Serum	*pH*	*Apparent TIBC (μg/100 ml)*
Fresh human	6·5	315
	8·4	383
	9·5	461
Fresh bovine	7·0	394
	8·0	551
	10·0	596
Freeze-dried human	7·5	338
	8·4	416
Freeze-dried bovine	7·5	608
	8·9	652

Table *Effect of initial pH on the apparent TIBC*

When ion-exchange resin is used, then the TIBC is virtually independent of the amount of resin added, when more than about 100 mg of resin is used. This independence has been found with both human and bovine sera, and is not dependent on pH. Serum TIBC is slightly affected, however, by the state of the resin used (Lehmann and Kaplan, 1971).

However, the apparent TIBC, when ion-exchange resin is used, is markedly affected by the initial pH of the serum. This has been demonstrated with both bovine and human sera, either fresh or freeze-dried; the serum pH was adjusted by drop-wise addition of N hydrochloric acid or N sodium hydroxide (table).

To overcome this problem, titration of each serum to the same initial pH and tonometry were attempted but were rather impractical for routine use. A simple satisfactory solution, which in our laboratory has led to improved precision and to improved inter-laboratory comparison, has been to add barbitone buffer pH 7·5 to each serum before saturating the transferrin with iron, as opposed to the final pH adjustment in the standard AutoAnalyzer methodology.

This factor, and other factors such as that studied by Leggate and Crooks (1972), can contribute markedly to the large inter-laboratory variation found in regional and national quality control schemes as many commercial freeze-dried and in house control sera have a high pH, and again emphasizes that caution should be exercised in the interpretation of quality control data.

C. G. FRASER
Department of Chemical Pathology, University of Aberdeen, Foresterhill, Aberdeen AB9 2ZD.

References

Koepke, J. S. (1965). Comparison of light magnesium carbonate and amberlite sponge adsorbents in the measurement of the latent iron-binding capacity of serum. *Amer. J. clin. Path.*, **44**, 77-81.

Leggate, J., and Crooks, A. E. (1972). Problems in quality control in determinations of serum total iron-binding capacity by the magnesium carbonate method. *J. clin. Path.*, **25**, 905-906.

Lehmann, P. H., and Kaplan, A. (1971). Use of an anion-exchange column to determine serum iron-binding capacity. *Clin. Chem.*, **17**, 941-947.

Williams, H. L., and Conrad, M. E. (1972). Problems in the measurement of iron-binding capacity in serum. *Clin. chim. Acta*, **37**, 131-140.

Standardization of Clinical Enzyme Assays

The publication of a definitive report on the standardization of clinical enzyme assays is a consummation devoutly to be wished by all clinical biochemists but it is doubtful if any such report would have their unreserved approval. However, the report by Wilkinson, Baron, Moss, and Walker (1972) can elicit little sympathetic response from any.

The first prerequisite of any standardization of clinical enzyme assays is to decide upon a standard temperature. This was speculated on by the authors, who 'first considered 37° as the most suitable' and then sought evidence against it, only finding comfort in a putative glucose-6-phosphate dehydrogenase instability which incidentally is contrary to our own experience (King, 1972). However, this apparently was sufficient to make for a decision in favour of 25°C for estimating transaminase activities. Certainly we cannot find any argument advanced by the authors in favour of 25°C 'there is often a lag phase of 2 to 5 min' is considered an advantage. How temperature control is readily accomplished by using tap water as an external cooling unit baffles us and our credulity is inordinately exercised to believe that the reason why 30°C has not been generally adopted in this country is because 25°C can be maintained by this device 'for most of the year'. Presumably for that part of the year when it is not practicable one must patiently await the return of more favourable climatic conditions.

The same logic is applied to the reactant concentrations. The substrate concentrations employed by Henry, Chiamori, Golub, and Berkman (1960) at 32°C, as modified for alanine transaminase by Arvan and Coyle (1969) at an unstated temperature, are proposed for use at 25°C, even though it is acknowledged that the L-alanine (and hence 2-oxoglutarate) concentration is suboptimal. We have found no solubility problems with 810 mM L-alanine nor have Boehringer or Merck in their commercial test kits. This problem has been artificially created by the use of separate buffer and substrate solutions, aided by the unnecessary suggestion that D,L-alanine can be used.

On 50 sera, using the Boehringer 'optimized' test combinations (Bergmeyer and Bernt, 1963), we have found very similar temperature correction factors between 25°C and 37°C, that is, 2·18 and 1·93, with coefficients of variation 4·45% and 7·8 (King, 1973a) for aspartate and alanine transaminase respectively. These figures indicate that considerably more than caution should be exercised in using temperature conversion factors. Our normal ranges for aspartate and alanine transaminase by optimized assay at 37°C are 13-42 mU/ml (females 10% lower) and 11-55 mU/ml (females 25% lower) with respective coefficients of variation of 2·0 to 5·0% and 1·5 to 5·9% depending upon the level of activity and whether the determinations were within-batch or between-batch (King, 1973b). With the lower activities obtained at 25°C the quality of our results was poorer but the normal range of activities approximated that given in the Boehringer literature and considerably higher than that obtained by Wilkinson and colleagues (1972). We can therefore emphatically state that the techniques recommended do not 'give maximal activities' as claimed by the Working Party.

