Original paper

Rotator cuff coactivation ratios in participants with subacromial impingement syndrome

Joseph B. Myers [a],*, Ji-Hye Hwang [b], Maria R. Pasquale [c],
J. Troy Blackburn [a], Scott M. Lephart [d]

[a] *Department of Exercise and Sport Science, University of North Carolina at Chapel Hill, Chapel Hill, NC, United States*
[b] *Department of Physical Medicine and Rehabilitation, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, South Korea*
[c] *Novel Electronics Inc., St. Paul, MN, United States*
[d] *Department of Sports Medicine and Nutrition, School of Health and Rehabilitation Sciences, University of Pittsburgh,
Pittsburgh, PA, United States*

Abstract

Coactivation of the rotator cuff is vital to glenohumeral joint stability by centralising the humeral head within the glenoid fossa. Yet in individuals with subacromial impingement, it is hypothesised that rotator cuff coactivation abnormalities are present that could contribute to their shoulder pain. The purpose of this study was to determine if abnormal rotator cuff coactivation and deltoid activation patterns exist in participants with subacromial impingement. Rotator cuff (supraspinatus, infraspinatus, and subscapularis) coactivation and middle deltoid activation was assessed during an elevation task. ANOVA models were used to compare muscle activation patterns in 10 participants with subacromial impingement and 10 control participants. Participants with impingement exhibited decreased rotator cuff coactivation (subscapularis–infraspinatus and supraspinatus–infraspinatus) and increased middle deltoid activation at the initiation of elevation (0–30° of humeral elevation). The participants with impingement also had higher subscapularis–infraspinatus and supraspinatus–infraspinatus coactivation above the level of the shoulder where pain is typically present (90–120° of humeral elevation). The results indicate that individuals with subacromial impingement exhibit rotator cuff muscle coactivation and deltoid activation abnormalities during humeral elevation that might contribute to the encroachment of the subacromial structures associated with subacromial impingement.
© 2008 Sports Medicine Australia. Published by Elsevier Ltd. All rights reserved.

Keywords: Shoulder; Impingement; Rotator cuff; Coactivation; Electromyography; Injury

1. Introduction

The primary purpose of the rotator cuff muscles is to create compressive forces through coactivation to stabilise the humeral head within the glenoid fossa [8,17,22]. During humeral elevation, compression by the rotator cuff creates a stable fulcrum for the humeral head on the scapula, allowing the deltoid to elevate the arm without superiorly translating the humeral head on the glenoid [13]. But in patients with subacromial impingement, superior translation during humeral elevation has been identified, contributing to the compression of the subacromial structures [4,6,19]. Potentially, poor coac-tivation by the rotator cuff muscles combined with increased activation by the deltoid could be contributing to this superior humeral migration and ultimately impingement. While activation of each rotator cuff muscle and humeral mover has been assessed in patients with subacromial impingement [14,20], it is the coactivation between the rotator cuff muscles that is most important functionally. To date, coactivation has not been quantified in individuals diagnosed with subacromial impingement. We hypothesise that individuals with subacromial impingement will demonstrate differences in rotator cuff coactivation and deltoid activation compared to individuals with no history of shoulder pain and injury. As such, the purpose of this study is to measure rotator cuff coactivation and middle deltoid muscle activation in participants diagnosed with subacromial impingement syndrome.

* Corresponding author.
E-mail address: joemyers@email.unc.edu (J.B. Myers).

Table 1
Study participant demographics

	Subacromial impingement participants (5 males, 5 females)		Control participants (5 males, 5 females)		*t*-Test [t(d.f.), p]
	Mean	±S.D.	Mean	±S.D.	
Age (years)	42.70	10.61	36.58	7.61	1.6(18), 0.13
Height (cm)	170.27	10.60	176.22	6.83	−1.6(18), .13
Mass (kg)	73.76	13.68	81.14	17.27	−1.8(18), 0.28
VAS pain level	5.00	1.54			

2. Methods

Twenty participants took part in this study including ten individuals diagnosed with subacromial impingement and ten control participants. Participants with subacromial impingement were recruited by providing information to patients with subacromial impingement seen within our orthopedic clinic. Interested participants contacted the investigators. Subacromial impingement was defined as localised pain lasting longer than 2 weeks in duration on the proximal anterolateral shoulder region, positive impingement signs, including positive Neer, Hawkins, and empty can tests, a painful arc of movement (60–120°), and/or tenderness to palpation in the region of the greater tuberosity or rotator cuff tendons. All diagnoses were made by an orthopedic surgeon and confirmed by lidocaine injection in the subacromial space to verify subacromial impingement. Subsequent follow-up with participant records indicated that nine out of the 10 participants with subacromial impingement eventually opted for subacromial decompression surgical intervention. Ten control group participants were matched with the impingement participants were matched according to gender, age, height, weight, and limb (dominance and involvement). All control participants had no self-reported history of shoulder pain or injury that required medical intervention. Control participants were recruited by posted advertisements. All study participant demographics are shown in Table 1.

