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Greater Lumbar Extension During Dolphin Kick and Psoas Major Tightness in Swimmers with Low Back Pain

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Abstract

Context: In competitive swimming, many swimmers experience lower back pain (LBP). Lumbar hyperextension may cause LBP and tight hip flexor muscle may cause lumbar extension during swimming.

Objective: The purpose of this study was to clarify the features of the elastic moduli of the muscles and the lumbar extension when swimmers with low back pain (LBP) perform a dolphin kick (DK).

Design: Cross sectional study.

Setting: Single center.

Participants: Eleven male college swimmers were enrolled as the LBP group (who have LBP when swimming and during a lumbar extension), and 21 male college swimmers were recruited as control group (no LBP).

Interventions: The elastic moduli of the psoas major, iliacus, teres major, latissimus dorsi, pectoralis major, and pectoralis minor were measured through ultrasonic shear wave elastography. The lumbar and hip extension angles during a DK were measured using a video camera. The passive hip extension and shoulder flexion range of motion (ROM) were measured using a goniometer.

Main Outcome Measures: Muscle elastic moduli and lumbar extension angles during DK.

Results: The characteristics, muscle elastic moduli, DK motion, and ROM were compared between the two groups. LBP group demonstrated significantly higher elastic modulus of the psoas major and lower modulus of pectoralis minor compared to the control group. Also, LBP group showed greater lumbar extension during a DK and less hip extension ROM than the control group.

Conclusion: The higher elastic modulus of the psoas major and greater lumbar extension during a DK may be related to the LBP in swimmers.

Keywords: prevention, swimming, ultrasound, posture, injury management

In various sports, low back pain (LBP) is frequently seen in athletes including volleyball players, rhythmic gymnasts, and competitive swimming.[1] This often leads to reductions in training time and/or competitive opportunities. It has been reported that about half of all competitive swimmers experience LBP.[2] Therefore, the prevention of LBP in competitive swimmers is important. The physical characteristics of competitive swimmers is different from those of ordinary people. To prevent LBP in swimmers, it is necessary to know their physical characteristics. Competitive swimmers have a high risk of spinal deformities such as spine asymmetry, thoracic kyphosis, or lumbar lordosis.[3] Intervertebral disc degeneration is frequently found in the lower area of the lumbar spine (mainly at the L5/S1 level, and occasionally at the L4/L5 level) and this tendency is not associated with the swimming style.[4]

Several studies have been conducted on the swimming kinematics of swimmers without LBP. It has been reported that the hip joint extends to about 10 degrees during a front crawl[5] and may be larger during a dolphin kick (DK) owing to its performance characteristics. In a previews study, it was reported that the tightness of the hip flexor muscle can reduce hip extension that create a lumbar hyperextension and pelvic anterior tilt in various movement in water.[6] Pelvic anterior tilting can make the pelvis at a lower position than normal in water.[6] A study examined the swimmers experiencing LBP and reported that repetitive lumbar hyperextensions during swimming may cause LBP.[7] The tight hip flexor muscle may extend lumbar spine during DK and cause LBP in swimmers. Furthermore, excessive training may also cause LBP in swimmers. However, the physical and kinematic characteristics of swimmers with LBP have not been clarified.

Competitive swimmers have more general joint laxity than normal, but the range of motion (ROM) of their internal and external shoulder rotations are smaller.[8] Furthermore, it has been reported that the ROM of the internal shoulder rotations decrease as competitive swimmers continue their careers from their youth level to the college level.[9] Therefore, although joint flexibility is required in competitive swimming, swimming training imposes a burden on the muscles around the shoulder joint, which can tighten. The basic posture in swimming used to achieve the least amount of water resistance, called a “streamline,” is a posture with the trunk and hip at a 0 degree extension, and a shoulder flexion of greater than 180 degrees, with both hands placed above the head. As described above, swimming training can burden the muscles and tighten shoulder muscles. If a swimmer experiences tightness in the shoulder muscles, a trunk extension will occur as a compensation of the shoulder flexion during a streamline posture, which is frequently seen in practice. As described above, repetitive lumbar extensions can cause LBP.[7] Therefore, tightness of shoulder muscles may cause wrong streamline posture that is related to LBP in swimmers.

