

Effect of Stretching Protocols on Glenohumeral-Joint Muscle Activation in Elite Table Tennis Players

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Context: Several studies report static-stretch-induced deficits and dynamic-stretch performance improvement after intervention. **Purpose:** To investigate the muscle activation of the forehand and backhand in table tennis players after experiencing static- and dynamic-stretching protocols. **Methods:** A total of 24 elite male table tennis players (age 22.7 [3.46] y, height 1.78 [0.03] m) were tested before and 0, 10, 20, and 30 min after the 3 conditions (dynamic stretch, static stretch, and no stretch). The MEGA ME6000 (Mega Electronics, Kuopio, Finland) was used to capture the surface EMG data of the anterior deltoid, middle deltoid, posterior deltoid, biceps, and triceps muscles. Muscle activation data of the pretest were compared with posttest 0, 10, 20, and 30 min. These data were also compared between 3 different conditions (dynamic stretch, static stretch, and no stretch). **Results:** A 2-way repeated-measures analysis of variance indicated significant differences in the forehand and backhand, and Bonferroni test as a post hoc comparison revealed significant differences between the pretest and posttests in several muscles (P < .05). Furthermore, there were significant differences in the posttest between the 3 conditions (P < .05). **Conclusions:** In general, there was a short-term effect of static- and dynamic-stretching protocols on glenohumeral-joint muscle activation in elite table tennis players. The static and dynamic stretching presented a decrease and increase, respectively, in muscle activation up to 30 min after stretching. In conclusion, the additive and subtractive effects of dynamic- and static-stretching protocols on muscle activation seem to persist after 30 min.

Keywords: forehand, backhand, warm-up, electromyography

Stretching techniques before physical activity are common practices aimed at increasing the flexibility of athletes, and static and dynamic stretching are the most common techniques used toward this goal.¹

Scientific evidence have shown that static stretching may temporarily reduce force and power.^{2,3} The acute reduction in force has been termed the "stretching-induced force deficit," and it may be harmful to athletic performance.⁴ Although the precise mechanism is unknown, research data have suggested that the stretching-induced force deficit could be caused by (a) neural factors (decrease in motor unit activation, firing frequency, and reflex inhibition from afferent types III and IV) that reduce muscle activation, (b) mechanical factors (changes in the viscoelastic properties of the musculotendinous unit or changes in the angle–torque relationship) that affect the transmittal of force, or a combination of factors.^{3,5}

Hypotheses attempting to explain the deleterious effects of acute stretching include reduced stiffness of the musculotendinous unit, resulting in less effective transfer of force from the muscle to the lever and greater autogenic inhibition and, therefore, fewer motor units activated in a stretched muscle.⁶

Studies indicate that stretching-induced performance changes were typically low to moderate in magnitude when testing was performed immediately after the stretching protocol. An initial assumption, based on the overall results, may be to not recommend static stretching within pre event warm-up activities when high performance is required immediately after stretching.^{7,8} In addition, the vast majority of studies have examined only the effect of stretching on lower-body performance; the different injury

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mechanisms of common sites of upper- or lower-extremity injury and differing motor pathways to upper- and lower-body performance stretching effects on the upper extremity need focused investigation. Furthermore, previous studies have examined the effect of stretching on maximal strength, explosive force production, vertical jump performance, concentric isokinetic peak torque, and isometric force production at different joint angles. Also, limited data are available regarding the electrical components of muscle production as a result of stretching. Therefore, the purpose of this study was to examine the acute effects of 2 different stretching techniques on the muscle activation of the glenohumeral joint during forehand and backhand movements in elite table tennis players. The main hypothesis considers that muscle activation decreases after static stretching when compared with the dynamic-stretching protocol in both tennis movements.

