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Effect of Graded Exercise on Esophageal Motility and Gastroesophageal Reflux in Nontrained Subjects

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The effects of graded exercise on esophageal motility and gastroesophageal reflux were evaluated in nine nontrained subjects, using a catheter with three strain-gauge transducers connected to a solid-state datalogger and an ambulatory intraesophageal pH monitor. Subjects exercised on a stationary bike at 45%, 60%, 75%, and 90% of peak O_2 uptake ($\dot{V}O_2$ max). Durations of exercise sessions and rest periods varied among subjects. Studies were performed after an overnight fast and subjects received only intravenous infusion of 5% glucose solution during the study. Plasma concentrations of gastrin, motilin, glucagon, pancreatic polypeptide (PP), and vasoactive intestinal peptide (VIP) were determined at rest and before and after each exercise session. The duration, amplitude, and frequency of esophageal contractions declined with increasing exercise intensity, and the differences were significant ($P \leq 0.05$) for all three variables at 90% $\dot{V}O_2$ max. The number of gastroesophageal reflux episodes and the duration of esophageal acid exposure were significantly ($P \leq 0.05$) increased during exercise at 90% $\dot{V}O_2$ max. Plasma regulatory peptide concentrations showed no significant changes between rest and the various exercise sessions. Thus, exercise has profound effects on esophageal contractions and gastroesophageal reflux, which are intensity dependent. These effects were not mediated by the hormones measured. The results were similar to those observed in highly trained athletes, suggesting that the effects of exercise on esophageal function are similar in trained and nontrained subjects performing at similar percentages of $\dot{V}O_2$ max, even though the absolute levels of exercise achieved in each group are different.

KEY WORDS: exercise; esophagus; motility; reflux; hormones.

Physical exercise has gained increasing popularity in recent years among fitness- and health-conscious individuals. Symptoms of heartburn, belching, regurgitation, and chest pain are common during exercise (1), however, the subjects studied were all trained athletes. Exercise, particularly running, was shown to induce gastroesophageal reflux in active healthy subjects, but with different levels of training (2). We have recently shown that intense exercise can depress esophageal contractile activity and induce gastroesophageal reflux in trained athletes (3). We have also shown that intense exercise can affect intestinal motility in trained athletes (4). The effects observed in both studies were intensity dependent, and it is not

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known whether the findings are applicable to nontrained subjects who perform at lower intensities. The purpose of this investigation was to study the effect of various exercise intensities on esophageal motor activity and gastroesophageal reflux in nontrained individuals. Furthermore, because exercise can result in increased plasma concentrations of a variety of regulatory peptides, including those known to affect lower esophageal sphincter pressure (5, 6), we studied the effect of exercise on plasma concentrations of gastrin, motilin, glucagon, pancreatic polypeptide (PP), and vasoactive intestinal peptide (VIP), and compared these to the results obtained in trained athletes.

MATERIALS AND METHODS

Subjects. Nine subjects (five females, four males age range 20–43 years, median 25 years) participated in the study. They had a sedentary lifestyle, both at work and at home and were not engaged in any physical exercise, beyond that required for daily routine activities. Females were studied in the first part of their menstrual cycle. They were all healthy nonsmokers with no gastrointestinal complaints. The protocol was approved by the Human Investigations Committee and informed consent was signed by each subject.

Exercise Testing. Maximal oxygen uptake ($\dot{V}O_2$ max) was obtained by having the subjects exercise on a treadmill (Quinton Instruments; AH Rubins, Seattle, Washington) with incremental increase in speed and elevation until reaching a plateau in heart rate and $\dot{V}O_2$. Forty percent, 60%, 75%, and 90% of $\dot{V}O_2$ max were then determined. They exercised on a bicycle attached to a stationary windload simulator (Road Machine, Findley Inc., Newport, Vermont). They maintained a cadence of 80–90 rpm and different gear combinations were used to achieve the above percentage of $\dot{V}O_2$ max (7).

