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體育運動論文評論(運動生理學) （本試題共六頁）

注意:答案一律寫在答案卷上，可用中文或英文作答，但必須橫寫，否則不予計分

(1) 分析本研究（Inbar 等，2000）內在效度（internal validity）的優缺點及改進方法。(50%)

(2) 分析本研究（Inbar 等，2000）外在效度（external validity）的優缺點及改進方法。(50%)

Specific inspiratory muscle training in well-trained endurance athletes

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ABSTRACT

INBAR, O., P. WEINER, Y. AZGAD, A. ROTSTEIN, and Y. WEINSTEIN. Specific inspiratory muscle training in well-trained endurance athletes. *Med. Sci. Sports Exerc.*, Vol. 32, No. 7, pp. 1233–1237, 2000. **Purpose:** It has been reported that arterial O_2 desaturation occurs during maximal aerobic exercise in elite endurance athletes and that it might be associated with respiratory muscle fatigue and relative hypoventilation. We hypothesized that specific inspiratory muscle training (SIMT) will result in improvement in respiratory muscle function and thereupon in aerobic capacity in well-trained endurance athletes. **Methods:** Twenty well-trained endurance athletes volunteered to the study and were randomized into two groups: 10 athletes comprised the training group and received SIMT, and 10 athletes were assigned to a control group and received sham training. Inspiratory training was performed using a threshold inspiratory muscle trainer, for 0.5 $h \cdot d^{-1}$ six times a week for 10 wk. Subjects in the control group received sham training with the same device, but with no resistance. **Results:** Inspiratory muscle strength (PI_{max}) increased significantly from 142.2 ± 24.8 to 177.2 ± 32.9 cm H_2O ($P < 0.005$) in the training but remained unchanged in the control group. Inspiratory muscle endurance (PmPeak) also increased significantly, from 121.6 ± 13.7 to 154.4 ± 22.1 cm H_2O ($P < 0.005$), in the training group, but not in the control group. The improvement in the inspiratory muscle performance in the training group was not associated with improvement in peak $\dot{V}_{Emax}$, $\dot{V}O_{2max}$, breathing reserve (B_R), or arterial O_2 saturation (%SaO_2), measured during or at the peak of the exercise test. **Conclusions:** It may be concluded that 10 wk of SIMT can increase the inspiratory muscle performance in well-trained athletes. However, this increase was not associated with improvement in aerobic capacity, as determined by $\dot{V}O_{2max}$, or in arterial O_2 desaturation during maximal graded exercise challenge. The significance of such results is uncertain and further studies are needed to elucidate the role of respiratory muscle training in the improvement of aerobic-type exercise capacity. **Key Words:** RESPIRATORY MUSCLE TRAINING, WELL-TRAINED ATHLETES, AND AEROBIC PERFORMANCE

A fundamental question in exercise physiology concerning the factor(s), which limits maximal oxygen uptake ($\dot{V}O_{2max}$), is still unresolved. However, growing evidence indicates that in trained athletes, oxygen delivery to the muscles limits the $\dot{V}O_{2max}$ during heavy exercise (1,33,35). O_2 delivery depends on blood flow, arterial O_2 saturation, and hemoglobin concentration. Traditionally, it was claimed that arterial O_2 saturation does not decline markedly in healthy individuals exercising at sea level, even at maximal effort (1,37,39). However, there are reports in the literature which suggest that arterial hypoxemia may occur during heavy exercise in healthy individuals with a high $\dot{V}O_{2max}$ (11,17,23,32). Aerobic performance of highly trained individuals may, therefore, be limited by this arterial hypoxemia.

Four possible mechanisms of exercise-induced hypoxemia have been proposed: 1) venoarterial shunt, 2) ventilation-perfusion inequality, 3) hypoventilation, and 4) diffusion limitations (13,31,36,39). It seems that venoarterial shunt does not play an important role in exercise-induced arterial hypoxemia, because oxygen breathing during heavy exercise was found to correct such hypoxemia (13,34). It was suggested that ventilatory capacity might play a role in the development of hypoxemia during maximal effort in highly trained endurance athletes (17,19,20,31). In a previous study performed by our group (17), we studied well-trained athletes during graded maximal aerobic exercise while breathing atmospheric air, normoxic helium, and oxygen-enriched mixture. It was concluded that the observed arterial O_2 desaturation is secondary to a relative hypoventilatory response and may limit $\dot{V}O_{2max}$ and aerobic performance at high work levels. It has also been shown that respiratory muscle fatigue occurred after both voluntary hyperpnea (2) and marathon running (20) in normal humans. Moreover, in at least one study (18), it was reported that increasing the stimulus to breathe during maximal exercise by inducing either hypercapnia or hypoxemia, but without parallel physiological modification in the respiratory system, failed to increase $\dot{V}_E$, $\dot{V}O_2$, as well as inspiratory or expiratory pressure.