Currently, shear wave elastography is known to be able to assess muscle stiffness noninvasively. However, no studies have compared the elastic moduli of the hip flexor and shoulder muscle or the lumbar extension angle during a DK between swimmers with and without LBP. Therefore, the aim of the present study was to compare the elastic moduli of the hip flexor and shoulder muscles as well as the ROM of the passive shoulder flexion and hip extension between swimmers with and without LBP, and to clarify the features of their muscle stiffness, hip and shoulder ROM, and lumbar motion during a DK. There were three hypotheses. First, the elastic modulus of hip flexor muscles is higher in swimmers with LBP than those without LBP. Second, the elastic modulus of shoulder muscles is higher in swimmers with LBP than those without LBP. Third, the lumbar extension angle during DK is greater in swimmers with LBP than those without LBP.

METHODS

Design

A cross sectional study was used.

Participants

Thirty-nine male college swimmers were initially enrolled in this study. The inclusion criteria of the LBP group were subjects with more than 30 mm of LBP in VAS while both swimming and during a lumbar extension.[10] The inclusion criteria of the control group were subjects currently with no LBP (0 mm on the VAS). As a result, 11 subjects were enrolled in the LBP group (21.1 ± 1.5 years), and 21 subjects were enrolled in the control group (20.6 ± 1.5 years). Three subjects were excluded because they experienced LBP when swimming but not during a lumbar extension, and four were excluded because their LBP was less than 30 mm in VAS. The subjects were provided a full explanation, based on the Helsinki declaration, regarding the aim, methods, and risks of the study, and their agreement was received. This study was conducted with the approval of the ethics committee of institution of authors.

Procedures

The subjects answered a questionnaire after an explanation of the study and an agreement to participate. In the questionnaire, the presence and intensity of their LBP, their swimming style, and the number of their training years were determined. The intensity of their LBP was assessed as being between zero and 100 based on the visual analogue scale (VAS). The elastic moduli of the psoas major, iliacus, teres major, latissimus dorsi, pectoralis major, and pectoralis minor muscles were measured using ultrasonic shear wave elastography. Their lumbar and hip extension angles during a DK were measured using an underwater camera. In addition, their passive hip extension and shoulder flexion ROM were measured using a goniometer.

In this study, the elastic moduli of the iliacus, psoas major were analyzed since they may restrict hip extension and the teres major, latissimus dorsi, pectoralis major clavicular, pectoralis major sternocostal, and pectoralis minor muscles were measured because they may restrict shoulder flexion. The elastic moduli of these muscles were measured using ultrasonic shear wave elastography (Aixplorer, Supersonic imagine, Aix-en-Provence, France) with a linear array probe (SL10-2, Super Sonic Imagine, Aix-en-Provence, France). Using this device, the muscle stiffness can be measured quantitatively based on elastic moduli, and the elastic moduli of the muscles as measured through ultrasonic shear wave elastography were found in a study on cadavers to be strongly related to the degree of muscle expansion.[11] In a living body, passive joint movement or passive tension of a muscle are strongly related to its elastic moduli.[12] The elastic moduli (G) reflects the elasticity or stiffness of a tissue, which is calculated based on the propagation velocity (V) of the shear wave in the tissue, as described in the following expressions.[13]

$$G=\rho V^2$$

In this expression, ρ is the density of the tissue, which for muscle has been reported to be 1000 ㎏/m³.[14]

Measurements of the elastic moduli of the psoas major and iliacus muscles were conducted in a relaxed supine position with the hip joint extended at 0 degrees, and the pelvis of the subject was fastened using a band to avoid anterior tilting. The measurement location of the psoas major and iliacus are shown in Figure 1. This location was determined to be 3 to 5 cm below the anterior superior iliac spine (ASIS) and distal of the inguinal ligament, based on a previous study.[15] The psoas major and iliacus were then identified as lateral to the femoral artery using ultrasonic images.

