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Authors: Thomas Frydendal, Henrik Eshøj, Behnam Liaghat, Pascal Edouard, Karen Sjøgaard, Birgit Juul-Kristensen



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**Sensorimotor control and neuromuscular activity of the shoulder in adolescent competitive swimmers with generalized joint hypermobility**

Thomas Frydendal<sup>a,b,\*</sup>, Henrik Eshøj<sup>c</sup>, Behnam Liaghat<sup>a,d</sup>, Pascal Edouard<sup>e,f</sup>, Karen Søgaard<sup>a</sup>, Birgit Juul-Kristensen<sup>a</sup>

<sup>a</sup> Department of Sports Science and Clinical Biomechanics, University of Southern Denmark, Odense, Denmark, <sup>b</sup> Department of Physiotherapy, Lillebaelt Hospital, Vejle Hospital, Vejle, Denmark, <sup>c</sup> Quality of Life Research Center, Department of Haematology, Odense University Hospital, Odense, Denmark, <sup>d</sup> Department for Health, University of Bath, Bath, United Kingdom, <sup>e</sup> Department of Clinical and Exercise Physiology, Sports Medicine Unit, University Hospital of Saint-Etienne, Faculty of Medicine, Saint-Etienne, France, <sup>f</sup> Inter-university Laboratory of Human Movement Science (LIBM EA 7424), University of Lyon, University Jean Monnet, F-42023, Saint Etienne, France

\* **Corresponding author:** Thomas Frydendal, Department of Physiotherapy, Hospital Lillebaelt, Vejle Hospital, Beriderbakken 4, DK-7100 Vejle, Denmark. Tel.: +45 79406159. Email: thomas.frydendal@rsyd.dk

**Co-authors:** Henrik Eshøj, Quality of Life Research Center, Department of Haematology, Odense University Hospital, Sdr. Boulevard 29, DK-5000 Odense C, Denmark, email: heshoj@health.sdu.dk; Behnam Liaghat, Department of Sports Science and Clinical Biomechanics, University of Southern Denmark, Campusvej 55, DK-5230 Odense M, Denmark, email: ir.liaghatbehnam@bath.edu; Pascal Edouard, Department of Clinical and Exercise Physiology, Sports Medicine Unit, Bellevue Hospital, University Hospital of Saint-Etienne, F-42055 Saint-Etienne Cedex 2, France email: pascal.edouard42@gmail.com; Karen Søgaard, Department of Sports Science and Clinical

Biomechanics, University of Southern Denmark, Campusvej 55, DK-5230 Odense M, Denmark, email: ksogaard@health.sdu.dk; Birgit Juul-Kristensen, Department of Sports Science and Clinical Biomechanics, University of Southern Denmark, Campusvej 55, DK-5230 Odense M, Denmark, email: bjuul-kristensen@health.sdu.dk

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## Highlights

- Swimmers with hypermobility showed no shoulder sensorimotor control deficits.
- Pectoralis major activity in swimmers with hypermobility was altered.
- Long-term effect of altered pectoralis major activity in hypermobility is unknown.

## Abstract

Introduction: Shoulder pain is highly prevalent in competitive swimmers, and generalized joint hypermobility (GJH) is considered a risk factor. Sensorimotor control deficiencies and altered neuromuscular activation of the shoulder may represent underlying factors.

Research question: To investigate whether competitive swimmers with GJH including shoulder hypermobility (GJHS) differ in shoulder sensorimotor control and muscle activity from those without GJH and no shoulder hypermobility (NGJH).

Methods: Competitive swimmers (aged 13-17) were recruited. GJHS or NGJH status was determined using the Beighton score (0-9) and Rotès-Quérol test (positive/negative).

Inclusion criteria for GJHS were a Beighton score  $\geq 5$  and minimum one hypermobile shoulder, while NGJH was defined as a Beighton score  $\leq 3$  and no shoulder hypermobility.

Three prone lying, upper-extremity weight-bearing shoulder stabilometric tests were

performed on a force platform: Bilateral upper-extremity support eyes open (BL-EO) and eyes closed (BL-EC) and unilateral upper-extremity support eyes open (UL-EO). Surface electromyography (SEMG) was measured from the upper trapezius, lower trapezius, serratus anterior, infraspinatus and pectoralis major muscles. SEMG was normalized using maximal voluntary isometric contractions and presented relative to maximal voluntary SEMG (%MVE). Co-contraction index (CCI) was calculated for the following muscle pairs: upper trapezius-lower trapezius, upper trapezius-serratus anterior, and infraspinatus-pectoralis major. Between-group differences in stabilometric parameters, %MVE, and CCI were analyzed with a mixed effects model.

Results: Thirty-eight swimmers were enrolled as GJHS (n=19) or NGJH (n=19). There were no group differences in stabilometric parameters or CCI. GJHS displayed significantly decreased (29%) pectoralis major activity during BL-EO compared to NGJH ( $5.35 \pm 1.77\% \text{MVE}$  vs.  $7.51 \pm 1.96\% \text{MVE}$ ;  $p=0.043$ ).

Significance: Adolescent competitive swimmers with GJHS displayed no shoulder sensorimotor control deficiencies and no generally altered shoulder muscle activity pattern, except for decreased pectoralis major activity in BL-EO. Longitudinal studies are needed to investigate whether decreased pectoralis major activation contributes to the development of shoulder pain in swimmers with GJHS.

