Effects of meal ingestion on blood pressure and regional hemodynamic responses after exercise

Masako Yamaoka Endo, Chizuko Fujihara, Akira Miura, Hideaki Kashima, and Yoshiyuki Fukuba

Department of Health Sciences, Prefectural University of Hiroshima, Hiroshima, Japan

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Endo MY, Fujihara C, Miura A, Kashima H, Fukuba Y. Effects of meal ingestion on blood pressure and regional hemodynamic responses after exercise. J Appl Physiol 120: 1343–1348, 2016. First published February 25, 2016; doi:10.1152/japplphysiol.00842.2015.—This study investigated the combined effects of consuming a meal during postexercise hypotension (PEH) on hemodynamics. Nine healthy young male subjects performed each of three trials in random order: 1) cycling at 50% of heart rate reserve for 60 min, 2) oral ingestion of a carbohydrate liquid meal (75 g glucose), or 3) carbohydrate ingestion at 40 min after cycling exercise. Blood pressure, heart rate, cardiac output, and blood flow in the superior mesenteric (SMA), brachial, and popliteal arteries were measured continuously before and after each trial. Regional vascular conductance (VC) was calculated as blood flow/mean arterial pressure. Blood pressure decreased relative to baseline values (P < 0.05) after exercise cessation. Blood flow and VC in the calf and arm increased after exercise, whereas blood flow and VC in the SMA did not. Blood pressure did not change after meal ingestion; however, blood flow and VC significantly decreased in the brachial and popliteal arteries and increased in the SMA for 120 min after the meal (P < 0.05). When the meal was ingested during PEH, blood pressure decreased below PEH levels and remained decreased for 40 min before returning to postexercise levels. The sustained increase in blood flow and VC in the limbs after exercise was reduced to baseline resting levels immediately after the meal, postprandial cardiac output was unchanged by the increased blood flow in the SMA, and total VC and SMA VC increased. Healthy young subjects can suppress severe hypotension by vasoconstriction of the limbs even when carbohydrate is ingested during PEH.

postexercise hypotension; postprandial hypotension; hemodynamics; blood pressure regulation

NEW & NOTEWORTHY

No study demonstrated the hemodynamic responses to meal ingestion during postexercise hypotension. This study investigated the combined effects of consuming a meal during postexercise hypotension on central and peripheral hemodynamics. Our results suggested that healthy subjects can suppress severe hypotension by vasoconstriction of the limbs even when a meal is ingested during postexercise hypotension. Our findings provide novel insight into the timing of exercise for the management of postprandial blood pressure in some individuals.

In most individuals, blood pressure decreases after aerobic exercise; this phenomenon is known as postexercise hypotension (PEH) (13, 14, 24). Blood pressure is also lowered by meal ingestion in some individuals (11, 21, 25). Therefore, a postexercise meal may enhance blood pressure reduction. However, it is unknown whether blood pressure is reduced when a meal is ingested during the period of hypotension after exercise. Blood pressure is determined by cardiac output and total vascular conductance (the sum of regional vascular conductances), but the simultaneous central and peripheral hemodynamic responses to a postexercise meal have not been reported.

In healthy individuals, an acute period of dynamic exercise elicits a reduction in blood pressure relative to preexercise levels for ~2 h (13, 14, 24). This PEH provides the basis for the antihypertensive effect of regular exercise training, especially in mildly hypertensive patients (24). However, it is possible that PEH may predispose patients to severe orthostatic hypotension and syncope after exercise (2, 15). Previous studies have suggested that PEH is induced by an increase in the total vascular conductance but not in cardiac output (6). This elevated total vascular conductance was mainly attributed to the increased vascular conductance of the skeletal muscles in exercised and nonexercised limbs, rather than to splanchnic and renal vasodilation (6, 27).

Postprandial hypotension (PPH) occurs frequently in older adults and those with autonomic nervous system disorders and diabetes, and it is now recognized as an important clinical problem (11, 21, 25). The pathophysiological mechanisms underlying PPH remain poorly defined although an increase in splanchnic blood pooling appears to be an important initial event (11, 21, 25). Thus PPH may result from an increase in total vascular conductance via vasodilation of the vessels supplying the gastrointestinal tract. However, the magnitude of the increase in mesenteric blood flow after meal ingestion is comparable in young and older subjects despite the lower blood pressure in older adults (17, 29). In healthy young subjects, meal ingestion results in increased vasoconstriction in the calf (30) and an increase in cardiac output attributable to increased heart rate that prevents a significant decrease in blood pressure (17). By contrast, in older subjects with PPH, inadequate cardiovascular responses fail to maintain blood pressure after eating (17, 22).

