wheelchair to bed

A cultural shift is needed to heighten awareness of wheelchair transfer safety, UE joint injury prevention and preservation of function. This review (1) describes UE use for transfers among individuals with SCI; (2) describes contributing factors associated with UE joint degeneration and loss of transfer independence; (3) summarizes and identifies gaps in existing research; and (4) provides suggestions for future research. Our goal is to stimulate transfer safety research for individuals with SCI.

Upper extremity function following SCI

Many individuals with SCI must re-learn how to perform ADL without the functional use of their lower extremities. Tasks such as mobility (transfers and wheelchair propulsion), weight transfer, and postural stability are now shifted to the UE. This functional shift results in UE joint degeneration from the cumulative effects of repetitious joint loading forces, dramatically affecting the quality of life of individuals with SCI, adding to their disability and diminishing their independence.[2–5] Because of its considerable mobility, multiple linkages, poor osseous stability, and predisposition to range of motion (ROM) and muscular strength deficiency or imbalance-induced postural changes, the shoulder is at particular injury risk among individuals with SCI. Lal,[4] in assessing the shoulder of 53 subjects at more than 15 years following SCI injury reported that 72% (38/53) had radiological evidence of degenerative changes. Older subjects with greater wheelchair dependence and females were more likely to develop degenerative changes in the shoulders.[4]

Individuals with SCI also experience a high incidence of carpal tunnel syndrome (CTS) or CTS-like symptoms.[6–10] Aljure *et al*[8] reported that 40% (19/47) of patients with paraplegia had clinical evidence of CTS. Electrophysiological evidence of CTS was found in 63% of these patients and 40% had evidence of concurrent ulnar neuropathy. Gellman *et al*[6] reported that 64.3% (54/84) of patients with paraplegia had CTS from the cumulative effects of transitory increased median nerve pressures from wheelchair transfers and propulsion. Sie *et al*[7] reported that 'repetitive contact neuropathy' may more accurately describe the 'CTS-like' bilateral thumb and index finger hyperesthesias experienced by individuals with SCI. Nichols *et al*[9] reported that 66% of patients with paraplegia who had significant UE pain also had CTS. Nemchausky & Ubilluz[10] reported that 16% (3/19) of male patients with acute or chronic SCI had clinical CTS and 26% (5/19) had 'CTS-like' neuropathies.

Factors contributing to UE joint degeneration and loss of function following SCI

To prepare wheelchair users for maximal functional independence, clinicians must address multiple UE joint degeneration and loss of function risk factors that may influence program outcomes, long-term wheelchair transfer independence, and safety of individuals with SCI (Figure 2). Factors which will be addressed include age and length of time from SCI onset, interface between subject-wheelchair, pain, UE joint ROM and muscle strength deficiencies or imbalances, exercise capacity and tolerance for the physical strain of ADL, body mass and composition, previous UE injury or disease history, and transfer techniques.

Age and length of time from SCI onset In assessing 708 individuals with paraplegia for the effect of age on self-care and mobility, Yarkony *et al*[11] reported that advancing age was associated with decreased independence in bathing, dressing, stair climbing, and transfers. Pentland and Twomey[12] reported that length of time from SCI onset was most predictive of developing UE complications secondary to wheelchair use. Wheelchair use in long term SCI is directly associated with UE pain and joint degeneration which interferes with the independent performance of ADL.[12,13] Subbarao *et al*[14] in comparing individuals with SCI who were either with or without shoulder pain, reported that time from SCI onset was the only variable that differentiated the two groups. Curtis *et al*[15,16] reported a strong relationship between age and the Wheelchair User's Shoulder Pain Index (WUSPI) score and a somewhat weaker relationship between years of wheelchair use and the WUSPI score. Although shoulder pain prevalence increases with age and years of wheelchair use, the age of the individual with SCI had the greater influence, particularly among extremely young or older subjects.[16] Gellman *et al*[6] in an UE evaluation of 84 individuals with paraplegia reported that shoulder pain frequency during transfers increased with the length of time from SCI onset with

