The Alpha (α)-Glucosidase Inhibitor, Acarbose, Attenuates the Blood Pressure and Splanchnic Blood Flow Responses to Intraduodenal Sucrose in Older Adults

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Background. Postprandial hypotension is an important problem in the elderly and may be triggered by the increase in splanchnic blood flow induced by a meal. Acarbose attenuates the fall in blood pressure (BP) induced by oral sucrose and may be useful in the management of postprandial hypotension. It is not known whether the effect of acarbose on postprandial BP reflects slowing of gastric emptying and/or carbohydrate absorption nor whether acarbose affects splanchnic blood flow. We examined the effects of intraduodenal (ID) acarbose on the BP, heart rate, superior mesenteric artery (SMA) flow, and glycemic and insulin responses to ID sucrose in older participants—this approach excluded any "gastric" effect of acarbose.

Methods. Eight healthy participants (four male and four female, age 66–77 years) received an ID infusion of sucrose (~6 kcal/min), with or without acarbose (100 mg), over 60 minutes. BP, heart rate, SMA flow, blood glucose, and serum insulin were measured.

Results. Acarbose markedly attenuated the falls in systolic (p < .01) and diastolic (p < .05) BP and rises in heart rate (p < .05), SMA flow (p < .05), blood glucose (p < .01), and serum insulin (p < .05). The maximum fall in systolic BP and peak SMA flow was inversely related on the control day (r² = −.53, p < .05) but not with acarbose (r² = .03, p = .70).

Conclusions. We conclude that in healthy older participants receiving ID sucrose, (a) acarbose markedly attenuates the hypotensive response by slowing carbohydrate absorption and attenuating the rise in splanchnic blood flow and (b) the fall in BP is related to the concomitant increase in SMA flow.

Key Words: Blood pressure—Elderly—Postprandial hypotension—Superior mesenteric artery flow—Ultrasound.

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Postprandial hypotension is a common and important problem, particularly in the elderly and patients with autonomic dysfunction (1) for which current management is suboptimal (1). The underlying mechanisms are poorly defined (1), but meal composition (1,2), the rate of small intestinal nutrient delivery and absorption (3–7), gastric distension (8), splanchnic blood flow (1), and neural and hormonal mechanisms (1) appear important.

The alpha (α)-glucosidase inhibitor, acarbose, is used frequently in the management of type 2 diabetes to reduce postprandial glycemia (9). It has been assumed that the latter effect reflects a delay in small intestinal carbohydrate absorption (9), but studies conducted by ourselves (10) and others (11,12) have demonstrated that acarbose may slow gastric emptying, which is a major determinant of the glycemic response to carbohydrate (13,14), substantially.

Acarbose also stimulates the secretion of the “incretin” hormone, glucagon-like peptide-1 (GLP-1) (15), and this may contribute to a reduction in glycemia by stimulating insulin (16), suppressing glucagon (17), as well as by slowing gastric emptying (18). A number of studies (10,19–23) have established that acarbose attenuates the postprandial hypotensive response to carbohydrate meals in patients with (19–23) and without (10) postprandial hypotension. We reported in healthy older participants that acarbose (100 mg) markedly attenuated the fall in systolic blood pressure (BP) induced by oral sucrose (10). More recently, Jian and colleagues (19) reported that acarbose (50 mg) attenuated the postprandial fall in BP after a semiliquid “meal” in a cohort of 43 older patients with postprandial hypotension. There are also three case reports suggesting that acarbose is beneficial in patients with symptomatic postprandial hypotension.
associated with type 1 (20) and type 2 (21,23) diabetes and evidence that another α-glucosidase inhibitor, voglibose, is also effective (24). The effect of acarbose on postprandial BP may relate to prolongation of gastric distension and a reduction in small intestinal nutrient delivery as a result of retardation of gastric emptying (25,26) and/or slowing of carbohydrate absorption as a result of inhibition of carbohydrate digestion (3,6,7). To allow discrimination between these potential mechanisms, it would be necessary to infuse carbohydrate directly into the small intestine, with and without acarbose, and thereby exclude any gastric effect. A substantial effect of acarbose in these circumstances would indicate that inhibition of carbohydrate absorption represents the dominant mechanism.

