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Acute, chronic, and combined pulmonary responses to swimming in competitive swimmers

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ABSTRACT

The combined effects of swimming on the inspiratory muscles and pulmonary functions are not well known. The aim of the present study was to determine the acute, chronic, and combined effects of swimming on the pulmonary functions and respiratory muscles of competitive swimmers. Thirty males (15 in the experimental group [EG] and 15 in the control group [CG]) participated in this study. The EG subjects participated in an 8-week swim training program and performed 1 day before and after an 8-week 100-m swimming event. Pulmonary functions and respiratory muscle strength were measured immediately before and after the swimming event in the EG and before and after an 8-week period in the CG. The obtained data were analyzed using repeated measures one-way analysis of variance, least significant difference, and independent- and paired-sample *t*-tests. Swimming exerted negative acute effects ($p < 0.05$) and positive chronic effects ($p < 0.05$) on respiratory muscle strength and pulmonary functions. Further, the negative acute effects decreased the combined effects of the chronic and acute effects of swimming on respiratory muscle strength and pulmonary functions ($p < 0.05$). The results indicated that swimming exerts negative acute, positive chronic, and combined effects on respiratory muscle strength and pulmonary functions.

1. Introduction

Pulmonary function is an important indicator of a healthy life and physical capacity (Schunemann et al., 2000). Previous studies have shown that an inadequate pulmonary function capacity increases mortality (Beaty et al., 1982, 1985). Recent conflicting research has also revealed that respiratory performance negatively (Romer and Polkey, 2008; Aliverti et al., 1997; Johnson et al., 1996) and positively (Özdal, 2016a, 2016b; Özdal and Bostancı, 2018; Özdal et al., 2016; Harms et al., 2000; Mancini et al., 1995) affects physical performance. Improvements in pulmonary functions and respiratory muscle strength may provide better physical performance and a healthy life (Özdal and Bostancı, 2018; Özdal et al., 2016; HajGhanbari et al., 2013; Illi et al., 2012; Kilding et al., 2010; Ross et al., 2008; Witt et al., 2007; McConnell and Lomax, 2006).

Swimming is a known stressor of the respiratory mechanism (Armario et al., 1991; Prince and Anisman, 1984). It exerts an acute collapsing effect on the pulmonary airway due to water pressure (Hobo et al., 1998; Armario et al., 1991) and is an acute reason for respiratory distress syndrome (Zareian et al., 2011; Koshinaka et al., 2009; Shen et al., 2004; Avital et al., 2001; Prince and Anisman, 1984). Additionally, the horizontal body position adopted during front crawl and the increased hydrostatic pressure result in pulmonary engorgement (Frangolias and Rhodes, 1995). The spaces in the chest wall are filled with blood as a consequence, causing both lung compliance and airway calibre to fall (Grassino et al., 1981; Johnson et al., 1996) with the horizontal body position, which loads both the respiratory muscles, such as the m. diaphragm, m. trapezius, m. sternocleidomastoid, m. intercostales exteni/interni, and the important postural muscles, such as the m. erector spinae and m. supraspinatus (Gupta and Sawane, 2012). Consequently, immersion increases the hydrostatic compression, which counteracts the forces of inspiratory muscles (Frangolias and Rhodes, 1996) around the chest (Withers and Hamdorf, 1989; Frangolias and Rhodes, 1995) and negatively affects respiratory muscle strength (Prince and Anisman, 1984; Hobo et al., 1998; Shen et al., 2004).

Several studies have reported the acute effects of swimming on respiration or the pulmonary system (Miller et al., 2010; Bernard et al., 2009; Biswas et al., 2004; Adir et al., 2004; Shupak et al., 2000;) and its chronic effects (Mickleborough et al., 2008; Matsumoto et al., 1999; Courteix et al., 1997; Fitch et al., 1976) related to decreased muscle stiffness, improved nerve conduction velocity, developed contractile

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activity, and increased metabolic enzyme activity in the respiratory muscles (Wright and Johns, 1961; Ranatunga et al., 1987; Cordain and Stager, 1988; Proske et al., 1993; Mickleborough et al., 2008). To date, however, no study has reported the combined effects, i.e., chronic and acute effects, of swimming on the respiratory muscles and pulmonary functions.