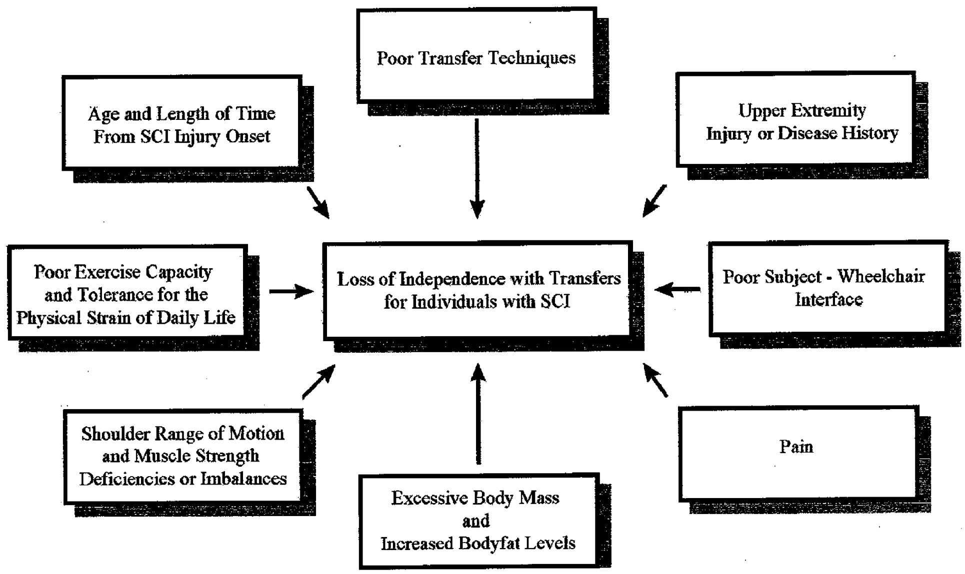

Figure 2 Risk factors associated with UE joint degeneration and loss of function following SCI

52% complaining of pain during the initial 5 years post-onset, and 100% complaining of shoulder pain by 20 years post-onset.

Interface between subject-wheelchair Seelen and Vuurman[17] reported that the complex sensorimotor impairments associated with SCI limit the dynamic control of sitting posture and task performance while sitting, likely serving as the precursor to the development of compensatory UE and trunk motor control mechanisms. Hobson *et al*[18] reported that decreased trunk stability necessitated individuals with paraplegia to assume a biomechanically abnormal sitting posture characterized by a 'C' shaped thoracic spine kyphosis, an extended cervical spine, a flattened lumbar spine, and a posteriorly tilted pelvis (approximately 15° more tilt than non-impaired subjects). Minkel[19] suggested that the increased passive stability provided by this sitting posture enabled more effective UE use during wheelchair propulsion and overhead reaching during the early years of wheelchair use.

Scapular positioning is controlled by multiple musculotendinous attachments and thoracic spine posture. When sitting, lumbar spine posture changes from its normal lordosis to a more straight or flat alignment, increasing thoracic spine kyphosis, prompting downward rotation and protraction of the scapulae.[20] Prolonged sitting and wheelchair propulsion among individuals with SCI further promotes this scapular orientation. Downwardly rotated and protracted scapular positioning contributes to abnormal forces impinging the subacromial tissues (glenohumeral joint capsule, subacromial bursa, rotator cuff tendons).[20] Because of this predisposition, individuals with compromised neuromuscular function are at particular risk for developing subacromial impingement when attempting overhead tasks from a sitting posture.[20] The direct relationship between lumbar and thoracic spine postures and their influence on scapular orientation suggests that maintaining a more neutral lumbar spine posture may help alleviate shoulder pain related to impingement and overhead reaching tasks.