PEH is induced by vasodilation of the skeletal muscles in both the exercised and nonexercised limbs. Thus, if a meal is ingested during PEH, severe hypotension may occur because of additional gastrointestinal vasodilation and a subsequent rise in total vascular conductance. To our knowledge, no studies have examined the effect of meal ingestion on the central and peripheral hemodynamic responses during PEH. We hypothesized that a postexercise meal would exacerbate the reduction in blood pressure that occurs after exercise because of insufficient cardiovascular adjustment. Thus the aim of this study was to evaluate the influence of oral carbohydrate ingestion on central, appendicular, and small intestinal hemodynamic responses during PEH in healthy young individuals.
MATERIALS AND METHODS

Subjects. Nine healthy, normotensive, nonsmoking male volunteers participated in this study. All gave their written informed consent before beginning the trial. The study was approved by the institutional ethics committee and was conducted in accordance with the guidelines in the Declaration of Helsinki. No subjects had a history of gastrointestinal disease or surgery, cardiovascular or metabolic disorders, or obesity, and none were taking medication. The participants’ characteristics are presented in Table 1.

Protocols. The participants arrived at the laboratory after having abstained from caffeine and exercise for at least 1 day. During their initial exercise session, the participants performed continuous graded cycle ergometer exercise (232CXL; Combi, Tokyo, Japan) at three different work rates (30, 60, and 90 W) for 5 min each at 60 revolution/min, followed by a 2-min rest in an upright position. Heart rate was monitored via a three-lead electrocardiogram (ECG) (BP-88S; Colin, Tokyo, Japan) during this incremental cycling exercise. The linear relationship between steady-state heart rate at the end of each stage and work rate was calculated, and the work rate corresponding to a heart rate of 50% of heart rate reserve, calculated as 0.5 × [(220 – age) – RHR] + RHR, where RHR is the resting heart rate, was estimated. This workload (88 ± 29 W) was used for the 60-min exercise in the main protocol.

The participants performed three different trials in random order and separated by at least 7 days. Each trial began at 0830 AM after an overnight fast. For the Meal and the Ex trials, subjects rested in a supine position for 30 min and then either ingested an oral carbohydrate liquid meal (Meal; partial hydrolysate of starch; 75 g glucose in a volume of 225 ml, this is the standard solution used for the oral glucose tolerance test; Trelan-G75g; Ajinomoto Pharmaceutical, Tokyo, Japan) or performed 60 min of upright cycling exercise (Ex). The participants then returned to a supine position just after completing the Meal (within 2 min) or Ex and remained in that position for 120 min. In the third trial, the subjects rested in a supine position for 30 min and then performed 60 min of upright cycling exercise. They were then repositioned supine for 160 min, and during this time they ingested a carbohydrate liquid meal at 40 min after the end of the cycling exercise (Ex + Meal). During all trials, the room temperature was maintained at 24 ± 1°C by a thermal feedback device.

Measurements. Heart rate was continuously monitored via a three-lead ECG throughout each protocol. Blood pressure was measured using an autonomic manometer (BP-306; Colin) with a left arm cuff every 10 min throughout the protocols, except during the 60-min cycling exercise. Blood pressure during exercise was measured using a mercury manometer at 20 and 50 min after the start of exercise with the left arm at heart level. Beat-to-beat stroke volume was calculated from continuously recorded beat-to-beat blood pressure measurements obtained using a Finometer finger blood pressure cuff (Finometer Pro; Finapres Medical Systems, Amsterdam, The Netherlands) placed on the right middle finger; blood pressure measurements were used to calculate stroke volume by the model-flow method (4, 16, 31). Cardiac output was calculated as stroke volume × heart rate every 10 min throughout the protocol, except during the cycling exercise. Total vascular conductance was calculated as cardiac output/mean arterial pressure.