Methods

Participants

Based on a statistical power analysis derived from surface electromyography (sEMG) data from a pilot study, a sample size of 21 participants was deemed necessary to achieve an alpha level of .05 and a power $(1-\beta)$ of .80. Therefore, a total of 24 elite table tennis players (age 22.7 [3.46] y, height 1.78 [0.03] m, and weight 75.43 [4.32] kg) were recruited to participate in the current study. They had at least 5 y of experience in playing tennis (at least 3 times a week) and no previous surgery or history of injury with residual symptoms (pain) in the upper limbs or spine within the last year. The Kharazmi University Research Ethics Committee approved this study (protocol no. DBSI22042018); we informed all participants about the procedures of the study, and they signed an informed consent form, in accordance with the Declaration of Helsinki.

Design

The research method was semiexperimental with a crossover design.

To control the learning effect, the participants were divided into 3 subgroups, and assessments were performed in 3 separate sessions (each session has a different condition) in nonconsecutive days with at least 48 h between them. All participants received specific instructions about warming up and stretching procedures. Afterward, the demographical characteristics of the participants were defined. The participants performed a general warm-up with 5 min of jogging and a specific warm-up with 5 min of playing table tennis without a ball, performing both the forehand and backhand in each session. 10 In the pretest, all participants performed a forehand and backhand, and the activation of the shoulder muscles was recorded during 3 trials of forehand and backhand. The participants performed 5 forehand and 5 backhand strokes in each trial. All balls were delivered to the player by a ball machine that was placed 1.2 m away from the opponent's court. Settings of the machine, including a projecting angle, a radian angle, velocity, and frequency, were consistent for all balls. The right-handed players performed strokes diagonally from the middle of the right and left sides of the table for the forehand and backhand, respectively. Before the test, the participants practiced the strokes 4 to 5 times so that they could become familiar with the test. At the beginning of the test, the instruction given to each participant was to hit the ball normally. The strokes were considered valid only if the ball landed over the net in the opponent's part of the table. 11,12 Then, all participants of each subgroup performed a single specific stretching protocol (static stretch, dynamic stretch, or no stretch). In the first session (condition number 1), subgroup number 1 performed static stretching, subgroup number 2 performed dynamic stretching, and subgroup number 3 was the control subgroup. In the next sessions, all of the subgroups changed, and each subgroup performed the exercise that they had not performed yet (Figure 1). After the stretching protocols, the same test was applied in different moments: immediately after (0 min), 10 min, 20 min, and 30 min.

Interventions

Depending on the assigned intervention, the participants performed a static-stretch protocol that involved lengthening the muscle until the point of discomfort was reached, and then the muscle was maintained in a lengthened position for 2 sets of 15 s with a 15-s rest interval. For dynamic stretch they performed 15 repetitions in a nonpainful range of motion with high speed while still maintaining control.¹³

The static exercises included were the head side to side (stand up straight; relax and straighten shoulders; using your hand, slowly pull the head toward the shoulder and hold it; perform the exercise without abrupt movements), overhead reach (extend arms overhead with elbows straight, interlock fingers if possible, keep back and neck straight), deltoid side press (position arm across neck, place opposite hand on elbow, push elbow toward neck, hold stretch), triceps square (lift left arm above head; bend elbow, bringing forearm and hand behind head; place right hand behind left elbow and pull backward), and finger interlock (take the arms behind and, interlocking the fingers, stretch the shoulders and the arms behind, opening up through the chest). 10,14

The dynamic exercises included were the head side to side (start with chin level and tilt head directly to the left, drawing left ear toward left shoulder, backward and go to the other side and repeat), overhead reach (with arms extended overhead and palms together, stretch arms upward and slightly backward, relax, and repeat), crossover arm swings (stand with feet shoulder-width apart and lift arms straight out to sides so they are parallel the ground; in a steady motion, move your arms across the center of your chest and