**Keywords:** Hypermobility, Competitive swimmers, Shoulder, Sensorimotor control, Neuromuscular control, Electromyography

## 1. Introduction

Shoulder pain is the most frequent disorder in competitive swimmers with an estimated point prevalence of 40-91% [1]. Multifactorial risk factors have been suggested in the development

of shoulder pain in swimmers including extrinsic (competitive level, swimming experience, training distance/volume/intensity, and swimming training equipment) and intrinsic factors (glenohumeral instability, abnormal range of motion [ROM], strength imbalance of the rotator cuff muscles, and scapular dyskinesis) [2, 3]. Glenohumeral instability (traumatic) and shoulder pain have been associated with generalized joint hypermobility (GJH) [4-6]. GJH is an inherent condition characterized with increased ROM in multiple joints due to excessive laxity of the connective tissue such as joint capsules and ligaments [7], with a prevalence of 32% in adolescent competitive swimmers [8]. Increased glenohumeral ROM, possibly present in swimmers with GJH allow a body position that reduces drag and provides greater stroke length, positively correlated with swimming performance [1]. In contrast, it may also predispose to muscle-tendon overload and muscle fatigue resulting in shoulder pain [1].

Shoulder stability is ensured by an advanced coordination and interaction between the static (joint capsules and ligaments) and dynamic (scapular and rotator cuff muscles) stabilizers, facilitated by the sensorimotor system [9]. Any dysfunction or injury to the stabilizing structures of the shoulder may inhibit sensory or proprioceptive information (afferent), neuromuscular responses (efferent), and central integration resulting in sensorimotor control deficiencies [9]. Sensorimotor control of the lower-extremities is frequently assessed using laboratory force platforms (FP) during standing positions [10, 11], incorporating neuromuscular adjustments represented as displacements in center of pressure path length (COPL) [12]. The method has been adapted for assessment of shoulder sensorimotor control and was found reliable during prone lying with the upper-extremities positioned at a FP and the lower-extremities supported on a treatment table [13]. Using this set-setup, shoulder sensorimotor control deficiencies was demonstrated in subjects with unilateral post-traumatic recurrent glenohumeral instability in the dominant side compared with healthy controls [14]. Furthermore, studies indicate sensorimotor control deficiencies and altered muscle activity of

the lower-extremities in subjects with GJH [11, 15, 16]. In addition, altered shoulder muscle activity has been shown in competitive swimmers with shoulder pain [3]. To our knowledge no studies have investigated shoulder sensorimotor control and muscle activity in swimmers with GJH including shoulder hypermobility (GJHS), which may represent underlying factors predisposing to shoulder pain.

Thus, the aim of this study was to investigate whether adolescent competitive swimmers with GJHS differ in shoulder sensorimotor control and muscle activity from those without GJH and no shoulder hypermobility (NGJH). The hypothesis was that GJHS swimmers would exhibit shoulder sensorimotor deficits (increased COPL displacements) and altered muscle activity (increased or decreased muscle activity) compared to NGJH.

## **2. Methods**

### **2.1 Subjects**

Eligible subjects, competitive swimmers (aged 13-17) were recruited prospectively and consecutively from five swimming clubs from October 2015 to January 2016. GJH status was determined using the Beighton score comprising apposition of the thumbs, dorsiflexion of the fifth fingers  $>90^\circ$ ; hyperextension of the elbows and knees  $>10^\circ$  and forward bending in standing. Each positive test equals one point (score range 0 to 9). Since the shoulder is not included in the Beighton score, a shoulder hypermobility test (Rotès-Quérol) was added. Rotès-Quérol involves an actively guided shoulder lateral rotation (positive score  $>90^\circ$ ) with the upper arm along the body, elbow in  $90^\circ$  flexion, and forearm in neutral [17]. The principal author performed the clinical screening in the swimming clubs, according to a standardized protocol with moderate to substantial reproducibility [17], approximately two months prior to the laboratory test session.

Inclusion criteria for GJHS: Beighton score  $\geq 5$  [16], and minimum one hypermobile shoulder. Inclusion criteria for NGJH: Beighton score  $\leq 3$  and no shoulder hypermobility. General exclusion criteria were: absence from swimming (training/competition), neurological disorders, history of surgery to the trunk, wrists, elbows and shoulders, previous serious trauma to the upper-extremities and lumbar spine, current pain in the upper-extremities or trunk  $>50$  mm on a Visual Analogue Scale (VAS), diagnosis of Marfan Syndrome, Ehlers-Danlos Syndrome and Osteogenesis Imperfecta. Included subjects were matched by age and sex on a ratio of 1:1.

The Regional Committees on Health Research Ethics for Southern Denmark determined that no ethics approval was required for this study, since the intervention was considered non-invasive. Oral and written informed consent was obtained prior to the test session from each subject and their parents or guardian, according to the Helsinki Declaration.