In the present study, we investigated the acute, chronic, and combined effects of swimming on pulmonary functions and inspiratory muscle strength and hypothesized that acute swimming exercise would negatively affect pulmonary function and maximal inspiratory mouth pressure, whereas chronic swimming exercise would positively affect these.

2. Materials and methods

2.1. Experimental design

The present study was designed as a randomized pre- and post-test study with a control group (CG). The study design included one familiarization visit followed by four testing sessions for the experimental group (EG; T1-pre, T1-post, T2-pre, and T2-post) and two testing sessions for the CG (T1 and T2). During the familiarization visit, all subjects underwent laboratory-based pulmonary functions and maximal inspiratory mouth pressure (MIP) tests as a surrogate measure of inspiratory muscle strength. The acute swimming performance of the EG was similar to the swim training sets (frequency, four times per week; volume, 800–1000-m front crawl swimming per session and 3200–3600-m front crawl swimming per week; intensity, at the maximum aerobic swimming speed; overload, rest decrement after fourth week and maximal aerobic swimming speed measured before every 2 weeks) that participants performed on a regular basis. After the familiarization visit, subjects of similar age, height, mass, and swimming age were assigned to the EG or CG according to a stratified random sampling method. No significant differences were detected between the groups in terms of age, height, mass, and swimming age.

The EG subjects participated in an 8-week swim training program, whereas the CG did not participate in this program. During 8-week experimental period, the EG and CG subjects continued their recreational aerobic training routine with walking, running, and swimming due to being in the transition period. The EG subjects performed 100-m sprints with a front-crawl style before (T1) and after (T2) the 8-week training period. All EG measurements were taken before (T1-pre and T2-pre) and after (T1-post and T2-post) two 100-m sprints in four testing sessions. The CG subjects participated in measurements only once before (T1-pre) and once after (T2-pre) the 8-week period, without the 100-m sprints and swim training, in two testing sessions; these subjects were instructed to perform their daily activities as usual, and measurements were performed at the same time (between 14:00 and 18:00).

The acute response was determined between before (T1-pre and T2-pre) and after (T1-post and T2-post) measurements of the 100-m sprint performances in the EG subjects, whereas the chronic response was determined between before (T1-pre) and after (T2-pre) the 8-week period in the EG and CG subjects. The combined response was revealed with differences between percent decrements in the 100-m sprint performances of the EG subjects both before and after the 8-week period.

2.2. Subjects

Approval was obtained from the Gaziantep University Clinical Research Ethical Committee (Protocol Reference No. 2017/92), and written informed consent was obtained from all participants. We recruited 30 healthy male national-level competitive swimmers with no pulmonary or other relevant health problems. The participants had no injuries at the time of data collection and training, and all were sprint distance specialists. All participants had received competitive swim training for at least 5 years prior to the beginning of the study. Descriptive characteristics (age, height, mass, and swimming age) of participants are shown in Table 1.

Table 1
Descriptive characteristics of participants.

	EG (n = 15)	CG (n = 15)
Age (years)	18.6 ± 1.38	18.4 ± 0.67
Height (cm)	175.75 ± 8.77	175.33 ± 9.75
Mass (kg)	65.67 ± 9.52	65.75 ± 10.12
PIF (L/s)	5.75 ± 1.01	5.09 ± 1.90
PEF (L/s)	6.91 ± 1.80	5.77 ± 1.56
MIP (cmH_2O)	113.08 ± 8.17	110.75 ± 23.09
FVC (L)	3.92 ± 0.51	3.90 ± 0.68
FEV1 (L)	3.36 ± 0.37	3.52 ± 0.70
FEV1/FVC (%)	86.07 ± 7.01	90.32 ± 5.58
MVV (L/min)	114.31 ± 18.94	123.80 ± 31.90
VC (L)	3.77 ± 0.77	4.16 ± 1.14
ERV (L)	1.83 ± 0.58	1.68 ± 0.45
IRV (L)	1.23 ± 0.29	1.61 ± 0.53
TV (L)	0.99 ± 0.28	1.44 ± 0.68
IVC (L)	2.79 ± 0.44	2.71 ± 0.64

