Any Questions?

We publish below a selection of those questions and answers which seem of general interest. It is regretted that it is not possible to supply answers to all questions submitted.

P.A.S. and Pulmonary Tuberculosis

Q.—*Is calcium B-P.A.S. as effective in the treatment of pulmonary tuberculosis as sodium P.A.S.?*

A.—The most important effect of P.A.S. and its analogues in the treatment of pulmonary tuberculosis consists in its ability, when given in combination with streptomycin or isoniazid, to diminish the liability to the emergence of strains of tubercle bacilli resistant to the other drugs. The therapeutic effect of P.A.S. alone is considerably less than that of the other two commonly used drugs. There is good evidence that tolerable doses of sodium P.A.S. are effective in this respect.[1,2] The best way of estimating the effectiveness of any alternative preparation to the sodium salt of P.A.S. is therefore to determine whether, when given in combination with streptomycin or isoniazid to patients whose organisms are known to be sensitive to the drugs used, it prevents the emergence of drug-resistant organisms. Evidence on this point is difficult to obtain, but Lewis[3] has reported 39 patients who were admitted to hospital with pulmonary tuberculosis and were treated with calcium benzamidosalicylate (Ca B-P.A.S.) with streptomycin or isoniazid for a period of at least two months. Of these, at least 20 subsequently produced tubercle bacilli with impaired sensitivity to one or more of the standard drugs. Since some of these patients had been treated as out-patients, it was possible that not all of them had taken the prescribed drugs conscientiously, and drug sensitivity tests had not been done in most of them before treatment was begun. However, seven of them were known to have taken the drugs conscientiously and to have had sensitive organisms before Ca B-P.A.S. was given. In three of these bacilli resistant to isoniazid or streptomycin emerged, and in two more sputum remained positive after six or eight months of treatment, although sensitivity tests were not done. These results indicate that, in the commonly used dose of 14 g. a day, Ca B-P.A.S. is ineffective in preventing the emergence of organisms resistant to streptomycin or isoniazid. Undissociated B-P.A.S. is very much less active than P.A.S.[4] and its therapeutic activity therefore depends upon its conversion to P.A.S. Lewis points out that, assuming complete hydrolysis of Ca B-P.A.S. and with the knowledge that only about 60% of an oral dose is absorbed from the gut, the dose of Ca B-P.A.S. which would correspond with the dose of sodium P.A.S. known to be effective in preventing emergence of resistance to streptomycin is no less than 48 g. daily. This is far above the customarily used dose, and it is doubtful if it would be tolerated any better than the normal dose of sodium P.A.S.

REFERENCES

1 Medical Research Council, *Brit. med. J.*, 1953, **2**, 1005.
2 —— ibid., 1955, **1**, 435.
3 Lewis, D. O., *Tubercle (Lond.)*, 1958, **39**, 247.
4 Schönholzer, G., Lauener, H., and Hurni, H., *Schweiz. med. Wschr.*, 1955, **85**, 222.

Oedema in Pre-eclampsia

Q.—*Is it necessary to restrict the fluid intake in the control of oedema in pre-eclampsia?*

A.—The best method of controlling the oedema of pre-eclampsia remains in doubt. Indeed, it is not established that the control of oedema *per se* by any method is of value. It is, nevertheless, usually attempted.

If the oedema is associated with sodium retention, as is generally believed to be the case in pre-eclampsia, the complete elimination of sodium from the diet is theoretically all that is necessary. In fact, however, a sodium-free diet is impracticable. It is therefore usual to compromise by restricting both sodium and fluid. The avoidance of fluid to the extent of causing dehydration could be harmful and even result in uraemia. This is unlikely to happen in a waterlogged patient, in whom experience shows it to be safe to reduce the intake of fluid to 20 oz. (0.6 l.) in 24 hours. Moreover, no harm can result when the amount of fluid allowed is related to the output of urine, this being the usual clinical guide. The recently introduced orally administered saluretics (e.g., acetazolamide and chlorothiazide), unlike the older mercurial diuretics given by injection, do control the oedema of toxaemia. Chlorothiazide acts by increasing chloride excretion and this in turn raises the sodium output. It also has a less important action as a carbonic anhydrase inhibiting factor. This new drug is therefore being used without restricting the patient's intake of either fluid or sodium. However, in the presence of liver damage (and especially if the sodium intake is restricted) chlorothiazide can produce dangerous hypokalaemia.[1] There is, therefore, some reason to question the advisability of using this drug in the treatment of pregnancy toxaemia unless the patient is having potassium supplements and is in a hospital, where the electrolyte balance can be constantly controlled. Even then its value is doubtful. Although it may improve maternal oedema it does not—as judged by foetal survival rates—benefit significantly the underlying disease.[2]

There is at present probably no better method of controlling the oedema of pre-eclampsia than by physical rest (which is indicated on other grounds and which in itself promotes elimination of fluid), by reasonable restriction of the intake of fluid and sodium, and possibly by administering a simple sodium replacement diuretic such as ammonium chloride.

REFERENCES

1 Read, A. E., Haslam, R. D., Laidlaw, J., and Sherlock, S., *Brit. med. J.*, 1958, **1**, 963.
2 Bayliss, R. I. S., *Practitioner*, 1958, **181**, 216.

Treatment of Rabies

Q.—*Where may supplies of rabies vaccine and serum be obtained in England and Wales?*

A.—A Ministry of Health memorandum (H.M.(58)66), August, 1958, lists the following centres as holding supplies of rabies vaccine (for active immunization) and serum (for immediate passive protection) for the treatment of rabies.

London.—Central Public Health Laboratory, Colindale Avenue, N.W.9 (Colindale 7041).

Newcastle.—Public Health Laboratory, or Institute of Pathology, General Hospital, Westgate Road, Newcastle, 4 (Newcastle 38811).

Liverpool.—Public Health Laboratory, 126, Mount Pleasant, Liverpool (Royal 3636).

Wales.—Public Health Laboratory, Institute of Preventive Medicine, The Parade, Cardiff (Cardiff 29110 and 23967).

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