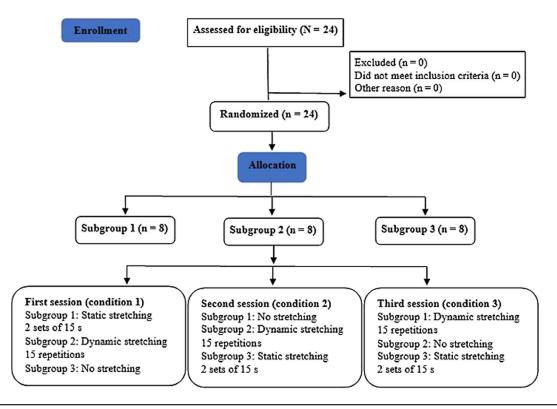


Figure 1 — Flowchart.

let them crisscross each other and repeat), arm circle (standing perpendicular to a wall, make big, slow circles with your arm; get as close to the wall as you can), and overhead arm swings (stand nice and tall, both arms straight down by sides, start to swing them upward until the fingers are pointing to the sky, and then quickly bring them back down to the starting position and repeat). ^{10,14} In the control condition, the participants were given a rest period as long as the longest stretching treatment.

Instrumentation

The participants' skin was prepared before placement of the sEMG electrodes. Hair at the site of the electrode placement was shaved and abraded, and the skin was cleaned with alcohol. Bipolar passive disposable dual Ag/AgCl surface electrodes were used (1 cm in diameter for each circular conductive area, with 2-cm center-to-center spacing). These were placed on the dominant upper extremity over the longitudinal axes of the short and long head of the biceps brachii (placed on the line between the medial acromion and the fossa cubit at 1/3 from the fossa cubit), long head of the triceps brachii (placed at 50% on the line between the posterior crista of the acromion and the olecranon at 2 finger widths medial to the line), anterior deltoid (placed at 1 finger width distal and anterior to the acromion), middle deltoid (placed from the acromion to the lateral epicondyle of the elbow), and posterior deltoid (2 finger breadths behind the angle of the acromion). About 2 reference electrodes were placed over C7 and around the elbow. Sensor placement was carried out according to SENIAM (Surface Electromyography for the Non-Invasive Assessment of Muscles) guidelines as far as possible. The sEMG signals were recorded by an EMG acquisition system (MEGA ME6000, Mega Electronics, Kuopio, Finland), with a sampling rate of 2 kHz. The sEMG activity was amplified (bipolar differential amplifier, input impedance = 2 M Ω , common mode rejection ratio > 100 dB min (60 Hz), gain \times 20, noise > 5 μ V) and analog-to-digitally converted (12 bits). The sEMG signals were collected during the forehand and backhand of table tennis. The digitized sEMG data were band-pass filtered at 20 to 400 Hz using a fourth-order Butterworth filter with a zero lag. The root mean square variables, in µV, were calculated.

Statistical Analyses

The normality and homogeneity of variances within the data were confirmed with the Shapiro–Wilk and Levene tests, respectively. The EMG data were analyzed with 2-way repeated-measures analysis of variance with 3 stretching protocols and (preintervention and 4 postinterventions) for all dependent variables. Post hoc comparisons were performed with the Bonferroni test. Cohen formula for effect size (*d*) was calculated, and the results were based on the following criteria: trivial (<0.2), small (0.2–0.6), moderate (0.6–1.2), large (1.2–2.0), and very large (>2.0) effects. Reliability was operationalized using the following criteria: <0.40 poor; 0.40 to <0.75 satisfactory; ≥0.75 excellent. The intraclass correlation coefficient ranged between .70 and .99 (excellent) for all dependent variables. An alpha of 5% was used to determine statistical significance.

Results

Table 1 shows the absolute mean values (SD) for the root-mean square variables before and after 3 stretching protocols.

The statistical analysis for EMG amplitude indicated that there were significant differences in the backhand (F = 12.859; P = .035)

and forehand (F = 2.893; P = .003). The post hoc test indicated that the differences were between the pretest and posttest 0 min (back [P = .037], fore [P = .009]), 10 min (back [P = .031], fore [P = .021]), 20 min (back [P = .032], fore [P = .027]), and 30 min (back [P = .036], fore [P = .033]) in the dynamic and between the pretest and posttest 0 min (back [P = .008], fore [P = .038]), 10 min (back [P = .049]), fore [P = .049]), 20 min (back [P = .053], fore [P = .045]), and 30 min (back [P = .041], fore [P = .013]) in the static protocol for the anterior deltoid muscle.