SD, standard deviation; MIP, maximal inspiratory mouth pressure; FVC, forced vital capacity, FEV1, forced expiratory volume in 1 s; MVV, maximal voluntary ventilation; VC, vital capacity; ERV, expiration reserve volume; IRV, inspiration reserve volume; TV, tidal volume; IVC, inspiratory vital capacity.

2.3. Eight-week training program

The CG subjects did not receive the 8-week swim training program, which the EG subjects received. The EG and CG subjects continued their recreational aerobic training routine with walking, running, and swimming due to being in the transition period. The EG subjects performed the 8-week swim training program four times a week for 8 weeks at a maximum aerobic swimming speed that was determined as the average swimming speed measured over the distance between 50 m and 350 m during the 400-m sprint swim with the front-crawl style and push start, including tumble turns, and after warming up. The maximum swimming speed was measured at the beginning of the study and once every 2 weeks to determine the overload principle. The EG executed 16 × 50 m front-crawl swimming with 1 min of passive rest to improve the anaerobic capacity. After the fourth week, passive rest was decreased to 30 s to support the overload principle (Dalamitros et al., 2016; Libicz et al., 2005). Each training session took 90 min with warming and cooling exercises that were performed in the water. The EG subjects performed swimming weekly at a distance of 3200–3600 m.

2.4. Front crawl 100-m swimming event

Participants completed 100-m front crawl swimming sprints, starting from a stationary position on the pool starting blocks, as in official competitions. Sprints were individually completed in a 25-m indoor pool, and performance times were measured using a single stopwatch by an experienced time-keeper (swimming referee) who was unaware of the study hypothesis. Participants were instructed to complete the sprint as fast as possible, and strong verbal encouragement was provided throughout the sprints (Parouty et al., 2010).

2.5. MIP measurement

MIP was measured to indicate inspiratory muscle strength using a respiratory pressure meter (MicroRPM, CareFusion Micro Medical, Kent, UK) according to the 2002 guidelines of the American Thoracic Society and European Respiratory Society (2002). Measurements started from the residual volume. The nose was occluded throughout the effort. To obtain the best values, all subjects performed three to five attempts with no more than a 5% difference between two attempts. An

Table 2
Analysis of acute pulmonary responses; repeated measures one-way analysis of variance results of before (T1-pre and T2-pre) and after (T1-post and T2-post) 100-m sprint performances of the EG subjects.