Pain In a survey of 130 individuals with SCI, Dalyan *et al*[21] reported that 58.5% (76 total, 38 with paraplegia, 38 with tetraplegia) experienced UE pain. Of these respondents, pain was identified at the shoulder (71%), wrist (51%), hand (43%) and elbow (35%).[21] Of the 10 functional activities that were assessed, UE pain was most associated with pressure relief, transfers, and wheelchair propulsion, with 65% (36/55) of the patients complaining of pain that interfered with transfer performance. Subbarao *et al*[14] reported that UE pain was more prevalent in SCI patients (72.7%, 582/800) than in non-impaired subjects. Pentland and Twomey[12,13] reported that 31% of individuals with long-term paraplegia had elbow pain, 39% had shoulder pain and 40% had wrist or hand pain associated with wheelchair use. Nichols *et al*[9] in surveying individuals with SCI for shoulder pain (76% response rate of 708 surveyed) reported that 51.4% experienced shoulder pain and 92% of these individuals cited primary reasons as wheelchair transfers and propulsion. Significant UE pain was evident in 64% (66/103) of the patients with paraplegia and 66% of these patients also had CTS.[9] The shoulder was the most common site of UE joint pain (36%) with 73% of cases involving soft tissue injury (tendonitis, bursitis, capsulitis) and 13% displaying pain referral from the cervical spine.[9]

Sie *et al*[7] in studying the relationship between SCI and shoulder pain, operationally defined 'significant pain' as that (1) requiring analgesic medication, (2) associated with two or more ADL, or (3) intense enough to warrant activity cessation. They reported that 32% of subjects with paraplegia had UE pain at 1-year following SCI and 66% had symptoms of CTS.[7] Curtis *et al*[15,16] designed a 15-item Wheelchair User's Shoulder Pain Index (WUSPI) to assess shoulder pain associated with the functional activities common to wheelchair users including transfers, postural stability, self-care, and wheelchair propulsion, reporting excellent measurement reliability. They advised that multiple factors including shoulder ROM and muscle strength deficiencies or imbalances, subacromial impingement positioning during ADL, overuse, and de-conditioning may also influence WUSPI scores.[16] The WUSPI was considered better able to quantify the influence of shoulder pain on the daily function of wheelchair users than conventional clinical shoulder dysfunction measurement methods.[16] In studying 64 long-term wheelchair users with SCI, Curtis *et al*[15] reported that shoulder pain was most intense during activities requiring high levels of UE strength such as transfers to non-level surfaces, ascending a ramp in a wheelchair, overhead reaching, and washing their backs. In a later comparative study between individuals with tetraplegia or paraplegia, Curtis *et al*[22] reported that less than 15% of subjects reported that they had experienced shoulder pain before becoming wheelchair users. In contrast, 78% of the subjects with tetraplegia and 59% of the subjects with paraplegia reported that their shoulder pain began after initiating wheelchair use.[22] Silfverskiold and Waters[23] reported that 46% of individuals with paraplegia and 60% of individuals with tetraplegia experienced shoulder pain during sleeping hours. Escobedo *et al*[24] used magnetic resonance imaging to study the shoulders of 37 subjects with paraplegia (26 with shoulder pain, 11 asymptomatic), reporting that 73% of the symptomatic shoulders and 59% of the asymptomatic shoulders had evidence of rotator cuff tears. Considering the intimate relationship between pain and joint dysfunction, ameliorating pain early via activity modification, therapeutic modalities, non-steroidal anti-inflammatory medication and gentle stretching should prevent the development and progression of an UE pain-muscle inhibition cycle.

Shoulder joint range of motion and muscle strength deficiencies or imbalances Calmels *et al*[25] reported that subjects with paraplegia had more symmetrical bilateral elbow flexor and extensor muscle strength and mass than non-impaired subjects, suggesting an attenuation of extremity dominance from wheelchair mobility demands. Silfverskiold and Waters[23] reported that unrestricted shoulder function is one of the major factors influencing functional independence and the ability of individuals with SCI to successfully participate in a rehabilitation program. A relatively minor shoulder problem can seriously impair the ability of an individual with a SCI to achieve independence in ADL such as transfers, moving from supine to prone, feeding, hygiene, relieving ischial pressure, dressing and wheelchair propulsion.[23] The amount of time in a sitting posture and UE dependence for functional mobility predisposes the shoulders of many individuals with SCI to developing ROM and muscle strength deficiencies or imbalances. In assessing the shoulder function of individuals with paraplegia, Burnham *et al*[26] reported that subjects who were symptomatic for subacromial impingement also had rotator cuff and glenohumeral joint adductor muscle weakness. Although the WUSPI was designed to measure shoulder pain among wheelchair users, Curtis *et al*[16] reported that total WUSPI scores were inversely related to shoulder ROM, indicating decreased active shoulder ROM as index pain scores increased. Lal[4] reported pre-mature acromioclavicular joint degeneration from the altered mechanical stresses created by acquired shoulder ROM and muscle strength deficiencies or imbalances among individuals with SCI 15 years after onset.

