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# Letter to the Editor

## Postprandial hypotension in the elderly: what is the metabolic chain of events?

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The recent paper by Visvanathan *et al.* (2005) elegantly demonstrates the increased risk of postprandial hypotension in older individuals following consumption of glucose or the glucose-releasing sugar, sucrose, compared with that of non-glucose sugars such as fructose or xylose (Da Costa *et al.* 1985).

It would be very interesting to know whether the authors of this paper have data on the relative insulin responses during the different studies in their subjects, since one of the explanations of postprandial postural hypotension that has been suggested is that insulin is a vasodilator in peripheral arteries, and in both resistance and capacitance vessels, whether given pharmacologically or secreted physiologically in response to glucose (Miles & Hayter, 1968; Page & Watkins, 1976; Takata *et al.* 1985). Increased distensibility of large arteries in response to physiological doses of insulin is also reported in healthy adults (Westerbacka *et al.* 1999). The normal protective adrenergic responses are reduced in subjects with increased insulin resistance, in older individuals and, most markedly, in subjects with autonomic neuropathy (Da Costa *et al.* 1985; Fagius *et al.* 1996). The vascular effects of insulin in man can appear before blood glucose levels change, suggesting possible glucose-independent mechanisms.

Avoidance of dietary glucose and sucrose can reduce the risk and severity of symptomatic episodes, but further understanding of the chain of events involved in post-glucose hypotension, including those induced by insulin, might lead to additional therapeutic approaches for the management of disabling postprandial hypotension, especially in autonomic neuropathy as a complication in diabetes where insulin therapy is usually unavoidable.

### References

Da Costa DF, McKintosh C, Bannister R, Christensen NJ & Mathias CJ (1985) Unmasking of the cardiovascular effects of carbohydrate in subjects with sympathetic denervation. *J Hypertens* **3**, Suppl., S447–S448.

Fagius J, Ellerfelt K, Lithell H & Berne C (1996) Increases in muscle nerve sympathetic activity after glucose intake is blunted in the elderly. *Clin Auton Res* **6**, 195–203.

Miles DW & Hayter CJ (1968) The effect of intravenous insulin on the circulatory responses to tilting in normal and diabetic subjects with special reference to baroreceptor reflex block and atypical hypoglycaemic reactions. *Clin Sci* **34**, 419–430.

Page MM & Watkins PJ (1976) Provocation of postural hypotension by insulin in diabetic autonomic neuropathy. *Diabetes* **25**, 90–95.

Takata S, Yamamoto M, Yagi S, Noto Y, Ikeda T & Hattori N (1985) Peripheral circulatory effects of insulin in diabetes. *Angiology* **36**, 110–115.

Visvanathan R, Chen R, Garcia M, Horowitz M & Chapman I (2005) The effects of drinks made from simple sugars on blood pressure in healthy older people. *Br J Nutr* **93**, 575–579.

Westerbacka J, Wilkinson I, Cockcroft J, Utriainen T, Vehkavaara S & Yki-Jarvinen H (1999) Diminished wave reflection in the aorta. A novel physiological action of insulin on large blood vessels. *Hypertension* **33**, 1118–1122.

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