Effects of acute graded exercise on human colonic motility

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Rao, Satish S. C., Jennifer Beaty, Mindi Chamberlain, Patrick G. Lambert, and Carl Gisolfi. Effects of acute graded exercise on human colonic motility. Am. J. Physiol. 276 (Gastrointest. Liver Physiol. 39): G1221–G1226, 1999.—Whether physical exercise stimulates colonic motility is unclear. Our aim was to determine the immediate effects of graded exercise on colonic motility. Colonic motility was recorded at six sites in 11 untrained subjects, by colonoscopically placing a solid-state probe. Subjects were free to ambulate. The next day, subjects exercised on a bicycle at 25, 50, and 75% of peak oxygen uptake for 15 min, with each followed by a 15-min rest. Motor patterns, motility indexes, and regional variations before, during exercise, during rest, and during postexercise periods were compared. During exercise, there was an intensity-dependent decrease (P < 0.001) in the number and area under the curve of pressure waves. The incidence of propagated or simultaneous pressure waves and cyclical events also decreased (P < 0.05). After exercise, the pressure activity reverted to baseline, but the number and amplitude of propagated waves increased (P < 0.01), whereas the simultaneous waves and cyclical events remained lower. Acute graded exercise decreases colonic phasic activity. This may offer less resistance to colonic flow, whereas the postexercise increase in propagated activity may enhance colonic propulsion.

ABDOMINAL CRAMPS AND diarrhea are reported more commonly in runners (19, 25), whereas constipation is reported more commonly in sedentary subjects (10, 27). These observations suggest that exercise may stimulate gut and colonic motor activity and facilitate defecation, but there is only scant evidence to support this notion, and the underlying mechanisms are not known.

In one human study, exercise stimulated postprandial motor activity in the stomach and small bowel, and this effect was related to the intensity of exercise (4, 18). In contrast, it has been shown that exercise decreased antral contractility (3) and mouth-to-cecum transit time (17). In untrained dogs, exercise also decreased jejunal myoelectric activity (14).

With regard to colonic motility, there has been only one human study that showed an increase in the 4–9 cycle/min pressure activity and a reduction in the 1–4 cycle/min activity (5). However, this study had several limitations: an athletic group of subjects was studied as opposed to normal subjects; a limited number of sensors were used; the intensity of exercise was not quantified; and a short colonic segment, i.e., only the rectosigmoid region, was examined. From this study, it was unclear whether colonic pressure activity increased or whether there was merely a shift in the frequency of pressure waves. Also, whether the pressure activity was propagated or nonpropagated was not clear. In contrast, physical exercise has been shown to induce giant migrating contractions in the dog's colon, which was associated with defecation in the postprandial period but not in the fasting state (9).

The net effect of colonic motility is to regulate stool transport, but the effects of exercise on colon transit are also controversial. Previous studies have shown that colonic transit was either unaffected (6) or significantly enhanced (8, 15, 20). Thus, there is limited, incomplete, and conflicting information regarding the effects of exercise on normal colonic motor activity in humans. Some of these discrepancies may be due to methodological issues, such as the duration and intensity of exercise and the method of recording colonic motor activity. Hence, the aim of our study was to investigate the effects of acute aerobic exercise, at different intensities, on colonic motor activity in healthy humans under more physiological conditions.

METHODS

Subjects. Twelve healthy volunteers [6 males, 6 females, mean age (range) 37 yr (23–55 yr)] were recruited for this study. All subjects gave written informed consent, and the study protocol was approved by the human subjects ethical committee. The volunteers had no previous history of gastrointestinal surgery, were not taking any medications, and had normal physical examinations. These subjects were recruited through hospital advertisement. None of these subjects was a trained athlete. They reported normal bowel habits with a stool frequency of 3–7/wk.

Experimental design. One week before the exercise study, for each subject we determined the peak maximal oxygen uptake (V\textsubscript{O\textsubscript{2}} peak) during cycle ergometry (The Bike; Cybex, Ronkonkoma, NY). A progressive protocol was used, and, based on the submaximal oxygen uptakes for each subject, the exercise intensities for 25, 50, and 75% V\textsubscript{O\textsubscript{2}} peak were determined. Expired gases and ventilatory rates were measured with a Quinton Q-Plex I Metabolic System (Seattle, WA).