Curtis *et al*[27] reported that 75% of individuals with SCI who performed an exercise protocol designed to alleviate shoulder ROM and muscle strength deficiencies or imbalances reported a 39.9% reduction in WUSPI scores. Although American Wheelchair Paralympians displayed a greater frequency of elbow-arm and forearm-wrist soft tissue injuries than athletes from other disabled sports organizations at the 1996 Summer Games, their shoulder injury frequency did not differ.[28] This further supports the efficacy of prescriptive conditioning programs as proposed by Curtis *et al*[27] for restoring appropriate shoulder ROM and muscle strength balance. Olenik *et al*[29] reported that rowing exercise produced greater scapular retractor muscle activation than backward wheeling and coupled with its efficacy as a cardiovascular exercise recommended its use for restoring normal scapular positioning.

Exercise capacity and the physical strain of daily life Noreau *et al*[30] in assessing the functional capacity (peak oxygen intake, UE strength, power and work) of 123 individuals with SCI (73 with paraplegia, 50 with tetraplegia) reported that functional capability related strongly to peak oxygen intake and UE muscle strength, particularly for individuals with high-level SCI lesions. They concluded that physical fitness directly correlated with functional capability and emphasized the importance of systematic rehabilitation and conditioning exercise programs. Noreau and Shephard[31] compared the physical fitness of employed and unemployed individuals (60 subjects) at least 3 years following SCI onset, reporting that the group that had returned to work weighed less, had a lower body mass index, and had greater aerobic power than the unemployed group. Isokinetic UE endurance

(25 biphasic elbow flexor-extensor contractions at 180°/s) was also greater among the employed group suggesting that endurance testing was more useful than peak strength testing in predicting vocational activity capability.[31]

Janssen *et al*[32] in evaluating 44 males following SCI reported large physical capacity variations during standardized ADL, although subjects with high-level lesions generally displayed greater physical strain (heart rate response). The physical strain experienced by individuals with SCI was inversely proportional to physical capacity (isometric UE strength, peak oxygen uptake, and maximal aerobic power) and these parameters were more accurate predictors of physical strain during ADL task than SCI lesion level.[32] Dallmeijer *et al*[33] in comparing the physical capacity changes of 20 subjects with SCI (nine with tetraplegia, 11 with paraplegia) between time of hospital discharge and an average of 1.2 years later reported that physical capacity (isometric UE strength, power output, peak oxygen uptake) increases coincided with decreased physical strain and improved performance times with ADL such as transfers and ascending a ramp in a wheelchair. Based on what is known regarding non-impaired individuals, during episodes of high physical strain, individuals with SCI who have low tolerance would more likely assume dysfunctional UE postures increasing or otherwise altering shoulder joint mechanical stress. These postures would likely promote UE joint degeneration and gradual loss of function.

Body mass and composition Blackmer and Marshall[34] reported on the detrimental influence of obesity on the functional capacities of two individuals with SCI. Kocina[35] reported that both physically active and sedentary individuals with SCI have greater fat mass than non-impaired subjects, with the sedentary group displaying body fat levels that placed them 'at risk' for cardiovascular disease. Using the Lyndburst Computerized Health Risk Assessment, McColl and Skinner[36] reported that more than 50% of individuals with SCI expressed concerns over increased bodyweight and the associated health risks. Jones *et al*[36] using dual energy X-ray absorptiometry (DEXA) reported 15% and 12% reductions in lean tissue mass and bone mineral content, respectively, and a 47% increase in fat mass for individuals with paraplegia compared to age matched non-impaired subjects. They suggested the need for further research on specific dietary and exercise interventions to maintain or improve lean tissue mass and bone mineral content among individuals with SCI, while reducing body fat accumulation.[37] Proportional UE lean body mass increases which maintain an adequate strength:mass ratio, and reduced body fat levels would improve UE muscular efficiency during transfers, thereby reducing UE degenerative forces from excessive loading and preserving UE function.