Following a meal, there is a considerable increase in splanchnic blood volume with an approximate doubling of superior mesenteric artery (SMA) flow (1). Although the magnitude of the postprandial increase in mesenteric blood flow is comparable in healthy young and older individuals, the latter experience a fall in BP, indicative of inadequate cardiovascular adjustment (27). Somewhat surprisingly, the relationship between the postprandial fall in BP with splanchnic blood flow has hitherto not been assessed. The beneficial effects of acarbose on the hypotensive response to carbohydrate may, accordingly, reflect changes in the splanchnic blood flow response to enteral glucose. This possibility has also not been evaluated.

The primary aims of this study were to determine in healthy older participants the effects of intraduodenal (ID) acarbose on the BP, heart rate (HR), and SMA flow responses to an ID sucrose infusion and the relationship between changes in BP and SMA flow. The broad hypotheses were (a) that acarbose would attenuate the fall in BP and rise in HR induced by ID sucrose by slowing small intestinal carbohydrate absorption and, thereby, also attenuating the rise in SMA flow and (b) the magnitude of the fall in BP and rise in SMA flow induced by ID sucrose would be related directly.

**Materials and Methods**

**Participants**

Eight healthy older participants, (four male and four female) with a median age of 70 years (range: 66–77 years) and body mass index of 24.6 kg/m² (range: 20.1–28.7 kg/m²), recruited by advertisement, were studied. Based on a mean difference in the maximum fall in systolic BP after oral sucrose, with and without acarbose [4.7 ± 5.7 mmHg (SD)] in our previous study (10), a sample size of eight participants with 80% power and α < .05 was required to detect a difference between treatments of 6.6 mmHg. All participants were nonsmokers. None had a history of gastrointestinal disease or surgery; diabetes; significant respiratory, renal, hepatic, or cardiac disease; chronic alcohol abuse; or epilepsy or were taking medication known to influence BP or gastrointestinal function.

We have reported the effects of acarbose on the hypotensive response to 300 mL water containing 100 g sucrose in eight healthy older participants (five male and three female with a median age of 71 years, range 65–79 years) (10). Data in this group were compared with those obtained in the current study.

The protocol was approved by the Human Research Ethics Committee of the Royal Adelaide Hospital, and each participant provided written informed consent prior to their inclusion. All experiments were carried out in accordance with the Declaration of Helsinki.

**Protocol**

Each participant was studied on two occasions, separated by 3–21 days. On each day, participants attended the University of Adelaide, Discipline of Medicine at the Royal Adelaide Hospital at 0830 hours following an overnight fast (10.5 hours for solids and 8.5 hours for liquids) (2,28). At that time, a silicone-rubber catheter (external diameter ~4 mm) (Dentsleeve International Ltd, Mui Scientific, Mississauga, Canada) was introduced into the stomach via an anesthetized nostril (2,28). The assembly included an infusion channel (internal diameter ~1 mm) and was positioned so that the infusion port was located ~10 cm distal to the pylorus (ie, in the duodenum) as well as two other channels that were positioned in the antrum (2.5 cm proximal to the pylorus) and duodenum (2.5 cm distal to the pylorus), respectively, and were perfused with 0.9% saline. The correct positioning of the catheter was maintained by continuous measurement of the transmucosal potential difference from the antral (~40 mV) and duodenal (0 mV) channels (2,28,29). For this purpose, a cannula filled with sterile saline was placed subcutaneously in the left forearm and used as a reference electrode (2,28,29). The tip of the catheter passed into the duodenum by peristalsis, which took between 10 and 180 minutes. Once intubated, the participant rested in the recumbent position. An automated BP cuff (23–33 cm Adult Sensa-cuf, Critikon BP Cuffs, GE Medical Systems, Milwaukee, WI) was placed around the right arm for measurements of BP and HR (2,28). Approximately 30 minutes after the tube was positioned correctly (ie, at t = 0 minutes), an ID infusion of 100 g sucrose dissolved in saline (0.9%) in a total volume of 300 mL was commenced and maintained at a rate of 5 mL/min for 60 minutes (ie, ~6 kcal/min). On one of the two days, 100 mg acarbose (Bayer Australia Ltd, Pymble, Australia) was added to the ID infusion in randomized double-blind order. ID infusions were performed using a volumetric infusion pump (Gemini PC-1; IMED Corp, San Diego, CA). BP (systolic and diastolic), HR, and SMA flow were measured for 60 minutes. At t = 60 minutes, the catheter was removed. On one day, cardiovascular autonomic nerve function was evaluated.
immediately after the completion of the study (30,31). On both study days, participants were given a light meal prior to leaving the laboratory.