There were significant differences between the pretest and posttest 0 min (back [P=.032], fore [P=.006]), 10 min (back [P=.028], fore [P=.027]), 20 min (back [P=.031], fore [P=.030]), and 30 min (back [P=.017], fore [P=.029]) in the dynamic and between the pretest and posttest 0 min (back [P=.025]), 10 min (back [P=.012]), 20 min (back [P=.032], fore [P=.045]), and 30 min (back [P=.036]) in the static protocol for the middle deltoid muscle.

In the posterior deltoid muscle, significant differences were between the pretest and posttest 0 min (back [P=.038], fore [P=.009]), 10 min (back [P=.006], fore [P=.009]), 20 min (back [P=.027], fore [P=.026]), and 30 min (back [P=.017], fore [P=.001]) in the dynamic and between the pretest and posttest 0 min (fore [P=.047]), 10 min (fore [P=.026]), 20 min (fore [P=.006]), and 30 min (fore [P=.023]) in the static protocol. In the biceps muscle, significant differences were between the pretest and posttest 0 min (back [P=.007], fore [P=.017]), 10 min (back [P=.001]), fore [P=.004]), and 30 min (back [P=.029], fore [P=.001]) in the dynamic and between the pretest and posttest 0 min (back [P=.018]), fore [P=.025]), 10 min (back [P=.03], fore [P=.028]), 20 min (back [P=.032], fore [P=.002]), and 30 min (back [P=.019]), fore [P=.048]) in the static protocol.

In the triceps muscle, significant differences were between the pretest and posttest 0 min (back [P=.042], fore [P=.015]), 10 min (back [P=.034], fore [P=.039]), 20 min (back [P=.004], fore [P=.006]), and 30 min (back [P=.042], fore [P=.036]) in the dynamic and between the pretest and posttest 0 min (back [P=.022], fore [P=.033]), 10 min (back [P=.032], fore [P=.047]), 20 min (back [P=.034]), fore [P=.046]), and 30 min (back [P=.027], fore [P=.045]) in the static protocol. There was also no significant difference between the pretest and posttests in the nostretch group for all muscles in the backhand and forehand.

Between-groups analysis indicated that, for the pretest, there were no significant differences between the 3 different protocols for all muscles in the backhand and forehand. Table 2 shows all of the significant differences in the backhand.

Between-groups analysis in the forehand indicated that there were significant differences between static and no stretch (P = .013) in the posttest 20 min for the posterior deltoid muscle. Between-groups analysis for the biceps muscle indicated that, in the posttest 0 min, the significant differences were between dynamic and static (P = .02) and dynamic and no stretch (P = .003). In the posttest 10 min, the significant differences were between dynamic and static (P = .022)and dynamic and no stretch (P = .015). In the posttest 20 min, the significant differences were between dynamic and static (P = .003), dynamic and no stretch (P = .02), and static and no stretch (P = .018). In the posttest 30 min, the significant differences were between dynamic and static (P = .03) and dynamic and no stretch (P = .033). It also indicated that, in the posttest 20 min, the significant differences were between dynamic and static (P = .048) and dynamic and no stretch (P = .002). In the posttest 30 min, the significant differences were between dynamic and no stretch (P = .049) for the triceps muscle.