Each participant attended one laboratory testing session. Before testing, each participant provided consent as required by the Institutional Review Board. Initially, each participant's maximum humeral elevation torque was recorded isometrically on a dynamometer (Biodex System III Isokinetic Dynamometer, Biodex Medical, Shirley, NY) with the participants seated and their limb positioned at 20° elevation in the scapular plane. Maximal elevation torque was used to calculate the load to be held during subsequent functional elevation tasks. The load held equalled 25% of their maximum elevation torque.

Dual finewire electrodes constructed with 0.05 mm nickel chromium alloy wire insulated with nylon (California Fine Wire Company, Grover Beach, CA) were prepared according to published recommendations [2,11] and inserted intramuscularly through a 1.5-in. 25-gauge needle into the subscapularis, supraspinatus, and infraspinatus. Insertion sites were sanitised using 70% isopropyl alcohol and an iodine solution before insertion. Silver–silver chloride surface electrodes (Medicotest Inc., Rolling Meadows, IL) were used to measure middle deltoid muscle activity. Two surface electrodes were placed side-by-side and perpendicular to the orientation of the muscle fibers with 2 cm separating the center of each electrode. Correct positions of all electrodes were confirmed through isolated manual muscle testing. Electromyographic data were collected with the Noraxon Telemyo (Noraxon, Scottsdale, AZ) electromyography system. Electromyographic signals collected were passed through a single ended amplifier (gain, 500) to an eight-channel FM transmitter. A receiver unit collected the telemetry signals from the transmitter, where the receiver amplified (gain, 500) and hardware filtered (range, 15–500 Hz band pass Butterworth filter; common mode rejection ratio of 130 dB) the signals. Signals from the receiver were then converted from analog to digital data at a rate of 1000 Hz. Additionally, all participants were fitted with electromagnetic receivers (MotionMonitor electromagnetic tracking device (Innovative Sports Training, Inc., Chicago, IL)) that were used for calculation of humeral elevation during the elevation trials [16].

Collection trials consisted of each participant elevating his/her arm in the scapular plane (30° anterior to the frontal plane) from 0° elevation (arm at the side) to maximum elevation and returning to 0° elevation. The participants were seated during the elevation tasks. Through the use of a metronome, each elevation–depression task lasted 4 s (2 s to complete humeral elevation and 2 s to complete humeral depression). Participants were provided with a period of trial practice to until they felt comfortable performing the elevation–depression task with the metronome. For data collection, each participant performed 10 continuous elevation–depression repetitions while holding the previously determined resistance (sandbags). Elevation in the scapular plane was maintained through the use of a guide tube.

To calculate rotator cuff and deltoid muscle activation and rotator cuff coactivation at the desired phases of elevation, humeral elevation relative to the thorax was calculated using recommended Euler angle sequence [23]. The phases of interest studied were 0–30°, 30–60°, 60–90°, and 90–120° humeral elevation (relative to the thorax) in the scapular plane.

All raw EMG data was smoothed by root mean square at a time constant of 50 ms. Mean activation of the supraspinatus, infraspinatus, subscapularis, and middle deltoid were

calculated for each phase of interest studied from the middle eight repetitions. All muscle activation data were normalised to mean activation of the entire 10 elevation–depression cycles [24]. Thus each data point for each muscle was divided by the average of the amount of activation during all 10 repetitions. We opted to use this normalisation technique for two reasons including concern for our patient group performing a maximum voluntary contraction given their impingement diagnosis and because it has shown better reliability for normalisation then maximum voluntary contractions [24]. The normalised mean activation of the supraspinatus, infraspinatus, and subscapularis were used to calculate coactivation ratios for each phase established for humeral elevation. All EMG processing was completed through a custom written analysis program within MatLab programming software (Mathworks, Natick, MA).