**Measurements**

**BP and HR.**—BP (systolic and diastolic) and HR were measured using an automated oscillometric BP monitor at \( t = -9, -6, \) and \(-3\) minutes prior to commencement of the ID infusions and, subsequently, every 3 minutes between \( t = 0 \) and 60 minutes (2,10,28). “Baseline” BP and HR, that is, \( t = 0\) minutes, were calculated as the mean of measurements taken at \( t = -9, -6, \) and \(-3\) minutes prior to the commencement of the ID infusion. Postprandial hypotension was defined as a fall in systolic BP of \( \geq 20 \) mmHg that was sustained for at least 30 minutes (1).

**SMA flow.**—SMA flow was measured by Duplex ultrasonography (ie, B-mode and Doppler imaging) using a Logiq 9 ultrasonography system (GE Healthcare Technologies, Sydney, Australia) as described previously (2,32). Each participant was scanned using a 3.5C broad spectrum 2.5–4 MHz convex transducer (2,32) at \( t = -2\) minutes and then at 15-minute intervals between \( t = 0 \) and 60 minutes. Blood flow (milliliters per minute) was calculated immediately using the formula: \( \pi \times r^2 \times \text{TAMV} \times 60 \), where \( r = \) the radius of the SMA and TAMV is the time-averaged mean velocity (32).

**Blood glucose and serum insulin concentrations.**—Venous blood samples (~7.5 mL) were obtained prior to the commencement of the ID infusion (ie, \( t = -2\) minutes) and at 15-minute intervals between \( t = 0 \) and 60 minutes (10,28). Blood glucose concentrations were determined immediately using a portable blood glucose meter (Medisense Precision Q·I·D System, Abbott Laboratories, Medisense Products Inc, Bedford, MA) (10,28). Blood samples for serum insulin were collected in ice-chilled serum tubes with clotting activator and stored at \(-70^\circ\)C for subsequent analysis. Insulin concentrations were measured by enzyme-linked immunosorbent assay immunoassay (Diagnostics Systems Laboratories Inc, Webster, TX) (10). Sensitivity was 0.26 mU/L, intraassay coefficient of variation was 2.6%, and interassay coefficient of variation was 6.2% (6).

**Autonomic function.**—Autonomic nerve function was assessed using standardized cardiovascular reflex tests (30,31). Parasympathetic function was evaluated by the variation (R–R interval) of the HR during deep breathing and the HR response to standing (30:15 ratio). Sympathetic function was assessed by the fall in systolic BP in response to standing. Each of the test results were scored according to age-adjusted predefined criteria as 0 = normal, 1 = borderline, and 2 = abnormal for a total maximum score of 6. A score \( \geq 3 \) was considered to indicate autonomic dysfunction (30,31).

