Editorial

Postprandial Hypotension—The Ultimate Big Mac Attack

John E. Morley

Division of Geriatric Medicine, Saint Louis University, and Geriatric Research, Education and Clinical Center, St. Louis VA Medical Center, Missouri.

It is now well recognized that when older persons eat a meal, they may have a dramatic drop in their blood pressure (1,2). This fall in blood pressure has been associated with syncope and falls (3,4). Other effects of postprandial hypotension include dizziness, fatigue, angina pectoris, stroke, and myocardial infarction (5,6). It does not, however, reduce the response to rehabilitation in frail older persons (7).

Postprandial hypotension occurs more commonly in certain groups of patients. These include persons with autonomic dysfunction such as is seen in multisystem atrophy (8), diabetes mellitus (9), Parkinson’s disease (10), and in patients on dialysis (11). In addition, persons with systolic hypertension tend to have worse postprandial hypotension (4,12,13,14). It has been suggested that very healthy older persons do not have postprandial hypotension (15). In this study, whereas forearm vascular resistance fell, it was compensated for by other alterations in the autonomic nervous system to maintain cardiac output. Others, however, have found this fall in healthy elderly persons (16). Postprandial hypotension is not associated with orthostasis, but the two effects can be additive (17,18).

Carbohydrate in the diet has previously been demonstrated to be the major factor involved in the production of postprandial hypotension (19). In this issue of the Journal, Vloet and colleagues (20) show that postprandial hypotension is directly related to the amount of glucose given. Xylose fails to have a similar effect on postprandial hypotension (21). This suggests that limiting the quantity of glucose in the diet will decrease the occurrence of postprandial hypotension in older persons.

There is evidence that the rate of gastric emptying determines the presence of postprandial hypotension (22). Xylose, which slows gastric emptying dramatically, does not produce postprandial hypotension (23). Guar gum, which slows gastric emptying, also alleviates postprandial hypotension (24). Similarily acarbose, an α-glucosidase inhibitor that slows gastric emptying by releasing glucagon-like peptide-1, has been used to treat postprandial hypotension in a diabetic patient (25). Whether the slowing of gastric emptying produces its alleviation of postprandial hypotension by slowing the presentation of glucose to the rest of the gastrointestinal tract or secondary to gastric distention is uncertain. Fundic distension results in muscle nerve sympathetic activity resulting in an increase in blood pressure (26). Older persons have a decrease in fundic compliance, which could lead to a decrease in this gastrovascular response (27,28).

Much data has suggested that the fall in blood pressure following a meal is not due to mesenteric artery vasodilation (4,29). Similarly, insulin does not appear to play a role in the pathogenesis of postprandial hypotension (4,30). This is despite the depressor effect of insulin, which can decrease the vasoconstriction in the calf seen after a high-carbohydrate meal (31). Insulin is thought to produce this effect through the modulation of nitric oxide release (32). Although there is some suggestion that postprandial hypotension is due to a decreased release of norepinephrine following food intake (30), this has not been confirmed in the majority of studies (4).

The somatostatin analog octreotide has been used to treat postprandial hypotension (33–35). This suggests that a peptide hormone may be involved in the pathogenesis of postprandial hypotension. Calcitonin gene-related peptide (CGRP) is a gastrointestinal peptide hormone that is released by glucose and inhibits gastric emptying (36). CGRP is a potent vasodilatory agent. Edwards and colleagues (37) found that CGRP circulating levels increase to a greater extent in those patients with postprandial hypotension. This suggests that CGRP may play a major role in the pathogenesis of postprandial hypotension.

From the clinical perspective it is important to document the blood pressure following a meal in all persons whose falls have occurred within 2 hours of the meal. This may need to be done on more than one occasion because individuals with postprandial hypotension show day-to-day variation (38). In addition, blood pressure should be measured before and after breakfast because it is after this meal that postprandial hypotension is most likely to occur (39). Similar day-to-day variability in blood pressure has been shown to occur in older persons with orthostasis (40). In these subjects up to three measurements have been necessary to document orthostatic hypotension.

Management of postprandial hypotension requires appropriate treatment of hypertension and limitation of the use of nitrates and farusemide (4,39,41). Although caffeine has been suggested to ameliorate postprandial hypotension through an effect on the adenosine receptor (4), recent studies have suggested that neither tea nor coffee will prevent the meal-
induced fall in blood pressure (42). Limitation of carbohydrate load is the major therapeutic option. Other approaches are to utilize octreotide (4) or the norepinephrine precursor 3,4-DL-threo-dihydroxyphenylserine (41,43).

In conclusion, postprandial hypotension represents an important geriatric condition. Not only can it result in falls, syncope, stroke, dizziness, and myocardial infarction, but it also can be a precursor to frailty (44), malnutrition through a learned conditioned aversion to food (45), and hip fracture (46).

Acknowledgments

Address correspondence to John E. Morley, MB, BCh, Editor, Journal of Gerontology: Medical Sciences, Division of Geriatric Medicine, Saint Louis University School of Medicine, 1402 S. Grand Blvd., M238, St. Louis, MO 63104. E-mail: jgeronmed@slu.edu

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