Previous upper extremity injury or disease In the individual with SCI a 'minor' UE injury may cause a marked decrease in functional independence, particularly when the shoulder is involved.[6] Based upon increased dependence on the UE for wheelchair transfers and propulsion, individuals with SCI who have a previous history of UE injury or disease generally have a greater disability than the non-spinal cord injured. Although the literature is lacking as to there being a direct relationship between previous UE injury or disease and life expectancy among individuals with SCI, the result may be analogous to the effects of long-term immobility caused by lower extremity injury or disease among the non-spinal cord injured.

Transfer techniques Bayley *et al*[3] reported that 63% (15/23) of patients with paraplegia who were diagnosed with shoulder impingement actually had rotator cuff tears. In studying the glenohumeral intra-articular pressures of individuals with paraplegia who experienced shoulder pain, they reported pressures 2.5 times greater than arterial pressure, particularly during lateral transfers at the mid-transfer position. They postulated that pressure increases during transfers promoted glenohumeral joint degeneration. During wheelchair to bed transfers the weight of the body appears to shift from the trunk through the clavicle and scapula across the subacromial tissues to the humeral head. Although these reaction forces are significantly less than those that normally occur at the hip joint, the glenohumeral joint is less capable of withstanding repetitious compression without degenerative processes occurring.[3] These investigators suggested that high glenohumeral intra-articular pressures in conjunction with increased mechanical stresses across the subacromial region contributed to the high shoulder injury frequency among individuals with SCI.[3]

Kirby *et al*[38] reported that 57.4% of wheelchair users in Nova Scotia reported falling at least once and 66% reported experiencing at least a 'partial tip'. Subjects with paraplegia and lateral transfers were strongly associated with the risk of sustaining a fall.[38] In examining the epidemiology of 2066 non-fatal wheelchair-related accidents (1986–1990) using the National Electronic Injury Surveillance System, Ummat and Kirby[39] reported that elderly women were most likely to sustain an injury, with most accidents related to falls and tips (73.2%), ramps (41.1%), and transfers (16.9%). Spasticity may increase the risk of falls during ADL such as transfers, wheelchair re-seating after a fall, and repositioning of the lower extremities. Little *et al*[40] reported that individuals with incomplete SCI lesions had more pain and more frequent spasticity interfering with ADL than patients with complete lesions. Lower extremity spasticity may increase both UE mechanical stress and physical strain during transfers. Minkel[19] suggested that individuals with SCI who perform a weight relief raise might lean forward or to either side to decrease shoulder stress. Research on the relationships between wheelchair transfer styles, mechanisms of falls or 'near falls', lower extremity spasticity, UE joint degeneration and loss of UE function among individuals with SCI is greatly needed.

Research on UE function during transfer-related tasks
In evaluating 12 asymptomatic patients with low level paraplegia for the electromyographic (EMG) activity of 12 shoulder muscles during depression transfers (using indwelling, fine wire electrodes) Perry *et al*[41] reported three distinct phases; Preparation, Lift and Descent. The Lift phase required the greatest muscular effort by the lead arm, with peak activity of the pectoralis major muscle requiring approximately 81% manual muscle test (MMT) values and moderate action of the serratus anterior, latissimus dorsi, and infraspinatus (37% to 47% MMT). In the trailing arm considerable serratus anterior activity (54% MMT) was evident, while the pectoralis major, infraspinatus, anterior deltoid, and supraspinatus exerted moderate effort (38% to 49% MMT). The Descent phase displayed the least intense muscle activation, consistent with the greater efficiency of eccentric muscular activation. In the trailing arm the sternal portion of pectoralis major (39% MMT) and the lower portion of serratus anterior (34% MMT) were most active. The leading arm displayed moderate and simultaneous pectoralis major (36% MMT) and latissimus dorsi activation (26% MMT). Perry *et al*[41] proposed that the sternal portion of pectoralis major, via its direct attachments at the arm and thorax, helped circumvent glenohumeral joint compression. Using similar methods to evaluate 13 asymptomatic patients with low-level paraplegia during the performance of depression raises, Reyes *et al*[42] reported that it took an average of 2.6 s for a subject to complete the three phases of the maneuver (Load, Lift, and Hold). Trunk elevation and bilateral elbow extension (from 85° to 17° flexion) were the primary movements while the latissimus dorsi (58% MMT), the long head of triceps brachii (54% MMT), and the sternal portion of pectoralis major (32% MMT) were the most active muscles. Considerable latissimus dorsi activity was also noted during the Hold phase (51% MMT) while the triceps and sternal pectoralis major displayed only moderate activation. With the exception of the subscapularis (16% MMT during the Load phase) and the lower serratus anterior (12% MMT during the Lift phase), neither the rotator cuff, deltoid, or scapular muscles exceeded 10% MMT.