Subsequently, at 7:00 AM, subjects were admitted to the Clinical Research Center and received a tap water enema. At 7:30 AM, a flexible, 6-mm-diameter manometry probe with six strain gauge microtransducers (Gaeltec Limited, Isle of Skye, UK) was placed in the colon, with the help of a colonoscope, such that the tip of the probe was located in the midtransverse colon. The technique has been described in detail previously (23). The probe was taped securely to the gluteal region. Immediately after probe placement and again after completion of the study, the location of each sensor was checked under fluoroscopy. The pressure sensors were approximately located at 7, 14, 25, 35, 45, and 60 cm from the anus. The total radiation exposure did not exceed 1,144 μrad/sentinel. The probe was connected to a 8-mHz, multiple-
channel portable recorder (MPR-7; Gaeltec Limited) that was placed and carried in a shoulder bag. The subjects were free to ambulate and to return to their room in the Clinical Research Center. Pilot studies were performed to determine whether a treadmill or a stationary bicycle provided a more optimal recording. We found that our subjects found the bicycle exercise easier, and there was less movement artifact on the recording.

The following morning, the location of each microtransducer was checked under fluoroscopy. The subjects were then escorted to the cardiovascular rehabilitation center. At 7:45 AM, while fasting, the subjects performed 15-min bouts of exercise at 25, 50, and 75% peak VO2, which alternated with 15-min periods of rest (Fig. 1). Subjects were exercised on a stationary cycle ergometer. The pulse rate and blood pressure were recorded every 5 min throughout the exercise test and for the 15-min period before (control) and after (recovery) exercise. After exercise, sensor location was checked again under fluoroscopy. If the probe migrated >10 cm, the subject was excluded.

Measurements and analyses. The motor activity was analyzed at each pressure channel and for each 15-min period, beginning with a preexercise control period. Thereafter, the pressure activity was analyzed for each level of exercise intensity, i.e., 25, 50, or 75% VO2 peak, and for each rest period. After the 75% VO2 peak rest period, we measured motor activity for the next 30 min (recovery). These data are presented as recovery 1 (first 15-min period) and recovery 2 (second 15-min period). Exercise produced both movement and respiratory artifacts. The software program was unable to consistently distinguish these artifacts from pressure waves. Hence, the pressure activity was displayed on a monitor at variable speeds (range: 30 s/page to 4 min/page) and at variable gain settings to identify the individual colonic pressure waves from those induced by bodily or diaphragmatic movements. Only pressure waves with an amplitude $\geq 8$ mmHg and duration $\geq 3$ s were included in the study (23).

The following parameters were measured: the number of pressure waves, the amplitude of pressure waves, the area under the curve of each pressure wave, and the changes in the proximal and distal three channels.

The patterns of motor activity were assessed independently by one investigator who was unaware of the results of other motor parameters. The following patterns were recognized: 1) isolated pressure waves that occurred randomly; 2) propagated pressure waves that were defined as pressure waves that propagated aborad across three or more consecutive channels, with a velocity of propagation between 0.5 and 2.4 cm/s (Fig. 2); 3) simultaneous pressure waves defined as waves that occurred simultaneously in three or more consecutive channels.

Fig. 1. Schematic diagram of the study protocol. VO2 max, maximal oxygen uptake.

Fig. 2. Typical example of motility changes after exercise. Left: movement artifacts can be seen during exercise at 75% VO2 peak; right: rest period, 2 high-amplitude propagating pressure waves (p1–p4) and a propagating cyclical event (p4–p5) can be seen after exercise. D. colon, descending colon; R. sigmoid, rectosigmoid.
tive channels with a phase lag of <1 s between channels; and 4) cyclic activity defined as a burst of phasic pressure waves at a frequency of 2–4 waves/min that last for at least 3 min (Fig. 2) and occurred in one or more channels. The number of cyclical events and their duration were measured.

Statistics. The statistical differences between the exercise and recovery period and between the preexercise, exercise, and postexercise periods for the number of waves, the area under the curve, and mean amplitude were compared by using multifactorial analysis of variance. These data are expressed as means ± 95% confidence interval. The differences in the incidence of wave patterns and the duration of cyclical activity were compared by applying a nonparametric test (Freidman's test). Because the incidence of wave patterns was not normally distributed, the data are expressed as median with the 25th and 75th percentiles.

RESULTS

Subjects. All subjects completed the protocol satisfactorily. However, data from one female subject were excluded because the probe had migrated significantly. Thus data from 11 subjects (6 males/5 females) were analyzed. No adverse events were noted.

Effects of exercise on cardiopulmonary function. Compared with either the control period or the recovery period, heart rate and respiratory rate increased during exercise (P < 0.05; Table 1). Also, during exercise, the systolic blood pressure increased but was significantly higher (P < 0.05) only at 75% of VO₂ peak when compared with the control period. Diastolic blood pressure did not change significantly (Table 1).

