Effect of postprandial hyperglycemia and hyperinsulinemia on vascular responsiveness

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Recent clinical study**,** reported that treatment with an α-glucosidase inhibitor in patients with impaired glucose tolerance (IGT), which have postprandial hyperglycemia, resulted in significant reduction of hypertension, leading to hypothesis that transient postprandial hyperglycemia and hyperinsulinemia should be partly attribute the development of hypertension.

Therefore, to investigate influence of acute hyperglycemia and hyperinsulinemia on vascular responsiveness, we studied in vivo vascular responses to spinal cord stimulation (SCS) and intravenous (i.v.) bolus injections of noradrenaline (NA), calcitonin gene-related peptide (CGRP) using pithed rats, which had no vasoreflex. Euglycemic male Wistar rat was pithed and blood pressure (BP) was measured. Continuous glucose (15-25%) infusion, which increased both blood glucose and serum insulin level, significantly potentiated adrenergic nerve-mediated pressor response to SCS without affecting pressor response to NA. In pithed rats with artificially increased BP and blockade of autonomic outflow, CGRPergic nerve-depressor response to SCS was attenuated by glucose infusion. These results suggest that acute hyperglycemia associated with hyperinsulinemia increases adrenergic nerve-mediated vasoconstriction, which is partly associated with the blunted CGRPergic nerve function.

In conclusion, transient postprandial hyperglycemia associated with hyperinsulinemia elcites abnormal neuronal regulation of vascular tone, which may contribute in part the development of hypertension.