Although there is ample evidence that inspiratory muscles' ability to generate force may be reduced, especially a

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work rates associated with high ventilatory requirements (19), it is well established that respiratory muscles can be trained like other skeletal muscles (4,29), both in healthy subjects and in patients with dyspnea (23,27,28,34,38). We, therefore, hypothesized that if respiratory constraints resulting in a relative hypoventilation plays a role in arterial O_2 desaturation at high work levels, then specific respiratory muscle training might result in improvement in exercise performance and in aerobic capacity in well trained endurance athletes.

METHODS

Subjects. Twenty well-trained endurance athletes mean ($\pm$SD) ages 28.9 $\pm$ 8.9 yr (range 18–40), active participants in national track events were recruited to the study. All participants had normal spirometry and a $\dot{V}O_{2max}$ higher than 55 mL·kg^{-1}·min^{-1}. All subjects were informed of the intent and procedures of the study, and a written informed consent was obtained before any data collection. The institution's research committee approved the study. Subjects were requested to refrain from any form of heavy exercise and the consumption of alcohol or caffeine in the 24 h before testing.

Subjects continued their regular aerobic training programs and were required to record the training load [velocity (running, swimming, and cycling) and distance per week] they attained during the study period.

Study design. After baseline measurements (the pretraining tests), the athletes were randomized into two groups: 10 subjects comprised the experimental group and received specific inspiratory muscle training (SIMT), and 10 athletes were assigned to the control group and got sham training.

Tests. All tests were performed before and at the end of the training period. The subjects had previously been exposed to all study procedures and therefore did not need any habituation sessions before the present study.

Spirometry. Pulmonary function was assessed by spirometry (Vitalograph, Compact, Buckingham England), performed before and at the end of the training period. The forced vital capacity (FVC) and the forced expiratory volume in one second (FEV_1) were measured four to five times (3–4 min interval between tests) with the subjects in a standing position wearing a nose clip. The average of the three best trials was taken as the value for each measurement (25). The pulmonary function assessment also included an isocapnic 60-s (B_f = 60 $\pm$ 3 breaths·min^{-1}) measurement of maximal voluntary ventilation in 60 s (MVV_{60}) (5,17).

Respiratory muscle strength. Respiratory muscle strength was assessed by measuring the maximal inspiratory mouth pressure (PI_{max}) at residual volume (RV) and the maximal expiratory mouth pressure (PE_{max}) at total lung capacity (TLC) as previously described by Black and Hyatt (6). The values recorded were the best of at least three efforts.

Inspiratory muscle endurance. To determine inspiratory muscle endurance, a device similar to that proposed by Nickerson and Keens (26) was used. Subjects inspired through a two-way Hans-Rudolph valve, whose inspiratory port was connected to a chamber and plunger to which weights could be added externally. Inspiratory elastic work was then increased by the progressive addition of 25- to 100-g weights at 2-min intervals, as previously described by Martyn and coworkers (22), until the subjects were exhausted and could no longer inspire. The pressure achieved with the heaviest load (tolerated for at least 60 s) was defined as the peak pressure (PmPeak).

Exercise capacity test. The test was carried out on a treadmill (Quinton 65®, Seattle, WA), using a modified Balke protocol (3). The running speed was individually adjusted (based on each subject's half marathon pace), and the work load was then incremented, at the conclusion of each min of exercise, by increasing the treadmill slope by 1.5%, until voluntary exhaustion (verified by age-predicted maximal HR, RER > 1.15, RPE > 18) (a typical test consisted of 8–11 1-min stages). All tests were conducted at the Ribstein Center for Sports Medicine and Research at the Wingate Institute, in an air-conditioned laboratory at sea level (mean room temperature 25 $\pm$ 2°C, relative humidity 45 $\pm$ 5%, and barometric pressure 760 $\pm$ 5 mm Hg). The pretraining tests were performed at the end of September, when all athletes concluded their preparatory training phase. This timing was selected in an attempt to minimize further improvement of the subject's aerobic capacity. During the study period regular training was continued but was devoted mainly to technical and tactical components.