Blood flow measurements in the superior mesenteric artery (SMA), right brachial artery, and right popliteal artery were obtained using simultaneous pulsed and echo Doppler ultrasound (LOGIQ6s; GE Healthcare, Tokyo, Japan) to measure mean blood velocity and arterial blood vessel diameter every 10 min throughout the protocols, except during the cycling exercise. The mean velocity of the blood within each vessel was obtained on a beat-by-beat basis; for the SMA, a convex 3.3-MHz probe was positioned at a depth of 2 cm from the aorta by the anterior abdominal approach, and a linear 5.0-MHz probe was positioned at >3 cm proximal to the right oelectralon process for the brachial artery and at the right popliteal fossa for the popliteal artery with an insonation angle of 45-60°. The mean blood velocity in the SMA was measured during breath holding at the spontaneous end-expiration for 20 s, as previously described (6). Mean blood velocities were always measured by the same experimenter in identical locations to minimize measurement error. The diameter of each vessel was measured simultaneously using an imaging frequency of 5.5 MHz for the SMA and 12.0 MHz for the brachial and popliteal arteries. Blood flow in each vessel was calculated from the mean blood velocity and cross-sectional area of the vessel. Blood flows were always measured in the same order (SMA, then brachial artery, then popliteal artery) with an interval of ~10 min between repeated measurements. The vascular conductance of each vessel was calculated as the ratio of blood flow to mean arterial pressure.

Statistical analysis. To test the significance of time-serial changes in each variable, the effect of time on the variables was examined by repeated-measures ANOVA. When a significant F value was detected, Dunnett’s post hoc test was used to perform multiple comparisons. The effects of each protocol on the measured hemodynamic parameters were examined by two-way ANOVA. Data are expressed as means ± SD in the table and as means ± SE in the text and in the figures. Differences were considered statistically significant if P < 0.05 (SPSS 12.0; IBM, Chicago, IL).

RESULTS

Postexercise hemodynamic responses. Systolic and mean blood pressure significantly increased during exercise relative to preexercise levels (P < 0.05), whereas diastolic blood pressure during exercise did not change from the preexercise level. Heart rate significantly increased from 58 ± 3 beats/min before exercise to 135 ± 4 beats/min at 50 min into the exercise protocol (P < 0.05). Mean arterial pressure consistently decreased in all subjects from ~30 min to 80 min after exercise cessation (Fig. 1). Heart rate and cardiac output remained elevated until ~30 min and 20 min after the cessation of exercise, respectively, and then returned to preexercise levels (Fig. 1). Total vascular conductance was significantly increased until 40 min after exercise cessation (Fig. 1). Blood flow and vascular conductance in the calf and arm remained elevated throughout the recovery period relative to preexercise levels (Figs. 2 and 3). Blood flow and vascular conductance in the small intestine did not change after exercise (Figs. 2 and 3).

Hemodynamic responses postcarbohydrate ingestion. During the 120 min immediately after oral carbohydrate ingestion, the mean arterial pressure, heart rate, cardiac output, and total vascular conductance did not change from preingestion levels (Fig. 1). Blood flow and vascular conductance in the arm significantly decreased just after carbohydrate ingestion; this decrease was maintained for the full 120-min measurement period (Figs. 2 and 3). Blood flow and vascular conductance in the calf significantly decreased from 90 min to 120 min after

Table 1. Participant characteristics

<table>
<thead>
<tr>
<th>Participant Characteristics</th>
<th>Value</th>
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<tr>
<td>Age, yr</td>
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<tr>
<td>Height, m</td>
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<tr>
<td>Weight, kg</td>
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<td>59 ± 5</td>
</tr>
<tr>
<td>Resting heart rate, beats/min</td>
<td>58 ± 10</td>
</tr>
</tbody>
</table>

All values are means ± SD; n = 9.
carbohydrate ingestion (Figs. 2 and 3). In contrast, blood flow and vascular conductance in the small intestine remained increased for the full 120-min postcarbohydrate ingestion measurement period (Figs. 2 and 3).

Comparison of postexercise and postcarbohydrate ingestion hemodynamic responses. The mean arterial pressure, heart rate, and cardiac output responses to oral carbohydrate ingestion were significantly different than the responses of these parameters to exercise ($P < 0.05$). There was a trend toward a significant difference between the Meal and Ex protocols in total vascular conductance after the intervention ($P = 0.067$). Blood flow and vascular conductance in the leg and arm were significantly lower after oral carbohydrate ingestion relative to postexercise values; however, blood flow and vascular conduc-

tance in the small intestine were both significantly greater after ingestion of oral carbohydrate ($P < 0.05$).

