should have been observing in large part for several years.

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Serum fructosamine—the pH factor

SIR,—The answer to one of the questions posed by Drs E J Hindle and Rostron Glenise (7 September, p 676) is contained in a letter to me from Dr John Baker dated 4 June 1985. I had written to ask for clarification about the pH of the fructosamine assay, which had changed from pH 10·8 in the original paper[1] to pH 10·35 in their most recent publication (2 February, p 352). They replied "The fructosamine assay described by Roger Johnson[1] was incorrect. The pH of the carbonate buffer should have read 10·35 [not pH 10·8]. . . . Unfortunately, we did not recognise this mistake and the discrepancy was not pointed out to us until relatively recently."

During the three years since the original "mistake" was described by the original authors, papers have appeared by Lloyd and Marples[2] and Hindle *et al*[3] using the incorrect pH 10·8. Although both publications were known to the New Zealand group, no comment had been made by them about the differences in pH or in reference ranges described.

It is difficult to believe that authors may be deliberately misleading, but it is clear that a great deal of time and money can be wasted in trying to reproduce work that contains errors in its publication. Perhaps authors should be more careful when publishing their work, and be more willing to admit publicly their mistakes.

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1 Johnson RN, Metcalf PA, Baker JR. Fructosamine. A new approach to the estimation of serum glycosyl protein. An index of diabetic control. *Clin Chim Acta* 1982;127:87-95.
2 Lloyd D, Marples J. Simple colorimetry of glycated serum protein in a centrifugal analyser. *Clin Chem* 1984;30:1686-8.
3 Hindle EJ, Rostron GM, Gatt JA. The estimation of serum fructosamine: an alternative measurement to glycated haemoglobin. *Ann Clin Biochem* 1985;22:84-9.

**The authors reply below.—ED, *BMJ*.

SIR,—Throughout we have used a buffer comprising 75 mmol Na_2CO_3 plus 25 mmol $NaHCO_3$ per litre because it is simple to prepare and minimises interference from glucose and other serum analytes. In 1982 we believed the pH of this buffer to be 10·8 as reported, and it is only since that time that others have redetermined it as pH 10·35. Presumably this serves to illustrate the difficulties of making accurate pH measurements under alkaline conditions. Nevertheless, a different reagent pH between 10 and 11 should still allow a clinically useful assay, although the numbers will be different as Lloyd *et al* and Hindle *et al* have described in their publications.

We also recognised the effects of albumin concentration on the reaction between primary standard and reagent and the variation in reducing activity of different albumin preparations as noted by Drs E J Hindle and Rostron Glenise (7 September, p 352). We opted to keep albumin concentration in the standards constant at 40 g/l and to correct for reducing activity in the zero standard by using the slope of the standard plot as a calibration factor. As a result, our reference interval (mean and 2SD) derived from 502 non-diabetic blood donors is now 2·0 to 2·7 mmol/l. Moreover, we use secondary standards of serum or glycosylated albumin in routine practice to improve the precision of the method and facilitate its transfer to different automated analysers.

Far from attempting deliberately to mislead, we have taken steps to set the record straight. Following publication of Dr Hindle's paper we wrote to the editor of the *Annals of Clinical Biochemistry*. Moreover, we have recently published a more complete description of our assay.[1]

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1 Baker JR, Metcalf PA, Johnson RN, Newman D, Rietz P. Use of protein-based standards in automated colorimetric determinations of fructosamine in serum. *Clin Chem* 1985;31:1550-4.

Development of new renal scars

SIR,—Dr J F B Dossetor (21 September, p 826) concludes his comments on detecting renal scarring in children with two recommendations: accurate diagnosis of all urinary tract infections and adequate follow up of all those not investigated. While we accept the logic of Dr J M Smellie and her colleagues (29 June, p 1957) that all children with proved urinary tract infection should be investigated, our experience is that when the standard of general practice and the index of suspicion are high investigation of all the children detected would put an impossible strain on hospital resources.