Motility Recording. Esophageal motility was studied by a thin (2.5-mm OD), flexible probe (Gaeltec Limited, Dunvegan, Isle of Skye, Scotland) with three pressure transducers spaced 5 cm apart. The probe was passed transnasally. The distal sensor was positioned at the upper margin of the lower esophageal sphincter (LES), which was located beforehand by a standard slow pull-through technique. The probe was connected to a small solid-state datalogger (Cavendish Automation, Huntingdon, Cambridgeshire, United Kingdom) providing each channel with 512 kilobyte recording capability. The datalogger was 11 × 18 cm and weighed 950 g. It was carried in a shoulder bag, allowing continuous recording while not interfering with the physical activity of the subjects. At the end of each study, an analog recording was obtained with a digital-to-analog converter and a Gould chart recorder.

pH Recording. Intraesophageal pH monitoring was used to evaluate gastroesophageal reflux. An antimony pH electrode (Synectics Medical) was calibrated with pH 4.0 and pH 7.0 buffers and then placed transnasally 5 cm above the upper margin of the LES, its location determined manometrically prior to placement of the pH probe. An ambulatory recorder (Synectics Medical) was used to obtain continuous pH monitoring. At the end of the study, the recorder was connected to a computer for analysis of the data.

Plasma Hormone Assays. Venous blood was drawn for all samples. It was collected in chilled EDTA-containing glass tubes with aprotinin at 500 KIU/ml of whole blood. The tubes were centrifuged at 4° C and the plasma was stored at −20° C for subsequent analysis. Plasma concentrations of motilin, gastrin, glucagon, PP, and VIP were determined by established methods (8).

Experimental Protocol. Studies lasted 5–6 hr and were performed the morning after an overnight fast. Subjects rested for an hour after being fitted with the manometry and pH probes, and this hour was considered the control period. The subjects then exercised at different intensities, and the duration of each exercise session varied according to the ability of each individual. The cadence was continuously monitored by a cyclocomputer (Cat Eye Co., Osaka, Japan) attached to the gear. The exercise sessions were separated by at least 30-min rest periods. All studies started with a 1-hr rest period, but the sequence of exercise sessions was randomized. Blood for the measurement of plasma concentrations of the various hormones was drawn at rest and before and after each exercise session. The subjects assumed a sitting position during all rest periods, and leaned forward so as to simulate the body posture during exercise. Subjects were kept fasting during the study but were given 1.5 liters of 5% glucose solution by intravenous infusion over the study period. As a result, primary contractions represent the response to dry swallows. Protocol and equipment were similar to those employed in the study of trained athletes (3), so as to make a meaningful comparison between the two studies.

Analysis. Esophageal manometric records were analyzed visually, using the last 10 min of each exercise session (or less, when subjects were not able to exercise for this length of time) and compared to the last 10 min of the control rest period. Only contractions observed at the level of the proximal and middle sensor were analyzed. Contraction amplitude was measured from the expiratory intraesophageal baseline to the peak of the contraction wave. Wave duration was measured from the start of the upstroke to the point of return to baseline. We did not use a sensor for deglutition, because of motion artifacts.

A gastroesophageal reflux episode was considered to occur when pH dropped below 4. The percent durations of pH < 4 (esophageal acid exposure time) during each exercise session and during all rest periods were assessed.

Log transformations were applied to the reflux data to obtain normal distributions (9). Results were expressed as means ± 1 SEM. Comparisons between the various sessions were made by one-way analysis of variance, using Duncan's test for multiple comparisons and also by the McNemar test for the reflux data (10), and $P \leq 0.05$ was selected as the level of significance.

TABLE 1. EFFECTS OF EXERCISE ON ESOPHAGEAL CONTRACTIONS*

Contraction	Rest	VO_2 max 40%	VO_2 max 60%	VO_2 max 75%	VO_2 max 90%
Duration (sec)	3.2 ± 0.2	2.8 ± 0.2	2.3 ± 0.1†	2.0 ± 0.1†	1.7 ± 0.3†
Amplitude (mm Hg)	51.2 ± 6.0	45.8 ± 7.4	35.7 ± 4.0	31.4 ± 3.3†	27.3 ± 4.7†
Frequency (contractions/min)	1.1 ± 0.2	1.0 ± 0.2	0.7 ± 0.2	0.4 ± 0.1†	0.2 ± 0.1†

*Values are mean ± SEM.
†$P \leq 0.05$ compared to rest.