Coactivation ratios for the rotator cuff force were calculated for the supraspinatus–infraspinatus, supraspinatus–subscapularis, and subscapularis–infraspinatus. We defined coactivation, according to Rudolph et al. [21], as the simultaneous activation of two muscles using the following equation:

$$\frac{EMG_{Low}}{EMG_{High}} \times (EMG_{Low} + EMG_{High})$$

where EMG_{Low} is the mean activation of the less active muscle and EMG_{High} is the mean activation in the more active muscle. Using this equation, high coactivation values signify a high level of activation of both muscles, whereas low coactivation values indicate either low level activation of both muscles or a high level activation of one muscle along with a low level activation of the other muscle in the pair [21].

One within, one between ANOVA models were utilised to analyse each muscle coactivation pair and the middle deltoid activation. The within factor was level of humeral elevation (0–30°; 30–60°; 60–90°; 90–120° of elevation) while the between factor was group (impingement participants vs. control participants). Bonferroni post hoc analyses were utilised when significant findings presented from the ANOVA models, to determine group differences at each elevation angle. An alpha level of 0.05 was set before analyses. Descriptive statistics for the each rotator cuff activation level was calculated to assist with interpretation of the coactivation findings.

3. Results

Significant humeral elevation by group interactions were present for middle deltoid mean activation ($F[3, 54] = 6.29$; $p = 0.005$), supraspinatus–subscapularis coactivation ($F[3, 54] = 10.64$; $p < 0.001$), supraspinatus–infraspinatus coactivation ($F[3, 54] = 5.30$; $p = 0.01$), and subscapularis–infraspinatus coactivation ($F[3, 54] = 15.09$; $p < 0.001$). From the post hoc analyses, it was determined that participants with impingement had less subscapularis–infraspinatus (post hoc $p = 0.012$) and supraspinatus–subscapularis (post hoc $p = 0.018$) coactivation at the initiation of elevation (0–30°). At 30–60° of elevation, less supraspinatus–infraspinatus (post hoc $p = 0.011$) coactivation was shown in the impingement group. The participants with impingement had higher subscapularis–infraspinatus (post hoc $p = 0.033$) and supraspinatus–infraspinatus (post hoc $p = 0.022$) coactivation at 90–120° of humeral elevation. In addition to the coactivation differences shown in the impingement participants, these individuals also had increased middle deltoid activity at 0–30° (post hoc $p = 0.038$). The descriptive statistics for rotator cuff coactivation, rotator cuff activation, and middle deltoid activation and post hoc p-values for rotator cuff coactivation and middle deltoid mean activation are presented in Table 2.

4. Discussion

Humeral elevation results from muscle activation and resulting force generated by the deltoid [9,10]. Yet at the initiation of elevation, the deltoid's lines of action results in a majority of the force from contraction causing superior translation rather than the rotary force needed to elevate the humerus [5]. The use of cadaveric models has shown that the middle portion of the deltoid is most capable of causing superior humeral head migration in this position [7,12]. This superior humeral migration would be problematic in patients with subacromial impingement. In the current study, the results indicate that participants with subacromial impingement have increased middle deltoid activity at the initiation of motion, where the tendency for superior migration is high [7].

To counteract this tendency for superior migration, the rotator cuff must coactivate, creating force couples to centralise the humeral head within the glenoid. Some authors have reported that supraspinatus, infraspinatus, teres minor, and subscapularis play an equally substantial role in maintaining position [3,18] while others suggest that only the subscapularis and infraspinatus play a substantial role [9,17]. In either case, it appears that the force couple function of the supraspinatus, subscapularis, and infraspinatus play a vital role in opposing the superior migration force generated by the deltoid at the initiation of humeral elevation. In the current study, not only did the participants with impingement have increased middle deltoid activation that may contribute to humeral head superior migration, but the coactivation between the subscapularis–infraspinatus, supraspinatus–infraspinatus, and subscapularis–supraspinatus was suppressed as well at the initiation of motion when the tendency for deltoid shearing is highest. These suppressed coactivation ratios most likely result from observed decreases in supraspinatus, infraspinatus, and subscapularis mean activation (from Table 2).