Carbohydrate ingestion during PEH. Blood pressure and heart rate responses during exercise in the Ex + Meal trial were similar to those obtained during exercise in the Ex trial. In addition, after exercise the mean arterial pressure, cardiac output, heart rate, total vascular response, individual vascular responses, and blood flow in the small intestine, calf, and arm were similar in the Ex + Meal trial to the responses seen in the Ex trial for the first 40 min after exercise, until just before oral carbohydrate ingestion (Figs. 1–3).

When carbohydrate was ingested during PEH, the mean arterial pressure decreased and remained low for 40 min after ingestion (Fig. 1). The time course of the changes in mean arterial pressure was similar to that seen in the Ex trial. Heart rate significantly increased throughout the protocol after carbohydrate ingestion; thus cardiac output tended to be higher than during the rest period before exercise (Fig. 1). Total vascular conductance significantly increased after oral carbohydrate ingestion and remained higher throughout the measurement period (Fig. 1). This increased total vascular conductance was due to the increase in the vascular conductance of the small intestine (Fig. 2). Concomitantly, the sustained increase in the vascular conductance and blood flow in the limbs seen during PEH.
thought to be predominantly derived from increased vascular conductance via vasodilation in both exercised and nonexercised skeletal muscles (13, 14, 24). Previous studies have shown that vascular conductance in the splanchnic and renal arteries did not change after moderate cycling exercise (6, 27). These data support our finding that the vascular conductance in the calf and arm remained greater than resting levels for 1.5 h after exercise, whereas vascular conductance in the small intestine did not change after exercise (Figs. 1 and 2).

PPH is an important clinical problem and is associated with syncope, falls, and even coronary events, stroke, and cerebrovascular accidents (11, 21, 25). The magnitude of the reduction in blood pressure after a meal is affected by many factors including meal composition, volume, temperature, body posture, and medications (11, 17, 21, 25). Of the macronutrients, carbohydrate has been shown to induce a greater decrease in postprandial blood pressure than protein, fat, or water (18, 36). In particular, glucose and sucrose have a more pronounced effect on blood pressure than do fructose and xylose (5, 19, 35). Blood flow in the SMA following oral and intraduodenal carbohydrate and fat ingestion has been reported to be greater than after protein ingestion, and the increase in SMA blood occurs more rapidly following carbohydrate ingestion than after fat ingestion (10, 28, 29). Therefore, the degree of PPH may relate to the magnitude of the postprandial increase in splanchnic blood flow although there are some contrasting reports in the literature (10, 28, 29).

The magnitude of the postprandial increase in SMA blood flow is comparable in healthy young and older individuals despite a greater fall in blood pressure in older subjects (29). In healthy young individuals, consumption of a meal is associated with a rapid rise in heart rate; this baroreflex may prevent a significant postprandial fall in blood pressure by increasing cardiac output (11, 17). It has been previously reported that the reduction in blood pressure after a liquid meal resulted from increased total vascular conductance via SMA vasodilation, which was partially offset by a concomitant increase in cardiac output and heart rate after a meal in healthy subjects (26). However, in the present study, blood pressure was maintained mainly by vasoconstriction in the arm, whereas heart rate and cardiac output did not increase after carbohydrate ingestion (Figs. 1 and 2). A novel finding of our study was that powerful vasoconstriction after a meal occurred in the arm, but not the calf, which helped to maintain blood pressure in healthy young subjects. It has been previously shown in young subjects that blood flow in the calf was reduced for 60 min after a high-fat meal, but only for 15 min after a high-carbohydrate meal (29).

In the present study, changes in blood flow in the popliteal artery after oral carbohydrate ingestion were similar to the changes in vascular conductance (i.e., there was a significant decrease at 90–120 min after ingestion; Fig. 3). The difference between our findings and those of previous work may be partly attributed to the use of different methods for measurement of blood flow. Our present study used Doppler ultrasonography, which has been shown to be a valid and accurate method for noninvasive measurement of blood flow in a variety of vessels, whereas previous studies used venous occlusion plethysmography.