Table 1 Absolute Mean Values (SD) of the Root Mean Square Variables Before and After 3 Stretching Protocols

Protocol	Muscle	Pre		Post 0		Post 10		Post 20		Post 30	
		Back	Fore								
Dynamic	AD	30.795 4.092	34.945 5.352	39.241 4.150	37.814 5.708	39.988 5.208	39.157 4.289	40.859 4.446	38.711 5.503	39.630 4.373	38.191 4.401
	MD	31.444 4.422	30.635 3.327	40.975 4.772	39.156 4.432	38.059 5.060	40.220 6.115	38.280 6.289	37.620 4.813	38.541 5.581	39.760 3.136
	PD	31.532 4.735	29.083 3.371	39.516 3.130	35.125 4.325	41.629 5.746	36.520 3.891	39.866 5.529	34.814 3.521	38.594 5.098	36.253 3.814
	BC	28.830 4.194	32.158 2.632	36.923 3.075	39.588 4.056	34.835 4.414	38.001 2.928	35.291 4.637	38.619 2.537	34.698 3.237	37.257 2.361
	TC	33.978 4.424	32.273 3.838	41.785 2.270	35.455 4.195	42.412 2.396	36.201 5.181	39.393 4.961	36.045 3.399	41.070 4.293	37.306 5.085
Static	AD	31.317 3.626	34.874 3.240	27.639 4.148	30.381 3.378	26.975 4.081	31.406 4.563	26.165 3.104	29.660 2.809	26.654 3.130	31.881 3.685
	MD	31.583 2.078	30.530 4.488	29.388 2.445	27.654 4.559	28.594 1.966	28.574 4.914	28.236 2.818	27.770 4.767	28.393 3.160	27.754 4.670
	PD	31.968 2.106	29.494 3.461	26.631 2.118	27.718 3.253	27.590 2.636	27.312 3.581	28.802 2.447	27.339 3.310	28.699 2.339	27.854 3.355
	BC	28.412 2.650	32.675 2.697	25.228 2.080	27.486 2.875	24.995 2.017	28.484 3.412	24.265 2.419	27.032 2.735	24.945 2.632	27.709 2.392
	TC	33.307 4.116	32.606 3.531	30.664 3.699	29.853 3.623	30.262 4.826	29.521 3.734	30.072 4.037	28.269 3.964	31.637 4.040	28.667 4.721
No stretch	AD	31.428 3.186	34.087 4.072	31.990 3.481	34.014 4.571	30.777 3.122	34.722 4.343	30.814 3.440	33.890 4.676	31.363 2.580	34.002 3.312
	MD	30.459 3.455	30.728 3.906	30.661 3.507	31.059 3.660	30.569 4.254	30.979 3.629	30.699 3.049	31.529 3.792	30.092 3.362	30.926 4.001
	PD	31.025 2.732	29.732 3.216	30.684 2.463	29.770 4.049	31.772 2.664	29.925 3.409	31.814 2.191	31.393 3.544	30.472 3.607	29.926 4.278
	BC	28.722 3.246	32.307 3.639	28.731 3.451	32.071 3.421	28.806 3.728	31.711 3.540	29.486 3.587	32.615 3.838	29.395 3.418	32.337 3.776
	TC	32.913 4.802	33.032 3.001	32.621 4.677	33.144 2.186	32.050 4.523	32.482 3.732	33.043 3.913	32.551 3.132	33.266 4.345	32.810 3.410

Abbreviations: AD, anterior deltoid; back, backhand; BC, biceps; fore, forehand; MD, middle deltoid; PD, posterior deltoid; TC, triceps.

Discussion

The purpose of this study was to examine the acute effects of static and dynamic stretching on muscle activation of the upper extremity. These data support the hypothesis of a decrease in muscle activation following static stretching⁶ that has been extensively observed in several studies. Furthermore, the present findings supported the hypothesis of an increase in muscle performance followed by dynamic stretching.