The shoulder position and locations of the elastic moduli measurements of the shoulder muscles are shown in Figures 2A–2C. The teres major and latissimus dorsi were measured during a relaxed sitting position and with the shoulder flexed at 90 degrees, rotated at 0 degrees, and the elbow flexed at 90 degrees. The measurement location of the teres major and latissimus dorsi was defined as 10 cm below the acromion (Figure 2A). The pectoralis major and minor were measured during a relaxed sitting position and with the shoulder abducted at 90 degrees, rotated at 0 degrees, and the elbow flexed at 90 degrees. The pectoralis major clavicular and sternocostal parts were measured at the midpoint between the greater tubercle and the acromioclavicular joint and at midpoint between the greater tubercle and the fourth sternocostal joint, respectively. The pectoralis minor was measured at the midpoint between the coracoid process and the fourth sternocostal joint in the same way as in a previous study.[16]

Before measuring the elastic moduli, the state of each muscle was confirmed through ultrasonic imaging. For all muscle measurements, the region of interest (ROI), in which area the elastic moduli can be measured, was set at the center of the muscle. The elastic moduli of all muscles were measured by placing an ultrasonic probe parallel to the muscle fiber. The elastic moduli of all muscles considered were measured three times, and the mean value was used for a statistical analysis.

To evaluate the reliability of the elastic moduli measurement using ultrasonic elastography with a liner probe, the elastic moduli of all muscles for ten of the asymptomatic subjects were measured twice to assess the intra-class correlation coefficients (ICC 1.1). Consequently, the ICC 1.1 value of each muscle was as follows; psoas major = 0.888, iliacus = 0.924, teres major = 0.622, latissimus dorsi = 0.756, pectoralis major clavicular = 0.755, pectoralis major = 0.897, and pectoralis minor = 0.642. According to a report by Landis et al., if the ICC value is higher than 0.81, the reliability of the measurement may be nearly perfect.[17] Therefore, the number (k) of measurements to meet the requirement of the lowest ICC value of the teres major, i.e., greater than 0.8, was calculated using the Spearman-Brown formula (a).

$$k = \frac{\rho 1(1 - \rho 2)}{\rho 2(1 - \rho 1)} \qquad \text{(a)}$$

In this formula, ρ1 is the target ICC value, and ρ2 is the resulting ICC value. As a result, all measurements of the elastic moduli were conducted three times because k was approximately 2.57.

An underwater video camera (O10-SC13001-W-1, Lerving Technology, Kwai Chung N.T., Hong Kong; 1920×1080 resolution, at 60 fps) and a Kinovea video player (version 0.8.15, available for download at http://www.kinovea.org) were used to measure the lumbar angle during a DK. The reliability of the joint angle measurement in the sagittal plane as assessed from a digital image was obtained in a previous study.[18] The subjects performed an underwater DK for 15 m with full effort, and the video was taken at a point 7.5 m away along the sagittal plane. The subjects were instructed to perform the DK at around 0.5 m from the surface of the water. The camera was set at 0.5 m below the surface of the water, 7.5 m from the starting side wall of the pool, and 4 m from the subject's swimming lane.[19] Markers were put on the subject's spinous process at T10, L3, S2, iliac crest (IC), greater trochanter (GT), and lateral epicondyle of the femur (LEF) to measure the angle of the lumbar and hip joints. The lumbar angle (θ_1) was defined as an angle made by lines A and B. Line A was produced by the markers at T10 and L3, whereas line B was produced by the markers at L3 and S2 (Figure 3).[20] The hip angle (θ_2) was defined as the angle made by two lines, one produced by the IC and GT, and the other produced by the GT and LEF.[19] The lumbar angle was measured when the angle of the hip extension was largest when performing a DK. The average values from three appropriate trials were used in the statistical analysis and false trials in which the subjects did not pass in front of the video camera were excluded. The time for the 15 m DK was measured with a stopwatch.

The passive shoulder flexion and hip extension ROM were assessed using a goniometer. The shoulder flexion ROM was assessed in a sitting position, and the hip extension ROM was assessed in a prone position. All measurement of joint angle during DK and passive ROM were analyzed by the same researcher.

Statistical Analyses

A statistical analysis was conducted using SPSS (version 22.0, SPSS Japan, Inc., Tokyo, Japan). The normality of all data obtained was confirmed through a Shapiro-Wilk test. The age, height, weight, number of training years, elastic modulus of each muscle, lumbar extension, and hip extension angle applied when performing a DK, as well as the passive ROM of the shoulder flexion and hip extension between the LBP and asymptomatic groups were compared using a Mann-Whitney U-test. Spearman's rank correlation coefficient test was conducted to investigate the relationship between swimming performance (best record of 50 m crawl or 15 m DK time) and degree of lumbar extension or muscle elastic modulus. The differences were shown to be statistically significant at an alpha level of 0.05.