		Mean	SD	Effect size	SE	95% CI	
						LB	UB
MIP (cmH_2O)	T1-pre	113.08[a]	8.17	0.919	2.36	107.89	118.28
	T1-post	98.33	6.49		1.87	94.21	102.45
	T2-pre	123.50[acd]	8.20		2.37	118.29	128.71
	T2-post	117.33[bd]	8.37		2.42	112.02	122.65
FVC (L)	T1-pre	3.92[a]	0.51	0.615	0.15	3.60	4.25
	T1-post	3.68	0.65		0.19	3.26	4.09
	T2-pre	4.27[acd]	0.75		0.22	3.80	4.75
	T2-post	3.82	0.64		0.19	3.41	4.22
FEV1 (L)	T1-pre	3.36[a]	0.37	0.729	0.11	3.12	3.59
	T1-post	2.95	0.48		0.14	2.65	3.26
	T2-pre	3.98[acd]	0.69		0.20	3.53	4.42
	T2-post	3.36[b]	0.53		0.15	3.03	3.70
FEV1/FVC (%)	T1-pre	86.07[a]	7.01	0.623	2.03	81.61	90.52
	T1-post	80.80	6.30		1.82	76.79	84.80
	T2-pre	93.09[acd]	2.01		0.58	91.81	94.36
	T2-post	88.24[b]	2.23		0.64	86.83	89.66
MVV (L/min)	T1-pre	114.31[a]	18.94	0.697	5.47	102.27	126.34
	T1-post	108.78	16.29		4.70	98.43	119.12
	T2-pre	128.57[acd]	16.60		4.79	118.02	139.11
	T2-post	123.62[bd]	17.57		5.07	112.45	134.78
VC (L)	T1-pre	3.77[a]	0.77	0.755	0.22	3.28	4.26
	T1-post	3.27	0.79		0.23	2.77	3.78
	T2-pre	4.20_{acd}	0.60		0.17	3.82	4.58
	T2-post	3.89[b]	0.57		0.17	3.53	4.26
ERV (L)	T1-pre	1.83	0.58	0.192	0.17	1.46	2.20
	T1-post	1.92	0.61		0.18	1.53	2.30
	T2-pre	1.98	0.51		0.15	1.66	2.31
	T2-post	2.16	0.50		0.15	1.84	2.47
IRV (L)	T1-pre	1.23	0.29	0.143	0.08	1.04	1.41
	T1-post	1.25	0.33		0.10	1.04	1.46
	T2-pre	1.39	0.39		0.11	1.14	1.64
	T2-post	1.44	0.44		0.13	1.16	1.72
TV (L)	T1-pre	0.99	0.28	0.379	0.08	0.81	1.17
	T1-post	1.30[a]	0.30		0.09	1.11	1.49
	T2-pre	1.24[c]	0.33		0.10	1.03	1.45
	T2-post	1.28[bd]	0.27		0.08	1.11	1.46
IVC (L)	T1-pre	2.79[a]	0.44	0.919	0.13	2.51	3.07
	T1-post	2.39	0.34		0.10	2.17	2.60
	T2-pre	3.42[acd]	0.42		0.12	3.15	3.69
	T2-post	3.14[bd]	0.38		0.11	2.90	3.38

[a] significant difference between pre and post tests; [b] significant difference between T1-post and T2-post tests; [c] significant difference between T1-pre and T2-pre tests; [d] significant difference between T1-pre and T2-post or T2-pre and T1-post; SD, standard deviation; SE, standard error; CI, confidence of interval; LB, lower bound; UB, upper bound; MIP, maximal inspiratory mouth pressure; FVC, forced vital capacity; FEV1, forced expiratory volume in 1 s; MVV, maximal voluntary ventilation; VC, vital capacity; ERV, expiration reserve volume; IRV, inspiration reserve volume; TV, tidal volume; IVC, inspiratory vital capacity.

average of three acceptable attempts was used as the MIP value (Polkey et al., 1995).

2.6. Pulmonary function assessment

Pulmonary functions were evaluated using a spirometer (PocketSpiro USB100, Medical Electronic Construction R&D, Brussels, Belgium). Slow and forced vital capacity (FVC) tests were selected for pulmonary function assessment and measured according to the 2002 guidelines of the American Thoracic Society and European Respiratory Society (2002). FVC, forced expiratory volume in 1 s (FEV1), FEV1/FVC, and calculated maximal voluntary ventilation (MVV), vital capacity (VC), expiratory reserve volume (ERV), inspiratory reserve volume (IRV), tidal volume (TV), inspiratory vital capacity (IVC) were recorded using the pulmonary function test (Miller et al., 2005).

2.7. Statistical analyses

SPSS version 22.0 (SPSS Inc., Chicago, IL, USA) was used for statistical analyses. Data were expressed in terms of mean, standard deviation, standard error, effect size, lower and upper bounds of the 95% confidence of interval, and percentage of mean difference. The effect size was obtained from partial eta-squared data. The Shapiro–Wilk test was used to assess normality. Significance was defined as $p \leq 0.05$.