## **2.2 Experimental procedure**

The total duration of procedures lasted approximately 90 minutes. Firstly, anthropometric and demographic data (self-reported sports participation, pain rating [VAS], and The Western Ontario Shoulder Instability Index [WOSI]) were obtained. WOSI covers four domains (physical symptoms, sport/recreation/work, lifestyle, and emotions) comprising 21 items [18]. Each item is scored on a 0-100 point VAS-scale (score range 0 [best] to 2100 [worst]) [18]. WOSI was found to be reliable and valid in the Danish population [19]. Secondly, surface electromyography (SEMG) electrodes were placed onto five shoulder muscles. To normalize SEMG signals, subjects performed 10-minutes of standardized warm-up prior to completing maximum voluntary isometric contractions (MVIC) for each muscle. Thirdly, three prone lying, shoulder stabilometric tests were performed on a FP with concurrent SEMG recordings. Finally, three clinical tests for glenohumeral instability were performed. Except for the principal author, all examiners were blinded for group status.



### 2.3 Electromyography

Initially, hair removal, light abrasion and disinfection were performed to ensure an inter-electrode impedance of  $<10\text{ k}\Omega$ . Bipolar silver/silver chloride (Ag/AgCl) surface electrodes (Ambu Blue Sensor, N-00-S/25, Ballerup, Denmark) with a 10 mm conducting area were placed in line with the muscle fiber direction using an inter-electrode distance of two cm on the upper trapezius, lower trapezius, serratus anterior, infraspinatus and pectoralis major muscles, according to standardized locations (Table 1) [20-23]. SEMG was registered from the dominant shoulder in GJH with bilateral shoulder hypermobility (n=15), and the hypermobile shoulder in GJH with unilateral shoulder hypermobility (n=3 dominant and n=1 non-dominant shoulder). For NGJH the tested shoulder was equivalent to their respective match. The dominant side was the side used for handwriting [14]. An examiner blinded for group status conducted SEMG procedures.

Then subjects performed bilateral MVICs in five different positions in a fixed order to determine maximum voluntary muscle activity (MVE) of each muscle (Table 1). Resistance was applied with an external load of 40kg, and isometric manual resistance was applied to the non-measured arm to obtain symmetry during testing. In prone and supine lying MVIC positions subjects were secured with straps around the thighs (10 cm proximal to the knee) and the lower back/abdomen (cranially to the pelvis). Three trials of five sec duration were performed with verbal encouragement, separated by one minute of rest.

SEMG signals were preamplified (gain 500) and band-pass filtered with cut-off frequencies at 10-500 Hz using a wireless 16-channel Noraxon Telemetry DTS system (Noraxon, Scottsdale, USA). Signals were A/D converted (16-bit, Cambridge Electronic Design Limited, UK) and recorded via a laboratory interface (Spike2 software) with a sampling frequency of 1000 Hz.

## 2.4 Stabilometric measurements

Shoulder stabilometric tests were recorded using an AMTI-FP (OR6-7-1000, Advanced technologies, MA, USA) with an amplifier (MiniAmp MSA-6) in an undisturbed environment with no deviation in luminosity [24]. The ground reaction forces were A/D converted (16-bit, National Instruments, TX, USA), low pass filtered (Butterworth 4<sup>th</sup> order) with a cut-off value at 10 Hz and recorded with a sampling frequency of 125 Hz.

Subjects were placed in a prone lying, upper-extremity weight-bearing position, with the lower-extremities resting on a treatment table from the anterior superior iliac spines and down. Shoulders and wrists were positioned in 90° of flexion and extension, respectively, with the scapulothoracic joint in neutral position and the head in neutral aligned with the trunk axis [14]. Three test conditions were used: Bilateral upper-extremity support with eyes open (BL-EO) and eyes closed (BL-EC), and unilateral upper-extremity support with eyes open (UL-EO) (Figure 1) [13, 14]. Subjects were instructed to remain steady during testing [14]. In BL-EO and BL-EC the hands were pressed together at the center of the FP with the thumbs positioned behind the x-axis and the y-axis in between. In UL-EO the testing hand was placed with the third finger along the y-axis at the center of the FP and the thumb behind the x-axis, with the contralateral hand pressed against the abdomen [14].

In total, seven trials of 30 sec duration with 30 sec rest in between were conducted in a fixed order, corresponding to one trial for BL-EO followed by three trials for BL-EC and UL-EO, respectively [11, 14]. Trial progression of time was verbally announced after 10 sec, 20 sec and at the end of each trial. In case of failure during testing (e.g. movement of the lower-extremities, taking support with the non-supported hand, rotation of the head, speaking) a new trial was permitted, with maximum three unsuccessful attempts [25].

## 2.5 Clinical tests

Three clinical glenohumeral instability tests, including Gagey, Sulcus sign, and Load-and-shift were performed. The Gagey test, assessing the inferior glenohumeral ligament, was considered positive with shoulder abduction  $>105^\circ$ . The Sulcus sign, testing for inferior glenohumeral instability, was rated positive with  $>2$  cm widening between the acromion and humeral head. The Load-and-shift, evaluating anterior and posterior glenohumeral instability on a four-point scale ranging from 0 (little to almost no movement) to 3 (humeral head moves beyond the glenoid labrum and remains dislocated), was considered positive with a score  $\geq 2$  [26].

