Letters to the Editor

RED CELL SUSPENSION AS A WORKING STANDARD

In response to a number of requests, we would like to make available details of the preparation of a red cell suspension which we have found satisfactory as a working standard. This preserved red cell suspension can be used for manual methods as well as for electronic counters and multichannel equipment such as the SMA-4/7a.

The suspension is prepared as follows:

One litre of Alsever's solution is modified by the

40 to 45%. The suspension is dispensed into 25 ml aliquots and stored at 4°C. It has been found important to resuspend the red cells by inversion daily, to ensure constant contact of the cells with the medium.

Aliquots of this preparation have been examined at weekly intervals by estimating the parameters which it is designed to control, *ie*, haemoglobin, red cell count, and packed cell volume, and the results are given in Table I. Mechanical fragility has also been studied and shows no significant alteration during the four-week period.

In our department, daily standardization of the SMA 7a requires 2 ml of the suspension, and a similar amount is passed through after every 20 blood samples in order to detect instrumental drift. Thus with a daily turnover of about 200 specimens, 20 to 25 ml of the red cell suspension is required for each working day.

TABLE I

STABILITY DURING STORAGE AT 4°C

Week	Haemoglobin (g/100 ml)	Red Cell Count (10 ⁶ /cmm)	Haematocrit (%)	MCV (cμ)	MCHC (%)
1	13.1 ± 0.21	4.34 ± 0.23	40.0 ± 0.00	90.0 ± 3.4	32.6 ± 1.3
2	13.0 ± 0.24	4.43 ± 0.54	40.0 ± 0.00	90.0 ± 1.6	32.4 ± 1.1
3	13.1 ± 0.12	4.46 ± 0.26	40.0 ± 0.00	90.0 ± 1.9	32.8 ± 1.2
4	13.1 ± 0.28	4.48 ± 0.27	40.0 ± 0.00	89.0 ± 1.8	32.6 ± 1.3

¹Mean of 25 replicate observations ± 2 SD

addition of 3.6 g of inosine and 240 mg of adenine. From this solution 200 ml is transferred to a standard 550 ml blood bottle and autoclaved. In order to maintain sterility an aseptic technique is employed at all subsequent stages. Next 340 ml of blood from a healthy donor is collected into this anticoagulant. The blood is then centrifuged at 1,200g for 20 minutes and approximately half the supernatant is removed to give a packed cell volume of

This standard has the advantage of being easily prepared, and of maintaining viable red cells apparently unaltered for a period of at least four weeks. This limited storage life does not present a problem in our hands, since each batch prepared as described is sufficient for about three weeks of routine use.

J. M. CARVILLE
D. LEE

FALSE POSITIVE CURRY'S TEST

We wish to draw attention to a cause for a false positive Curry's test for barbiturates in gastric contents which appears not to have been previously reported.

Recently we suspected barbiturates to be present in gastric contents because of a strongly positive colour reaction with Curry's test (ACP Broadsheet no. 52, 1966).

Subsequent investigation showed that Slow K (Ciba) contains a substance which reacts with the reagents in Curry's test to give an orange colour. This substance is extracted in the A₂ fraction of Clarke (1969) so that this preliminary procedure used to separate barbiturates from the other acid and neutral drugs is of no help in removing the contaminant. This interfering substance is not potassium chloride and is detectable at a concentration of 1 tablet in 10 ml water but not at half this concentration.

The substance does not give a colour reaction with cobalt acetate-lithium hydroxide nor does it interfere with barbiturate ultraviolet spectrographic absorption

bands (Clarke, 1969).

A. W. MATTHEWS and P. G. I. STOVIN

REFERENCE

Clarke, E. G. C. (1969). *Isolation and Identification of Drugs*, pp. 8 and 908. Pharmaceutical Press, London.

DR A. S. CURRY replies:

Curry's test to which the writers refer is for barbiturates in blood and not in gastric contents. This fact is implied in the title of the original paper and specifically pointed out on page 30 of Curry's 'Poison detection in human organs', 2nd edition, 1969 (Charles C. Thomas, Publisher). ED.

MONOVALENT TYPHOID VACCINE

We felt that it might be timely to remind readers of the Journal that a monovalent typhoid vaccine is being marketed and used in this country (Burroughs Wellcome & Co.), and that its use may give rise to confusion in interpreting the results of a single Widal reaction. Most pathologists are accustomed to interpreting a serological

Book reviews

FETAL MALFORMATIONS CAUSED BY AMIOTIC RUPTURE DURING GESTATION. By Richard Torpin. (Pp. 159 + xii; illustrated. \$11.50.) Springfield, Illinois: Charles C. Thomas. 1969.