RESULTS

All measurements were completed for each subject. The characteristics of the subjects are shown in Table 1. There were no differences in age, height, weight or number of training years between both groups.

The results of the elastic moduli are shown in Table 2. The LBP group showed a higher value in the elastic modulus of the psoas major compared with the control group. For the pectoralis minor, the LBP group showed a lower value compared with the control group. There were no differences in the elastic moduli of the other muscles between both groups.

The results of the lumbar and hip angle when performing a DK, along with the passive shoulder flexion and hip extension ROM, are shown in Table 3. The LBP group showed a larger lumbar extension angle when performing a DK than the control group.

The passive hip extension ROM of the LBP group was smaller than that of the control group. There were no significant differences in the other joint angles between both groups.

The result of Spearman's rank correlation coefficient test is shown in Table 4. No significant correlation between swimming performance and lumbar extension angle or muscle elastic modulus was found (Table 4).

DISCUSSION

The LBP group showed a higher elastic modulus of the psoas major and lower elastic modulus of the pectoralis minor than control group. Additionally, LBP group indicated a larger lumbar extension angle when performing a DK and smaller passive hip extension angle than the control group.

This is the first study comparing the elastic moduli of the muscles and the joint angle when performing a DK between swimmers with and without LBP.

The first hypothesis of this study was that the LBP group would show a higher elastic modulus of the hip flexor muscle than the control group. This hypothesis was partially supported because the results of this study demonstrated that, for the psoas major, the LBP group showed a higher elastic modulus than the control group, although no significant difference was shown in the elastic modulus of the iliacus. The psoas major orients from the lumbar spine, and attaches to the lesser trochanter. This muscle is a hip flexor and has an extension moment arm at levels L1 through L3 of the lumbar spine at standing and lumbar extended positions.[21] Furthermore, because the spine is extended to maintain a streamline position, which is a basic position for swimming, the extension stress from the psoas major increases at a higher level of the lumbar spine. In addition, because the psoas major has a flexion moment arm at a low level of the lumbar spine, and produces a lumbar moment in a different direction between the high and low levels of the lumbar spine, it is thought that the L3 vertebra moves forward and the L1 vertebra moves backward, such that the entire lumbar spine becomes extended.[21, 22] Therefore, LBP may likely occur if the elastic modulus of the psoas major is high because the lumbar spine will be more extended. Moreover, the psoas major causes a stronger shear force at the low level area of the lumbar spine than at a high level.[21] It is known from studies on rats and rabbits that lumbar intervertebral disc degeneration occurs if the vertebral disc is stressed from a continual shear force.[23, 24] In humans, an overload of the intervertebral disc may be related to disc degeneration.[25] A previous study reported that intervertebral disc degeneration, such as a decrease in the intervertebral disc thickness, is related to the frequency, duration, and intensity of the LBP.[26] With the subjects of our current study, an increase in the shear force at the low level of the lumbar spine may have occurred owing to the high elastic modulus of the psoas major, which could be the cause of their intervertebral disc degeneration and LBP. Although the iliacus has a hip flexion moment arm, as with psoas major, the iliacus is oriented from the pelvis and not from the lumbar spine. Therefore, if the elastic modulus of the iliacus is high, a pelvic anterior tilt and subsequent lumbar extension stress will occur; however, the stress to the lumbar spine might be less compared to that of the psoas major muscle. For this reason, it is rational for the iliacus to not be related to LBP in the present study, unlike with the psoas major.

