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CORRESPONDENCE

Because we receive many more letters than we have room to publish we may shorten those that we do publish to allow readers as wide a selection as possible. In particular, when we receive several letters on the same topic we reserve the right to abridge individual letters. Our usual policy is to reserve our correspondence columns for letters commenting on issues discussed recently (within six weeks) in the BMJ.

Letters critical of a paper may be sent to the authors of the paper so that their reply may appear in the same issue. We may also forward letters that we decide not to publish to the authors of the paper on which they comment.

Letters should not exceed 400 words and should be typed double spaced and signed by all authors, who should include their main degree.

Are problem drinkers dangerous drivers?

SIR,—The findings of Dr James A Dunbar and others (16 March, p 827) that many of those convicted of driving under the influence of alcohol appear to have significant drinking problems should come as no surprise to those of us with an interest in this problem. Their fears that "the Department of Transport criteria [may] be missing a large proportion of drinking and driving offenders with alcohol problems and should be reviewed" are surely justified. Indeed, the incidence of problem drinking among their subjects is almost certainly higher than their results indicate. First of all, the evidence suggests that biochemical and haematological tests are a relatively inefficient way of identifying problem drinkers and that questionnaires give a much higher rate of identification. Even so, I am surprised that they did not measure the mean corpuscular volume of their subjects since in practice one encounters many patients in whom the mean corpuscular volume is raised even when liver function values are normal, and vice versa. Drunk driving accounts for considerably more deaths each year than the total number of homicides for the entire United Kingdom (including that part of it in which a miniature civil war is taking place), and many of the dead and injured, whether pedestrians, passengers, or drivers, are the sober and innocent victims of the most lethal antisocial activity which our society has ever produced. In the face of this perennial blood bath the response of the Department of Transport, the police, and, most particularly, parliament is really quite supine.

This results not only in failure to prevent a number of preventable deaths on the roads but also in the failure to point some of these offenders towards treatment for their drinking problems, which experience elsewhere suggests at least some of them will accept with beneficial consequences to themselves and their families. In several states in the USA, all those convicted of driving under the influence of alcohol undergo mandatory screening for drinking problems, which they themselves have to pay for. Those who seem likely to have a problem (as opposed to an isolated instance of carelessness or bad luck, depending on one's point of view) have to undergo a further mandatory course of alcoholism education and treatment, which generally includes a period of probation-supervised disulfiram. These measures are being carefully monitored and the evidence so far is that they reduce both the incidence of recidivism and the size of the prison population. Since offenders also have to pay for their own treatment, the system is self supporting and in at least one city even makes a small profit. The cost of treatment represents a further deterrent. Surely this is a more promising approach than the present timid attempts to "assess" only those whose drinking problems are so obvious that they hardly need assessing.

COLIN BREWER

Community Alcoholism Treatment Service,
All Saints' Hospital,
London SE11 4SL

Serum fructosamine concentration as measure of blood glucose control in insulin dependent diabetes

SIR,—Dr John R Baker and his colleagues described significant correlations between fructosamine and the integrated plasma glucose concentration as well as between fructosamine and glycosylated haemoglobin (HbA_{1c}) drawn from patients on the same day (2 February, p 352). They stated that in their experience the fructosamine concentrations had been useful for monitoring short term control (3-6 weeks). Based on the correlations found they concluded that this index would be useful for evaluating diabetic control.

The data in fig 1 reveal that there is too wide a scatter among the fructosamine concentrations to make them a reliable index of diabetic control. In fig 3 two subjects (case 1 and case 2) were excluded from their calculations, but these two cases were included in the statistical analyses given for fig 1. We are also interested to know the correlation coefficient between the fasting glucose value and the mean glucose value as well as reviewing the scattergrams for the correlations given in table II.

Many variables have long been known to be abnormal in uncontrolled diabetes. These include growth hormone, aminoacids, creatinine, and lipids. All these variables have either improved or become normal with the control of the diabetic state.

Fig 1 also shows that in those diabetic subjects whose fructosamine values were $<2{\cdot}5$ mmol/l (63 mg/100 ml), who constituted over 85% of the group, no correlation was likely to be found between the fructosamine value and HbA_{1c} even when cases 1 and 2 were included. Interestingly six subjects (14%) in fig 1 had raised fructosamine values when compared with the controls despite their normal HbA_{1c}.

In conclusion, we feel that the data presented simply suggest that fructosamine values are raised in subjects with diabetes mellitus, including those with normal HbA_{1c}. If the authors wish to use fructosamine concentration as an index of control we would suggest that they correlate it with integrated plasma glucose values at variable antecedent time intervals. Their object should be to show the linear relation and the variances between fructosamine and indices of control within the diabetic subjects rather than attempting