This monograph is concerned with foetal malformations which are thought to result from amniotic rupture during pregnancy. The first two-thirds of the text are devoted to the consideration of a wide variety of such malformations, ranging from gross deformities in which there may be direct attachment of the placenta and its membranes to the foetal skull, to less serious deformities such as club-foot, clubhand, and syndactylism. The majority of cases referred to in this section have been culled from the literature, and illustrations consist of line drawings which are not always very informative. An attempt has been made to separate malformations resulting from genetic malfunction or teratogenic agents from those of a more mechanical origin consequent upon amnion rupture. It is thought that the latter may occur at any stage during gestation, and although the amniotic membrane may be severely damaged the chorion remains intact. Remnants of the detached amnion, often in the form of constricting bands, may attach themselves to parts of the foetus and may even lead to amputation of a limb.

Another consequence of amniotic rupture may be a transient oligohydramnios. Compression effects of the uterine wall on the foetus in the absence of a cushion of amniotic fluid are alleged to result in a high incidence of clubfoot deformities in these cases. This is closely analogous to the theories regarding clubfoot deformities held by the late Sir Denis Browne.

The remainder of the monograph is devoted to a description of a method devised by the author for the study of the placenta and its membrane after delivery, and a fairly detailed description of 14 cases of foetal deformity studied personally. This section is well illus-

trated with clinical photographs, radiographs, and photomicrographs of normal and abnormal amniotic membrane.

The author has rightly drawn attention to a fascinating series of lesions which may culminate in severe foetal malformation or even intrauterine death of the foetus. It is possible, however, that not all the deformities result only from rupture of the amniotic membrane, and the cause of the latter remains obscure.

A. E. CLAIREAUX

MALIGNANT LYMPHOMAS AND THEIR MANAGEMENT By M. Jacobs. In 'Recent Results in Cancer Research', Vol. 16. (Pp. vii + 48; 7 figures. DM.18). Berlin, Heidelberg, and New York: Springer-Verlag. 1968.

The author has compressed into 44 pages most of the essential information about the common lymphomas. The subject is treated with a deep historical perspective—31 out of 95 references precede 1930—leading up to an admirable, concise summary of modern ideas on histological classification and staging. The prospect of general and fairly permanent agreement in these aspects of the lymphomas gives this part of the book a lasting value.

The same is unlikely to be the case with sections devoted to management where practice is changing and finality is many years away. The author gives an excellent account of the radiotherapeutic controversy in Hodgkin's disease but is less than adequate in dealing with chemotherapy. Drugs such as vinblastine, steroids, and methyl hydrazine are scarcely mentioned and there is no reference to the principles of combination chemotherapy.

Thus, despite many virtues, this presentation is insufficient and already out of date as a guide to the complete management of patients with lymphomatous disease. It compels one to question the real value of monographs of this sort. The 'Union internationale contre le cancer' have no less than 27 in preparation. How many will be of more than ephemeral value? Will any of them perform a service not already supplied by existing journals and new editions of textbooks? And if they do not, are they perhaps more of a liability than an asset upon the overloaded library shelves?

H. E. M. KAY

VISTAS IN CONNECTIVE TISSUE DISEASES Edited by J. Claude Bennett. (Pp. xii + 314). Springfield, Illinois: Charles C. Thomas. 1968.

Dr Bennett and 11 co-authors have aimed to 'acquaint young physicians with research opportunities in connective tissue diseases and to make them excited about this work'. They have succeeded admirably and this collection of 11 essays should appeal to all laboratory workers concerned with connective tissue.

Inevitably, there is bias in the selection of subjects for critical discussion. Immunology rates six of 11 chapters (152 of 268 text pages). The remaining five chapters are very diverse. The outstanding contribution (in terms of the Editor's stated aims) is a sparkling account by McCarty of his researches on urate and pyrophosphate crystal arthritis, an account which is sufficiently enthralling to be recommended reading for medical students as well as researchers. The kinetics of antigen/antibody

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reaction to *S. paratyphi A* as indicative of previous vaccination; when monovalent vaccines are used, this marker is no longer available. The finding of high-titre typhoid antibody, whether or not accompanied by other antibody, is not an indication, in itself, for initiating epidemiological investigations; careful and prompt enquiry into vaccination history is required. It should be borne in mind that the patient's general practitioner or physician is not always *au fait* with the full vaccination history, as vaccinations may be advised and undertaken by firms or employing authorities or airline medical services. The patient should be asked about all previous immunizations.

ROSALIND HURLEY

Secretary, Microbiology Committee,
Association of Clinical Pathologists