Effects of exercise on colonic manometry. The number of pressure waves decreased (P < 0.001) during each exercise period compared with its corresponding rest period (Table 2). Furthermore, during each intensity of exercise, the number of pressure waves was less (P < 0.05) than those of the control (preexercise) period or the recovery (postexercise) period. The area under the curve of pressure waves was lower (P < 0.001) during each exercise period compared with either its corresponding rest period or the control or recovery periods (Fig. 3). There was also an intensity-dependent decrease (P < 0.05) in the area under the curve of pressure waves (Table 2).

Effect of exercise on regional variation in colonic motility. During exercise, the area under the curve of pressure waves was lower (P < 0.05) in the proximal (transverse/descending colon) than the distal (rectosigmoid) colon, but, during the control and recovery periods, there was no difference between the two regions (Table 2).

Effect of exercise on colonic motor patterns. Although all subjects exhibited propagating pressure waves during the control and recovery periods, fewer subjects showed these pressure waves during exercise (Fig. 4). At 25% VO₂ peak 6 of 11 (55%) subjects, at 50% VO₂ peak 0 of 11 (0%) subjects, and at 75% VO₂ peak 3 of 11 (27%) subjects showed propagating pressure waves. The median number of propagating waves decreased (P < 0.001) during each level of exercise compared with the

<table>
<thead>
<tr>
<th>Table 1. Effects of graded exercise on cardiopulmonary function</th>
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<tbody>
<tr>
<td><strong>Pulse rate/min</strong></td>
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<td></td>
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<tr>
<td><strong>Respiratory rate/min</strong></td>
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<tr>
<td><strong>Systolic pressure, mmHg</strong></td>
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<tr>
<td><strong>Diastolic pressure, mmHg</strong></td>
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</table>

Values are means ± 95% confidence interval. VO₂, rate of oxygen uptake. Repeated-measures ANOVA: *P < 0.05, exercise vs. rest; †P < 0.05, control vs. exercise; and ‡P < 0.05, recovery vs. control.
control period (Table 3). During recovery, the median number of propagating waves was higher compared with exercise ($P < 0.001$) or control ($P < 0.05$) periods. During the second part of the recovery period, the amplitude of propagating pressure waves was significantly greater ($P < 0.001$) than that observed during exercise or the control period (Table 3).

Similarly, all subjects exhibited simultaneous waves during the control and recovery periods, but during exercise at 25% $V\dot{O}_2$ peak 6 of 11 (55%) subjects, at 50% $V\dot{O}_2$ peak 6 of 11 (55%) subjects, and at 75% $V\dot{O}_2$ peak 1 of 11 (9%) subjects showed simultaneous waves. The median number of simultaneous pressure waves was lower ($P < 0.05$) during exercise at 75% $V\dot{O}_2$ peak and during recovery compared with the control period (Table 3). No difference was observed between exercise and rest. During exercise at 75% $V\dot{O}_2$ peak, the number of isolated waves decreased ($P < 0.05$) when compared with the control period (Table 3).

All subjects showed runs of cyclical activity varying between 2 and 4 waves/min, both during the control and recovery periods. During exercise, at 25% $V\dot{O}_2$ peak 9 of 11 (82%) subjects, at 50% $V\dot{O}_2$ peak 5 of 11 (45%) subjects, and at 75% $V\dot{O}_2$ peak 2 of 11 (18%) subjects showed cyclical activity. Thus there was an intensity-dependent decrease ($P < 0.05$) in cyclical events. The median duration of cyclical activity was less ($P < 0.05$) during exercise at 50 and 75% $V\dot{O}_2$ peak compared with the control period (Table 3), but there was no difference between exercise and the corresponding rest periods (Table 3).

**DISCUSSION**

There is conflicting information regarding the effects of exercise on colonic motor function (5, 19, 27). This could be in part due to technical and methodological limitations that include colonic cleansing immediately before recording motility, the use of limited sensors, short-segment and short-duration recordings, the performance of studies in athletic individuals (5) rather than normal untrained subjects, and the use of transit markers such as red dye (6). To better understand the changes in colonic motor function, here, we examined the effects of acute graded aerobic exercise in healthy, untrained individuals. Also, our subjects were tested 24 h after probe placement, which possibly allowed sufficient time for the colonic milieu to return to the basal state.