## 2.6 Data processing

MVE for each muscle was defined as the highest SEMG activity ( $\mu\text{V}$ ) measured during a MVIC calculated as the mean root-mean-square (RMS) in a one sec moving time-interval, incremented in 100 ms steps [11]. SEMG activity during shoulder stabilometric tests was calculated as the mean RMS values using a one sec moving time-interval, incremented in one sec steps, and each muscle was normalized and presented relative to MVE (%MVE) [10]. SEMG sampling was synchronized with the stabilometric measurements with a manual trigger marking the trial start and end [10]. Co-contraction index (CCI) was calculated according to the formula:  $\text{CCI} = (\%MVE_{\text{min}}/\%MVE_{\text{max}}) \times (\%MVE_{\text{min}} + \%MVE_{\text{max}})$  [27], for the following muscle pairs of upper trapezius-lower trapezius, upper trapezius-serratus anterior, and infraspinatus-pectoralis major. CCI provides an estimate of the relative simultaneous neuromuscular activation of two muscles, as well as the magnitude of the co-contraction [27].

Stabilometric data were analyzed with custom-made software, and values for COPL (mm), COPL velocity ( $\text{mm s}^{-1}$ ), and body weight-load (kg) for each test were calculated. COPL and

COPL velocity reflect the net neuromuscular activity essential to obtain stability with low values indicating greater stability, while high values signify less stability [14].

## 2.7 Statistical analysis

Data were tested for normality with Shapiro-Wilks, histograms, and QQ-plots and %MVE (except infraspinatus activity during UL-EO), CCI, VAS, and WOSI were not normally distributed. %MVE and CCI were log-transformed. To test for between-group differences in demographic variables and self-reported outcomes an un-paired t-test was used for continuous data, Mann-Whitney for continuous data not normally distributed, and Fisher's exact test for dichotomous data. A paired t-test was used to test for within-group differences in stabilometric outcomes, to evaluate the assumption of BL-EO being a reference test. Between-group differences for COPL, COPL velocity, %MVE, and CCI were analyzed with a mixed effects model, with stabilometric variables, %MVE or CCI as dependent variables (individually), and group (GJHS/NGJH), trial, sex, age, weight and height as fixed factors, and subject as random factor.

Sample size was calculated using previous stabilometric values [14], resulting in a group size of 19. The minimum mean difference was estimated to be 30% difference in COPL with a standard deviation (SD) of 67.8 mm,  $\alpha=0.05$ , and  $\beta=0.20$ .

Means and SD were calculated for each continuous variable. P-values  $<0.05$  were reported as statistically significant. All statistical analyses and calculations were performed using STATA 14.2 (Statacorp, College Station, Texas, USA).

## 3. Results

In total, 97 subjects were screened for eligibility with 38 enrolled as either GJHS or NGJH with 11 females and eight males in each group. The groups were comparable on demographics (sex, age, height, weight, BMI, sports participation, clinical tests for

glenohumeral instability, and self-reported pain [VAS] and shoulder instability [WOSI]), except for Beighton scores ( $p<0.001$ ) and number of positive Rotès-Quérol tests ( $p<0.001$ ), which was in consistency with the inclusion criteria (Table 2).

### **3.1 Sensorimotor control**

There were no group differences in stabilometric parameters. COPL and COPL velocity were significantly lower in BL-EO compared with BL-EC and UL-EO in both groups ( $p<0.001$ ) (Table 3).

### **3.2 Neuromuscular activity**

For the majority of shoulder muscles there were no group differences in muscle activity, except for significantly decreased (29%) pectoralis major activity during BL-EO in GJHS compared to NGJH ( $5.35\pm 1.77\%$  MVE vs.  $7.51\pm 1.96\%$  MVE;  $p=0.043$ ) (Figure 2). There were no between-group differences in CCI (Figure 3).

## **4. Discussion**

Adolescent competitive swimmers with GJHS showed equivalent shoulder sensorimotor control during each shoulder stabilometric test, but with significantly decreased pectoralis major activity during BL-EO compared to NGJH.

### **4.1 Sensorimotor control**

There were no group differences during shoulder stabilometric tests, indicating no shoulder sensorimotor control deficiencies in swimmers with GJHS compared to NGJH. These findings are in contrast to a previous study using the same shoulder stabilometric set-up, displaying shoulder sensorimotor control deficiencies in subjects with post-traumatic recurrent anterior glenohumeral instability in the dominant side compared to healthy controls [14]. Several reasons may explain this. Firstly, it may be due to different mechanisms resulting in increased glenohumeral laxity (GJHS vs. traumatic injury). Studies indicate traumatic shoulder instability to the capsuloligamentous structures (static stabilizers) may lead

to decreased mechanoreceptor stimulation resulting in proprioceptive and sensorimotor control deficits [9], while findings for GJH regarding the shoulder are inconsistent [28, 29]. Secondly, group demographics were different between the studies, including the current subjects performing competitive swimming, presenting with fewer males (42% vs. 88%), lower mean age (14.8 years vs. 29.5 years), and lower weight (64.3kg vs. 74.2kg). Thirdly, the current stabilometric test protocol comprised three test positions with the hands orientated  $0^\circ$  to the sagittal plane, repeated three times (except BL-EO) in a fixed order. This was in contrast to the previous study [14] using four different test positions with hands orientated  $15^\circ$  laterally to the sagittal plane, performed once and in random order, resulting in larger variation.

