CE echocardiography for global left ventricular function when MRI is the reference standard.

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http://dx.doi.org/10.1016/j.jacc.2012.06.020

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**Dysglycemia and Cardiovascular Risk**

I read with interest the “state-of-the art” paper by Grundy (1) that was recently published in the Journal. The paper demonstrated that the pre-diabetic range of glucose levels do not directly cause atherosclerosis or its complications, on the basis of a systemic review of the relationship between pre-diabetes and cardiovascular disease (CVD). The paper also indicated that the major targets for the prevention of CVD are elevations of cholesterol and blood pressure, but not elevations of plasma glucose, because certain clinical trials have suggested that the glucose-lowering drugs did not retard atherogenesis. My question is whether it is in fact reasonable to exclude the clinical worth of lowering glucose, especially in postprandial hyperglycemia, for the prevention of CVD in subjects with pre-diabetes.

Both epidemiological and interventional studies have shown that pre-diabetes, especially in post-prandial hyperglycemia, is a direct and independent risk factor for CVD (2–4). In the DECODE (Diabetes Epidemiology: Collaborative Analysis of Diagnostic Criteria in Asia) study, the risk for CVD and stroke increased progressively with the change from impaired fasting glucose to impaired glucose tolerance (IGT) to type 2 diabetes (5), indicating that hyperglycemia is a risk factor for cardiovascular (CV) mortality. The relation between glycemia and CV risk started within the normal blood glucose range, with a linear relationship (4,5). In addition, post-challenge hyperglycemia is strongly associated with future macrovascular events and total mortality in patients with acute myocardial infarction and/or patients with coronary disease who received angiography (6). The results of the STOP-NIDDM (Study to Prevent Non-Insulin-Dependent Diabetes Mellitus) trial demonstrated that treating IGT with acarbose, an alpha-glucosidase inhibitor, which specifically reduces postprandial hyperglycemia, significantly reduces the conversion rate of IGT to type 2 diabetes associated with reductions in the development of CV events (by 49%), the incidence of new cases of hypertension (by 34%) (7), and the annual increase of carotid intima–media thickness (by 50%) (8). Moreover, pioglitazone significantly reduces diastolic blood pressure and the rate of the intima–media thickness associated with a reduction in the conversion of IGT to type 2 diabetes (9).

Thus, dysglycemia, especially in the case of postprandial hyperglycemia, is related to CV risk and mortality, and a variety of pharmacological and nonpharmacological therapies should be targeted to the management of postprandial blood glucose independent of blood pressure and/or cholesterol.

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http://dx.doi.org/10.1016/j.jacc.2012.03.078

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