RESULTS

Subjects were encouraged to exercise for as long as they could in each session; however, no fixed time was set, and durations varied among subjects: 40% $\dot{V}O_2$ max = range 25–60 min, median 40 min; 60% $\dot{V}O_2$ max = range 20–35 min, median 20 min; 75% $\dot{V}O_2$ max = range 5–10 min, median 10 min; 90% $\dot{V}O_2$ max = range 2–3 min, median 2 min. Resting periods between exercise sessions varied greatly between subjects, ranging from 33–143 min, with a median of 50 min.

Peak heart rate values during the four levels of exercise were significantly higher than resting values, and the four exercise sessions resulted in heart rates that were significantly different from one another (control = 80.5 ± 4.4, 40% $\dot{V}O_2$ max = 130.1 ± 5.3, 60% $\dot{V}O_2$ max = 160.2 ± 6.2, 75% $\dot{V}O_2$ max = 179.7 ± 5.3, 90% $\dot{V}O_2$ max = 194.4 ± 2.4). This confirmed that the athletes were exercising at different work loads. They experienced no gastrointestinal symptoms during the exercise session; however, one subject complained of leg cramps at the end of the study.

The effect of exercise on variables of esophageal contractions is shown in Table 1. The duration, amplitude, and frequency of contractions decreased progressively as the intensity of exercise increased, the difference being significant ($P \leq 0.05$) at the higher intensities of 75 and 90% $\dot{V}O_2$ max. The effect of exercise at 75% $\dot{V}O_2$ max on contractile activity is shown in Figure 1.

The distribution of percent durations of esophageal pH < 4 in each subject in each exercise intensity is shown in Figure 2. Because of a technical problem with the pH recorder in one subject, the data represent results from eight subjects. Mean percent duration was significantly longer during exercise at 90% $\dot{V}O_2$ max compared to rest (rest = 0.9 ± 0.5, 40% $\dot{V}O_2$ max = 13.5 ± 11.3, 60% $\dot{V}O_2$ max = 12.4 ± 11.0, 75% $\dot{V}O_2$ max = 22.9 ± 13.9, 90% $\dot{V}O_2$ max = 29.4 ± 14.6). The mean number of reflux episodes (normalized per hour) increased with increasing exercise intensity and was significantly higher at 90% $\dot{V}O_2$ max when compared to rest (rest = 0.7 ± 0.2, 40% $\dot{V}O_2$ max = 2.4 ± 1.0,

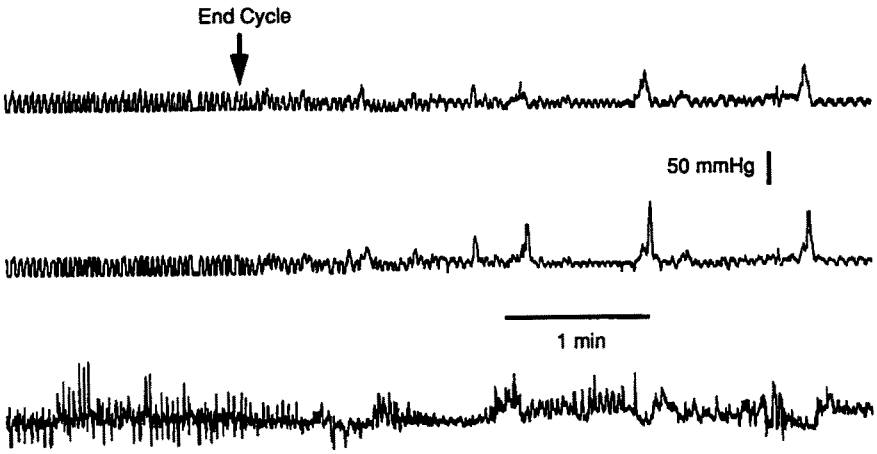

Fig 1. The final phase of exercise at 75% $\dot{V}O_2$ max. Esophageal contractions are suppressed during exercise and resume soon after it stops. The lower sensor is located at the level of the lower esophageal sphincter, and the middle and proximal sensors are located 5 and 10 cm above the LES, respectively. The exaggerated baseline represents respiratory oscillations.