Adolescent girls with GJH and knee hypermobility have shown decreased sensorimotor control during bilateral standing with eyes open and eyes closed [11], which is also in contrast to the current stabilometric results of the shoulder. A direct comparison between the studies is limited due to sensorimotor control assessments of different joints. Nevertheless, the different results may potentially be explained by demographic differences with the current subjects performing competitive sports, which may indicate regular training being a protecting factor for shoulder sensorimotor control deficits. Furthermore, trial duration of the stabilometric tests in the current study was 30 sec compared with 60 sec used in the previous study [11], indicating that shorter trial duration may not be challenging enough for detecting group differences.

#### **4.2 Neuromuscular activity**

Swimmers with GJHS exhibited no generally altered shoulder muscle activity pattern, except for decreased pectoralis major activity during BL-EO displayed as the less challenging, indicating altered muscle activation in the medial rotation agonist. Previous studies of GJH including knee hypermobility have found generally altered knee muscle activation strategies

during isometric strength, dynamic jump, and standing balance tests compared to controls, [11, 15, 16], which suggests the current shoulder stabilometric tests may not be appropriate for investigating differences in shoulder muscle activity. Moreover, the lack of group differences in muscle activity may be due to group size was calculated using stabilometric data (COPL), in addition to large inter-individual in SEMG measurements, increasing the risk of a type II error.

In line with current results, decreased pectoralis major activity has been shown in subjects with multidirectional shoulder instability [20, 30], which may influence glenohumeral kinematics and stability in swimmers with GJHS. Furthermore, competitive swimmers with shoulder pain display alterations in shoulder muscle activity [3]. However, whether decreased pectoralis major activity contributes to the development of shoulder pain in swimmers with GJHS should be investigated in longitudinal studies.

In contrast to previous studies [11, 15, 16], there were no group differences in CCI. This may be explained by the current stabilometric tests being less challenging compared to previous tests [11, 15, 16]. Also, the current swimmers may be a well-trained group with the ability to compensate for decreased pectoralis major activity as shown in GJHS during BL-EO.

#### **4.3 Limitations and strengths**

One of the limitations is the current shoulder stabilometric tests represent assessment of sensorimotor control during static closed-chain kinetic positions in a specific part of ROM. Thus, the tests do not specifically represent the functional demand of a swimming stroke. However, static sensorimotor control is anticipated to be a prerequisite for dynamic control. Furthermore, the shoulder stabilometric analysis was confined to COPL and COPL velocity, which were the only variables displaying satisfactory reliability in a previous study [13]. Consequently, whether group differences are present in other stabilometric variables (e.g.

COP area, anterior-posterior and medial-lateral displacements, rambling and trembling) is unknown.

Another limitation is AMTI-FP generates levels of COPL values, specifically when weight loads are below 20kg as in the present UL-EO test (Table 3), referred to as background noise [25]. However, whether COPL displacements from subjects are contained in or added to the produced background noise is unknown [25]. Nonetheless, weight-load was equal in both groups in all tests and thus unlikely to have biased the stabilometric data.

The strengths of the current study were the standardized, reliable, and valid procedures utilized for screening subjects for eligibility, objective shoulder stabilometric and SEMG measurements performed in a noise-free environment. Further, the same examiner blinded for group status conducted SEMG procedures, reducing inter-tester variability. Lastly, fixed external load (40kg) were used for normalizing SEMG data to %MVE.

## 5. Conclusion

In conclusion, adolescent competitive swimmers with GJHS displayed no shoulder sensorimotor control deficiencies and no generally altered shoulder muscle activity pattern, except for decreased pectoralis major activity in BL-EO. Longitudinal studies are needed to investigate whether decreased pectoralis major activation contributes to the development of shoulder pain in swimmers with GJHS.

### **Authors' contribution:**

TF, HE, BL, PE, KS and BJK contributed to the design of the study. TF and BL collected the data. TF and BL performed the data management. TF performed the data analysis and TF and BJK were in charge of data interpretation. TF and BJK wrote the manuscript. All authors participated in data interpretation and contributed to manuscript revision. All authors read and approved the final version.



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## **Conflict of interest**

The authors have no financial or personal conflict of interest in relation to submission of this paper.

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## Legend to figures

**Figure 1** The shoulder stabilometric tests and corresponding hand placements. (A) Bilateral upper-extremity support with eyes open (BL-EO). (B) Bilateral upper-extremity support with eyes closed (BL-EC). (C) Unilateral upper-extremity support with eyes open (UL-EO). (D) Hand placements for BL-EO and BL-EC. (E) Hand placement for UL-EO.

**Figure 2** Electromyography measurements (%MVE) of five shoulder muscles during shoulder stabilometric tests for swimmers with generalized joint hypermobility including shoulder hypermobility (GJHS) and swimmers without GJH and no shoulder hypermobility (NGJH) (A) Bilateral upper-extremity support with eyes open (BL-EO). (B) Bilateral upper-extremity support with eyes closed (BL-EC). (C) Unilateral upper-extremity support with eyes open (UL-EO). Values are mean (standard deviation). \*=Significant difference.

**Figure 3** Co-contraction index (CCI) of the upper trapezius-lower trapezius, upper trapezius-serratus anterior, and infraspinatus-pectoralis major during shoulder stabilometric tests for swimmers with generalized joint hypermobility including shoulder hypermobility (GJHS) and swimmers without GJH and no shoulder hypermobility (NGJH). (A) Bilateral upper-extremity support with eyes open (BL-EO). (B) Bilateral upper-extremity support with eyes closed (BL-EC). (C) Unilateral upper-extremity support with eyes open (UL-EO). Values are mean (standard deviation). \*=Significant difference.