The second hypothesis of this study was that the LBP group will show higher elastic moduli in the shoulder muscles than the control group; however, a lower elastic modulus was found in the pectoralis minor muscle for the LBP group than for the control group, and there were no significant differences in the elastic moduli of the other shoulder muscles. Therefore, our hypothesis was not supported. The pectoralis minor is oriented from the second to the fifth rib, and attached to the coracoid process, and the scapula is anterior tilted, rotated downward, protracted, and depressed when this muscle is activated.[27] Although, the normal scapula movement during a shoulder flexion has an upward rotation, a posterior tilt, and an external rotation,[28] the short length of the pectoralis minor normalized by height can cause a scapula anterior tilt and an internal rotation compared with the longer length of the pectoralis minor normalized by height.[29] To the best of our knowledge, there have been no studies investigating the relationship between the stiffness of the pectoralis minor and scapula kinematics; however, if the pectoralis minor is tight, the scapula kinematics may be similar to the kinematics under a short pectoralis minor. Conversely, the scapular kinematics under less stiffness of the pectoralis minor may be similar to that of a long pectoralis minor. In this study, a low elastic modulus of the pectoralis minor in the LBP group may help the scapula tilt at the posterior and rotate externally during a shoulder flexion, and the LBP subjects may be able to compensate for a glenohumeral flexion by increasing the movement of their scapula. Therefore, to prevent LBP from occurring owing to a lumbar extension, the LBP group may compensate for the lumbar extension during a shoulder flexion while in the streamline position through an increased scapula movement. We initially hypothesized that the LBP group would show higher elastic moduli of the shoulder muscles than the control group because the LBP subjects would compensate for a shoulder flexion occurring from the high elastic moduli of their shoulder muscles by increasing their trunk extension, thereby causing LBP. However, the LBP group showed a lower elastic modulus of the pectoralis minor than the control group, and accordingly, it is thought that the low elastic modulus of the pectoralis minor in the LBP group is not the cause of, but rather a compensation for, LBP. In addition to the pectoralis minor, the elastic moduli of the latissimus dorsi, teres major, and pectoralis major, as the shoulder muscles considered in this study, were measured. These muscles have a shoulder extension moment in a shoulder flexed position,[30] and are thought to restrict a shoulder flexion, induce a lumbar extension, and result in LBP if their elastic moduli are high. However, there was no significant difference in the elastic moduli of the latissimus dorsi, teres major, and pectoralis major between the LBP and control groups. Therefore, it is thought that these muscles did not affect the LBP in the swimmers considered in this study.

The third hypothesis was that the LBP group would show a greater lumbar extension when performing a DK than the control group. The LBP group showed a significantly higher lumbar extension angle during a DK than the control group, which is congruent with this hypothesis. Because repetitive lumbar hyperextensions while swimming can induce LBP,[7] the large angle of the lumbar extension during a DK can cause LBP. In this study, the passive hip extension ROM in the LBP group was significantly lower than that in the control group, and the subjects in the LBP group may have compensated for a hip extension when performing a DK through a lumbar extension. However, because the angle of the hip extension during a DK was about 9 degrees in both groups, it is thought that the hip was not extended to the limit of a passive hip extension ROM when performing a DK. Therefore, the significant difference in the lumbar extension angle during a DK may be influenced by another factor. The results of this study indicate that the elastic modulus of the psoas major was higher in the LBP group than in the control. Because the high elastic modulus of the psoas major can cause a lumbar lordosis, and the hip will extend the psoas major when performing a DK, thereby increasing the elastic modulus of psoas major, the high lumbar extension angle during a DK in the LBP group may be caused by the high elastic modulus of the psoas major. An approach to decrease the elastic modulus of the psoas major, or exercising the hip extension without a trunk extension may be useful in treating or preventing LBP.

There was no correlation between swimming performance and degree of lumbar extension during DK, as demonstrated by the Spearman’s rank correlation coefficient test. Therefore, increased lumbar extension during DK does not necessarily improve the swimming performance. However, it may increase the lumbar spine load. Thus, higher lumbar extension degree during DK is not an appropriate technique to improve swimming performance or reduce lumbar spine load.

There may be several factors causing LBP in swimmers; extended training period being one of them. However, no significant relationship was found between LBP and number of training years using Mann-Whitney U-test. These results may be partially explained by the participants’ team. In this study, the participants were the members of a swimming club. Thus, their practice frequency and training year were similar. So, the effects of extended training on swimmers with LBP are unclear.