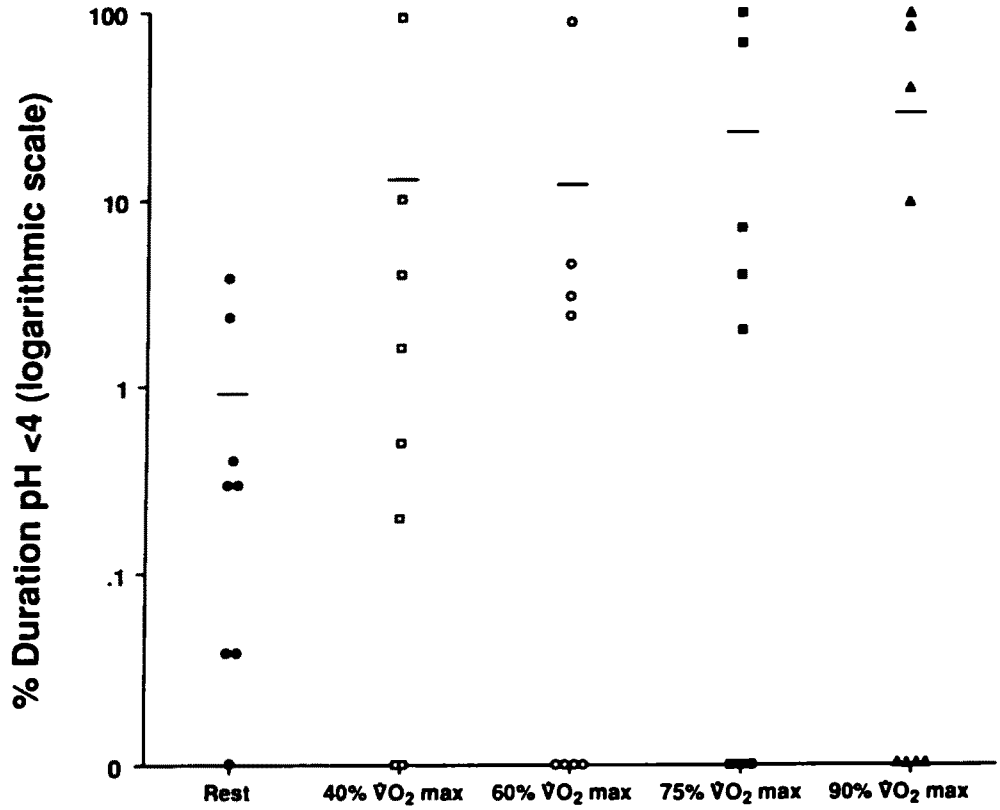

Fig 2. Distribution of percent durations of pH < 4 in each subject, in each study period. Horizontal lines represent the mean.

60% $\dot{V}O_2$ max = 2.7 ± 0.8, 75% $\dot{V}O_2$ max = 4.5 ± 1.9, 90% $\dot{V}O_2$ max = 17.5 ± 7.7).

The plasma concentrations of the various hormones were not affected by exercise (Table 2). Blood glucose levels at rest and after each exercise session were not significantly different (rest = 78.9 ± 5.8, 40% $\dot{V}O_2$ max = 76.2 ± 3.5, 60% $\dot{V}O_2$ max = 74 ± 5.1, 75% $\dot{V}O_2$ max = 81.5 ± 7.4, 90% $\dot{V}O_2$ max = 80.5 ± 8.5) (mg/100 ml).

DISCUSSION

This study shows that exercise in nontrained subjects resulted in changes in esophageal motor activity and gastroesophageal reflux that were similar to those observed in trained athletes.