This would suggest that patients with subacromial impingement may not only have the tendency for increased humeral head superior migration attributable to increased

Table 2
Rotator cuff coactivation ratios and muscle activation mean amplitudes for the study participants

	Subacromial impingement participants		Control participants		t-Test [t(d.f.), p]
	Mean	±S.D.	Mean	±S.D.	
Phase 0–30°					
Subscapularis–infraspinatus*	116.73	25.60	143.74	20.55	−3.6(18), 0.012
Supraspinatus–infraspinatus	124.36	36.11	136.36	27.12	−0.9(18), 0.384
Supraspinatus–subscapularis*	107.50	20.99	133.28	26.89	−3.1(18), 0.018
Middle deltoid*	60.32	21.18	44.68	14.43	2.3(18), 0.038
Supraspinatus	64.62	11.27	92.10	15.34	
Infraspinatus	75.57	17.53	82.58	12.29	
Subscapularis	62.41	9.05	91.75	18.22	
Phase 30–60°					
Subscapularis–infraspinatus	162.25	29.39	175.67	25.41	−1.2(18), 0.264
Supraspinatus–infraspinatus*	149.09	17.84	170.25	18.32	−3.1(18), 0.011
Supraspinatus–subscapularis	164.68	13.55	170.99	16.99	−0.9(18), 0.354
Middle deltoid	95.82	21.07	80.61	17.39	1.2(18), 0.229
Supraspinatus	93.20	12.91	103.79	9.84	
Infraspinatus*	92.00	15.09	105.36	13.00	
Subscapularis	101.09	10.26	102.45	19.47	
Phase 60–90°					
Subscapularis–infraspinatus	168.61	16.10	184.30	24.35	−1.9(18), 0.097
Supraspinatus–infraspinatus	223.57	86.47	190.33	20.84	1.3(18), 0.211
Supraspinatus–subscapularis	188.69	23.30	183.99	13.41	0.6(18), 0.560
Middle deltoid	110.42	9.35	121.86	9.10	0.7(18), 0.469
Supraspinatus	133.56	37.44	98.90	8.38	
Infraspinatus	135.40	44.56	110.02	7.94	
Subscapularis	110.20	17.58	102.79	10.63	
Phase 90–120°					
Subscapularis–infraspinatus*	191.28	24.78	160.85	35.36	2.7(18), 0.033
Supraspinatus–infraspinatus*	204.95	35.01	169.22	32.52	2.7(18), 0.022
Supraspinatus–subscapularis	188.92	15.22	178.22	31.02	1.0(18), 0.333
Middle deltoid	132.37	36.00	146.38	30.80	−1.0(18), 0.340
Supraspinatus	126.13	23.77	101.37	14.60	
Infraspinatus	130.63	13.58	101.73	18.66	
Subscapularis	120.19	8.36	104.11	21.66	

* Significant difference between the impingement and control group.

activation of the deltoid, but a compromised ability to oppose this migration because of suppressed rotator cuff coactivation. Therefore, the encroachment of the subacromial structures and resulting pain that occurs during the painful arc may be attributed to these muscle activation alterations that are occurring during the pain free initiation of humeral elevation.

As the participants with impingement continued to elevate their humerus into the region that corresponds with the commonly described painful arc, there were coactivation alterations in shoulder muscle activation similar to those in previous reports [14,20]. Toward the end of the tested elevation (90–120°), the participants with impingement had increased coactivation of both subscapularis–infraspinatus and supraspinatus–infraspinatus. The posterior orientation of the infraspinatus and supraspinatus [3,18] as well as action by the transverse force couple (subscapularis–infraspinatus) [9,17] suggests that combined action by these muscles may be suitable for counteracting any increased anterior humeral translation on the glenoid that may be present in participants with subacromial impingement at 60–120° elevation [15]. In the current study, this increased coactivation in this position may be an attempt to minimise the superior humeral translation that may be present at this position (90–120° elevation) [6,22].

The implications of these results are that patients with subacromial impingement have muscle activation alterations at the initiation of humeral elevation. These abnormalities may facilitate encroachment of the subacromial structures as a patient continues to elevate the arm overhead. The deltoid shearing which could result from the increased middle deltoid activation and poor opposing force-couple action) may be contributing to the pain associated with impingement later in the phase of elevation. This is important information for clinicians when implementing therapeutic treatment regimens. Clinicians may better treat impingement by facilitating the coactivation of the rotator cuff, counteracting any potential humeral shearing by the deltoid. By facilitating coactivation in positions where the alterations are present (0–30° elevation), patients can most likely perform therapeutic exercise in this position, given that patients with impingement rarely have pain at the initiation of motion.

