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Effect of Graded Exercise on Esophageal Motility and Gastroesophageal Reflux in Trained Athletes

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We evaluated the effect of graded exercise on esophageal motility and gastroesophageal reflux. We studied eight trained cyclists using a catheter with three strain-gauge transducers connected to a solid-state datalogger and an ambulatory intraesophageal pH monitor. Each study lasted 4 hr during which subjects exercised on a stationary bike for 1 hr at 60% of peak O_2 uptake (O_2 max), 45 min at 75% of O_2 max, and for 10 min at 90% of O_2 max. Subjects rested 1 hr before exercise (control period) and for 30 min between exercise sessions. 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% 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% O_2 max. Plasma hormone 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 are not mediated by the hormones measured.

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

Physical exercise has become popular in recent years. Although symptoms of heartburn, belching, regurgitation, and chest pain are common during exercise (1), there is very little data concerning the effect of exercise on esophageal motor function. Running induces gastroesophageal reflux, usually associated with belching (2), more so than bicycling or weight lifting (3). One study of esophageal motility during exercise showed no change in the amplitude of contractions, but there was an increase in disordered motor activity (4). Unfortunately, the intensity of exercise was not determined. Exercise does affect intestinal motor activity, and the effect is intensity dependent (5). The purpose of this investigation was to study the effect of exercise intensity on esophageal motor activity and gastroesophageal reflux. Furthermore, because exercise can result in increased plasma concentrations of a variety of hormones, including those known to affect the lower esophageal sphincter pressure (6, 7),

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we studied the effect of exercise on plasma concentrations of gastrin, motilin, glucagon, pancreatic polypeptide (PP), and vasoactive intestinal peptide (VIP).

MATERIALS AND METHODS

Subjects. Eight trained male cyclists (age range 20–32 years, mean 24.7 years) participated in the study. All participated in regional or national races. They were all healthy nonsmokers with no gastrointestinal complaints. The protocol was approved by our Human Investigations Committee and informed consent was signed by each subject.

Exercise Testing. Peak oxygen uptake ($\dot{V}O_2$ max) was obtained by having the subjects walk on a treadmill (Quinton Instruments; AH Rubins, Seattle, Washington) with incremental increase in speed and elevation until reaching a plateau in heart rate and $\dot{V}O_2$. Sixty percent, 75%, and 90% of $\dot{V}O_2$ max were then determined. Subjects attached their own bicycle to a stationary windload simulator (Road Machine; Findley Inc., Newport, Vermont). They then maintained a cadence of 90 rpm while different gear combinations were tried to determine the workload necessary to achieve the above percentage of $\dot{V}O_2$ max (8).

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, UK) 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, which was determined manometrically. 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 trasylol 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 (9).

Experimental Protocol. Studies lasted 4 hr and were performed the morning after an overnight fast (Figure 1). Subjects rested for an hour after being fitted with the manometry and pH probes. They exercised for 60 min at 60% of $\dot{V}O_2$ max, 45 min at 75% of $\dot{V}O_2$ max and 10 min at 90% $\dot{V}O_2$ max. The cadence was continuously monitored by a cyclocomputer (Cat Eye Co., Osaka, Japan) attached to the gear. The exercise sessions were separated by 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 whole 4 hr of the study. As a result, primary contractions represent the response to dry swallows.

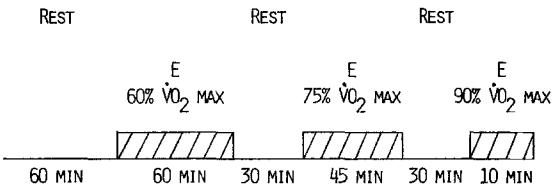

Fig 1. The experimental design. E, exercise.

Analysis. Esophageal manometric records were analyzed visually, using the last 10 min of each exercise session 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 (10). Results were expressed as means ± 1 SE. Comparisons between the various sessions were made by one-way analysis of variance. Scheffe's test for multiple comparisons was used, and $P \leq 0.05$ was selected as the level of significance.

RESULTS

Peak heart rate values during the three levels of exercise were significantly higher than the resting value, while the value at 90% $\dot{V}O_2$ max was significantly higher than those at 60% and 75% $\dot{V}O_2$ max (control rest = 64.5 ± 3.0; 60% $\dot{V}O_2$ max = 137.0 ± 6.6; 75% $\dot{V}O_2$ max = 155.2 ± 6.1; 90% $\dot{V}O_2$ max = 184.7 ± 4.1). This confirmed that the athletes were exercising at different work loads. The cyclists experienced no gastrointestinal symptoms during the exercise sessions.