**Statistical analysis.**—Data were evaluated using repeated measures two-way analysis of variance, with “treatment” and “time” as within-subject factors. Systolic and diastolic BP and HR were analyzed as changes from baseline. SMA flow and blood glucose and serum insulin concentrations were analyzed as absolute values. Data were analyzed from \( t = 0 \) to 60 minutes (systolic and diastolic BP and HR) and \( t = -2 \) to 60 minutes (SMA flow, blood glucose, and serum insulin concentrations) to determine the effects (“treatment” and “time”) of sucrose and acarbose. One-way analysis of variance was used to analyze the effects of “time” on systolic and diastolic BP, HR, SMA flow, blood glucose, and serum insulin concentrations. In all analyses, contrasts were used to examine point-by-point comparisons to test preplanned hypotheses of interest. In the event that the analysis of variance demonstrated a statistically significant “Treatment \times Time” interaction (both treatments in the same model), post-hoc tests were used to examine point-by-point comparisons between treatments with Bonferroni correction for multiple comparisons. The maximum falls in systolic and diastolic BP and rises in HR, blood glucose, and serum insulin concentrations were defined as the greatest mean changes from baseline in each participant at any given time point for each treatment. Relationships between variables were assessed using linear regression analysis. Systolic BP in the first 60 minutes after ID sucrose, without acarbose, was compared with those observed in the first 60 minutes after oral sucrose without acarbose (10). All analyses were performed using Statview (version 5.0; Abacus Concepts, Berkeley, CA) and SuperANOVA (version 1.11, Abacus Concepts). Data are presented as mean values ± standard error of the mean. A \( p \) value < .05 was considered significant in all analyses.

**Results**

The studies were well tolerated. Looseness of stools and/or flatulence was reported by all participants during (two participants between \( t = 30 \) and 53 minutes) or after (8 participants from \( t = 60 \) minutes) acarbose infusion. In all cases, the symptoms were mild and had resolved within 11 hours. No participant volunteered gastrointestinal symptoms during the control infusion. No participant had definite autonomic neuropathy; median score 0.6 (range: 0–2). No participant had experienced postprandial hypotension on the control day or after acarbose.

**Baseline BP and HR**

There was no significant difference in baseline (ie, \( t = 0 \) minutes) BP or HR between the study days (control vs acarbose): systolic BP (124.3 ± 6.2 mmHg vs 125.9 ± 8.3...
mmHg; \( p = .59 \), diastolic BP (70.3 ± 2.5 mmHg vs 72.0 ± 3.4 mmHg; \( p = .39 \)), and HR (58.1 ± 3.0 beats per minute vs 57.0 ± 2.8 beats per minute; \( p = .42 \)).

Systolic BP.—Between \( t = 0 \) and 60 minutes, there was a fall in systolic BP after control (\( p < .0005 \)) but not after acarbose (\( p = .35 \); Figure 1a). Systolic BP fell promptly during control, and the maximum decrease was 11.2 ± 2.0 mmHg. In seven of the eight participants, the maximum fall in systolic BP was less during ID infusion of acarbose (\( p < .05 \); Figure 2). There was a significant “treatment × Time” interaction for systolic BP (\( p < .0001 \)) between the study days. At \( t = 60 \) minutes, there was no difference in systolic BP from baseline after control (\( p = .39 \)) or acarbose (\( p = .49 \)).

When compared with our previous study (10), overall systolic BP was lower after ID than after oral sucrose both without acarbose (\( p < .05 \)) and with acarbose (\( p < .05 \); Figure 3).

Diastolic BP.—Between \( t = 0 \) and 60 minutes, there was a fall in diastolic BP during both control (\( p < .0001 \)) and acarbose (\( p < .05 \); Figure 1b). The maximum fall in diastolic BP was greater (\( p < .05 \)) on the control day (10.9 ± 0.9 mmHg) compared with after acarbose (8.1 ± 1.5 mmHg). There was a significant “Treatment × Time” interaction for diastolic BP (\( p < .0001 \)) between the study days. At \( t = 24 \) minutes and between \( t = 36 \) and 60 minutes, diastolic BP was less (\( p < .05 \)) on the control day when compared with acarbose. At \( t = 60 \) minutes, diastolic BP was lower than baseline after control (\( p < .01 \)) but not after acarbose (\( p = .46 \)).

HR.—Between \( t = 0 \) and 60 minutes, there was rise in HR during both control (\( p < .0001 \)) and acarbose (\( p < .0001 \); Figure 1c). The maximum increase in HR was greater
(p = .002) on the control day (18.8 ± 1.8 beats per minute) compared with after acarbose (10.2 ± 1.7 beats per minute). There was a significant “Treatment × Time” interaction for HR (p < .0001) between the study days. At t = 21 minutes and between t = 33 and 60 minutes, HR was greater (p < .05) on the control day compared with acarbose. At t = 60 minutes, HR was greater than baseline after control (p < .0005) and acarbose (p < .001).