To determine the acute response, repeated measures one-way analysis of variance and least significant difference correction were used to analyze differences in measurements among the four testing sessions (T1-pre, T1-post, T2-pre, and T2-post) of the EG. To determine the chronic response, percent differences before and after the 8-week period between the EG ([T2-pre/T1-pre] × T1-pre) and CG ([T2-pre/T1-pre] × T1-pre) were identified and analyzed using independent samples *t*-test. To determine the combined response, differences between percent decrements at both 100-m sprint performances of the EG subjects before ([T1-post/T1-pre] × T1-pre) and after ([T2-post/T2-pre] × T2-pre) the 8-week period were identified and analyzed using paired samples *t*-test.

3. Results

Table 2 shows a comparison of all testing sessions of the EG subjects to analyze acute pulmonary responses to swimming. Significant differences in terms of MIP, FVC, FEV1, FEV1/FVC, MVV, VC, TV, and IVC

Table 3
Analysis of chronic pulmonary responses; independent samples *t*-test results between the EG ([T2-pre/T1-pre] × T1-pre) and CG ([T2-pre/T1-pre] × T1-pre) as percent differences before and after the 8-week period.

		EG (mean ± SD)	CG (mean ± SD)
MIP	T1-pre (cmH_2O)	113.08 ± 8.17	110.75 ± 23.09
	T2-pre (cmH_2O)	123.50 ± 8.20[b]	111.50 ± 25.07
	Percent Diff. (%)	9.33 ± 4.50[a]	−3.49 ± 4.22
FVC	T1-pre (L)	3.92 ± 0.51	3.90 ± 0.68
	T2-pre (L)	4.27 ± 0.75 [b]	3.73 ± 1.01
	Percent Diff. (%)	8.48 ± 7.39[a]	−1.27 ± 2.83
FEV1	T1-pre (L)	3.36 ± 0.37	3.58 ± 0.68
	T2-pre (L)	3.98 ± 0.69 [b]	3.38 ± 0.92
	Percent Diff. (%)	18.33 ± 15.56[a]	−5.29 ± 13.88
FEV1/FVC	T1-pre (%)	86.07 ± 7.01	91.75 ± 5.66
	T2-pre (%)	93.09 ± 2.01 [b]	90.50 ± 8.07
	Percent Diff. (%)	8.84 ± 9.52[a]	−4.04 ± 13.95
MVV	T1-pre (L/min)	114.31 ± 18.94	120.78 ± 31.74
	T2-pre (L/min)	128.57 ± 16.60 [b]	118.36 ± 32.29
	Percent Diff. (%)	13.59 ± 11.07[a]	−0.45 ± 5.53
VC	T1-pre (L)	3.77 ± 0.77	4.00 ± 1.28
	T2-pre (L)	4.20 ± 0.60 [b]	4.17 ± 0.88
	Percent Diff. (%)	13.38 ± 12.69[a]	−1.85 ± 10.34
ERV	T1-pre (L)	1.83 ± 0.58	1.74 ± 1.12
	T2-pre (L)	1.98 ± 0.51	2.00 ± 1.10
	Percent Diff. (%)	14.09 ± 33.36	7.74 ± 45.10
IRV	T1-pre (L)	1.23 ± 0.29	1.20 ± 0.87
	T2-pre (L)	1.39 ± 0.39	1.56 ± 0.70
	Percent Diff. (%)	17.39 ± 37.41	10.13 ± 41.30
TV	T1-pre (L)	0.99 ± 0.28	1.44 ± 0.84[b]
	T2-pre (L)	1.24 ± 0.33 [b]	0.92 ± 0.61
	Percent Diff. (%)	25.47 ± 8.83[a]	−19.44 ± 51.87
IVC	T1-pre (L)	2.79 ± 0.44	2.71 ± 0.64
	T2-pre (L)	3.42 ± 0.42 [b]	2.75 ± 0.59
	Percent Diff. (%)	23.42 ± 10.41[a]	3.88 ± 17.01

[a]significant difference between the EG and CG; [b] significant difference between the T1-pre and T2-pre of each group; SD, standard deviation; MIP, maximal inspiratory mouth pressure; FVC, forced vital capacity; FEV1, forced expiratory volume in 1 s; MVV, maximal voluntary ventilation; VC, vital capacity; ERV, expiration reserve volume; IRV, inspiration reserve volume; TV, tidal volume; IVC, inspiratory vital capacity.

were observed between the testing sessions ($p < 0.05$). Significant decrements were observed in these pulmonary measurements after the 100-m sprint.