There are certain limitations to the present study. First, as a cross-sectional study, it is impossible to indicate the cause and effect relationship between LBP and the elastic moduli of the muscles or the lumbar angle during a DK. Second, the inclusion criteria for the LBP in this study differ from those of many other previous studies because many other studies include LBP patients whose LBP has continued for longer than 3 months, and thus the results of this study are incommensurable with those of other studies. Third, the stiffness of the psoas major is thought to cause a lumbar extension when swimming; however, swimming styles other than a DK were not assessed. In competitive swimming, swimmers spend a significant amount of their training time using a crawl stroke regardless of their particular swimming style. Therefore, the lumbar motion during a crawl stroke may be related to LBP. To clarify the cause and effect relationship between the elastic moduli of the muscles and LBP, or the same criteria of LBP as applied in other studies, further longitudinal researches are needed.

CONCLUSION

This study indicated that swimmers suffering from LBP during lumbar extension have a higher elastic modulus of their psoas major, lower elastic modulus of their pectoralis minor, and a greater lumbar extension during a DK than swimmers without LBP. This study also showed that lumbar extension during DK and muscle elastic modulus are not related to swimming performance. Therefore, these issues in swimmers with LBP must be resolved to prevent LBP.

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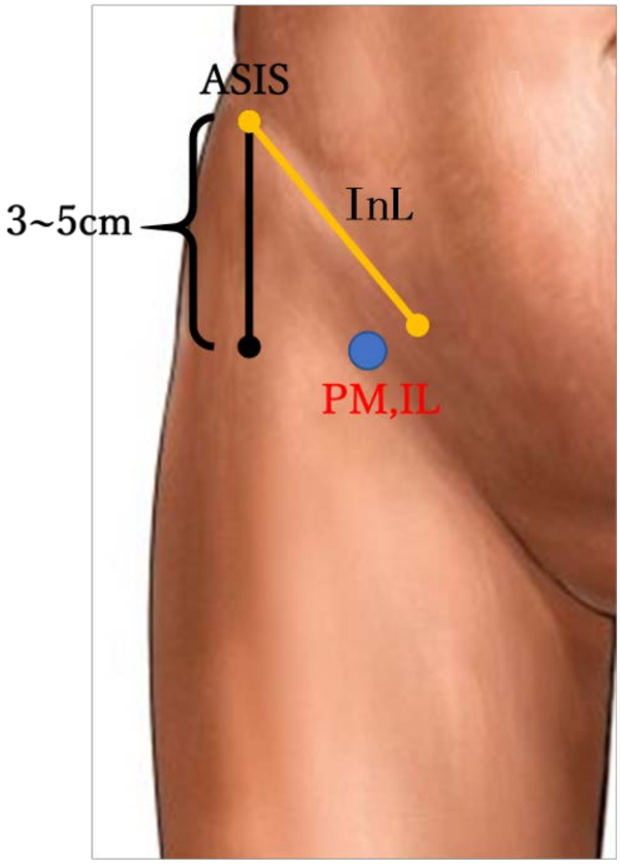

Figure 1. Measurement cites of shear elastic modulus for psoas major and iliacus muscles

The measurement locations of the psoas major (PM) and iliacus (IL) are shown. First, the femoral artery was identified through ultrasonic imaging as 3 to 5 cm below the anterior superior iliac spine (ASIS) and distal of the inguinal ligament (InL). These muscles were then identified lateral to the femoral artery. At this location, the probe was set parallel to the muscle fiber, and the elastic moduli were measured.

