

diagnose this most easily resectable of all abdominal neoplasms before life is threatened by the advent of acute obstruction.—I am, etc.,

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REFERENCES

- ¹ Haggie, M., *Lancet*, 1952, 1, 21.
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SIR,—In assessing a patient with gastro-intestinal disturbance the present tendency to hasty reference for barium meal or enema examination is to be deprecated. Such action increases the already significant waiting-list for these procedures which are, moreover, increasingly expensive and time-consuming as techniques elaborate and improve. Furthermore, as Drs. A. N. Exton-Smith and G. Osborne indicate (June 24, p. 1799), the aged or frail patient may find the investigation an ordeal and occasionally, in the case of the barium enema examination, a peril. Nor can one disregard the recommendations of the Adrian Committee on reducing radiation dosage.

I agree with Drs. Exton-Smith and Osborne that tests for faecal occult blood give some guidance in the selection of cases for such examinations. Unfortunately they do not state which method they employ. The modern orthotoluidine tablet tests of the Ames Company (London) Ltd. are simple and inexpensive and their use makes for a rapid, efficient, and relatively inoffensive examination of the patient's faeces. They are therefore of particular value in the investigation of an iron deficiency anaemia which may be caused by gastro-intestinal bleeding.

In general practice such anaemias are often severe, and a search for occult blood loss will coincide with the administration of oral iron. As I previously indicated,¹ this is correct procedure with all preparations except ferrous fumarate tablets, which lead to the development of false-positive results in the faeces. Drs. T. D. S. Halliday and I. M. Cuthill substantiated this belief,² recorded the confusion which such misleading results had caused, and advised that published information on that preparation should carry a warning of this obstacle to its use. I have now appraised a newcomer to the field of iron therapy: a preparation of ferrous carbonate and ascorbic acid ("ferrodic"). This product also causes false-positive results for occult blood, *in vivo* and *in vitro*, with either the obsolescent benzidine test or the orthotoluidine tablet tests. In view of the experiences of Drs. Halliday and Cuthill, I feel that awareness of this fact may help to reduce the number of patients referred for barium examination.—I am, etc.,

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- ¹ Illingworth, D. G., *Brit. med. J.*, 1959, 2, 1099.
- ² Halliday, T. D. S., and Cuthill, I. M., *ibid.*, 1960, 2, 1310.

Antimony and the Heart

SIR,—Your annotation, "Antimony and the Heart" (June 10, p. 1665), very properly underlines the possibility of cardiovascular damage following treatment of schistosomiasis with trivalent antimonial substances. To this may be added the possibility of liver damage. While few would disagree that tartar emetic or antimony sodium tartrate *given in the classical course* are the most active (and most toxic) schistosomicides at present

available, it must be appreciated that the conditions under which they are used materially affect their efficacy and practical value in treatment.

In areas endemic for schistosomiasis, hospital facilities are usually grossly inadequate and only a small proportion of those requiring treatment can be admitted. The majority must be treated as ambulatory out-patients pursuing their normal occupations. Under these conditions, a thrice-weekly course of intravenous injections of tartar emetic may extend over four weeks. Cardiovascular toxicity apart, the side-effects are frequently severe, the interruption to earning capacity is considerable, and, unless under compulsion, many patients may fail to complete the prescribed course. The over-all efficacy of tartar emetic may be seriously affected by the lengthy course of treatment and the severity of side-effects.

Unlike bacteria, schistosomes do not multiply within the body and, in general, the pathological effects of the disease are related directly to the weight of infection and to its duration. Thus, in an endemic area where reinfection is likely, the virtue of striving after radical cure through the prolonged use of highly toxic drugs is open to doubt. It is in this context that the use of other trivalent antimonials such as sodium antimonyl-gluconate, B.P., may offer the advantages of reduction in length of treatment and reduced severity of side-effects, leading to a lower incidence of absenteeism and, consequently, a lower level of infection in the population under treatment.

The absence of more effective and less toxic schistosomicides is only too apparent, but, meanwhile, the need to treat demands the use of available drugs to best advantage in the prevailing circumstances. Trivalent antimony has been in use for the treatment of schistosomiasis for more than 40 years, but there is still need for controlled quantitative studies with the available preparations to define the least toxic and most effective regimen.—I am, etc.,

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Cataract and Steroids

SIR,—Following a report by Black *et al.*¹ and also your annotation (January 14, p. 116) on the occurrence of posterior subcapsular lens opacities as a possible complication of steroid therapy; we have examined 98 asthmatic patients receiving steroids. Fifty-two of these had received prednisone or an equivalent analogue in a daily dose of 10 mg. or more for one year or more. Of these, 41 received continuous therapy for between one and three years and 11 for between four and nine years. All of these patients were examined under a mydriatic by ophthalmoscopy and slit-lamp microscopy.