In 1974, 332 children with urinary tract infection were detected as a result of mid-stream specimens of urine sent to this laboratory by general practitioners.[1] Since then the use of the laboratory for urine culture by general practitioners has roughly doubled, almost certainly as a result of vocational training and of grouping of doctors in health centres. At present it is not realistic to suggest that all children found to have a urinary tract infection should be referred to hospital for investigation. As a compromise it is our practice to suggest referral of children in certain categories: those with proved recurrent infections, those infected with resistant or unusual organisms, those with persistent pyuria, and those in whom infection fails to respond to appropriate antibacterial treatment. For the rest Dr Dossetor's recommendations would seem to be a reasonable compromise.

Our purpose in writing this letter is to point out the effect that the improved management of urinary tract infection in general practice has had, and continues to have, on the laboratory workload. Diagnosis can and should be improved by culture of mid-stream specimens of urine, general practitioners may opt for regular monitoring before deciding on referral, and outpatient visits of patients under hospital supervision can be less frequent if general practitioners arrange regular urine cultures.

Hitherto it has been possible to offset such justifiable increases in laboratory workload by eliminating some of the unnecessary requests or procedures: so called screening of groups of patients when there is no evidence that it is necessary or that the findings are acted on, routine culture of postoperative catheter specimens, and culture of specimens from patients with long term catheters when they are well. In this laboratory we have now eliminated most of these tests and those that we now get asked for are usually necessary and well requested; the standard of general practitioner use of the laboratory is high. Laboratory workload still grows relentlessly, and budgets, far from being increased, have been reduced over the past few years. Perhaps concerned clinicians who continue to advise the use of the laboratory for the diagnosis and mangement of urinary tract infection should throw their weight behind the need for adequate financial provision.

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1 Maskell R, Pead L. Urinary infection in children in general practice: a laboratory view. *J Hyg Camb* 1976;77:291-8.

Prolonged use of nitrazepam for epilepsy in children with tuberous sclerosis

SIR,—The general conclusion of Dr Jennifer Dennis and Ms Ann Hunt (14 September, p 692) that anticonvulsant drugs including nitrazepam may be used for longer than necessary and that children may feel rather better when the drug is removed are unexceptionable, but to suggest a specific motor and cognitive side effect on the evidence presented is misleading. Although tuberous sclerosis may show a degree of genetic homogeneity, there is wide pathological variation even within that group with early onset epilepsy. No allowance seems to have been made for this in the study. Thus an alternative explanation of the relation between long term nitrazepam therapy and not walking could be that those with worse epilepsy have rather more wrong with their central nervous system and that their lack of both motor and cognitive development is an independent result of worse underlying disease. It would be a shame if nitrazepam fell into disrepute as a specific anticonvulsant for myoclonic epilepsy as a result of this study.

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Does the underprivileged area index work?

SIR,—I read with interest the article by Mr Ralph Leavey and Ms Jo Wood (14 September, p 709). I am glad that several groups have been comparing the score with morbidity and mortality data for wards in various cities.

The score is a measure of general practitioners' assessments of the effects on their workload or pressure on their services of certain characteristics of the populations that they serve. It is made up of a combination of census data relating to the percentages in the general population of the single elderly, the unemployed, the unskilled, those living in overcrowded conditions, highly mobile and ethnic groups, under 5 year olds, and lone parent families—each of these variables being weighted according to a national survey of general practitioners. Similar results are found from surveys of other primary care workers[1] (which are now being extended nationally).

From the way that it was derived one would expect the score to correlate with the use of primary and secondary health care resources, on the one hand, and with measures of morbidity and mortality, on the other. Scores calculated would be expected to relate to potential rather than actual workload. The score is not meant to be a measure of the present distribution of resources.[1-3] I have suggested that information about the scores and general practitioner services should be collected for Medical Practices Committee areas,[1] and a study along these lines has been started.[4]

The association which the authors found between higher scores and lower list sizes is