Dynamic stretch involves the performance of a controlled movement through the range of motion of the active joint. ¹⁷ Based on some reasons, dynamic stretch is preferable to static stretch in the preparation for sports. First, there is a high similarity between the stretching and exercise movement patterns. ¹⁸ Second, dynamic stretching is able to elevate the core temperature, which can increase nerve conduction velocity, muscle compliance, and enzymatic cycling, and accelerate energy production. ¹⁹ Lastly, dynamic stretching increases the central drive, as may occur as with a prolonged static-stretching protocol. ²⁰

Scientific literature indicates that shorter duration of dynamic stretching might not facilitate performance as well as longer durations of dynamic stretching,²¹ whereas some studies with shorter durations of the dynamic stretching demonstrated facilitation in

performance. Herda et al³ have shown that 4 sets of 3 dynamic stretches of 30 s each increased EMG and mechanomyogram activity.³ Also, Fletcher and Monte-Colombo¹⁷ indicated that 100-beat dynamic stretching resulted in significantly greater countermovement jump and drop jump heights than 50-beat dynamic stretching. Even 50-beat dynamic stretching showed significantly greater performance in the jumps than the no-stretch condition.¹⁷ Although there is no clear distinction regarding the duration of dynamic stretching needed to enhance performance, there is clarity that dynamic stretching does not impair performance.²²

The mechanisms of muscular performance improvement after dynamic stretching have been suggested to be related to the elevation of muscle and body temperature, ¹⁹ postactivation potentiation in the stretched muscle, ²¹ stimulation of the nervous system, and decreased inhibition of antagonist muscles. ²³ As a result of these effects, dynamic stretching may enhance force and power development. ^{14,21,23} It is hypothesized that the increases in force output after dynamic stretching are caused by an enhancement of neuromuscular function, and it is suggested that the dynamic stretching had a postactivation potentiation effect on performance by increasing the rate of cross-bridge attachments. ²³ Consequently, it allows a greater number of cross-bridges to form and results in an increase in force production. ²⁴ However, Herda et al³ reported that

Table 2 Between-Group Significant Differences for Backhand

Muscle	Test	Dynamic-static				Dynamic-no st	retch	Static-no stretch		
		P	95% CI	Effect size	P	95% CI	Effect size	P	95% CI	Effect size
AD	Pre	.874	-1.116 to 0.846	-0.135	.505	-1.154 to 0.809	-0.172	.963	-1.012 to 0.947	-0.032
	Post 0	.112	1.418 to 4.174	2.796	.059	0.713 to 3.072	1.893	.254	-2.192 to -0.080	-1.136
	Post 10	.075	1.407 to 4.155	2.781	.074	0.915 to 3.375	2.145	.080	-2.091 to -0.001	-1.046
	Post 20	.042*	2.182 to 5.482	3.832	.057	1.212 to 3.841	2.527	.027‡	-2.515 to -0.322	-1.419
	Post 30	.057	1.876 to 4.948	3.412	.035†	1.039 to 3.566	2.302	.103	-2.774 to -0.508	-1.641
MD	Pre	.939	-1.020 to 0.939	-0.040	.363	-0.735 to 1.232	0.248	.514	-0.595 to 1.383	0.394
	Post 0	.017*	1.613 to 4.498	3.056	.007†	1.163 to 3.762	2.463	.208	-1.411 to 0.569	-0.421
	Post 10	.035*	1.165 to 3.765	2.465	.005†	0.476 to 2.728	1.602	.293	-1.597 to 0.405	-0.596
	Post 20	.084	0.848 to 3.273	2.061	.056	0.419 to 2.648	1.534	.294	-1.861 to 0.183	-0.839
	Post 30	.023*	0.988 to 3.487	2.237	.049†	0.666 to 3.001	1.833	.095	-1.517 to 0.475	-0.520
PD	Pre	.843	-1.099 to 0.861	-0.119	.722	-0.849 to 1.112	0.131	.528	-0.602 to 1.375	0.386
	Post 0	.003*	4.821 to 6.758	4.821	.034†	1.672 to 4.599	3.136	.139	-2.919 to -0.609	-1.764
	Post 10	.035*	1.676 to 4.605	3.140	.032†	0.959 to 3.442	2.201	.068	-2.700 to -0.455	-1.578
	Post 20	.026*	1.259 to 3.916	2.587	.075	0.731 to 3.098	1.914	.107	-2.374 to -0.218	-1.296
	Post 30	.034*	1.188 to 3.801	2.494	.044†	0.670 to 3.008	1.839	.457	-1.583 to 0.417	-0.583
BC	Pre	.692	-0.861 to 1.1	0.119	.943	-0.951 to 1.008	0.028	.758	-1.085 to 0.876	-0.104
	Post 0	.002*	2.626 to 6.283	4.455	.003†	1.197 to 3.815	2.506	.055	-2.298 to -0.160	-1.229
	Post 10	.020*	1.472 to 4.263	2.867	.010†	0.370 to 2.581	1.475	.081	-2.346 to -0.197	-1.271
	Post 20	.013*	1.557 to 4.405	2.981	.038†	0.306 to 2.493	1.400	.027‡	-2.851 to -0.562	-1.706
	Post 30	.012*	1.798 to 4.813	3.306	.047†	0.468 to 2.717	1.593	.010‡	-2.561 to -0.356	-1.458
TC	Pre	.465	-0.824 to 1.138	0.157	.139	-0.752 to 1.213	0.230	.691	-0.892 to 1.068	0.088
	Post 0	.006*	2.031 to 5.216	3.623	.043†	1.186 to 3.799	2.492	.321	-1.457 to 0.529	-0.464
	Post 10	.018*	1.712 to 4.665	3.189	.031†	1.468 to 4.257	2.863	.172	-1.371 to 0.606	-0.382
	Post 20	.003*	0.848 to 3.273	2.061	.138	0.324 to 2.518	1.421	.332	-1.760 to 0.266	-0.747
	Post 30	.012*	1.007 to 3.518	2.263	.042†	0.644 to 2.969	1.806	.587	-1.377 to 0.600	-0.388