Contrary to popular belief (5, 9), the data suggest that exercise decreases colonic motility. Both the incidence of pressure waves and the area under the curve of pressure waves were significantly lower during exercise. Moreover, a higher intensity of exercise was associated with a greater reduction in motor activity. During rest, the motor activity recovered promptly. These findings differ significantly from those reported previously (5). In the study by Cheskin et al. (5), an

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**Table 2. Effects of acute graded exercise on colonic manometric measurements**

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>25% $V\dot{O}_2$</th>
<th>Rest</th>
<th>50% $V\dot{O}_2$</th>
<th>Rest</th>
<th>75% $V\dot{O}_2$</th>
<th>Rest</th>
<th>Recovery 1</th>
<th>Recovery 2</th>
</tr>
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<tbody>
<tr>
<td>No. of waves</td>
<td>21 ± 6</td>
<td>7 ± 4†</td>
<td>21 ± 7</td>
<td>3 ± 3†</td>
<td>23 ± 8</td>
<td>1.3 ± 2†</td>
<td>19 ± 7</td>
<td>39 ± 13‡</td>
<td>40 ± 13‡</td>
</tr>
<tr>
<td>Mean amplitude, mmHg</td>
<td>20 ± 4</td>
<td>32 ± 15†</td>
<td>20 ± 5</td>
<td>21 ± 22</td>
<td>21 ± 5</td>
<td>12 ± 17</td>
<td>20 ± 5</td>
<td>19 ± 5</td>
<td>23 ± 8</td>
</tr>
<tr>
<td>AUC $\times 10^2$, mmHg²</td>
<td>43 ± 15</td>
<td>8 ± 5†</td>
<td>58 ± 22</td>
<td>4 ± 3†</td>
<td>60 ± 33</td>
<td>0.2 ± 0.2†</td>
<td>38 ± 22</td>
<td>40 ± 24</td>
<td>45 ± 22</td>
</tr>
<tr>
<td>Proximal AUC $\times 10^2$, mmHg²</td>
<td>43 ± 22</td>
<td>7 ± 4†</td>
<td>57 ± 24</td>
<td>2 ± 2†</td>
<td>61 ± 31</td>
<td>0.1 ± 1†</td>
<td>34 ± 25</td>
<td>32 ± 29</td>
<td>40 ± 23</td>
</tr>
<tr>
<td>Distal AUC $\times 10^2$, mmHg²</td>
<td>44 ± 17</td>
<td>8 ± 4†</td>
<td>59 ± 27</td>
<td>7 ± 4†</td>
<td>60 ± 35</td>
<td>0.4 ± 2†</td>
<td>41 ± 26</td>
<td>47 ± 19</td>
<td>50 ± 19</td>
</tr>
<tr>
<td>AUC, area under the curve</td>
<td></td>
<td></td>
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</table>

Values are means ± 95% confidence interval. AUC, area under the curve. Repeated-measures ANOVA: *$P < 0.05$, exercise vs. rest; †$P < 0.05$, exercise vs. control; and ‡$P < 0.05$, control vs. recovery.

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**Fig. 4. Effects of exercise on the incidence of propagating pressure waves (median + 75th percentile).**
increased incidence of 4–9 cycles/min was reported. However, participants of this study were well-trained athletic individuals, 60% of whom reported loose stools with exercise. Also, the increased motor activity was essentially seen in those who reported diarrhea. Hence, their findings may apply to a select group of individuals with exercise-induced diarrhea and not necessarily to all individuals who exercise. Also, these changes may be a consequence of prolonged physical conditioning. In contrast, during exercise, we not only found a reduction in pressure waves but also a reduction in the incidence of individual patterns of pressure waves. We did not observe cyclical events that exceeded a frequency of 4 waves/min. Also, the number of cyclical events and duration of cyclical activity decreased during exercise.

In dogs, it has been reported that exercise stimulated colonic activity by increasing the incidence of giant migrating contractions (9). However, during exercise, we did not identify giant migrating contractions in the human colon. In contrast, during exercise, we found that the incidence of propagating and simultaneous pressure waves and that of cyclical events decreased. However, immediately after exercise there was a significant increase in the number of propagating pressure waves and in the amplitude of these pressure waves. This unique increase in the propagating pressure waves during the postexercise period may be a consequence of increased parasympathetic drive to the gut (8), but the underlying mechanism remains to be elucidated.

Exercise increases heart rate, respiratory rate, and systolic blood pressure. These effects are due to the activation of the sympathetic nervous system and the release of catecholamines (1, 11, 12, 16). In the gut, sympathetic stimulation inhibits smooth muscle phasic activity (13). This concurs with our finding of decreased colonic motor activity during exercise. In addition to the autonomic effects of exercise on the gut, blood is usually shunted away from the digestive tract toward the skeletal muscles and toward the skin (26). The reduced blood supply may further inhibit gut function. After exercise, the sympathetic drive ceases, and blood flow is restored. These physiological changes may reestablish colonic motor function, as confirmed in our study.