TABLE 1. EFFECTS OF EXERCISE ON ESOPHAGEAL CONTRACTIONS*

Contraction	*Rest*	*60% $\dot{V}O_2$ max*	*75% $\dot{V}O_2$ max*	*90% $\dot{V}O_2$ max*
Duration (sec)	3 ± 0.2	2.5 ± 0.2	1.5 ± 0.3	1.1 ± 0.2†
Amplitude (mm/Hg)	44 ± 5.3	38.2 ± 1	26.9 ± 5.9	17.0 ± 4.9†
Frequency (contractions/min)	1.2 ± 0.2	0.7 ± 0.2	0.7 ± 0.2	0.3 ± 0.1†

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

The effect of exercise on the 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 ($P \leq 0.5$) at the highest intensity. The effect of exercise at 90% $\dot{V}O_2$ max on contractile activity is shown in Figure 2.

The distribution of percent durations of esophageal pH < 4 in each subject in each exercise intensity is shown in Figure 3. Mean percent duration was significantly longer during exercise at 90% $\dot{V}O_2$ max (rest = 0.5% ± 0.3, 60% $\dot{V}O_2$ max = 0.2% ± 0.1, 75% $\dot{V}O_2$ max = 0.9% ± 0.6, 90% $\dot{V}O_2$ max = 13.6% ± 8.2). 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 (rest = 0.4 ± 0.2, 60% $\dot{V}O_2$ max = 0.6 ± 0.4, 75% $\dot{V}O_2$ max = 1.2 ± 0.5, 90% $\dot{V}O_2$ max = 3.7 ± 1.0).

The plasma concentrations of the various hormones were not significantly changed by exercise (Table 2).

DISCUSSION

This study shows that exercise has a profound effect on esophageal motor activity and gastroesophageal reflux, and the effect is intensity dependent in both cases. The amplitude, duration, and frequency of esophageal contractions decreased steadily with increasing exercise intensity. The effect could be the result of two mechanisms.

First, exercise may induce esophageal ischemia. Visceral blood flow in humans can decrease significantly during exercise (11, 12), probably the result of the diversion of blood flow from the splanchnic viscera to the working muscles (13). The blood supply of most of the esophagus is the descending aorta, while the lowest part is supplied by the left gastric artery (14). Thus, blood flow to the organ could possibly be affected by changes in splanchnic flow induced by exercise. Second, the volume of saliva in each swallow could be reduced. Although we did not assess salivary flow, drying of the mouth is common during intense exercise. This could result in a decreased frequency of swallowing, and

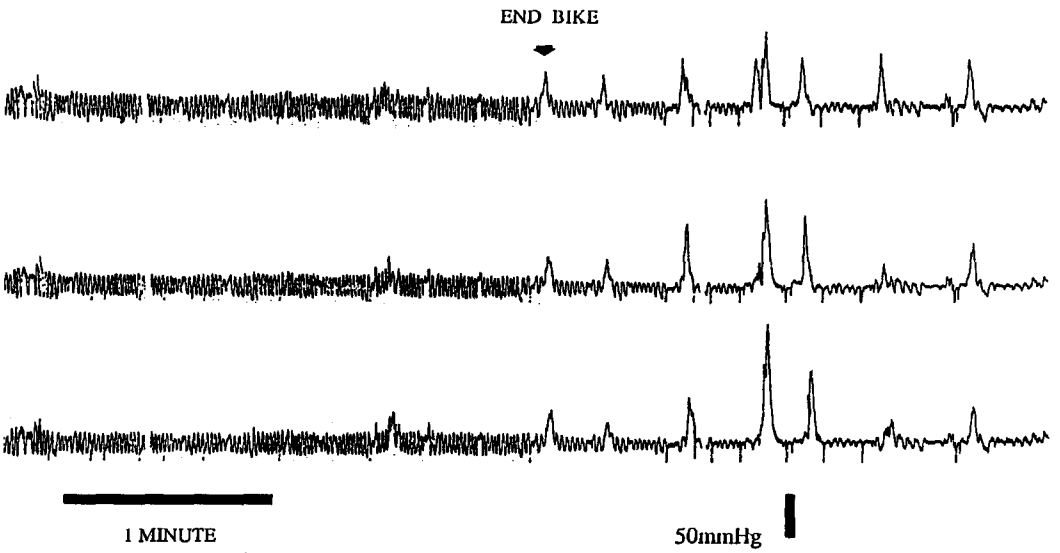

Fig 2. Exercise at 90% $\dot{V}O_2$ max for 10 min. Esophageal contractions are suppressed during exercise but resume soon after exercise stops. The exaggerated baseline represents respiratory oscillations.

