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LETTER COMMENT

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To: Strey CH, Young J, Collier M, Florkowski CM, Shand BI, Scott RS (2004) The postprandial state does not impair endothelial function in women with Type 2 diabetes irrespective of glycaemic control. Diabetologia 47:1838–1846

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To the Editor: In a recent article, Strey and co-workers reported that the postprandial state has no negative effect on endothelial function in postmenopausal women with type 2 diabetes [1]. However, the same authors also stated that their results were not consistent with many other reports [1]. We, the International Prandial Glucose Regulation (PGR) Study Group, have several concerns with the results of the study.

Firstly, no information was available on the concomitant therapy undertaken by the diabetic women; however, the mean blood pressure was significantly increased in the diabetic group, particularly pre-intervention (104.5±4.0 vs. 91.2±3.2 mmHg, $p<0.05$, diabetic and non-diabetic respectively), and was consistently high in the post-intervention period (100.3±3.8 mmHg). This suggests that some, or many, of these patients were hypertensive and, presumably, undergoing treatment. As it is well known that many compounds used in the treatment of hypertension, e.g. calcium channel blockers, ACE inhibitors and angiotensin 1 blockers, have a protective effect against endothelial dysfunction, one could hypothesise that the deleterious effect of the postprandial state may simply have been counter-balanced by the hypertensive therapy [2]. Secondly, patients showed the same levels of LDL as the control group, as well as reduced levels of HDL, but, surprisingly, HDL increased following intervention. This suggests that a statin may have been used during the study, which has been shown to protect the endothelium in postprandial conditions [3]. Thirdly, eight patients (42%) were taking sulphonylurea. This is another interesting point, as gliclazide has been shown to have a protective effect on the endothelium [4].

Finally, the most important reason why the intensive glucose control had no effect on postprandial endothelial dysfunction is that the investigators were unable to control effectively the postprandial glucose excursions. In this respect, their failure supports our hypothesis that perfect control of postprandial hyperglycaemia is essential in protecting the endothelium. The postprandial glucose value in the diabetic group was still 12.89 mmol/l, and the high-sensitivity C-reactive protein level was 6.85 mg/l (pre-intervention level 5.82 mg/l). Glycaemic control was,

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therefore, far from optimal. It has been shown that only near-normal glucose control leads to a normalisation of impaired endothelial function in the postprandial state [5].

We, the International PGR Study Group, believe that studies of this sort need careful evaluation, as they may create confusion about the role of prandial glucose regulation and its benefits in lowering the risk of cardiovascular disease and achieving optimal glycaemic control. It has been reported that cardiovascular mortality increases steadily with increasing post-challenge glucose [6]. Prandial glucose regulation is of great relevance to clinical practice and is included in many guidelines all over the world [7].

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