In some individuals, acute dynamic exercise or meal ingestion can elicit a reduction in blood pressure via changes in central and regional hemodynamic responses. However, sur-

**Fig. 3. Blood flow in the small intestine, leg, and arm at baseline (resting value) and after exercise (●), carbohydrate ingestion (○), and postexercise carbohydrate ingestion (▲) (n = 9). Values are means ± SE. The dashed line indicates the time of carbohydrate ingestion in the Ex + Meal trial. #P < 0.05 vs. baseline value in the Ex trial. +P < 0.05 vs. baseline value in the Meal trial. *P < 0.05 vs. baseline value in the Ex + Meal trial.**

**DISCUSSION**

The aim of this study was to determine the central and peripheral hemodynamic responses to meal ingestion during PEH. Our major findings were that the blood pressure response to oral carbohydrate ingestion after exercise was similar to that after exercise only and that a postexercise meal immediately induced an increase in small intestinal vasodilation and blood flow while simultaneously decreasing blood flow in the calf and arm via vasoconstriction. These data suggest that healthy young subjects can suppress severe hypotension by vasoconstriction in the exercised and nonexercised limbs, even if carbohydrate is ingested during PEH.

A single exercise training session results in an acute reduction in blood pressure (23). Thus the antihypertensive effects of PEH may be relevant to the management of hypertensive patients (14). In fact, the antihypertensive effects of exercise in such patients can last more than half a day, longer than their duration of 1–3 h in healthy young individuals. In the majority of individuals, PEH is caused by an increase in total vascular conductance that is not fully offset by a rise in cardiac output (13, 14, 24). This increased total vascular conductance is...
prisingly little is known about these central and regional hemodynamic effects on systemic blood pressure. Thus, for patients with PPH and individuals presenting symptoms of orthostatic intolerance after exercise, it is important to understand the precise mechanism(s) by which a postexercise meal can affect blood pressure. We hypothesized that carbohydrate ingestion during PEH would result in lower blood pressure than after exercise alone because of increased vascular conductance in the SMA and a subsequent rise in total vascular conductance. However, contrary to our hypothesis, we found that a postexercise carbohydrate meal did not affect total vascular conductance. Rather, a concomitant constriction of the vasodilated vascular beds in the arm and calf prevented a fall in blood pressure attributable to SMA vasodilation. One limitation of our study is that our cardiac output and total vascular conductance results were obtained from stroke volumes that were estimated using a Finapres finger manometer and model-flow methods. However, these methods have been validated in previous studies for the estimation of stroke volume at rest and during exercise (4, 16, 31), and we believe that the changes in blood pressure and peripheral hemodynamics that we report are reliable results.

Vasconstriction in the limbs after oral carbohydrate ingestion is thought to be related to sympathetic vasoconstrictor nerve activity in skeletal muscle (1, 3, 7). This is supported by previous findings that muscle sympathetic nerve activity (MSNA) was significantly greater following carbohydrate ingestion than after fat and protein ingestion (7). Furthermore, the increase in MSNA after oral glucose ingestion has been reported to be blunted in older adults (8). Thus a blunted MSNA may result in a failure of vasoconstriction of the limb vessels during meal ingestion and may be responsible for severe hypotension during meal ingestion after exercise in older individuals and in patients with autonomic failure. Indeed, older adults showed no change in skeletal muscle vascular resistance after meal ingestion (22). The present study showed that vasodilation in the limbs after exercise lasted for at least 1.5 h, suggesting that older individuals should not eat a meal for several hours after exercise. This needs further investigation in older subjects and PPH patients.

Although we did not determine the mechanism underlying the skeletal muscle vasoconstriction that occurred with a postexercise meal, this vasoconstriction is likely associated with selective sympathetic vasoconstrictor nerve activity. In the regulation of postprandial blood pressure, the roles of an attenuated baroreflex (11, 17), an attenuated reflex increase in sympathetic activity by activation of stretch receptors in the stomach (gastrovascular reflex) (9, 20, 33), sympathetic dysfunction (8, 34), decreased cardiac output (17), nitric oxide (12, 32), and various vasoactive peptides released from the small intestine (32) have been evaluated. Nevertheless, the mechanism of PPH remains unresolved. Although we used young healthy subjects, we believe that our findings also provide novel insight into the management of postprandial blood pressure in patients with PPH through appropriate timing of exercise.

GRANTS

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

Author contributions: M.Y.E. conception and design of research; M.Y.E. performed experiments; M.Y.E. and C.F. analyzed data; M.Y.E., A.M., and H.K. interpreted results of experiments; M.Y.E. prepared figures; M.Y.E. drafted manuscript; M.Y.E. edited and revised manuscript; M.Y.E. and Y.F. approved final version of manuscript.

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