in its longest diameter was the only lesion detected. No evidence was found by which the latter could be aetiologicaly linked to the hydrops. In particular, there were no "vulnerable" blood vessels of the placenta that might have been obstructed by the tumour, nor was this excessively vascular, so that an arteriovenous shunt effect was unlikely. Explanations such as protein sequestration by the tumour or unduly rapid increase in the vascular bed are within the realms of speculation.

This association has not been reported before so far as I am aware (although Macafee *et al*⁵ mention a case of mediastinal teratoma associated with hydrops) and I should be grateful to hear from any of your readers who know of a similar observation.

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¹ Lie-Injo, L E, *et al*, *British Journal of Haematology*, 1962, 8, 1.

² Halle, H, *Zentralblatt für Gynäkologie*, 1972, 94, 1487.

³ Turbeville, D F, *et al*, *Obstetrics and Gynecology*, 1974, 43, 567.

⁴ Kohler, H G, Unpublished observation.

⁵ Macafee, C A J, *et al*, *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1970, 77, 226.

⁶ Alexandrowsky, A, Inaugural Dissertation, University of Berne, 1916.

⁷ Tait, L, *Transactions of the Obstetric Society of London*, 1875, 17, 307.

⁸ Jones, C E M, *et al*, *Pediatrics*, 1972, 50, 901.

Death from asthma

SIR,—I am guilty—according to Dr E Posner (17 July, p 179)—of a most heinous crime: that of not referring every asthma patient to a "specialist." While I fully agree that patients with severe recurrent symptoms require specialist supervision, I am rather annoyed by the implication that we general practitioners are incapable of dealing with mild to moderate asthma.

Do we really need to send this latter group to a consultant chest physician for diagnosis and follow-up for "at least five years"? Perhaps Dr Posner should be reminded that we have access to simple but useful investigations such as differential white cell counts, chest x-rays, peak flow meters, and Vitalographs—and some of us even use them. If it is not justified to send them to a chest physician, then should they perhaps see a general physician? Consultant physicians probably see no more cases of mild to moderate asthma than do most GPs. Certainly this is true of the junior hospital doctors—in many cases housemen and senior house officers—who see the majority of the "follow-ups."

Perhaps Dr Posner did not really mean every patient, but none the less he might add to his list of quotations as follows: "Not all asthma that is treated in general practice alone is done so negligently" (Woodward, 1976).

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"Comprehensive Psychiatric Care"

SIR,—Your reviewer of *Comprehensive Psychiatric Care*, my old colleague and opponent Dr H R Rollin (24 July, p 242), has been a distinguished contributor for many years. His review will appeal to those who still regret the passing of the medical superintendent and the

style of life in mental hospitals which that system represented. Criticising the present concept of multidisciplinary team work Dr Rollin says, "in the bad old days pretty much the same relationship existed between those parties who worked together towards a common end." In the "old days" the medical superintendent controlled the nurses, administrators, social workers, and doctors. Perhaps Dr Rollin has not noticed that the first three are now independent professions and that doctors prefer a more democratic system. Whether doctors like it or not, decisions on patterns of care for patients can be reached only by agreement with the other professions and not because the doctor has authority over them. His criticisms are of importance because they stem from a failure to come to terms with the new relationships between the professions. This failure is not uncommon and explains many of the current complaints by doctors about nurses, social workers, and administrators and complaints of the latter about doctors.

His comments on leadership do not correspond with the opinions expressed in the book—for example, page 211 commences with, "The consultant has a major leadership role in many situations." He also criticises the nurse's and the administrators' chapters, but it will be of interest to see whether nurses and administrators make the same judgment.

May I suggest that readers compare Dr Rollin's review with that in the *Lancet*¹? Better still, they might read the book for themselves to see whether or not it represents the realities of psychiatry in 1976.

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¹ *Lancet*, 1976, 2, 129.

New look at monoamine oxidase inhibitors

SIR,—Your leading article on this subject (10 July, p 69) practically repeats what we reported over 10 years ago in *Physical Methods of Treatment in Psychiatry*¹ on the value of the monoamine oxidase inhibitors (MAOIs). This knowledge was obtained from the bedside; double-blind testing is useful only for leading astray chairborn professors who are not active bedside observers. What caused so much trouble was that a later Medical Research Council double-blind trial of phenelzine on "Bedlam melancholics," for whom the MAOIs alone are useless, was reported² and this specialised finding got applied to the rest of the anxiety states and lesser depressions.

And now Dr C L Brewer's figures (10 July, p 110) on the increasing number of tricyclic antidepressant suicides and deaths also repeat our similar bedside observations. If a patient feels much worse when given tricyclics during the day don't go on giving them till he tries to kill himself; for this worsening with tricyclics is often an important indication for the substitution of an MAOI, just as it is when a patient with depression still sleeps deeply.

Finally, the results of the trials on combined antidepressants which, you state, are now in progress will depend on the clinical types of depression chosen for them. The combination of MAOIs with other drugs will not work if the patient sleeps deeply but only if he has the type of depression characterised by early morning waking. And the doses of both drugs

must be individually adjusted to the patient's depth of symptoms. How can you double-blind test all this?

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¹ Sargent, W, and Slater, E, *An Introduction to Physical Methods of Treatment in Psychiatry*, 4th edn. London and Edinburgh, Livingstone, 1963.

² MRC Clinical Psychiatry Committee, *British Medical Journal*, 1965, 1, 881.

Detection of scanty blood parasites

SIR,—Mr M Conradie and Professor P Jacobs (17 July, p 181) point out that buffy coat smears are superior to thin films, thick films, and cytocentrifuge techniques for the identification of protozoa in the blood, particularly in the diagnosis of malaria. They ask for comment.

I would like to support their observation and add a few points. Firstly, it is my impression that in buffy coat preparations gametocytes are even better concentrated than trophozoites, and among the latter *Plasmodium vivax* appears also to be preferentially concentrated as compared with *P. malariae* or *P. falciparum*; this must be due to differences in density, and it is known that red cells containing *P. vivax* are swollen. Secondly, as mentioned by your correspondents, the morphology of the parasites themselves and of the surviving erythrocytes containing them is much clearer in buffy coat films than in thick films, which lyse the latter. Thirdly, malarial pigment in phagocytes (monocytes and neutrophils) is more readily identified in such preparations and, especially in partly treated cases, this may be an aid to diagnosis. Finally, giant nuclear masses, which may well be of endothelial origin and indicative of microvascular damage, can best be seen in buffy coat preparations and, given several buffy coat smears from blood samples of the order of 1 ml, can possibly be used to assess the presence of such damage in subtertian, malignant malaria.¹

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¹ Goodall, H B, *Lancet*, 1973, 2, 1124.

Management of acute myocardial infarction

SIR,—Professor J F Pantridge and Dr J S Geddes (17 July, p 168) draw attention to the installation of life support systems in American factories and office blocks, and quote the recent report of the Royal College of Physicians and the British Cardiac Society¹ which advises that lay or medical personnel working in factories should be trained in cardiac resuscitation techniques. Information on the extent of the problem would be useful to those who may be considering the matter. In a recent study of myocardial infarction in an Oxford car assembly plant² we were able to obtain some data on the number of heart attacks occurring at work.

In the seven years 1966-72, with a mean of 8300 male employees, there were six fatal heart attacks at work. The factory operated day and night shifts with cover from a medical centre that was not equipped with facilities for cardiac resuscitation, but another 23 attacks