None of our subjects reported cramps or experienced an urge to defecate within 1 h after exercise. Whether these symptoms can be induced by higher intensities of exercise or with prolonged exercise remains unclear. We did not examine the effects of a higher intensity of exercise such as 90% \( \dot{V}O_2 \) peak or a more prolonged duration of exercise, i.e., >15 min, because in pilot studies we found that not all of our untrained subjects could comply with the higher intensity or the prolonged duration of exercise.

Because the colon exhibits regional variation (24) and several complex motor patterns (21), we performed a qualitative analysis of colonic motor activity that included a separate assessment of the pressure activity in the proximal and the distal three channels. This analysis revealed that the transverse/descending colon showed less activity during exercise than the rectosigmoid region. Also, during the recovery period, the pressure activity in the rectosigmoid region reverted to control values, whereas in the transverse/descending colon there was prolonged inhibition of motor activity. The reduced incidence of phasic pressure waves together with a higher incidence of propagating waves may facilitate a greater movement of stool in the transverse/descending colon.

Our study, using ambulatory recording of colonic motility, showed that acute graded aerobic exercise did not increase but in fact decreased phasic colonic motor activity. However, after exercise there was a preferential increase in the number and amplitude of propagating pressure waves and a decrease in the simultaneous waves and cyclical events. It is possible that the reduced phasic activity may offer less resistance to colonic flow (7, 22), whereas the increased propagating activity may propel stool.

We thank Amber Ludlow, Renea Kavelock, and Kristopher Acker-son for superb technical assistance. We are grateful to Bridget Zimmerman for help and advice with statistical analysis. We also acknowledge the support of Dr. Ellen Gordon and Patti Lounsbury from the cardiovascular rehabilitation center for allowing us to use their facilities, Karen Johnson for help with the exercise tests, and Susie McConnell for typing the manuscript.

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Table 3. Effects of graded exercise on colonic motor patterns

<table>
<thead>
<tr>
<th>No. of propagating waves</th>
<th>Amplitude of propagating waves, mmHg</th>
<th>No. of simultaneous waves</th>
<th>No. of isolated waves</th>
<th>No. of cyclical events</th>
<th>Duration of cyclical events, min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>2.5 (2–4)</td>
<td>72 (56–91)</td>
<td>2 (0–3)</td>
<td>1 (0–2)</td>
<td>8 (4–14)</td>
</tr>
<tr>
<td>25% ( \dot{V}O_2 )</td>
<td>1.0 (0–1)†</td>
<td>71 (55–92)</td>
<td>1.0 (0–2)</td>
<td>1 (0–2)</td>
<td>5 (2–10)</td>
</tr>
<tr>
<td>Rest</td>
<td>1.0 (1–4)†</td>
<td>85 (71–97)</td>
<td>1 (0–2)</td>
<td>0.5 (0–2)</td>
<td>6 (3–6)</td>
</tr>
<tr>
<td>50% ( \dot{V}O_2 )</td>
<td>0.0 (0–1)†</td>
<td>58 (0–63)</td>
<td>1 (0–1)</td>
<td>0.5 (0–2)</td>
<td>1 (0–6)†</td>
</tr>
<tr>
<td>75% ( \dot{V}O_2 )</td>
<td>0 (0–1)†</td>
<td>88 (79–109)</td>
<td>1 (0–1)</td>
<td>0 (0–2)</td>
<td>2 (0–9)</td>
</tr>
<tr>
<td>Rest</td>
<td>1.0 (0–3)†</td>
<td>0 (0–64)†</td>
<td>1 (0–2)</td>
<td>0 (0–1)†</td>
<td>0 (0–1)†</td>
</tr>
<tr>
<td>Recovery 1</td>
<td>1.0 (0–3)†</td>
<td>81 (0–93)</td>
<td>1 (0–2)</td>
<td>1 (0–2)†</td>
<td>0 (0–2)†</td>
</tr>
<tr>
<td>Recovery 2</td>
<td>3 (0–3)</td>
<td>77 (38–99)</td>
<td>0.5 (0–1)†</td>
<td>1 (0–2)†</td>
<td>0.5 (0–1)†</td>
</tr>
</tbody>
</table>

Values are medians with 25th–75th percentiles in parentheses. Friedman's test: *P < 0.05, exercise vs. rest; †P < 0.05, exercise vs. control; and ‡P < 0.05, control vs. recovery.
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