In a literature review, symptoms of heartburn, chest pain, or belching associated with exercise were reported by 8–36% of individuals (1). The

TABLE 2. EFFECT OF EXERCISE ON PLASMA CONCENTRATIONS OF HORMONES*

	Hormone concentration (pmol/liter)								
		40%		*60%*		*75%*		*90%*	
Hormone	*Rest*	*B*	*A*	*B*	*A*	*B*	*A*	*B*	*A*
VIP	8.0 2.2	8.0 1.9	7.6 1.5	6.8 1.8	8.0 2.2	6.8 1.5	8.0 1.6	7.4 1.6	8.4 2.4
Motilin	42.7 11.3	25.7 6.4	29.4 5.0	25.0 4.6	24.2 2.5	28.8 8.8	31.3 6.7	32.7 7.0	40.6 7.4
Gastrin	11.7 3.5	10.6 3.6	13.2 3.9	9.8 2.3	10.9 2.7	9.9 2.5	11.9 3.4	12.5 3.6	11.2 2.8
PP	20.2 2.3	21.6 2.7	30.6 6.2	24.3 4.4	33.2 8.9	21.9 3.2	37.6 8.4	28.1 5.3	31.3 4.6
Glucagon	9.9 1.8	10.6 2.9	11.4 2.8	9.0 2.3	11.0 2.2	9.6 2.0	9.8 2.1	9.4 1.9	8.3 2.4

*Values are mean ± SEM. B, before exercise; A, after exercise. The percentages in the upper panel represent fractions of $\dot{V}O_2$ max.

participants in the various studies, however, were all highly trained athletes. In a study involving regularly exercising individuals, running was shown to induce gastroesophageal reflux (11). In a previous study we have shown that in trained cyclists the amplitude, duration, and frequency of esophageal contractions steadily decreased, while gastroesophageal reflux increased with increasing exercise intensity (3). The most profound changes in that study were seen at very high exercise intensity, 90% of $\dot{V}O_2$ max. In another study with trained cyclists, we have found that highly intense exercise, at 90% $\dot{V}O_2$ max, has a profound effect on postprandial small bowel motility (4). Can these findings be applied to nontrained individuals? If surveys on symptoms involved endurance athletes, and if studies on esophageal and small bowel motor function, performed on trained athletes, showed the gastrointestinal tract to be affected at high exercise intensity, could it be assumed that in nontrained subjects, who perform at lower levels of intensity, no changes in gut motility should be expected?

The differences between trained and nontrained subjects are not limited to differences in exercise intensities that can be achieved. Training results in cardiovascular adjustments that improve the oxygen transport capacity of the circulatory system (12). Training attenuates the hypoglycemia and catecholamine response induced by prolonged exercise (13). These adjustments may offset in part the effects of the reduction in mesenteric blood flow induced by exercise (12, 14–15). These "protective" adjustments induced by sustained exercise may imply that in nontrained individuals, who lack them, changes in gut motility could be anticipated at lower percentages of $\dot{V}O_2$ max. An ideal protocol would be to design a longitudinal study, in which nontrained subjects are studied before and after a long training period that can last weeks or months, thus each subject serves as his own control. While such studies were performed in protocols investigating exercise effects on cardiovascular and hormonal function (13, 16, 17), only scant data exist with regard to gastrointestinal function. In one such study on the effect of exercise on colonic function, colonic transit was not changed by training (18). Studies of this nature, however, are very difficult to carry out.

Comparable to the previous study in trained athletes, plasma concentrations of gastrin, motilin, PP, glucagon, and VIP were not affected by exercise (3). Since exercise can result in mild hypoglycemia (19), it is possible that the intravenous infusion of glucose during exercise prevented hypoglycemia in our subjects, which could have otherwise induced a rise in PP plasma levels (20). On the other hand, the glucose infusion did not result in elevated blood glucose levels, which could have attenuated a rise in motilin or glucagon plasma levels (21, 22). The results show that hormone changes, which in other studies may have been due, in part, to changes in blood glucose, cannot account for the motor and reflux effects induced by exercise.

In summary, in nontrained individuals intense exercise depresses esophageal contractile activity and enhances gastroesophageal reflux, findings that are comparable to those we observed in highly trained athletes. These findings suggest that with increasing intensity of exercise, gastrointestinal motor function may be similarly affected in nontrained as compared to highly trained individuals, although absolute workloads vary greatly.

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