Gas exchange, heart rate, and estimated O_2 saturation (Physiocontrol Lifestat 1600 pulse-oxymeter, Redmond, WA) data were collected, using a computerized data analysis system that included an Applied Electrochemistry S-3A O_2 analyzer (Sunnyvale, CA), Beckman I, B-2 CO_2 analyzer, (Fullerton CA) and Hewlett-Packard Fleisch-type pneumotachometer (no. 3, Ventura, CA). The gas analyzers were calibrated with precision gas mixtures and the pneumotachometer with precision 3.0-L Vacumed calibration syringe (Waltham, MA), immediately before each test. Basic gas and flow measurements were corrected for ambient temperature, barometric pressure, and water vapor. Measured and calculated cardiopulmonary variables (every 20 s) included: oxygen uptake ($\dot{V}O_2$), carbon dioxide output ($\dot{V}CO_2$), heart rate (HR), blood pressure (BP), minute ventilation ($\dot{V}_E$), respiratory exchange ratio (RER), ventilatory equivalent for oxygen ($\dot{V}_E/\dot{V}O_2$), and for carbon dioxide ($\dot{V}_E/\dot{V}CO_2$), and breathing reserve (B_R) (B_R = MVV_{60} − $\dot{V}_{Emax}$).

Respiratory training protocol. Subjects in both groups trained daily, six times a week. Each session consisted of 0.5-h training for 10 wk. The training was performed under the supervision of a trained physiotherapist. The subjects received SIMT with a threshold inspiratory muscle trainer (Threshold™ Inspiratory Muscle Trainer, Healthscan, NJ). The subjects started breathing at a resistance equal to 30% of their maximal inspiratory strength (Pi_{max}) for 1 wk. The resistance was then increased incrementally, 5% each session, to reach 80% of their PI_{max}, a

TABLE 1. Pre and post training anthropometric and pulmonary characteristics of the subjects in the training and in the control groups.[a]

	Training		Control	
	Pre	Post	Pre	Post
Height (cm)	175.4 ± 3.9	175.4 ± 3.9	175.0 ± 4.8	175.0 ± 4.8
Weight (kg)	66.6 ± 3.8	67.1 ± 4.3	65.0 ± 3.9	65.6 ± 7.9
Fat (%)	7.7 ± 1.4	7.0 ± 3.3	7.9 ± 1.2	7.2 ± 2.7
FVC (L)	5.1 ± 0.9	5.1 ± 0.8	5.2 ± 0.6	5.1 ± 0.5
FEV_1 (L)	4.1 ± 0.7	4.1 ± 0.8	4.3 ± 0.5	4.2 ± 0.5
MVV_{60} ($L \cdot min^{-1}$)	140.4 ± 27.6	140.4 ± 26.3	156.5 ± 20.6	50.4 ± 25.5

[a] Mean ± SD.

the end of the first 4 wk. SIMT was then continued for the next 5 wk at 80% of their PI_{max}, adjusted every week to the new PI_{max} achieved. Subjects in the control group received sham training with the same device, but with no resistance.

Data analysis. The results are expressed as means ± SD. A repeated measure analysis of variance (ANOVA) was applied to individual variables in order to investigate differences within (pre to post) and between the training and the control groups. A Tukey test was applied to compare the mean values. Statistical significance is accepted at $P < 0.05$.

RESULTS

The anthropometric, pulmonary functions, and peak exercise physiological characteristics of the subjects, as obtained before and after the training period, are summarized in Tables 1 and 2. Before training there were no differences between the groups for any of the variables studied.

Respiratory muscle strength and endurance. The effects of the respiratory muscle training on the inspiratory muscle strength and endurance are shown in Figure 1. All patients in the training group showed an increase in the inspiratory muscle strength. Mean PI_{max} increased significantly from 142.2 ± 24.8 to 177.2 ± 32.9 cm H_2O ($P < 0.005$). In the control group, mean PI_{max} remained unchanged (137.3 ± 22.2 and 138.6 ± 29.3, pre vs post training). Similarly, the respiratory muscle endurance (Pm-Peak) also increased significantly, from 121.6 ± 13.7 to 154.4 ± 22.1 cm H_2O ($P < 0.005$), in subjects of the training group, but not in subjects of the control group (128.2 ± 8.7 and 129.1 ± 16.8, respectively) (Fig. 1).