Abbreviations: AD, anterior deltoid; BC, biceps; CI, confidence interval; MD, middle deltoid; PD, posterior deltoid; post, posttest; pre, pretest; TC, triceps.

dynamic stretching did not improve muscular strength, although EMG amplitude increased,³ which may reflect a potentiating effect of the dynamic stretching on muscle activation.

Static stretching involves lengthening a muscle until either a stretch sensation or a point of discomfort is reached and then holding the muscle in a lengthened position for a prescribed period of time.²⁵ Several studies have reported decreases in the forcegenerating capacity of a muscle or muscle group following a bout of static stretching.5-7,26 The results of this study supported these previous findings and indicated a reduction in muscle activation as a result of the static stretching. About 2 mechanisms have been determined to explain the stretching-induced decrease in force production: the first one refers to mechanical factors, such as decreases in musculotendinous stiffness, which may affect the muscle's length-tension relationship and sarcomere shortening velocity, and the second one refers to neural factors, such as decreases in muscle activation.^{5,6} Fowles et al⁸ reported that, after 15 min of recovery from intense stretching, most of the decreases in muscular force-generating capacity were related to intrinsic mechanical properties of the musculotendinous unit. They hypothesized that the stretching might alter the length-tension relationship and the plastic deformation of connective tissues such that the maximal force-producing capabilities of the muscle could be limited.8 The literature seems to indicate that neural effects are

more transient or play a smaller or insignificant role than viscoelastic properties in static stretch-induced impairments.²

Changes of musculotendinous unit stiffness might be expected to affect the transmission of force, the rate of force transmission, and the rate at which changes in muscle length or tension are detected. A more slack of parallel and series elastic components by slowing the period between myofilament cross-bridge kinetics and the exertion of tension by the musculotendinous unit on the skeletal system could increase the electromechanical delay.²⁷ Furthermore, a lengthened muscle due to an acute bout of static stretching could have a less than optimal cross-bridge overlap which, according to the length-tension relationship, could diminish muscle force output.²⁷ Previous studies involving prior static stretching to the point of discomfort have resulted in impairments of force^{7,8,22,26} and muscle activation.^{7,26} There has been some evidence in the literature to suggest that less than maximal intensity stretching might not produce these deficits.²⁸ High-intensity stretch-induced stress might have a detrimental effect on neuromuscular activation. Guissard et al²⁹ reported that the attenuation of reflex responses with small stretching amplitudes was mainly attributed to premotoneuronal or presynaptic mechanisms, whereas a large amplitude of the stretch-induced motoneuron excitation decreases was dominated by postsynaptic mechanisms.²⁹ It has been suggested that the decrease in the excitation of the motoneuron pool resulted from a reduction in excitatory drive from

