fraction was higher during hyperglycaemia. The response to dynamic exercise was similar to that of a non-diabetic control group and was unrelated to blood glucose concentration. Thus while the *resting* left ventricular ejection fraction may be related to prevailing blood glucose, the response to *dynamic* exercise appeared to be independent of glycaemia. Furthermore, Goldwit et al. demonstrated that the response of the ejection fraction to hyperglycaemia or dynamic exercise was not affected by 6–25 weeks of improved glycaemic control [4]. In a previous study [5] we showed that the response to dynamic exercise was normal in a group of young Type 1 diabetic patients with diabetes of short duration. In our recent study [2] we chose to examine the cardiac response to dynamic exercise which is apparently unaffected by hyperglycaemia.

Other studies have shown that the response of the left ventricular ejection fraction to dynamic exercise is abnormal in patients with longstanding diabetes [6, 7] and this was confirmed by our own investigation [2]. Two recent reports have suggested that the responses to dynamic exercise may be more abnormal in diabetic patients with advanced retinopathy [8] and autonomic neuropathy [9]. The abnormalities provoked by dynamic exercise were consistent, and were associated with the coexistence of microvascular disease.

In our study we performed endomyocardial biopsy and documented the presence of fibrosis, small vessel changes, and basement membrane thickening [2]. The sampling problems inherent with endomyocardial biopsy coupled with the small number and size of specimens, may explain why histological changes were not found in all patients. The right ventricle was biopsied because of the greater safety of the procedure compared with left ventricular biopsy. Dr. Harrower [1] states that these histological changes have been reported previously. Fischer et al. [10] obtained ventricular biopsies from diabetic patients during coronary artery bypass grafting, and observed myocardial fibrosis and basement membrane thickening. All of these patients had severe coronary heart disease unlike the asymptomatic diabetic patients in our study. None of our patients had coronary heart disease demonstrable by coronary arteriography, and none had hypertension, so no obvious pathophysiological explanation can be advanced for these histological changes except that they are a direct consequence of long-standing diabetes.

By demonstrating abnormalities of left ventricular function on exercise in diabetic patients with normal coronary arteries, and by showing histological changes in the hearts of several of these patients, we believe that our study provides substantial support for the concept of a specific heart disease in diabetes.

Yours sincerely,
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Erratum

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The following abstract was misprinted in the July and Abstracts issues of the journal. The correct version should read:

352. Combined therapy with sulphonylurea (gliclazide) and insulin for Type 1 (insulin-dependent) diabetic patients: one year follow-up

A. Maldonato, D. Bloise, M. Pittaluga, F. Barbetti, F. Marani, O. Falla, P. Gandolfo, P. D'Ottavi, E. Sciullo and F. Fallucca. Diabetes Unit, "La Sapienza" University, Rome, Italy

We have recently described that the association of gliclazide (SU) for six months to the usual insulin treatment (INS) in Type 1 (insulin-dependent) diabetic patients was accompanied by a manifold improvement of diabetes control. To evaluate the persistence of such effects, we continued the study for six more months. The same dose of SU (160 mg/24 h) was left in association with INS in 8 Type 1 diabetic volunteers and plasma glucose, other intermediate metabolites, C-peptide (CPR), glucagon (IRG) and growth hormone (GH) were measured in basal conditions and after arginine challenge (ATT). Moreover, the insulin need, HbA1c, and the erythrocyte filtration index were evaluated. At the end of the 12-month trial, the plasma glucose values were maintained at the 6-month levels (mean: 12.10 ± 2.21 at time 0 vs 9.28 ± 1.77 at 6 months vs 9.56 ± 1.60 mmol/l at 12 months and the CPR was still increased (delta area: -0.14 at time 0 vs 0.93 at 6 months vs 1.18 ng/ml at 12 months. Growth hormone and IRG values were similar to those obtained at 6 months. Insulin need and HbA1c were significantly decreased. Red cell deformability was improved. In conclusion, the association of SU to INS in Type 1 diabetic patients induced metabolic, hormonal and haemorheological improvements, and a reduction of insulin need, which persisted after 12 months.