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AUTHORS'REPLY.—The points made by Dr Arnold concerning the different half lives of C peptide and insulin are well known and well taken. They are not relevant, however, to within group or between group comparisons of either variable at the same time points. Consistent differences between cases and community controls for both white and Asian groups reinforce the greater excess relative risk for C peptide as an indicator of infarction. Samples taken at two hours may not reflect peak C peptide concentrations, but Dr Arnold confuses measures such as areas under the curve to express absolute secretion with relative measures to compare group characteristics. His suggestion of ethnic variation in C peptide clearance is interesting but again is not relevant to the differences found within each ethnic group between the cases and controls. Epidemiological studies are designed to compensate for between subject variability in such variables by adequate sample size.

All the index cases of both groups had "normal" renal function as judged by plasma urea and creatinine concentrations, which we should have mentioned. This point was also raised by Drs Simmons and Coppack in their somewhat inaccurate letter. They are surprised that insulin did not persist as a risk indicator between Asians and white groups but failed to note that insulin levels were consistently higher in Asians, whatever the glucose tolerance state (tables II and III). Previous studies have not compared the two groups for relative risk. We accept that an unsupervised glucose loading at home is not ideal and may account for lesser effects of insulin (with its shorter half life). There would, however, have to be consistent bias between cases and controls and between Asian and white subjects to affect the results, and there was no such evidence of this, a point Dr Simmons will be familiar with in his own Coventry prevalence study, where screening was postprandial and only a subsample were given glucose loads. In the separate study we referred to by one of us (JKC), in which glucose tolerance by standard challenge was studied in three ethnic groups randomly sampled from the community, C peptide and the ratio of C peptide to glucose remained greatest two hours after challenge in Gujaratis compared with whites and Afro-Caribbeans. We await results of highly specific assays to assess whether insulin itself, proinsulin and its split products, or C peptide remain the markers they appear to be from this and another recent study.¹

We accept that the suggestion of excess hepatic extraction of insulin is somewhat speculative, but as this is the first time that C peptide rather than

insulin has been suggested as the risk indicator such an explanation is a clear possibility.

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- 1 Hamsten A, Efendic S, Walldius G, Szamosi A, de Faire U. Glucose tolerance and insulin response to glucose in non-diabetic young male survivors of myocardial infarction. *Am Heart J* 1987;113:917-27.

SIR.—The first observation in 1965 that survivors of a myocardial infarct had increased plasma concentrations of insulin¹ (in the light of recent data² it must be emphasised that insulin in this context embraces proinsulin and other peptides) has been repeatedly confirmed and similar findings have been reported in subjects with cerebral or peripheral vascular disease. Dr L O Hughes and colleagues failed to observe higher insulin levels in male Asian and white survivors of a myocardial infarct but did find that C peptide concentrations were increased.³ From this they concluded that insulin secretion was increased together with hepatic extraction of insulin. Given that their single sampling time was two hours after the insulinogenic stimulus it is a bold assumption to make. Thus Faber *et al*, using multiple sampling during 60 minutes after an oral glucose load and measuring molar ratios of insulin to C peptide, concluded that in obese hyperinsulinaemic subjects the hepatic extraction of insulin was reduced.⁴

Dr Hughes and colleagues further concluded that insulin resistance may be an important risk factor for coronary artery disease and partly responsible for the increased risk of diabetes and coronary artery disease in a migrant Asian population. The case in favour of insulin resistance predisposing to diabetes is respectable, but for coronary artery disease it is dubious. Dr Hughes and colleagues state that "the only data not fully supporting the hypothesis" arise from studies in Afro-Caribbeans. In fact there are many data that flatly contradict the hypothesis.⁵ To take but one example from ethnic comparisons: in Pima Indians, who have the highest prevalence of non-insulin dependent diabetes associated with obesity and insulin resistance so far recorded, only among diabetics with proteinuria was cardiovascular mortality increased. Indeed, total mortality was not different between diabetics without proteinuria and non-diabetics.⁶

One major risk factor for coronary artery disease, a low concentration of high density lipoprotein cholesterol, is consistently associated with high insulin concentrations and it might therefore be expected, if insulin is mediating the deranged lipoprotein metabolism, that prospective studies would consistently implicate insulin, as they do high density lipoprotein cholesterol. But they do not.

The reason for the increased risk of coronary artery disease in Asian migrants remains an enigma. The popularity of the insulin resistance or hyperinsulinaemia hypothesis seems to reside in the failure to find differences in the accepted risk factors, such as diet, lipids, or blood pressure, rather than in any inherent validity. There are, however, numerous differences in rates of coronary artery disease between populations or trends over time within populations that remain unexplained. As usual more research is required, but new hypotheses are a greater need.

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Hunterian Institute and dental research

SIR.—Dental research at the Royal College of Surgeons having been successively savaged by the effects of the Flowers report and by the Department of Health, it is perhaps understandable that Dr Tony Delamothe should be led to assume its impending demise.¹ Closer consideration might have persuaded him to be less dirgeful.

It is ironical that Flowers and the University of London, having abandoned a longstanding commitment to the support of the college's contribution to postgraduate education, should have precipitated the possibility of closing a department that at no time received financial support from the university or its institute of basic medical sciences. Thus do resounding policy documents produce unintended knock on effects.

Withdrawal of financial support by the Department of Health is responsible for the abandonment of the caries research programme. Some recent surveys indicating a decline in the incidence of caries have persuaded the department to believe that this disease has gone away. If it did nothing else the research at Downe, substantially supported by the department in earlier years, clearly reiterated the neglected realisation that caries is an infectious disease—and innumerable examples exist to show that such diseases do not merely go away. Whether the search for a vaccine was as unsuccessful as Dr Delamothe suggests is a matter on which judgment should be postponed until the damage caused by dental caries calls for renewed effort from a future generation of scientists. They will be better placed than today's cost conscious bureaucrats to assess the value of the extensively documented results of the Downe project.

This, as well as all other dental research at the college over the past 32 years, has been funded predominantly by grants and donations from sources outside the college. It was possible to do this because the department of dental science was underpinned by the college, but at the very time that this safety net has been called on the college itself has been forced into the dire situation of requiring £500 000 of its own funds annually to shore up the departments deprived of university support.

The future of dental research at Lincoln's Inn Fields lies in the hands of the college council and the board of its faculty of dental surgery. Their decision, unlike that of the university or the Department of Health, will take into account the total relation of dental research to college activities and aspirations and the extent to which it is able to fulfil and advance the purposes for which the faculty was first established.

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- 1 Delamothe T. Hunterian Institute and dental research. *Br Med J* 1989;299:757. (23 September.)

SIR.—Your news report on the Hunterian Institute and dental science¹ is incorrect. The faculty of dental surgery is not preparing itself for the passing of the department of dental science. This is indeed one of its most cherished creations, and the