Spirometry. There were no significant differences in FVC, FEV_1, or MVV_{60} either between the two groups of subjects or when comparing the post to pretraining results within each group (Table 1).

Exercise capacity test. The results of the post training exercise test showed that there were no significant differences in the $\dot{V}_{Emax}$, $\dot{V}O_{2max}$, arterial O_2 saturation, breathing reserve (B_R), and in the ventilatory equivalents for O_2 or CO_2 measured during and at the peak of the exercise test, for either the experimental or the control groups (Table 2). It should be pointed out that in both groups arterial O_2 saturation (%SaO_2) as well as breathing reserve (B_R) at maximal effort was indicative of respiratory limitation (36), both before and after the respiratory muscle training period.

DISCUSSION

Our study showed that in well-trained endurance athletes the SIMT significantly increases the inspiratory muscle strength and endurance. The magnitude of the improvement of the inspiratory muscle performance was similar to that previously reported for patients with COPD (28,31,38). The increase in respiratory muscle performance in the current study was not associated with increases in $\dot{V}O_{2max}$, $\dot{V}_{Emax}$, arterial O_2 saturation, or in the ventilatory equivalents for O_2 or CO_2 at peak exercise levels.

It has previously been shown that elite endurance athletes have greater inspiratory muscle strength and endurance than untrained subjects (21). The present findings as well as previously reported results (12,22,23) indicate that both strength and endurance of the inspiratory muscles in trained athletes can still be improved by specific inspiratory muscle training. The impressive improvement (25% for inspiratory muscles strength and 10% for endurance) shown in our study can be explained, at least in part, by the relatively long duration and high intensity of the respiratory training sessions carried out by the training group.

Ventilatory limitation during exercise may be quantitated, among other variables, by %SaO_2 values or by the breathing reserve (B_R) index (30,36,37). Although pulse oximeter has been shown to be both valid and reliable in estimating trends in %SaO_2 during exercise (30), its sensitivity to minute changes, especially at maximal effort, is somewhat compromised (30). The B_R index is based on the comparison of maximal voluntary ventilation at rest (MVV) (usually determined during 12 or 15 s) with maximal exercise ventilation ($\dot{V}_{Emax}$). In healthy individuals, B_R, expressed as a difference, MVV − $\dot{V}_{Emax}$, is normally > 15 $L \cdot min^{-1}$ (36). In patients with advanced lung disease, and in highly trained endurance athletes, this difference is often lower than normal (sometime even < 1), which reflects the requirement to breathe closer to their maximal breathing capacity (17,26,37). In an attempt to better mimic ventilatory demand during maximal aerobic effort, we employed a 60-s MVV (isocapnic) instead of the more commonly used 12 s MVV protocol (5) Test-retest reliability of the MVV_{60} maneuver in our subjects showed highly reliable values (r's = 0.93–0.95, unpublished data).

In the present study, both %SaO_2 and breathing reserve (MVV_{60} − $\dot{V}_{Emax}$) were indicative of hypoventilatory response at peak exercise, in both groups, before and remained so even after the respiratory muscle-training period (see Table 2). These findings support previous reports from our group (17), and from others (10,11,18,32), suggesting ventilatory limitation at peak aerobic exercise of such high levels.

It is reasonable to assume that if respiratory hypoventilation plays any role in arterial O_2 desaturation at high work levels, then improved respiratory muscle performance could result in improvement of ventilatory and possibly in the aerobic capacity of the well-trained athletes (10,17,32). Nevertheless, arterial O_2 desaturation, $\dot{V}_{Emax}$, and $\dot{V}O_{2max}$, breathing reserve (B_R) and spirometric pulmonary function

TABLE 2. Pre and post respiratory muscle training peak physiologic variables in the training and in the control groups.*

	Training		Control	
	Pre	Post	Pre	Post
$\dot{V}O_2$ ($mL \cdot kg^{-1} \cdot min^{-1}$)	58.0 ± 4.6	58.1 ± 5.4	61.2 ± 4.7	59.7 ± 7.1
HR $(beat \cdot min)^{-1}$	184.3 ± 7.7	188.9 ± 7.9	184.5 ± 3.2	182.1 ± 8.8
$\dot{V}_E$ $(L \cdot min)^{-1}$	145.6 ± 18.7	145.4 ± 21.7	150.2 ± 15.6	146.2 ± 14.3
B_R $(L \cdot min)^{-1}$	−5.2 ± 4.5	−5.0 ± 5.2	5.8 ± 5.1	4.1 ± 3.9
$\dot{V}_E/\dot{V}O_2$ $(L \cdot L)^{-1}$	37.8 ± 4.0	37.3 ± 4.6	37.6 ± 3.4	37.5 ± 4.5
$\dot{V}_E/\dot{V}CO_2$ $(L \cdot L)^{-1}$	29.3 ± 2.6	28.8 ± 3.0	29.7 ± 0.4	29.5 ± 3.6
SaO_2 (%)	93.9 ± 1.1	90.7 ± 4.4	92.2 ± 1.4	92.9 ± 2.8