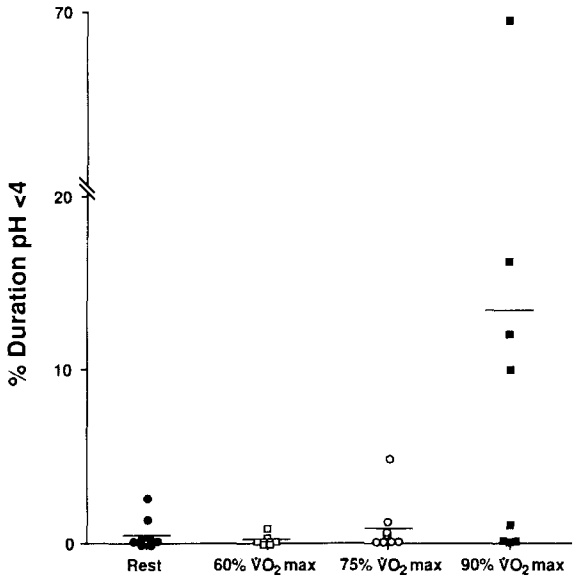

Fig 3. Distribution of percent durations of pH < 4 in each subject, in each study period. Horizontal lines represent the mean.

consequently a decrease in the frequency of contractions. Moreover, a smaller size of the salivary bolus is expected to produce contractions of lower amplitude and duration (15).

Gastroesophageal reflux episodes and esophageal acid exposure were significantly increased during exercise at the highest intensity. These effects too could be the result of a number of mechanisms.

First, the decrease in amplitude and frequency of esophageal contractions, and the possible decrease in the volume of saliva swallowed, might hamper esophageal acid clearance, which is believed to occur in two phases. First, peristaltic contractions rapidly clear fluid volume from the esophagus, following which the remaining residual acid is gradually neutralized by swallowed saliva (16, 17). With contractile activity reduced to a minimum during intense exercise, and saliva likely so, refluxed gastric acid would escape the esophagus mostly by gravity, the subjects being in an upright position, and the residual acid will be effectively neutralized when normal contractile activity and salivary flow resume at the end of the exercise. A second important antireflux factor is LES pressure, which we could not measure. LES pressure, however, can be affected by changes in the hormonal milieu and possibly by changes in visceral blood flow. A possible decrease in LES pressure could explain the increased number of reflux episodes during high-intensity exercise.

Since exercise in fasting subjects was reported to result in elevated plasma concentrations of a variety of hormones (6, 7), some with opposing effects on LES pressure, we measured plasma concentrations of gastrin, motilin, and PP, which tend to increase LES pressure, and those of glucagon and VIP, which tend to lower it (18, 19). We found no significant changes in the plasma concentrations of these hormones during any of the exercise sessions, although a potential increase in glucagon, motilin, and PP in response to exercise could have been attenuated by a possible increase in blood glucose levels induced by the intravenous infusion of a glucose solution (20–22). However, since endurance athletes routinely take carbohydrate loads prior to exercise, and also may consume carbohydrates during exercise, the protocol we used resembles the usual physiologic conditions during exercise.

Gastric emptying and gastric acid secretion can also influence gastroesophageal reflux. The effect of exercise on these two functions has been evaluated in studies that involved different techniques, different meals, and various exercise intensities. Gastric acid secretion is either unchanged or decreased, and gastric emptying of liquids is not affected by moderate exercise (70% $\dot{V}O_2$ max) (1).

In summary, we found that highly intense exercise depresses esophageal contractions and enhances gastroesophageal reflux. These effects are probably multifactorial in origin. The results also

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

	Hormone concentration (pmol/liter)						
Hormone	*Rest*	*B 60%*	*A 60%*	*B 75%*	*A 75%*	*B 90%*	*A 90%*
VIP	5.9 ± 0.9	6.5 ± 0.6	7.1 ± 1.3	6.5 ± 0.8	8.8 ± 1.1	6.9 ± 0.9	8.1 ± 0.9
Motilin	34.6 ± 6.3	26.9 ± 3.0	26.6 ± 5.5	26.6 ± 3.9	33.3 ± 10.0	27.8 ± 7.7	33.0 ± 6.9
Gastrin	16.1 ± 2.0	18.4 ± 4.1	18.8 ± 2.9	17.4 ± 2.1	16.8 ± 2.9	19.3 ± 4.1	14.6 ± 2.6
PP	25.3 ± 4.7	28.6 ± 5.2	33.3 ± 4.3	27.7 ± 5.5	34.6 ± 5.7	27.3 ± 4.9	34.1 ± 5.7
Glucagon	12.9 ± 1.3	8.8 ± 1.6	10.8 ± 1.9	7.6 ± 1.5	16.4 ± 2.0	9.8 ± 1.3	12.9 ± 2.1

*Values are mean ± SEM. B, before exercise; A, after exercise.

indicate that in exercise research exercise intensity should be a carefully controlled variable.

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