Bayley *et al*[3] suggested that patients with low-level paraplegia were able to partially support their bodyweight during transfers using functional abdominal or low back muscles. They considered this capacity vital to relieving the shoulder complex of excessive loading forces. Seelen *et al*[43] and Seelen and Vuurman[17] using center of sitting pressure and surface EMG assessments of trunk muscles reported increased compensatory use of the latissimus dorsi, trapezius, and pectoralis major muscles by individuals with high-level SCI lesions to maintain sitting balance. They reported that increased EMG activation levels signified compensatory postural stability adjustments rather than activity related solely to UE movement.[17,43] Potten *et al*[44] reported increased latissimus dorsi and trapezius EMG activation levels during a bilateral forward reaching task to maintain sitting balance among individuals with SCI.

Wang *et al*[45] in assessing wheelchair transfers to seats of varying height among six non-impaired subjects reported that transfers to lower height seats produced greater ground reaction forces and increased triceps brachii and posterior deltoid EMG activation levels (surface electrodes) to overcome the force of gravity. Transfers to seats positioned higher than wheelchair seat height resulted in a shift of the 'friction force' from a primarily anterior-posterior direction to a more medial-lateral direction, requiring greater biceps brachii EMG activation levels. Wang *et al*[45] reported that equal wheelchair seat and transfer destination heights enabled subjects to perform transfers with considerably less UE muscular effort. Allison and Singer[46] assessed the changes in center of sitting pressure location of a patient with tetraplegia during transfer and reaching tasks with and without a specially designed orthosis, but failed to identify significant differences between conditions. Curtis *et al*[47] using kinematic analysis reported that subjects with paraplegia were more effective in performing a modified functional reach task when they used a stabilization strap.[47] Kamper *et al*[48] used kinematic and center of pressure analysis to compare the lateral postural stability of individuals with SCI and non-impaired subjects during sudden perturbations. They reported that all non-impaired subjects maintained stability with all perturbations, while all individuals with a SCI failed to maintain stability when exposed to higher-level perturbations.[48]

Limitations of previous investigations
Several reports have examined UE kinematics,[49–58] EMG activation levels,[59,60] and kinetics (ground reaction forces, pressures)[45,57,60] among individuals with SCI during wheelchair propulsion. Sitting balance characteristics among individuals with SCI have also been evaluated using kinematic,[46–48] EMG activation level[17,43] and kinetic measurements.[17,46,48] Considerably less research has focused on transfers among any group of subjects, including individuals with SCI.[41,42,45] Increasing our understanding of UE function during wheelchair transfers to any of several destinations (chair, bed, commode, automobile) would increase the likelihood of intervening appropriately to decrease UE injury risk and preserve UE function. Existing studies of transfers among individuals with SCI have relied upon small groups of asymptomatic[41,42] or non-impaired subjects[45] using component UE EMG[41,42,45] or ground reaction force[45] measure-

ments. Additionally, the studies by Perry *et al*[41] and Reyes *et al*[45] relied on insertional fine wire EMG methods which may not have adequately described the total contributions of the larger, superficial trunk and UE muscles as effectively as surface EMG methods. Our review of existing literature revealed no investigation of transfers that integrated each of these data collection modes, or that analyzed joint moments.

Transfer research recommendations

Degenerative changes at the UE joints (particularly at the shoulder) and loss of UE function has a strong negative impact on the functional independence of individuals with SCI. Investigations focusing on transfers are needed to guide intervening clinicians and ergonomic engineers who design and modify assistive and adaptive devices to better serve individuals with SCI. Although effective methods of estimating UE and trunk contributions to transfer performance exist, and are steadily improving, our review of literature found a paucity of clinically relevant findings.