One case only showed bilateral, posterior, polar, subcapsular cataracts. This was a female patient aged 60 years who had received more than 10 mg. of prednisone per day for three years. However, elsewhere, she had also been given many courses of gold intramuscularly in the treatment of her asthma. This may be relevant to the development of her cataracts. Moreover, in this case, in addition to subcapsular changes, opacities were present in the posterior cortex of the lens mainly localized to the axial region. Therefore the changes in this one case are of doubtful aetiology, nor quite typical of those described by Black *et al.*

Four other patients, all of whom were receiving prolonged steroid treatment of more than 10 mg. per day, showed minimal changes at the posterior pole amounting to coarsening of the pattern of the lens fibres. These changes have been seen in patients not receiving steroids and their significance is doubtful.

Apart from the 98 patients in the above group, two patients have been seen in the Eye Department for other reasons and found to have changes typical of those described by the American workers. Both of these patients were suffering from rheumatoid arthritis and had received large doses of steroids over periods of one year and four years.

Our findings would seem to indicate that from the point of view of cataract formation there is no contra-indication to the prolonged use of steroids in the treatment of asthma. The posterior subcapsular cataracts described by Black *et al.*, the single case found by Dr. F. Dudley Hart and his colleagues (June 10, p. 1680), and our two cases occurred in patients with rheumatoid arthritis treated with steroids. It is possible, therefore, that besides prolonged exhibition of steroids an additional factor in the disease process, perhaps peculiar to a collagenosis, is necessary for the development of this particular lesion.—We are, etc.,

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Prevention of Rh Haemolytic Disease

SIR,—Dr. Frank Marsh (June 10, p. 1682) draws attention to the fact that ABO-incompatible foetal cells, even if rapidly destroyed, should still be capable of stimulating Rh antibodies, as the products of the cell destruction will enter the maternal reticulo-endothelial system and will not just “go up in smoke.” There is, however, a very considerable body of evidence to show that Rh-antibody formation is not in fact stimulated by such ABO-heterospecific pregnancies. One explanation of this anomaly is that the foetal cells are rapidly removed to phagocytic cells which break down the antigens before they have time to sensitize the antibody-producing tissue. On the other hand, a dosage effect may be involved—one large dose of antigen being less effective in sensitizing than repeated small doses. It is therefore reasonable to postulate that if foetal cells can be rapidly destroyed on entering the maternal circulation, then sensitization will not occur. If we can effect this and antibodies do not develop, there will be support for this hypothesis. On the other hand, if antibodies develop in spite of rapid elimination, the whole mechanism by which ABO-incompatibility protects will have to be reviewed.

We do not know if Race and Sanger¹ had any particular mechanism in mind when they used the word “elimination,” but we use it without any specific connotation, not necessarily meaning haemolysis, as we do not know precisely how foetal cells are removed from the maternal circulation by different kinds of antibodies.

Dr. Marsh is no doubt aware that steroids have been used extensively with a view to protecting the baby in sensitized mothers, but according to Wiener² they are not effective. Whether or not these compounds would prevent initial Rh-sensitization is another matter, but

we very much doubt whether even very large doses of steroids would induce a “total immunological paralysis,” and the widespread use of massive steroid doses would entail a considerable degree of risk to the mother.

We entirely agree that it would be unwise and probably ineffective to inject incomplete antibodies into pregnant women, but what we suggested in the paper was that it *might* be helpful to give anti-D after the birth of the child to those women in whom transplacental haemorrhage of some size had been demonstrated.—We are, etc.,

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REFERENCES

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² Wiener, A. S., *J. Amer. med. Ass.*, 1954, **155**, 63.

Cortical Mastoidectomy

SIR,—With reference to the article by Mr. R. S. McAlary entitled “Cortical Mastoidectomy is Still Necessary” (June 24, p. 1802), I object to the diagnosis of acute mastoiditis in some of the cases presented—namely, 5, 6, 7, 8, 9, and 12. These “failed antibiotic” cases had only a persistent aural discharge from which sensitive organisms were isolated. I have time and again admitted young patients similarly treated with oral penicillin, and achieved dry ears and normal hearing with parenteral penicillin and attention to the upper respiratory tracts where the cause is often found. From a pathological standpoint, mastoid air-cells are often involved in the course of an acute otitis media, but as long as infection is confined to the lining membrane, even to the extent of forming granulations, we do not speak of mastoiditis because the process is still reversible. Mastoiditis means bone necrosis.

Mr. McAlary's description of some of his cases and criteria to operate is at times confusing. I am singling out: (1) The acute flare-up of a chronic otitis media followed by a brain abscess. Was the disease of the attico-antral type, in which case a form of radical mastoidectomy and not a cortical one should have been done? (2) The mastoid which became reinfected after operation suggests that a timely adenoidectomy would have been more adequate. On these grounds I think Mr. McAlary's paper fails to make a case for more frequent cortical mastoidectomies.—I am, etc.,

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Geographical Pathology

SIR,—Dr. Richard W. Payne's paper on “Pernicious Anaemia and Gastric Cancer in England and Wales” (June 24, p. 1807) was of interest to me as I was recently stimulated into making a similar inquiry regarding post-cricoid carcinoma. I have come across several patients with pernicious anaemia who developed post-cricoid cancer from two to fifteen years after their anaemia had been diagnosed, and the possibility arises of an association between the two conditions.

Since 1957 the Registrar-General has coded post-cricoid carcinoma as a distinct cause of death, and with the aid of figures he has kindly provided it has been possible to determine the mortality from this condition