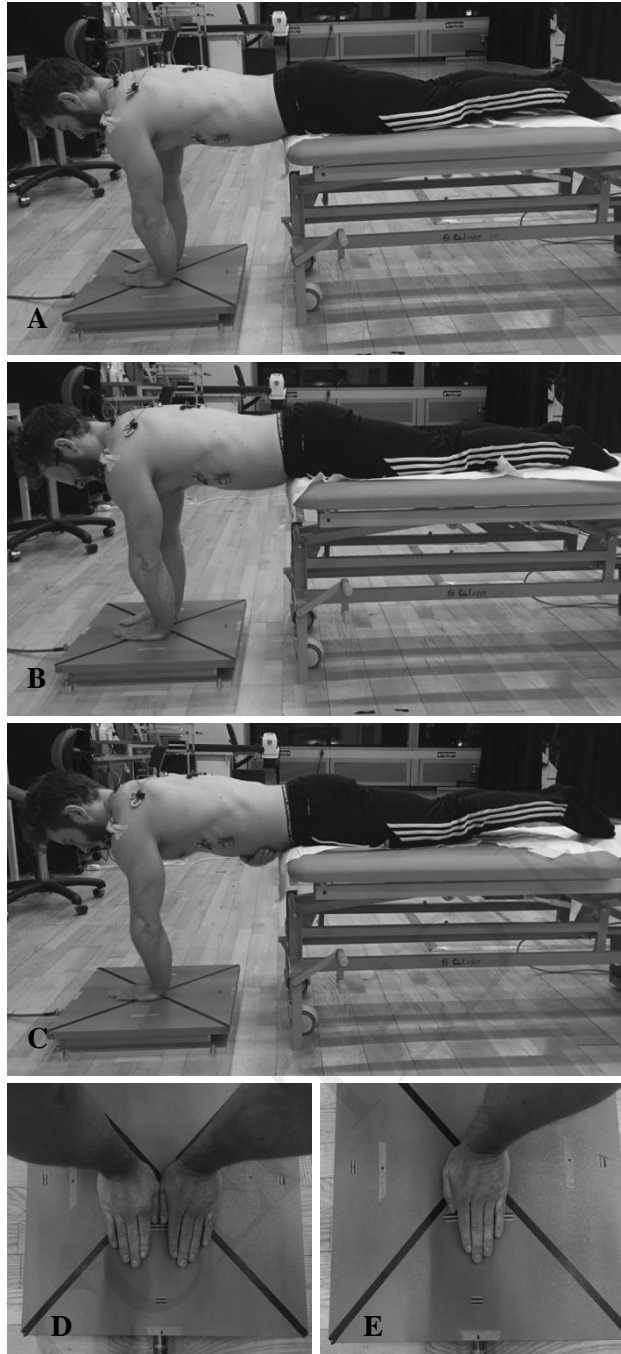
**Figure 1**

Figure 2