Significant increments were observed between the T1-pre and T2-pre measurements of the EG in terms of MIP, FVC, FEV1, FEV1/FVC, MVV, VC, TV, and IVC ($p < 0.05$). Table 2 also shows significant improvements between the T1-post and T2-post measurements of the EG, particularly in terms of MIP, FEV1, FEV1/FVC, MVV, VC, TV, and IVC ($p < 0.05$).

Table 3 provides a comparison of percent differences between the T1-pre and T2-pre measurements of the EG and CG to analyze chronic pulmonary responses. Significant differences were found in terms of MIP, FVC, FEV1, FEV1/FVC, MVV, VC, TV, and IVC between the groups ($p < 0.05$). Pulmonary responses of the EG subjects improved after the 8-week training period, whereas those of the CG subjects did not.

Fig. 1, shows a comparison of percent differences between the T1-pre/post and T2-pre/post measurements of the EG subjects to analyze combined pulmonary responses. Significant differences were found in terms of MIP, FEV1, VC, TV, and IVC between the groups ($p < 0.05$). Decrements in pulmonary responses improved after the 8-week training period.

The front crawl 100-m swimming performances of the EG subjects were 64.35 ± 2.14 s in T1 and 63.83 ± 1.83 s in T2. Significant improvement was found in the front crawl 100-m swimming performances of the EG subjects between the T1 and T2 measurements ($p < 0.05$).

4. Discussion

The aim of the present study was to determine the acute, chronic, and combined effects of swimming on pulmonary functions and inspiratory muscle strength of competitive swimmers. There were three major findings of the present study: (1) after the acute swimming exercise, inspiratory muscle strength and pulmonary functions showed a large decrement as the acute response ($p < 0.05$); (2) inspiratory muscle strength and pulmonary functions showed significant increment after the 8-week swimming training program as the chronic response ($p < 0.05$); and (3) the most important finding occurred in the combined response that positive effects of the 8-week swim training reduced the negative effects of the single-effort swimming.

In the present study, negative acute pulmonary responses to swimming were observed to determine the acute effects. We found significant decrements in MIP, FVC, FEV1, FEV1/FVC, MVV, VC, TV, and IVC after both 100-m sprint performances of the EG subjects ($p < 0.05$). These results illustrate the negative acute effects of swimming on pulmonary responses. Significant improvements were found in the pulmonary responses of the EG subjects after the 8-week training period. When percent differences were compared between the EG and CG, significant differences were observed in terms of MIP, FVC, FEV1, FEV1/FVC, MVV, VC, TV, and IVC ($p < 0.05$). Pulmonary responses of the EG subjects improved after the 8-week training period, whereas those of the CG subjects did not. These results show the positive chronic effects of swimming on pulmonary responses. The combined response, identified as the effect of chronic responses on acute responses, seemed to reduce the negative effects of the acute effects of swimming. Significant differences were found in terms of MIP, FEV1, VC, TV, and IVC between the groups ($p < 0.05$). Observed decrements in pulmonary responses improved after the 8-week training period. These results expose the positive combined effects of swimming on pulmonary responses.

Previous studies have demonstrated the negative acute effects of swimming on respiratory muscle strength (Lomax and McConnell, 2003) and respiratory mechanism (Hobo et al., 1998). However, no

