

factors, including developmental and genetic, may underlie panacinar emphysema, which sometimes affects coal workers and becomes pigmented. Acquired fibrocystic disease, which in coal workers is often heavily pigmented, must also be distinguished from emphysema. On present evidence it is difficult to envisage a primary role for alveolar macrophages in the genesis of emphysema. Even in α_1 -antitrypsin deficiency, an uncommon state in which leucocytic protease activity may be directed against connective tissue, other factors may co-operate in the genesis of emphysema. The occurrence of alveolar fenestrae and capillary damage may merely represent the early stages of secondary non-specific disruption, which when advanced and widespread and possibly complicated by infection renders it difficult to assess the relative significance of the different pathogenetic processes in emphysema.—I am, etc.,

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- ¹ Heppleston, A. G., *Annals of the New York Academy of Sciences*, 1972, **200**, 347.
² Ryder, R. C., Dunnill, M. S., and Anderson, J. A., *Journal of Pathology*, 1971, **104**, 59.

Psychiatry in the Soviet Union

SIR,—Dr. G. Morozov offers us evidence (6 July, p. 40) of the objectiveness of Soviet forensic psychiatry by referring to five dissidents whose mental illness was confirmed by an international team of psychiatrists. We must accept that judgement with respect. Unfortunately, as we are not told how or by whom these five patients were selected it proves nothing about the diagnosis made on other dissidents. Consequently it cannot refute in any way the complaints from distinguished Soviet citizens that many dissidents are still being interned in mental hospitals purely on account of their dissent.—I am, etc.,

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Sex for Medical Students

SIR,—Dr. Ronald Fletcher's analysis of the problems surrounding the introduction of teaching on sex and family planning, outlined in your leading article (29 June, p. 686), is as true for those subjects as it is for many other important aspects of medicine which are still outside the normal medical curriculum.

The problems, though, are not just lack of space in the curriculum and resistance by departments but also a complete lack of communication, not only between the departments themselves but also between them and the students they purport to teach. A telling example of this was in my own medical school, where the department of community medicine gave a lecture on the population crisis followed no less than three months later by a lecture from the department of obstetrics and gynaecology on the subject of contraception, to all intents and purposes a completely separate entity.

The introduction of a multidisciplinary course in contraceptive methods, sex counselling, demography, and population dynamics should not just be an excuse for more lectures. It is hardly surprising that medical students faced with slide after slide of

statistical evidence find more stimulation in a cup of coffee. More use must be made of teaching aids, films, and discussion groups related to clinical situations before students should be expected to take an active interest.

In fact, an extracurricular course on "Understanding Sex" was organized by students at Guy's consisting of lectures, discussion groups, and a film. It was well attended and appreciated by the students, which perhaps indicates that medical students, contrary to the picture of an un-receptive group needing to be cajoled into attendance, feel that sex education does occupy an important place in their course that is not yet filled.—I am, etc.,

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Jaundice after Halothane

SIR,—We wish to comment on the letters from Dr. W. H. W. Inman and Professor W. W. Mushin (27 April, p. 220) and Dr. D. Mansel-Jones (p. 221).

In their report (5 January, p. 5) Dr. Inman and Professor Mushin note that "no technique is available which will with certainty distinguish viral hepatitis from post-anaesthetic jaundice," while in their letters they state that "sufficient information to exclude other causes of jaundice was obtained for 111 (85%) of the patients." We are unable to reconcile these apparently contradictory statements. The three points made by Dr. Mansel-Jones in the fourth section of his letter apply to adverse reactions in general, but they should not apply to this specific one.

We are concerned that Dr. Inman and Professor Mushin note in their report that the more rapid onset of jaundice after multiple exposures to halothane provides "strong evidence of a cause-effect relationship between the use of halothane and jaundice." As the ability to differentiate halothane hepatitis from viral hepatitis was essential in the generation of their data, this conclusion is to some extent the result of circular reasoning. It is essential to remember that Klatskin¹ and Trey *et al.*² stressed the importance of multiple exposures to halothane in 1967 and 1968 respectively. Klatskin's uncontrolled data also suggested that there might be a shorter latent period to the onset of jaundice after multiple exposures to halothane compared with that after single exposures to the drug. It is interesting to observe from table II in Dr. Inman's and Professor Mushin's report that the percentage of cases in which the patient suffered multiple exposures to halothane rose in 1969 (1964-1968=78%, 1969-1972=90%, χ^2 test $P < 0.01$). In view of this it would be important to know if the picture relating to the rapidity of onset of jaundice also changed in 1969.

Dr. Inman and Professor Mushin have compared the risk of death in any one year after multiple exposures to halothane with the risk of "death in any one year from infectious hepatitis and acute or subacute hepatic necrosis in the general population." It would have been more appropriate—if indeed it is appropriate to compare numerical data derived from cases submitted voluntarily with anything—to compare the former with the risk of death in the same

year after multiple exposures to non-halothane anaesthetics.

The Mann-Whitney U test seems satisfactory, but while acknowledging that the result would remain "statistically significant" would it not have been more appropriate to use the two-tailed test? We would also like to ask Dr. Inman and Professor Mushin why they assumed that the multiple exposures to halothane rather than another variable, such as the medical condition requiring the additional surgery or the exposure to multiple operations, was the cause of the statistically significant difference. The population exposed to single administrations of halothane also differed from that formed by pooling the patients exposed to two and three administrations of the drug with regard to sex distribution (χ^2 test $P < 0.01$). Perhaps the populations differed in several other respects as well.—We are, etc.,

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- ¹ Klatskin, G., in *Toxicity of Anesthetics*, ed. B. R. Fink p. 159. Baltimore, Williams and Wilkins, 1968.

- ² Trey, C., *et al.*, *New England Journal of Medicine*, 1968, **279**, 798.

Infections in Asplenic Adults

SIR,—It is of interest that one of the adults with *Haemophilus influenzae* meningitis described by Dr. Susannah J. Eykyn and her colleagues (1 June, p. 463) had previously undergone splenectomy. The authors indicate that splenectomy has been mentioned as a predisposing condition, and I would like to emphasize certain points about the risk of life-threatening infection in asplenic adults which have a bearing on management.

As in children, asplenic adults have a peculiar susceptibility to fulminant pneumococcal infection presenting as meningitis or the Waterhouse-Friderichsen syndrome,¹ with a mortality of 60% in the 40 cases reported in the literature.² In recent reports of serious infection following splenectomy for staging of Hodgekin's disease *H. influenzae* infection has also been prominent, though less frequent than *Streptococcus pneumoniae*, and these two organisms account for the large majority of reported cases of serious sepsis in asplenic patients. The risk is said to be maximal in the first 2-3 years after splenectomy,^{3,4} but this is based on paediatric experience, which is biased by short follow-up after splenectomy.⁵ Excluding those who were followed up for only 2-3 years after splenectomy, eight of 20 cases of serious pneumococcal infection in asplenic adults occurred more than five years after splenectomy.² Fourteen (70%) occurred later than two years after splenectomy, suggesting that penicillin prophylaxis for two years⁶ is unlikely to solve the problem.

These devastating infections are an uncommon complication of the asplenic state, and it seems that the practical approach to management is prompt and effective treatment when they arise. It has been suggested that increased awareness of the risk may have contributed to the lower mortality in