^{*}Significant differences between dynamic and static groups. †Significant differences between dynamic and no-stretch groups. ‡Significant differences between static and no-stretch groups.

the Ia afferents onto the alpha motoneurons, possibly due to decreased resting discharge of the muscle spindles via increased compliance of the musculotendinous unit. Less responsive muscle spindles could result in a reduction in the number of muscle fibers that are subsequently activated.⁵ Moreover, auto compensation for the decrease in force production is a greater activation rate that resulted in a faster rate of neural fatigue. Further, inhibitory influences on the motoneuron could arise from types III (mechanoreceptor) and IV (nociceptor) afferents.8 Beyond neuromuscular effects, higher-intensity stretching has also been shown to impair blood flow through a muscle during the stretch.²⁹ Hence, performance could also be affected by changes in blood circulation to the muscle. Reduction of blood flow and tissue oxygen availability resulted in the accumulation of metabolic end products and reactive oxygen and nitrogen species. Trajano et al²⁰ observed that intermittent stretching caused notable perfusion and reperfusion of plantar flexor muscles and a greater magnitude of and longer-lasting force loss than did the same volume of continuous stretching, even though the absolute level of deoxygenation was greater during continuous stretches. Thus, ischemia-reperfusion cycles induced by intermittent stretching appear to be particularly problematic. The mechanism by which these cycles impair force production is not known specifically, but it appears not to be caused by a decrease in intracellular free calcium concentration.20

Based on the majority of the scientific literature and our findings in this study, it would seem logical to recommend that prolonged static stretching not be performed prior to a high level of competitive athletic or training performance. It would also seem prudent, based on the conflicting scientific literature, that even shorter-duration static stretching be minimized. There are many dynamic sports where enhanced static flexibility would be expected to affect performance. Although some studies have indicated that dynamic stretching can provide similar increasesin static flexibility as static stretching, other studies have indicated that dynamic stretching is not as effective at increasing static flexibility as static stretching within a single warm-up session or with prolonged training.³⁰ The results of this study indicated that dynamic stretching and static stretching before high-level or competitive athletic or training performance could increase and decrease the muscle activation, respectively, which could result in muscle imbalance and alter joint kinematic. Consequently, these stretch deficits could result in a dangerous circumstance for athletics.

The findings of the present study demonstrate that stretching (2 sets of 15-s static stretching per muscle group) held to the point of discomfort can adversely decrease sEMG amplitude even after 30 min of recovery. The stretch-induced impairments are hypothesized to be related to changes in muscle compliance with the stretching that may adversely affect the ability to detect and respond to the changes in muscle length and the rate of change in muscle length and forces. Furthermore, it was found that 15 repetitions of dynamic stretching in no painful range of motion can increase EMG amplitude even after 30 min of recovery.

Practical Application

Depending on athletic requirement, the duration and intensity of stretching can result in an advantageous warm-up prior to high level and competitive activities. In our study, we used 2 stretching protocols with specific duration and repetition. Thus, future research should investigate the effects of different stretching protocols with varied durations and repetitions. The result of this study demonstrated the addictive and subtractive effect of dynamic and

static stretching on muscle activation up to 30-min poststretching. It can provide useful data to elite table tennis players that could help improve their performance following correct warm-up and stretching procedures.

Conclusions

The results indicate that static stretching can decrease sEMG amplitude, while dynamic stretching can increase it. This suggests that, for many types of athletic performance that involve muscle actions of the upper extremity, static stretching as the sole activity during a warm-up routine should generally be avoided.

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