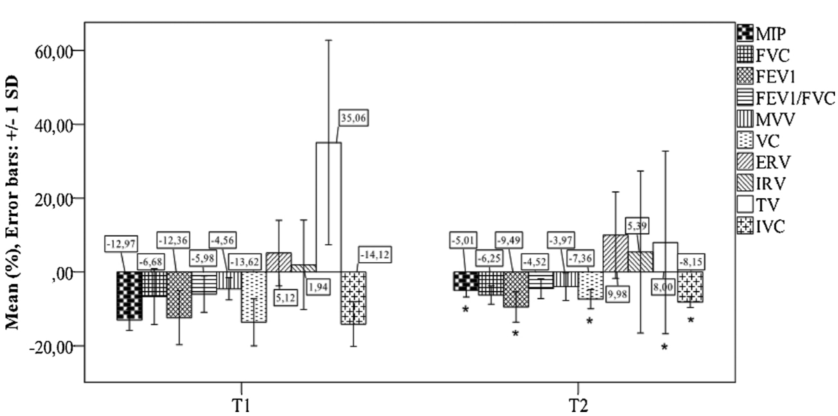

Fig. 1. Analysis of combined pulmonary responses; paired samples *t*-test results between the T1 ([T1-post/T1-pre] × T1-pre) and T2 ([T2-post/T2-pre] × T2-pre) as percent differences before and after the 8-week period of the EG subjects (* significant difference between T1 and T2 measurements. SD, standard deviation; MIP, maximal inspiratory mouth pressure; FVC, forced vital capacity; FEV1, forced expiratory volume in 1 s; MVV, maximal voluntary ventilation; VC, vital capacity; ERV, expiration reserve volume; IRV, inspiration reserve volume; TV, tidal volume; IVC, inspiratory vital capacity).

study has reported the negative acute effects of swimming on pulmonary functions. The acute results of the present study are significant in this aspect. Fatigue in the inspiratory muscles manifests as a decrement in inspiratory muscle strength (Özdal, 2016b). Increased temperature and acidosis with decreased pH in respiratory muscle tissues negatively affect muscle contraction (Lundin et al., 1993) and performance (Pacheco, 1957). Thus, the negative acute effects of swimming on respiratory muscles may be related to acidosis and decreased pH (Kapus et al., 2008; Gandevia, 2001; Bizid et al., 2009).

Hydrostatic compression (Frangolias and Rhodes, 1996; Withers and Hamdorf, 1989; Frangolias and Rhodes, 1995) and postural position (Gupta and Sawane, 2012) of swimming are unusual environments that create stressor effect (Grassino et al., 1981; Johnson et al., 1996; Armario et al., 1991; Prince and Anisman, 1984). Besides, fatigue of some trunk muscles occurs with swimming, which can be detected in MIP manoeuvres (Lomax et al., 2014, 2015). These perspectives can explain the observed reduction in inspiratory muscle strength due to swimming (Lomax, 2012; Lomax and Castle, 2011; Lomax and McConnell, 2003; Lomax et al., 2012, 2013).

Recent research has shown the positive effects of swimming on pulmonary responses (Wicher et al., 2010; Gupta and Sawane, 2012; Schaible et al., 1986; Clanton et al., 1987; Ide et al., 2005; Aysan et al., 2011). Moreover, some studies have demonstrated the effects of respiratory muscles on swimming performance (Ray et al., 2008, 2010, 2013; Wylegala et al., 2007; Lindholm et al., 2007). Chronic adaptation of the pulmonary system to the strain of swimming is related to decreased muscle stiffness, improved nerve conduction velocity, developed contractile activity, increased metabolic enzyme activity in respiratory muscles (Wright and Johns, 1961; Ranatunga et al., 1987; Proske et al., 1993), and increased elasticity of the lungs and chest wall (Gupta and Sawane, 2012; Pherwani et al., 1989; Lakhera et al., 1984) related to the thermal conductivity of water (Bowen, 1926). Respiratory muscles control some vital factors related to the airway and chest movements during respiration (Mehrotra et al., 1998). FEV1 measurements improve with increased airway clearance through increases in respiratory muscle strength (Joshi et al., 1992). Improvements in MVV could be derived from the positive effects of the strengthened respiratory muscles on the respiratory mechanism (Gopal et al., 1973). Inspiratory muscle strength is depended on the strength of the m. diaphragm, which is the most important respiratory muscle. Improvement in the inspiratory muscles such as the m. diaphragm positively affects expiratory forced works (Weiner et al., 2003).