Again we are perplexed to understand why the lack of specificity in the recommended transaminase assays brought about by the presence of glutamate dehydrogenase should only be mentioned in a footnote concerning liver extracts. The same source of error is present in sera where hepatic necrosis is present but is readily overcome by using lactate and

malate dehydrogenase in glycerol rather than ammonium sulphate solution. When the ability to overcome an obstacle is at hand there seems little point in continuing to flagellate oneself. The same applied to the presence of apotransaminase in the purified enzyme preparations. This was valid and indeed critical in 1959 (Rosalki and Wilkinson) but is no longer applicable to reputable sources today.

As previously recorded (King, Henderson, and McQueen, 1972), this report contradicts that earlier published by the German Society of Clinical Chemists (1970) and would have been better submitted to the Expert Panel on Enzymes of the International Federation of Clinical Chemistry for consideration during their deliberations on standard methods of assay. As it is we are somewhat apprehensive at this interference by a Government department through one of its advisory group's subgroup's working party in the international discussions and search for agreement by clinical biochemists.

J. KING
Department of Biochemistry, Royal Infirmary, Glasgow, G4 0SF
A. R. HENDERSON
Department of Clinical Biochemistry, University Hospital, London, Ontario, Canada

References

Arvan, D. A., and Coyle, M. (1969). Serum and tissue alanine aminotransferase (GPT) based on optimal substrate concentration. *Clin. Chem.*, **15**, 785.

Bergmeyer, H. U., and Bernt, E. (1963). In *Methods of Enzymatic Analysis*, edited by H-U. Bergmeyer, pp. 46-850. Academic Press, New York and London.

German Society of Clinical Chemists (1970). Standardization of methods for the estimation of enzyme activity in biological fluids. *Z. klin. Chem.*, **8**, 658-660.

Henry, R. J., Chiamori, N., Golub, O. J., and Berkman, S. (1960). Revised spectrophotometric methods for the determination of glutamic-oxalacetic transaminase, glutamic-pyruvic transaminase, and lactic acid dehydrogenase. *Amer. J. clin. Path.*, **34**, 381-398.

King, J. (1972). On a standard temperature for clinical enzymology. *Ann. Clin. Biochem.*, **9**, 197-202.

King, J. (1973a). In *Clinical Biochemistry: Principles and Methods*, edited by H-Ch. Curtius, and M. Roth. De Gruyter, Berlin. (In press).

King, J. (1973b). The LKB 8600 reaction rate analyzer in hospital practice. In *Proceedings of the 2nd Colloquium on Automatisation and Prospective Biology*, edited by G. Siest. (In press).

King, J., Henderson, A. R., and McQueen, M. (1972). Abbreviations for names of diagnostic importance. *J. clin. Path.*, **25**, 369.

Rosalki, S. B., and Wilkinson, J. H. (1959). A source of error in serum glutamic-oxaloacetic transaminase determination. *J. clin. Path.*, **12**, 138-139.

Wilkinson, J. H., Baron, D. N., Moss, D. W., and Walker, P. G. (1972). Standardization of clinical enzyme assays: a reference method for aspartate and alanine transaminases. *J. clin. Path.*, **25**, 940-944.

Book reviews

Pathology of Injury: Current Knowledge and Future Development. The report of a Working Party of the Royal College of Pathologists. Edited by A. C. Hunt. (Pp. 130. £2·00.) London: Harvey Miller and Metcalf, for the Royal College of Pathologists. 1972.

'Pathology of injury' is the report of the Royal College of Pathologists' working party on trauma. Originally announced as 'Pathology of trauma', the title has been changed to avoid confusion with the publication of the same name that has been produced by *this journal* as a supplement for the Royal College. This change is in itself confusing to those of us who cling to the belief that most of pathology represents reaction to injury! Thus we have two paperbacks of similar size and identical price and it is impossible to review 'Pathology of injury' (a blue book) in isolation, as 'Pathology of trauma' (an orange-coloured book) is the report of the symposium on trauma held in 1970. Drs Stoner and Sevitt were editors of this symposium and chairman and secretary of the working party. The blue book has an unadorned text on thick pages that have the irritating tendency to spring shut, like a continental paperback, unless the binding is put under severe strain; the orange book contains twice as much text and is well produced and illustrated in the style of the journal that you are now reading.

Comparisons are bound to be made but these two books are complementary. The blue 'Pathology of injury' divides traumatic injury into 17 chapters, many of which were subjects of the symposium reported in the orange 'Pathology of trauma'. This report reviews the state of knowledge of pathology of trauma, stating the facts concisely and quoting useful references. As a wide and general review, it is an excellent source of information that is not readily available elsewhere. In its terms of reference, the working party was required to assess those areas where knowledge is limited, and it has identified in the text a number of subjects that require research. From the first sentence, which is emotive, the report appears to accept that it is a political document and to appreciate that its recommendations for expanded interest and research into injury will have to compete for financial support and manpower