* Mean ± SD.

of our subjects did not change after the SIMT. These findings are in line with previous reports, which, similarly, did not find changes in metabolic, or cardiorespiratory responses at peak exercise after SIMT (9,12,23).

One might speculate that the oxygen cost of breathing for intense ventilatory efforts, such as during heavy muscular work, is high enough to hinder any increase in $\dot{V}O_{2max}$. It is also possible that central mechanisms were not stressed by the SIMT to the extent needed for a significant rise in $\dot{V}O_{2max}$. Furthermore, diffusion limitations across the blood gas interface and/or increased extracellular water volume, and not hypoventilation, may be the major mediator of exercise-induced arterial hypoxemia (1,8,11,14,15). It is well documented that during extremely high workload, red blood cell transit time in the pulmonary capillary could be reduced to as low as 0.2 s, time in which the diffusion of O_2 is restrained and arterial O_2 desaturation may occur (11,39).

The unexpected dissociation between the improvement of the inspiratory breathing capacity (in the training group) and enhancement of the expiratory capacity (as determined by $\dot{V}_{Emax}$ and B_R) could also be attributable to the specificity of training (7,23,27). Although respiratory muscle training was focused on the inspiratory muscles, the above mentioned ventilatory variables are predominantly expiratory and not inspiratory in nature. Thus, and as previously postulated (18), the improvement in the inspiratory muscle performance may not be instrumental in achieving higher levels of ventilation. This may be so because during very heavy exercise, at maximal lung volume, the elastic forces of the lungs are stretched to maximum and the elastic forces of the chest wall are compressed to maximum, both are exerted in an expiratory direction, in attempting to reexpand. Furthermore, because of the force-velocity relationship, the maximum inspiratory pressure that the muscles can generate falls, as flow increases (15). Howell et al. (16) have shown that uncompensated metabolic acidosis, typically present at heavy aerobic exercise, negatively affects the contractile mechanism and function of the canine's diaphragm in the face of increased metabolic demand. Hence, improving the capacity of the inspiratory muscles may paradoxically impede ventilatory response to maximal aerobic effort.

It is concluded that 10 wk of SIMT increases the respiratory muscles' capacity in highly trained athletes. This increase was not associated with an increase in aerobic capacity ($\dot{V}O_{2max}$), $\dot{V}_{Emax}$, or in arterial O_2 saturation at peak aerobic exercise. Furthermore, The SIMT, as used in this study, seems not to alleviate the respiratory constraints at peak exercise, or otherwise may suggest the need to reevaluate the B_R index (especially when using MVV_{60}), as a measure of respiratory limitation in general and in highly trained individuals in particular (24).

There are no clear answers here, but there appears to be a strong need for more work on the role of the respiratory mechanisms in limiting performance—including the interplay between respiratory muscle fatigue, muscle training, and ventilatory limitation.

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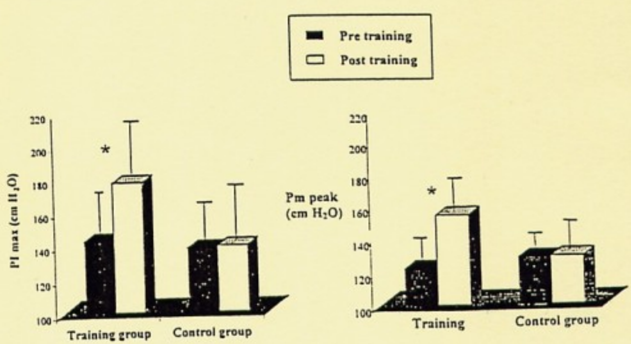

Figure 1—Effects of inspiratory muscle training on respiratory muscle strength (PI_{max}) and endurance (Pm peak): a comparison within each group. *Error bars* represent ± 1 SD; * denotes $P < 0.05$.

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