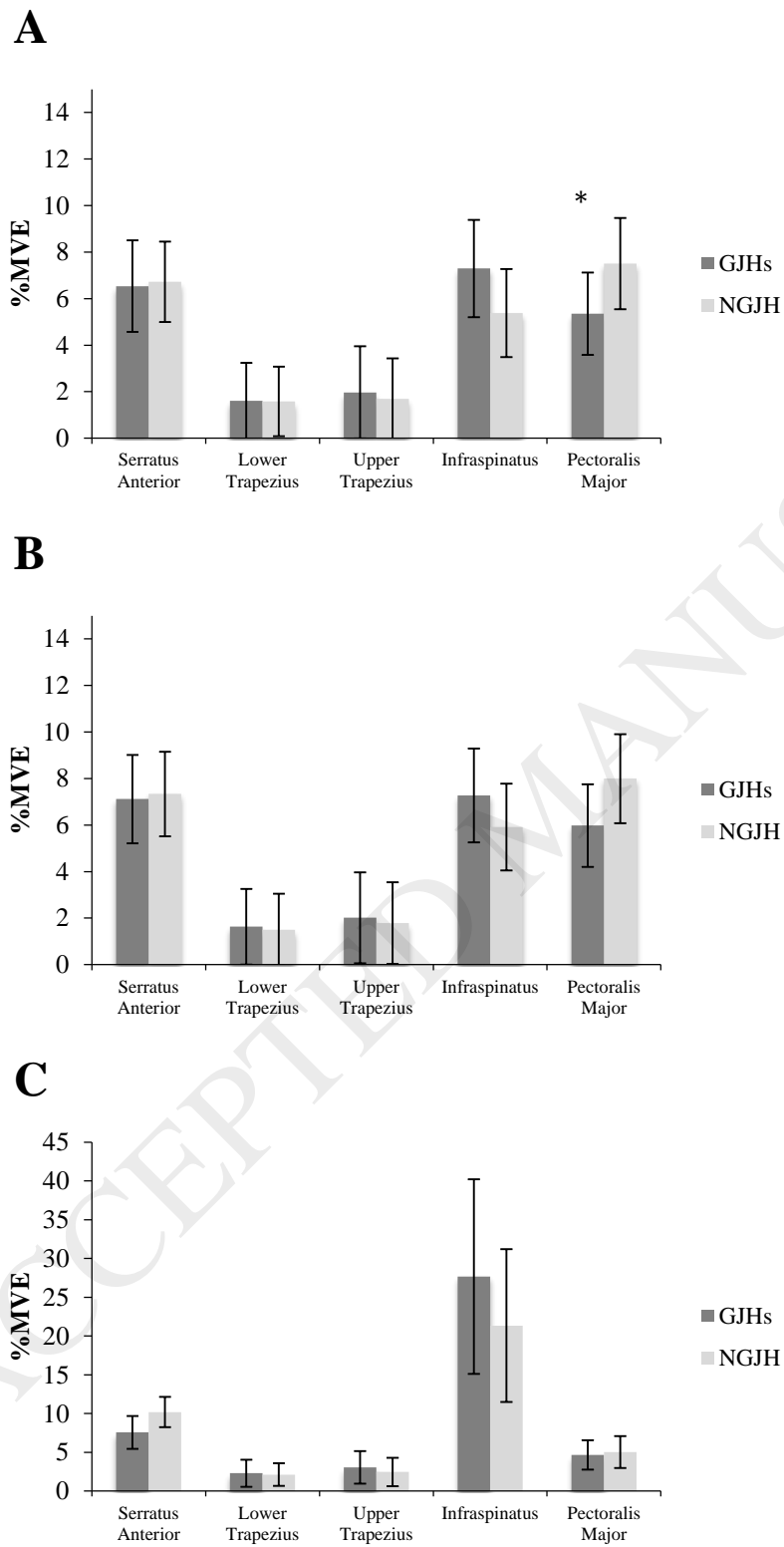
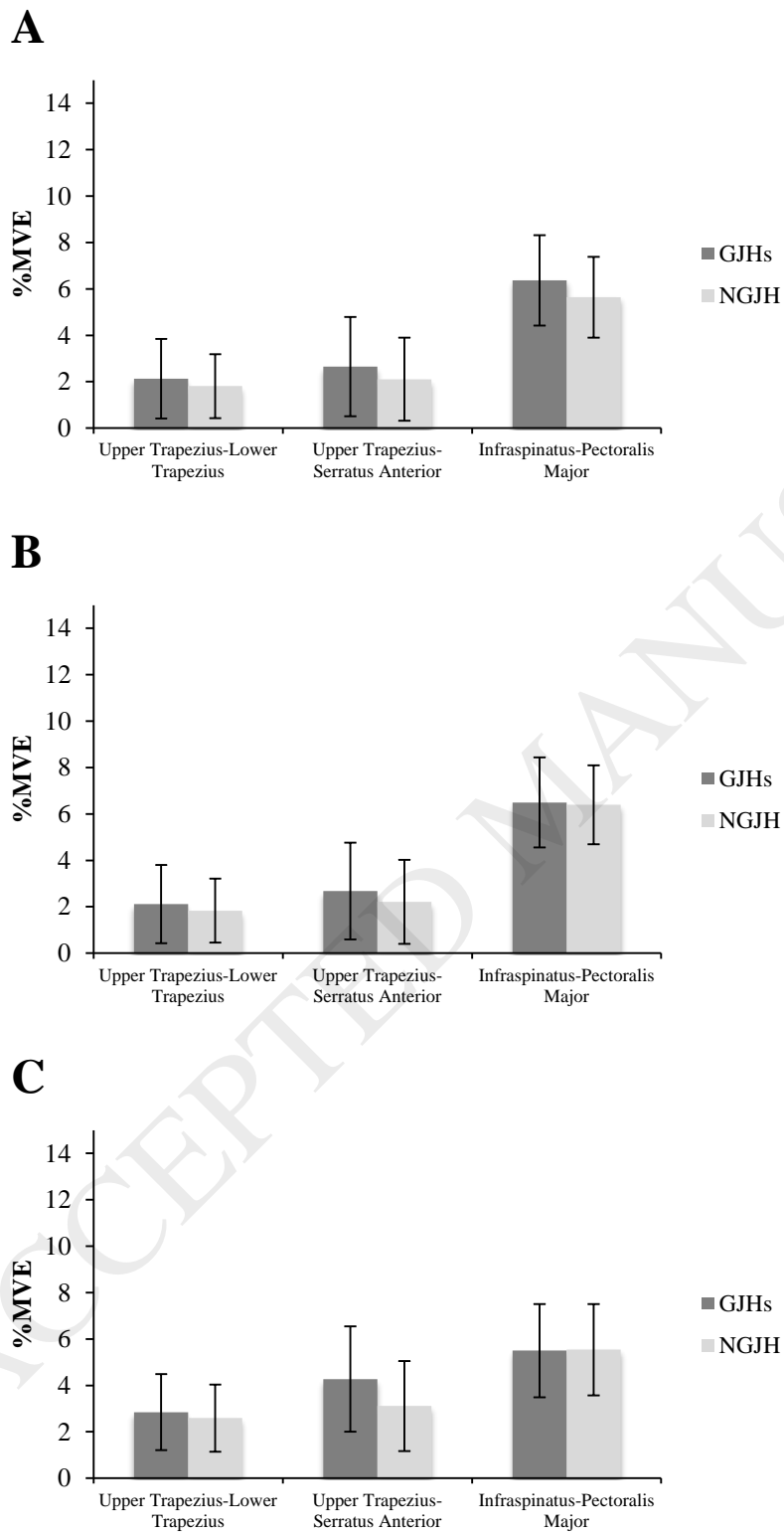