SMA Flow
There was no difference in baseline (ie, t = −2 minutes) SMA flow between the 2 days (control vs acarbose: 886 ± 72 ml/min vs 898 ± 88 ml/min; p = .88; Figure 4). Between t = −2 and 60 minutes, there was a rise in SMA flow during both control (p < .0001) and acarbose (p < .001), which was evident from t = 15 minutes during both control (p < .001) and acarbose (p < .05). Peak SMA flow was 3,000 ± 383 mL/min at 43 ± 5.0 minutes and 1,782 ± 225 mL/min at 56 ± 3.0 minutes (p < .001) for control and acarbose days, respectively. There was a significant “Treatment × Time” interaction (p < .0001) for SMA flow. Between t = 30 and 60 minutes, SMA flow was greater (p < .0005) on the control day compared with acarbose. At t = 60 minutes, SMA flow was greater than baseline after both control (p < .0001) and acarbose (p < .01).

Blood Glucose and Serum Insulin Concentrations
There was no difference in baseline (ie, t = −2 minutes) blood glucose or serum insulin between the two days (control vs acarbose): blood glucose (5.9 ± 0.2 mmol/L vs 5.8 ± 0.2 mmol/L; P = .67; Figure 5a) and serum insulin (9 ± 0.9 mU/L vs 10 ± 1.3 mU/L; p = .29; Figure 5b).

Between t = −2 and 60 minutes, there was a rise in blood glucose from baseline during control (p < .0001) and acarbose (p < .001), which was significant from t = 15 minutes during control (p < .001) and from t = 30 minutes during acarbose (p < .005). Peak blood glucose concentrations were 10.5 ± 0.7 mmol/L at 49 ± 5.0 minutes and 7.0 ± 0.2 mmol/L at 45 ± 5.0 minutes (p < .0005) for control and acarbose days, respectively. There was a significant “Treatment × Time” interaction (p < .0001) for blood glucose concentrations. Between t = 15 and 60 minutes, blood glucose concentrations were much greater (p < .05) on the control day compared with acarbose. At t = 60 minutes, blood glucose concentrations were greater than baseline after both control (p < .0005) and acarbose (p < .01).

Between t = −2 and 60 minutes, there was a rise in serum insulin from baseline during control and acarbose (p < .0001 for both), which was significant from t = 15 minutes during control (p < .0005) and from t = 30 minutes during acarbose (p < .01). Peak serum insulin concentrations were 130 ± 2.8 mU/L at 60.0 ± 0.0 minutes and 31 ± 5.3 mU/L at 52.5 ± 4.0 minutes (p < .005) for control and acarbose days, respectively. There was a significant “Treatment × Time” interaction (p < .0001) for serum insulin concentrations. Between t = 30 and 60 minutes, serum insulin concentrations were much greater (p < .0005) on the control day compared with acarbose. At t = 60 minutes, serum insulin concentrations were greater than baseline after both control (p < .005) and acarbose (p < .05).

Relationships Between Systolic BP With SMA Flow
There was no significant relationship between the maximum falls in systolic BP on the control day and with acarbose
There was a significant relationship between the maximum fall in systolic BP and peak SMA flow ($r^2 = -0.53, p < .05$) on the control day (Figure 6a) but not with acarbose ($r^2 = 0.03, p = .70$; Figure 6b).

**DISCUSSION**

Current management of symptomatic postprandial hypotension is less than optimal. Although strategies involving the use of somatostatin analogues, such as octreotide (33), caffeine (34), modifications to medications (1), and dietary permutations (35), have been reported to attenuate postprandial falls in BP, these are all associated with undesirable side effects, inconsistent efficacy, and/or high cost. Recent observations indicate that slowing the rate of small intestinal nutrient delivery and absorption may represent a novel and potentially effective approach to the treatment of postprandial hypotension (3–7). Consistent with this concept, oral ingestion of acarbose, which slows gastric emptying (10–12) and delays small intestinal absorption of carbohydrate (9), markedly attenuates the fall in postprandial BP in healthy older participants (10) and patients with postprandial hypotension (19–23).