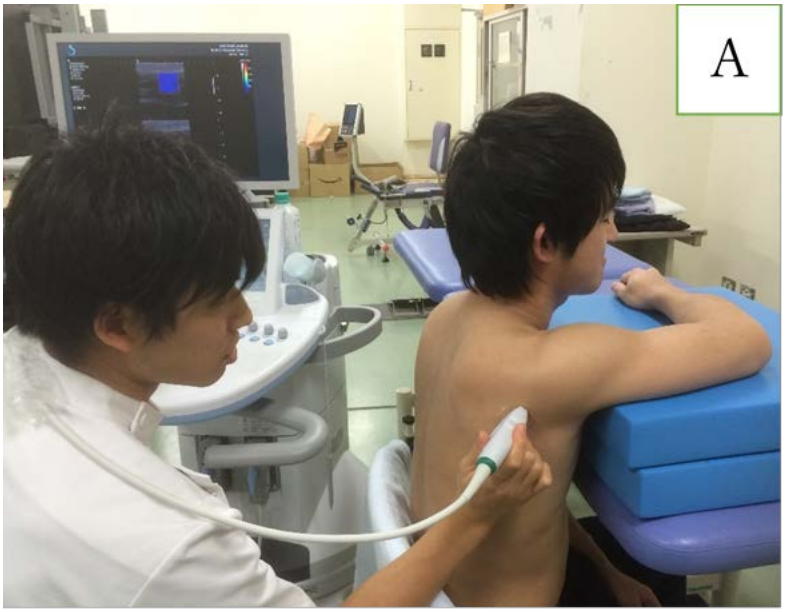

Figure 2A. Measurement position for the shear elastic modulus of the teres major and latissimus dorsi

The measurement position for the teres major and latissimus dorsi is shown in Figure 2A. The subject sat with a shoulder flexed at 90 degrees, rotated at 0 degrees, and the elbow flexed at 90 degrees.

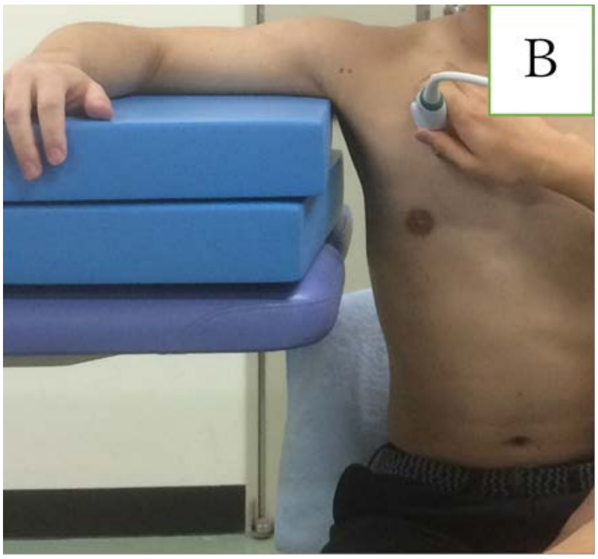

Figure 2B. Measurement position for the shear elastic modulus of the pectoralis major and minor shoulder muscles

The position used during the measurement of the pectoralis major and minor is shown in Figure 2B. The position was the shoulder abducted at 90 degrees, rotated at 0 degrees, and with the elbow flexed at 90 degrees.

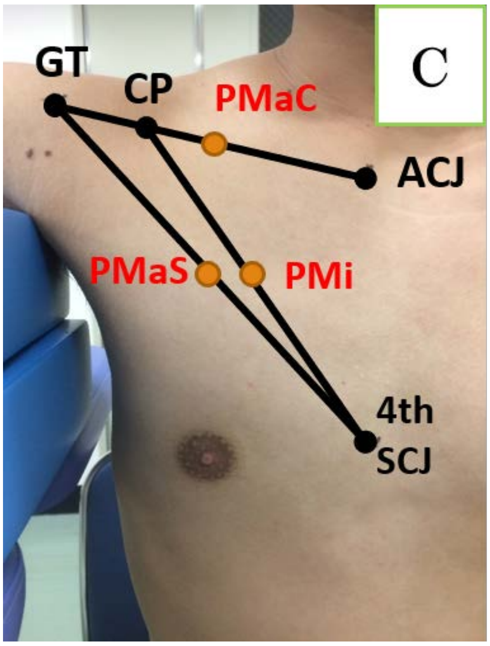

Figure 2C. Measurement sites of shear elastic modulus for pectoralis major clavicular part, sternocostal part, and pectoralis minor muscles

The measurement locations of the pectoralis major clavicular (PMaC), sterncostal (PMaS), and pectoralis minor (PMi) are shown in Figure 2C. The pectoralis major clavicular part was measured at the midpoint between the greater tubercle (GT) and acromioclavicular joint (ACJ), the pectoralis minor (PMi) was measured at the midpoint between the coracoid process (CP) and fourth sternocostal joint (4th SCJ), and the sterncostal (PMaS) was measured at the midpoint between the greater tubercle and the fourth sternocostal joint.