The positive effects of exercise on the lungs are well known. Among athletes, swimmers are ranked the highest in terms of pulmonary functions (Mehrotra et al., 1998). Forced pulmonary parameters depend on the performance of respiratory muscles located in the thorax and abdominal regions. Swimming affects the muscles of these regions because unlike other vertical exercises, this activity is performed in the horizontal position, which is beneficial not only to the known major respiratory muscles (e.g., m. diaphragm, m. trapezius, m. sternocleidomastoid, m. intercostales interni/externi) but also to the m. erector spinae and m. supraspinatus muscles, which play an important role in maintaining a horizontal position. In this sense, swimming naturally improves the respiratory mechanism (Gupta and Sawane, 2012). This exercise provides a high level of external pressure to the thorax area, and this pressure can act as a form of resistance exercise to the m. diaphragm. This resistance exercise provides an increased functional capacity to the respiratory muscles (Gupta and Sawane, 2012; Mehrotra et al., 1998; Pherwani et al., 1989; Lakhera et al., 1984). These perspectives can explain the muscular mechanism of the obtained chronic results. Additionally, chronic exercise has positive effects on the ventilator system, particularly on both respiratory (Özdal, 2016b) and muscular (Schwartz, 1999; Schwartz et al., 2001; Oldervoll et al., 2003; Mock et al., 2005) fatigue. Ventilation is limited during swimming, and this limitation is caused by intermittent hypoxemia. Hypoxemia initiates the anaerobic process, and lactic acid begins to build up in the blood. This process is perceived by the respiratory center in the medulla oblongata, and ventilation increases. During these processes, inter-alveolar hyperplasia (Gopal et al., 1973) can be attributed to increases in FVC, FEV1, and VC. These perspectives can explain the metabolic mechanism of the chronic responses.

A previous study has reported that improved pulmonary functions could be related to increases in inspiratory muscle strength, thereby indicating an interaction between inspiratory muscle strength and pulmonary functions (Özdal, 2016a). In this regard, the present findings on acute pulmonary responses could be explained by increases in inspiratory muscle strength.

As a combined effect, the 8-week swim training program reduced fatigue (decrease of force) in the inspiratory muscles after the single-effort swimming. During swimming, hypoxemia, which is caused by both effort and hydrostatic pressure, initiates the anaerobic process and triggers acidosis (Gopal et al., 1973). This development causes a decrease in forces in the inspiratory muscles responsible for ventilation, also called inspiratory muscle fatigue. Inspiratory muscle fatigue is defined as a reduction in the maximal power-production capacity of the inspiratory muscles (Coirault et al., 1995). The causes of this fatigue are increased ventilation load, acidosis, decreased arterial oxygen content, decreased blood volume in the inspiratory muscles, and decreased energy stores in the inspiratory muscles (Johnson et al., 1996; Macklem, 1980). Inspiratory muscle fatigue is an example of peripheral fatigue. Regular exercise is an important factor in the reduction of peripheral fatigue. Decrements in peripheral fatigue occur via increased local muscle strength (Ament and Verkerke, 2009). In the present study, decrements in inspiratory muscle fatigue could be explained by increases in inspiratory muscle strength, which are a chronic response to swim training. As a combined effect, the same mechanism can be considered to be effective on FVC, related to forced respiratory tasks, depending on the performance of the inspiratory muscles (Gupta and Sawane, 2012). Observed improvements in 100-m swimming performances between the before and after 8-week program in the EG subjects may suggest improvements in inspiratory muscle strength, fatigue, and pulmonary functions after the 8-week program.

In conclusion, the 8-week swim training significantly improved pulmonary functions and inspiratory muscle strength of subjects, whereas the single-effort swimming produced negative effects. The positive effects of the 8-week swim training reduced the negative effects of the single-effort swimming. The mechanism responsible for these positive findings is probably associated with increased inspiratory muscle strength.

Limitations

The present study was constructed on respiratory muscle strength; however, maximal expiratory pressure was not measured because of unexpected technical problems. Therefore, the term "inspiratory muscle strength" is used in the text.

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