In the current study, sucrose and acarbose were infused directly into the small intestine so that potential “gastric” effects of acarbose were eliminated, and we found that acarbose markedly attenuates the hypotensive (systolic and diastolic) response to ID sucrose—there was no significant fall in BP during acarbose infusion. This is despite the fact that sucrose was infused at a rate modestly greater than the normal range of gastric emptying of glucose, that is, 1–4 kcal/min (36). Given the recent use of acarbose and other α-glucosidase inhibitors in the management of postprandial hypotension (19,22,24), these mechanistic insights are important.

In considering the potential mechanisms underlying the effect of acarbose on BP, the concomitant changes in SMA flow are of considerable interest. On the control study, there was an inverse relationship between the magnitude of the hypertensive response and the rise in SMA flow. Although the demonstration of a relationship does not establish a causal association, that is, that the fall in systemic BP represents a response to the increase in splanchnic blood flow and further studies are required, this is to our knowledge the first time that changes in another “cardiovascular” parameter have been shown to account for the postprandial fall in BP. On the control day, a rise in SMA flow was evident at 15 minutes and a further increase at 30 minutes. That the decrease in systolic BP after 15 minutes was minimal may reflect that measurements of SMA flow were performed only at 15-minute intervals and the peak response in SMA flow may have occurred just after 15 minutes. Moreover, HR increased progressively. There was virtually no change in SMA flow after acarbose, consistent with the concept that increases in splanchnic blood flow are of central importance. The effect of acarbose on SMA flow may potentially reflect the delay in carbohydrate absorption and consequent reduction in the serum insulin response. However, although insulin has vasodilatory properties (1), both glucose and insulin are unlikely to play a major role in postprandial hypotension, given that intravenous glucose has little, if any, effect on BP (1,37) and postprandial hypotension occurs in type 1 patients who are insulin deficient (1,37). An alternative, and not mutually exclusive, possibility is the substantial stimulation of gut peptides by acarbose, particularly GLP-1 and glucagon-like peptide-2 (GLP-2) (38). Voglibose has been shown to inhibit the postprandial neurotensin response (24,39), and there is evidence that calcitonin gene–related peptide may be important (40) in postprandial hypotension. A role for all these hormones remains to be established. In the current study, differences in SMA flow were evident within 15 minutes arguing against a role for GLP-1 or GLP-2, given that their initial secretion after nutrient ingestion is modest (41).
Although the dose of acarbose (100 mg) is used therapeutically, oral acarbose would not be expected to be as effective, and this warrants evaluation. As discussed, the rate of sucrose infusion (−6 kcal/min) was supraphysiological (36), and it would be of interest to evaluate the effects of other sucrose loads. We have shown that the hypotensive response to small intestinal carbohydrate is dependent on the load and not on its concentration (28). In our previous study using oral acarbose, the mean rate of gastric emptying of sucrose (100 g) on placebo was 2.1 ± 0.2 kcal/min in healthy older participants (10). It is accordingly predictable, given that the protective effects of gastric distension (25) were “bypassed” and the rate of duodenal sucrose delivery was greater, that the fall in BP after ID, than oral, sucrose were greater both with and without acarbose. There is, however, evidence that the relationship between the hypotensive response to small intestinal glucose and the glucose load is nonlinear so that a specific “threshold” of duodenal glucose delivery needs to be exceeded to elicit a fall in BP, that is, in healthy older participants, the falls in systolic BP induced by ID infusions of 2 and 3 kcal/min are comparable, whereas an infusion at the rate of 1 kcal/min has no effect (42).

In conclusion, our observations establish, in healthy older participants, that (a) acarbose markedly attenuates the hypotensive response to ID sucrose by slowing carbohydrate absorption and thereby attenuating the rise in splanchnic blood flow and (b) there is a direct relationship between the hypotensive response to small intestinal sucrose and the rise in SMA flow. These observations support the use of acarbose, and presumably other α-glucosidase inhibitors such as voglibose and miglitol, in the management of postprandial hypotension. Studies to evaluate the effects of chronic administration in patients with postprandial hypotension are required.

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