Figure Diagram of segment of standard LKB sample rack (left) and modified rack (right). Note the extra height, longitudinal groove, and reagent cavity of the modified rack.

The operating procedure is simple. For addition of antiserum or labelled antigen, the reagent is placed in one of the cavities of the Teflon rack. The pumps are primed with buffer and the programme is initiated. The recipient tube racks are fed in automatically and, as the sampler cogs begin to turn,

the Teflon rack is pushed into the sampler position by hand. The sampler now operates repetitively to aspirate and dispense reagent with buffer into the recipient tubes as they move past normally. At the end of the run the Teflon rack is pushed out, again manually.

With this system only the sampler tip comes in contact with the reagent, and cleaning is easy and rapid. One possible problem is that the cavities in the rack might become contaminated and affect subsequent reagents. This could be avoided by using disposable plastic liners or containers in the cavities. We have found cleaning with a detergent, for example, Contrad (Hickman & Kleber; SA) to be adequate.

Another point to consider is whether or not significant evaporation occurs from the cavities. The mass of 20 ml of water in the large cavity (surface area $\pm 8.5 \text{ cm}^2$) at 22°C decreases by $\pm 0.2\%$ over 1 hour. As all the reagents used are aqueous and the processing time of 200 tubes is 30 minutes this does not create a problem.

All but $\pm 0.3 \text{ ml}$ of reagent is recovered from this rack. The reproducibility of sampling depends on the pumps and will not be affected by this modification.

Letter to the Editor

Unusual Megaloblastic Anaemia

Saary *et al* (1975) describe two cases of unusual megaloblastic anaemia and review the relevant literature. In discussing the mechanism of the changes they suggest that folic acid deficiency may have made a minor contribution to the blood abnormalities but they consider that some other disturbance in erythropoiesis is responsible for the bizarre blood and bone marrow appearances.

We have recently described two cases of rapidly developing megaloblastosis in our intensive therapy unit (Ibbotson *et al*,

1975). In addition, we have seen other patients who developed a similar though less florid megaloblastosis during intensive therapy. The syndrome has been associated with major surgery or trauma, renal failure and dialysis, and severe infection with the use of antibiotics. In three cases, megaloblastosis was diagnosed within three weeks of admission of previously healthy patients.

Diagnosis has been difficult because of a lack of macrocytosis, most of the patients having been transfused. Thrombocytopenia has been a constant feature and has been very severe in two cases. The white cell changes of megaloblastosis have been present in some cases but these could be attributed to renal failure (Hampers *et al*,

1967). Vitamin B12 and folic acid assay have been inhibited by antibiotics. One patient developed a leucoerythroblastic anaemia with circulating megaloblasts, as in Saary's cases, but the striking feature in common has been the morphology of the bone marrow megaloblasts including multilobed nuclei and basophilic stippling (figure, see page 1008).

In our two published cases we were able to give an adequate course of folic acid and both responded within seven days, the platelet count returning to normal and the bone marrow becoming normoblastic. We therefore feel that folic acid deficiency plays a major role in this problem but the aetiology of the deficiency remains uncertain.

Figure Bone marrow appearances showing multilobed erythroblast nuclei and basophilic stippling.

Book reviews

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This emphasizes an important therapeutic point although the megloblasts in the cases of Saary *et al* appear to be in a different class of bizarreness.—Ed.

Guide to the Laboratory Diagnosis of Trachoma. Prepared by the participants in a WHO Symposium. (Pp. 40; 16 figures; Sw. Fr. 12.) Geneva: World Health Organization. 1975.

This booklet is intended to provide 'a system of recommended procedures for use especially in the diagnosis of trachoma in countries where . . . laboratory facilities may be limited'. Methods are given for collecting specimens, detecting inclusion bodies by ordinary and fluorescence microscopy, isolating *Chlamydia*, and typing strains. The amount of detail makes this a useful working manual; but sophisticated techniques such as the use of irradiated cell cultures for isolation and serotyping by microimmunofluorescence are over-emphasized at the expense of simpler methods such as iodine-staining of inclusions and isolation in unirradiated cells. One of the most valuable features is the set of good colour photographs of Giemsa-stained inclusion bodies and other intracytoplasmic objects that may be mistaken for them.

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Human Viral Hepatitis. By A.J. Zuckerman. (Pp. x + 422; illustrated, \$49.95) Amsterdam: North Holland Publishing Company. 1975.

The second edition of *Human Viral Hepatitis* is twice the length of the first, which appeared only three years ago as *Hepatitis Associated Antigens and Viruses*. The new title does not reflect a major change in approach. The original material has been revised and pruned, while the general balance of the book has been improved by its expansion to include a description of the newly recognized particles found in the faeces of patients with hepatitis A.

As before, each chapter consists of a review of the published work on a limited topic, and the reference lists will provide useful guides to the English language literature. The new illustrations are well chosen but several of the electron micrographs of hepatitis B show similar features and could be deleted. The reviews are written from an essentially academic standpoint and consist largely of abstracts of the findings reported in individual