Kinematic descriptions of UE function are generally determined using an anatomically relevant marker system. With a system such as the 'Flock of Birds' electromagnetic tracking system (Ascension Technologies Corp., Burlington, VT, USA, Motion Monitor software version 3.5, Innovative Sports Training, Inc., Chicago, IL, USA) palpation is necessary only once to establish the orientations of anatomically relevant bony landmarks in the local coordinate systems of receivers positioned over the thorax, scapula and humerus.[61] By requiring only a single measurement to obtain accurate joint center description, the variability inherent in repeated bony landmark measurements in multiple arm positions is eliminated. By interfacing kinematic systems such as this with UE ground reaction force and EMG measurements, researchers have the necessary tools to acquire descriptive data of the UE joint forces, moments (Figure 3) and muscular contributions to transfers commonly performed by individuals with SCI between a wheelchair and multiple destinations (chair, bed,

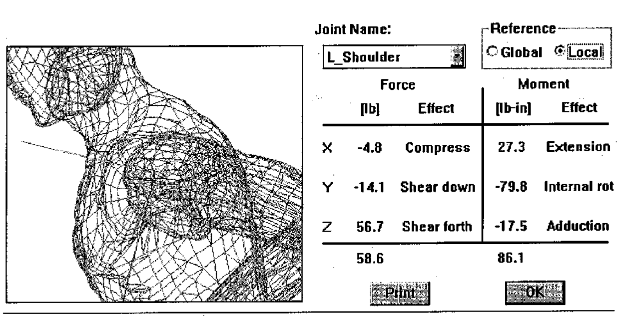

Figure 3 Glenohumeral joint kinetic description. Created using Mannequin Pro 7.0 software, HumanCAD, Melville, NY, USA

commode, automobile). Incorporating systems such as these to measure UE contributions to transfers among both symptomatic and asymptomatic individuals with SCI, with greater consideration for the aforementioned contributing risk factors, will provide more accurate and clinically relevant data for guiding intervention planning.

Biomechanical descriptions of shoulder function have progressed from qualitative 2D kinematic models with restricted motion patterns,[62,63] to models which attempt to estimate composite shoulder muscle forces as functions of 3D arm position and external load in static and quasi-static situations,[64–66] to more elaborate dynamic and 'muscle specific' descriptions of function.[67–70] Several reports have indicated that the glenohumeral joint can be accurately modeled as a ball-and-socket joint with 3° of freedom and with a center of rotation approximating the geometric center of the joint.[64,68,69] Biomechanical research on UE function among individuals with SCI is evolving to the evaluation of contributions of individual UE muscles to net shoulder forces and moments.[70] Due to the complex architecture of the shoulder muscles, Van der Helm and Veenbaas[70] employed 95 elements in their shoulder model to explain the function of 16 muscles. Appreciation of neurophysiological influences such as the physiological cross-sectional area of individual muscles in shoulder models adds further improvement in kinetic estimate accuracy. With developments in the field of magnetic resonance imaging, subject specific shoulder models will be developed, further increasing the accuracy of estimating UE function during the performance of transfer activities.

Conclusions

Upper extremity joint degeneration and loss of function has a tremendously detrimental effect on the functional independence, quality of life, and even the life expectancy of individuals following SCI. Researchers who investigate the transfer methods of individuals with SCI should consider factors including age and length of time from SCI onset, interface between subject-wheelchair, pain, UE ROM and muscle strength deficiencies and imbalances, exercise capacity and tolerance for the physical strain of ADL, body mass and composition, previous UE injury and disease history, and transfer techniques. Investigations of transfer techniques used by individuals with SCI should combine 3D kinematic, kinetic and EMG data to accurately describe UE function. We propose an immediate call for biomechanical studies to evaluate the shoulder joint forces, moments, muscle activation characteristics, and trunk-UE interactions of individuals with SCI during transfers. Biomechanical measurement and computer modeling innovations have provided increasingly accurate tools to acquire the data needed to guide intervention planning directed at preventing UE joint degeneration and preserving function. By identifying stressful sub-components of transfers, both intervening clinicians and engineers who design and modify assistive and adaptive devices will be better able to serve individuals with SCI.

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