While the findings of this study in combination with the current literature [14,20] clearly show that muscle activation abnormalities are present in participants with subacromial impingement, questions remain as to the source of these abnormalities. It is not known if these muscle activation abnormalities were present before the participants developed shoulder pain, thus contributing to the risk of impingement, or if the activation alterations manifested as a result of the impingement. To answer such questions, a true prospective study (before patients experience shoulder pain for the first time) to identify abnormal muscle activation as a risk factor for injury would have to be initiated. Currently, the literature is void of any such investigation. While a study of this type has not been performed, we believe that the muscle activation alterations present in patients with subacromial impingement result from the impingement syndrome and specifically from the pain manifestation. For example, Bandholm et al. [1] demonstrated that shoulder pain artificially induced with subacromial injection of hypertonic saline, showed potent pain effects on muscle activation. Specifically, activation of all muscles tested increased during humeral elevation, similar to deltoid activity patterns in the current study. Addressing questions related to whether muscle activation alterations result from or contribute to subacromial impingement would be a fertile area for future research.

The authors recognise several limitations in the current study. Throughout this paper, the authors discuss how muscle activation may contribute to the humeral head translation, which is suggested to contribute to impingement and to be present in patients with subacromial impingement [4,6,19]. However, humeral head translation was not measured in the current study. While the electromagnetic tracking instrumentation has the capability to indirectly measure if humeral translation is present [15], measurement of the small amounts of translation that are shown to exist [4,6,19] can be difficult because of the electromagnetic receiver movement at the receiver–skin interface [15]. As such, we speculate that muscle activation alterations may be contributing to abnormal humeral head translations present in patients with impingement [4,6,19]. A second limitation relates to the normalisation procedures used for the electromyography data. Commonly, electromyographic data are normalised to a maximum voluntary contraction to facilitate comparison between subjects. One assumption with this normalisation procedure is that the subjects are able to perform a muscle contraction that represents maximum force development within the muscle. In the current study we were concerned about our patient group performing such a maximum test, given the impingement diagnosis. Thus, we opted for normalising to the mean activation of the entire series of elevation–depression cycles, similar to what is reported as a valid and reliable method for cyclic motions like the task used in the current study and gait [24]. Because of the different means of normalisation, comparison with similar research [20] is difficult. An additional limitation is in the procedures for calculation of coactivation. In the coactivation calculation used in this study, only

two muscle amplitudes could be included at one time (such as subscapularis–supraspinatus, subscapularis–infraspinatus, or supraspinatus–infraspinatus). However, at the shoulder it is suggested that contraction of all of the rotator cuff muscles simultaneously plays a role in maintaining the humeral head within the glenoid rather than just two muscles opposing each other [3,18]. All possible rotator cuff coactivation combinations were measured in the current study, to appreciate the coactivation function of all of the rotator cuff muscles.

The results of the current study indicate that participants with subacromial impingement have coactivation and other muscle activation alterations at the initiation of humeral elevation. It is these alterations that may facilitate encroachment of the subacromial structures as a patient continues to elevate the limb over their head. Muscle activation alterations continued to manifest throughout elevation of the limb.

Practical implications

- Individuals with subacromial impingement exhibited abnormal rotator cuff muscle activation patterns.
- Abnormal muscle activation patterns may be contributing to their impingement syndrome.
- Clinicians should seek to restore normal muscle activation as part of the treatment for impingement syndrome.

Disclosures

No financial report was provided for this project. The authors have no conflicts of interest to disclose.

Acknowledgements

References

[1]. Bandholm T, Rasmussen L, Aagaard P, Diederichsen L, Jensen BR. Effects of experimental muscle pain on shoulder-abduction force steadiness and muscle activity in healthy subjects. *Eur J Appl Physiol* 2008;**102**(6):643–50.

[2]. Basmajian JV, DeLuca CJ. *Muscles alive. Their functions revealed by electromyography*. 5th ed. Baltimore: Williams and Wilkins; 1985.

[3]. Cain PR, Mutschler TA, Fu FH, Lee SK. Anterior instability of the glenohumeral joint: a dynamic model. *Am J Sports Med* 1987;**15**(2):144–8.

[4]. Deutsch A, Altchek DW, Schwartz E, Otis JC, Warren RF. Radiologic measurement of superior displacement of the humeral head in the impingement syndrome. *J Shoulder Elbow Surg* 1996;**5**(3): 186–93.