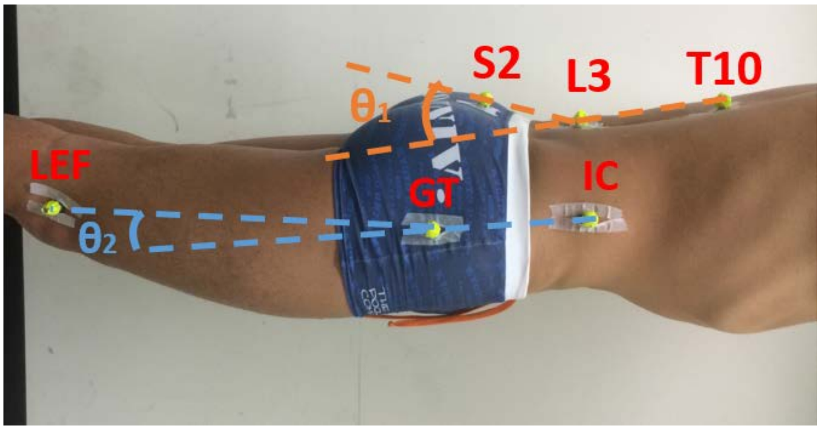

Figure 3. Measurement method of lumbar and hip joint angle during a DK

The measurement method of the lumbar and hip joint angle is shown in Figure 3. The lumbar angle (θ_1) was defined as the angle created by two lines, one produced by the markers at T10 and L3, and the other produced by the markers at L3 and S2. The hip angle (θ_2) was defined as the angle made by two lines, one produced by the iliac crest (IC) and greater trochanter (GT), and the other produced by the GT and lateral epicondyle of the femur (LEF).

Table 1. Characteristics of the participants

	LBP group (n = 11)	Control group (n = 21)	*P* value
age, y	21.1 (1.5)	20.6 (1.5)	.558
height, cm	176.4 (5.7)	173.2 (5.9)	.208
body mass, kg	68.3 (8.0)	65.9 (7.2)	.238
training years, y	12.6 (3.5)	12.1 (3.4)	.785

Abbreviations: LBP, low back pain.

Table 2. Shear elastic modulus of each muscle

Shear elastic modulus, kPa				
Muscles	**LBP group (n = 11)**	**Control group (n = 21)**	**Effect size (*r*)**	***P* value**
psoas major	17.9 (4.3)	13.1 (2.6)	0.516	.003
iliacus	14.2 (4.7)	11.6 (2.5)	0.228	.208
teres major	9.4 (3.6)	8.5 (1.7)	0.140	.434
latissimus dorsi	8.8 (2.6)	8.1 (1.6)	0.088	.639
pectoralis major clavicular part	10.3 (3.8)	10.4 (3.4)	0.025	.907
pectoralis major sterncostal part	8.5 (3.7)	8.3 (2.8)	0.025	.907
pectoralis minor	5.4 (2.1)	7.6 (3.5)	0.389	.027

Abbreviations: LBP, low back pain.

Table 3. Angle of each joint

Joint angle, deg	LBP group (n = 11)	Control group (n = 21)	Effect size (*r*)	*P* value
Lumbar extension during DK	22.1 (4.0)	15.3 (2.6)	0.701	<.001
Hip extension during DK	9.5 (4.3)	8.7 (5.3)	0.070	.690
Passive shoulder flexion	199.5 (11.4)	202.9 (8.8)	0.107	.546
Passive hip extension	23.6 (7.4)	29.3 (4.9)	0.364	.040

Abbreviations: DK, dolphin kick; LBP, low back pain.

Table 4. Correlation Between Swimming Performance and Lumbar Extension or Muscle Elastic Modulus

	Correlation coefficient		Correlation coefficient	
Variables	**Best records of 50m crawl**	***P* value**	**15m DK time**	***P* value**
Lumbar extension angle	.088	.630	.101	.583
Psoas major	-.033	.859	.037	.839
Iliacus	-.009	.962	.158	.387
Teres major	.175	.337	.031	.865
Latissimus dorsi	.219	.229	.229	.207
Pectoralis major clavicular part	-.084	.647	-.060	.745
Pectoralis major sterncostal part	.041	.826	-.012	.949
Pectoralis minor	.129	.483	.063	.734

Abbreviations: DK, dolphin kick.