Figure 3





**Table 1** Electrode placement and maximum voluntary isometric contractions (MVIC)

Muscle	Electrode placement	MVIC
Upper trapezius	20% medial to the mid distance between the seventh cervical vertebra and acromion	In standing, arm elevation was performed with resistance applied proximal to the elbow joint with the shoulder flexed to 90° in the scapular plane and thumb pointing upwards
Lower trapezius	33% medial to the midpoint between the medial border of the scapula and the eighth thoracic vertebra	In prone lying, horizontal abduction was performed in line with lower trapezius fibers with resistance proximal to the elbow joint perpendicular to the floor with the shoulder in 125° of abduction
Serratus anterior	On the bulky part of the seventh rib, below the axilla, posterior to pectoralis major and anterior to latissimus dorsi in line with the xiphoid process and anterior axillary fold	In standing, arm elevation was performed with resistance applied proximal to the elbow joint with the shoulder flexed to 135° in the scapular plane and thumb pointing upwards
Infraspinatus	2.5 cm below the midpoint of the spine of the scapula	In prone lying, shoulder external rotation was performed with resistance applied at the distal forearm with the shoulder in 90° abduction and 45° external rotation and elbow flexed 90°
Pectoralis major	33% of the distance from the greater tuberosity to the xiphoid process with the arm in 90° abduction	In supine lying, resistance against horizontal adduction was performed with resistance applied distal to the elbow joint with the shoulder in 90° flexion, elbow extended and a slight medial rotation of the humerus

**Table 2** Demographics for swimmers with generalized joint hypermobility including shoulder hypermobility (GJHS) and swimmers without GJH and no shoulder hypermobility (NGJH)

	GJHS (n=19)	NGJH (n=19)
Sex [female], n (%)	11 (57.9)	11 (57.9)
Age [years]	14.8 ± 1.3	14.7 ± 1.1
Height [cm]	172.6 ± 8.4	170.6 ± 9.8
Weight [kg]	65.8 ± 12.8	62.7 ± 10.9
BMI [kg/m <sup>2</sup> ]	22.0 ± 3.4	21.4 ± 1.9
Swimming experience at the competitive level [years]	4.3 ± 1.9	4.9 ± 1.7
Swimming practice duration [hours/week]	8.3 ± 3.9	8.7 ± 5.2
Other sports activities [hours/week]	6.1 ± 2.1	5.5 ± 3.0
Beighton score [0-9]	7.1 ± 1.1*	1.1 ± 1.2*
Rotès-Quérol [positive], n (%)	19 (100)*	0 (0)*
Gagey test [positive], n (%)	12 (63.2)	6 (31.6)
Sulcus sign [positive], n (%)	3 (15.8)	0 (0.0)
Load-and-shift test [positive], n (%)	6 (31.6)	1 (5.3)
VAS [0-100]	3.2 ± 6.1	2.7 ± 3.9
WOSI score [0-2100]	225.8 ± 248.9	232.5 ± 170.1
Physical symptoms [0-1000]	95.1 ± 106.6	102.5 ± 89.7
Sports/recreation/work [0-400]	46.6 ± 73.7	36.0 ± 39.8
Lifestyle [0-400]	27.4 ± 44.0	18.1 ± 17.8
Emotions [0-300]	56.7 ± 54.9	76.0 ± 64.8

BMI=Body Mass Index; VAS=Visual Analogue Scale; WOSI=Western Ontario Shoulder Instability Index.

Values are mean (standard deviation) unless otherwise is indicated (i.e. sex, Rotès-Quérol, Gagey test, Sulcus sign, and Load-and-shift). \*=Significant between-group difference.

**Table 3** Stabilometric variables displaying center of pressure path length (COPL), COPL velocity, and weight-load for swimmers with generalized joint hypermobility including shoulder hypermobility (GJHS) and swimmers without GJH and no shoulder hypermobility (NGJH)

	GJHS (n=19)	NGJH (n=19)	Between-group comparison (p-value)
BL-EO			
COPL [mm]	177.74 ± 42.15	187.25 ± 56.09	0.549
COPL velocity [mm s <sup>-1</sup> ]	5.93 ± 1.41	6.24 ± 1.87	0.549
Weight-load [kg]	25.31 ± 5.07	23.79 ± 4.31	0.293
BL-EC			
COPL [mm]	212.07 ± 66.43*	231.98 ± 73.61*	0.353
COPL velocity [mm s <sup>-1</sup> ]	7.07 ± 2.21*	7.73 ± 2.45*	0.353
Weight-load [kg]	25.78 ± 4.52	24.23 ± 4.23	0.223
UL-EO			
COPL [mm]	261.48 ± 80.32*	268.57 ± 70.74*	0.592
COPL velocity [mm s <sup>-1</sup> ]	8.72 ± 2.68*	8.95 ± 2.36*	0.592
Weight-load [kg]	18.86 ± 3.29	18.28 ± 3.51	0.656

Bilateral upper-extremity support with eyes open (BL-EO) and eyes closed (BL-EC); Unilateral upper-extremity support with eyes open (UL-EO). Values are mean (standard deviation). \*=Significant within-group difference compared to BL-EO.