[5]. Dvir Z, Berme N. The shoulder complex in elevation of the arm: a mechanism approach. *J Biomech* 1978;**1**:219.

[6]. Graichen H, Bonel H, Stammberger T, Haubner M, Rohrer H, Englmeier KH, et al. Three-dimensional analysis of the width of the subacromial space in healthy subjects and patients with impingement syndrome. *AJR Am J Roentgenol* 1999;**172**(4):1081–6.

[7]. Halder AM, Halder CG, Zhao KD, O'Driscoll SW, Morrey BF, An KN. Dynamic inferior stabilizers of the shoulder joint. *Clin Biomech* 2001;**16**(2):138–43.

[8]. Halder AM, O'Driscoll SW, Heers G, Mura N, Zobitz ME, An KN, et al. Biomechanical comparisons of effects of supraspinatus tendon detachment, tendon defects, and muscle retraction. *J Bone Joint Surg* 2002;**84-A**(5):780–5.

[9]. Halder AM, Zhao KD, Odriscoll SW, Morrey BF, An KN. Dynamic contributions to superior shoulder stability. *J Orthop Res* 2001;**19**(2):206–12.

[10]. Howell SM, Imobersteg AM, Seger DH, Marone PJ. Clarification of the role of the supraspinatus muscle in shoulder function. *J Bone Joint Surg* 1986;**68-A**:398–404.

[11]. Kelly BT, Cooper W, Kirkendall DT, Speer KP. Technical considerations for electromyography research on the shoulder. *Clin Orthop* 1997;**335**:140–51.

[12]. Kuechle DK, Newman SR, Itoi E, Morrey BF, An KN. Shoulder muscle moment arms during horizontal flexion and elevation. *J Shoulder Elbow Surg* 1997;**6**(5):429–39.

[13]. Levangie PK, Norkin CC. *Joint structure and function: a comprehensive analysis*. 3rd ed. Philadelphia: F.A. Davis Company; 2001.

[14]. Ludewig PM, Cook TM. Alterations in shoulder kinematics and associated muscle activity in people with symptoms of shoulder impingement. *Phys Ther* 2000;**80**(3):276–91.

[15]. Ludewig PM, Cook TM. Translations of the humerus in persons with shoulder impingement symptoms. *J Orthop Sports Phys Ther* 2002;**32**(6):248–59.

[16]. Myers JB, Laudner KG, Pasquale MR, Bradley JP, Lephart SM. Scapular position and orientation in throwing athletes. *Am J Sports Med* 2005;**33**(2):263–71.

[17]. Parsons IM, Apreleva M, Fu FH, Woo SLY. The effect of rotator cuff tears on reaction forces at the glenohumeral joint. *J Orthop Res* 2002;**20**:439–46.

[18]. Payne LZ, Deng XH, Craig EV, Torzilli PA, Warren RF. The combined dynamic and static contributions to subacromial impingement. A biomechanical analysis. *Am J Sports Med* 1997;**25**(6):801–8.

[19]. Poppen NK, Walker PS. Normal and abnormal motion of the shoulder. *J Bone Joint Surg* 1976;**58A**:195–201.

[20]. Reddy AS, Mohr KJ, Pink MM, Jobe FW. Electromyographic analysis of the deltoid and rotator cuff muscles in persons with subacromial impingement. *J Shoulder Elbow Surg* 2000;**9**(6):519–23.

[21]. Rudolph KS, Axe MJ, Snyder-Mackler L. Dynamic stability after ACL injury: who can hop? *Knee Surg Sports Traumatol Arthrosc* 2000;**8**(5):262–9.

[22]. Sharkey NA, Marder RA. The rotator cuff opposes superior translation of the humeral head. *Am J Sports Med* 1995;**23**(3):270–5.

[23]. Wu G, van der Helm FC, Veeger HE, Makhsous M, Van Roy P, Anglin C, et al. ISB recommendation on definitions of joint coordinate systems of various joints for the reporting of human joint motion. Part II. Shoulder, elbow, wrist and hand. *J Biomech* 2005;**38**(5):981–92.

[24]. Yang JF, Winter DA. Electromyographic amplitude normalization methods: improving their sensitivity as diagnostic tools in gait analysis. *Arch Phys Med Rehabil* 1984;**65**:517–21.