questioning. The story of unexplained abdominal pain in the mother or other members of the family tends to support the diagnosis.

Treatment with antidepressants, either the tricyclic group or monoamine oxidase inhibitors, is frequently effective in eliminating the abdominal discomfort. In most cases regardless of age the relief is permanent unless there is renewed stress causing repeated anxiety in the child. The condition may be disabling, since the depressed child cannot function adequately either socially or at school. The underlying emotional problems often remain unsuspected, and frequent short absences from school lead to poor school work and antipathy on the part of the teachers.

There is reluctance to use antidepressant drugs in children, but in this type of recurrent abdominal pain they are certainly no more dangerous than a laparotomy and may be much more beneficial.—We are, etc.,

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Paraquat Toxicity

SIR,—In paying tribute to Otto Warburg (obituary 15 August, p. 409), Drs. D. M. Stokes and D. A. Walker (22 August, p. 462) could have pointed out that while the "legendary biochemist" might well have been ignorant of the toxic effects of paraquat (methyl viologen) he would certainly have been aware of its unusual electrochemical properties. In this respect paraquat and some related dipyridyls have a long and honourable history as redox indicators.[1,2]

In support of their hypothesis on the mechanism of paraquat toxicity Drs. Stokes and Walker quote an article by Dr. J. C. Gage,[3] but in fact they do so out of context. Gage showed that the resting respiration of intact rat liver mitochondria was virtually unaffected by paraquat and diquat, probably because of their inability to penetrate the mitochondrial membrane. This finding appears to preclude the suggestion of Drs. Stokes and Walker that bypassing of the mitochondrial electron transport chain is a plausible mechanism of action of paraquat.

We agree with Drs. K. Fletcher and A. A. B. Swan (12 September, p. 646) that there is no parallel between Warburg's theory of carcinogenesis and paraquat toxicity. Any attempt to explain the apparently specific effect of paraquat, as opposed to diquat, on lung fibroblasts should take into account the substantial difference in their redox potentials,[4] since diquat is electrochemically more active than paraquat. However, since reduction might occur in vivo only as far as the free radicals, this consideration may not be important.

Dr. J. McEvoy (12 September, p. 647) is, of course, right to emphasize that his patient suffering from diquat poisoning showed no evidence of any lung lesion. A recent report[5] of fatal diquat poisoning, however, indicates that this compound may indeed produce "changes in the lungs similar to those reported for paraquat."—We are, etc.,

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Clofibrate, Fibrinolysis, and Platelet Stickiness

SIR,—Nobody would question the effects of clofibrate on serum lipids described in "Today's Drugs" (12 September, p. 632); but that clofibrate "corrects decreased fibrinolysis" is extremely doubtful, and that "abnormal platelet stickiness . . . [is] altered toward normal values" requires qualification.

The original Atromid (clofibrate plus androsterone) was reported by Srivastava *et al.*[1] to increase fibrinolytic activity in arteriopathic patients; subsequently Goodhart and Dewar,[2] using Atromid-S, stated that this effect occurred only in patients with hypercholesterolaemia. My colleagues and I found Atromid to increase fibrinolytic activity temporarily, the effect lasting for not more than three months.[3] When we studied clofibrate alone (Atromid-S)—that is, without androsterone, the dilute blood clot lysis times of five out of six patients which were within normal limits before treatment actually prolonged during treatment with the drug[4] in other words, fibrinolytic activity was reduced. Sweet *et al.*,[5] using the euglobulin lysis time, found clofibrate to have no effect on fibrinolytic activity, irrespective of hypercholesterolaemia. We believe that the temporary fibrinolytic effect of the original Atromid was due to its high content of androsterone, since we have shown that androgens temporarily increase fibrinolytic activity.[6]

Several workers, notably Carson *et al.*,[7] have shown that clofibrate reduces platelet stickiness over the short term but in none of these studies was the drug given for more than two months. In a study lasting nine months my colleagues and I found that while clofibrate initially reduced platelet stickiness, this effect was lost after six months' treatment. Our findings therefore fail to confirm that clofibrate has any worthwhile long-term effect on two of the "thrombogenic abnormalities" mentioned in your article; and also raise the possibility that in some patients the effect of the drug on fibrinolysis may be unfavourable.—I am, etc.,

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Cholera in Britain

SIR,—I found your leading articles on cholera (12 September, p. 601, and 3 October, p. 2) concise and useful. I was sorry, however, that you did not make more of the opportunity to remind the profession of the necessity in this jet age of making sure that a geographical history is taken from every patient. This could be the best protection against the consequences of spread of an imported disease such as cholera. The profession and the public seem to be becoming slightly more aware of the medical risks of going abroad, but it would appear that some of those concerned in the logistics of travel do not always face up to their responsibilities.

I have in front of me a cutting from a recent London evening paper in which it is said: "Tourists who ignored warnings to have inoculations, then picked up diseases like typhoid and cholera were criminally irresponsible, the Association of British Travel Agents said today."[1] It seems to me a bit hard to put the blame on the public in this way. Surely the agents should make sure that their passengers are informed and protected before they travel to any endemic or suspect area.

I detect some complacency about cholera appearing in Europe which, in this context, includes the United Kingdom. For example, another cutting, this time from a German paper, says: "Keine Cholera Gefahr für Europa." This is in keeping with the frequently expressed view that cholera is today not a serious community risk to the sophisticated world, where high standards of sanitation and hygiene and an adequate public health infrastructure make its spread unlikely. This may be so in the big cities and towns, where the chances of spread by infected water or food, or by personal contact are probably minimal. Nevertheless, I doubt its relevance in some slum areas and country villages in which the sanitation or lack of it sometimes seems to me to be as potentially encouraging to the vibrio as anything I have seen in the tropical world. Wherever there is dirt, squalor, and bad sanitation there could be some spread of cholera brought in by travellers from endemic regions or from areas where there are outbreaks.

The recent circular letter from the Chief Medical Officer (C.M.O. 16/70) has rightly drawn the attention of medical officers of health to the risks of imported cholera infection. The warning should be extended also to the general practitioners who are likely to see the suspect patients first, and to the travelling public, who should be informed about specific regions and vac-