be expected in patients with folic acid deficiency (clinical or subclinical). The findings of Kaimis, Summery, and Giles (personal communication to Marks and Shuster, 12 September, p. 618) and the demonstration of malabsorption in the proximal small bowel in which the site of absorption of folic acid, would seem to indicate that, in addition to an increased demand, there may be malabsorption of folic acid in these patients which would further exacerbate folic acid deficiency.

As correctable malabsorption of vitamin B₁₂ has been described in patients with folic acid deficiency, it would seem likely that the malabsorption of vitamin B₁₂ in patients with various skin diseases is also due to folic acid deficiency. I wonder whether the authors have repeated the study after a course of folic acid. It would also be interesting to see whether the small bowel dysfunction in dermatogenetic enteroopathy reverts to normal on folic acid therapy alone without local treatment of the rash. If it does (as I suspect) in addition to the local treatment of dermatosis early folic acid therapy would seem to be indicated in patients with enteroopathy, even when there are no obvious haematological changes.

Similar explanation probably holds true in enteroopathy associated with various chronic debilitating illnesses and also in conditions where increased cellular proliferation is a predominant feature.—I am, etc.,

B. N. SOMAYAJI.

Meharry Medical College,
Nashville, Tennessee, U.S.A.

REFERENCES

9. Lees, P., Quarterly Journal of Medicine, 1961, 32, 324.

Myocardial Infarction and the G.P.

Sr.—The hospital doctor (14 November, p. 433) who can include without explanation a delay of two days in seeing a patient with a coronary thrombosis in a discussion of factors influencing early death clearly needs to spend some time as a general practitioner's receptionist.

He would learn some of the form in which coronary thrombosis can be presented when first brought to the notice of the medical services. He might then be in a better position to inform general practitioners what information is useful to them so as to avoid once again finding that only five out of 41 of their letters contain any useful information. Those of us who work in hospital can make everyone's work easier by explanation rather than by damning comment.—I am, etc.,

John L. Struthers.

School of Tropical Medicine, Liverpool.

SIR:—We would like to draw the attention of surgeons to the habits of a man who has recently been admitted to Gulson and Walsgrave Hospitals in Coventry, and to St. Bartholomew's Hospital.

His initials vary between H.H. and E.H. and he gives his place of origin as Whiston, Manchester, where he has also been hospitalized on many occasions. On each of these numerous admissions he has claimed to have inadvertently swallowed a safety pin in the course of cleaning his teeth while travelling in a lorry. X-rays have shown the presence of two open safety pins in the stomach or small bowel. His abdomen has been opened a number of times, by his account for ulcer operations, but according to his previous hospital notes for the removal of open safety pins. On several recent occasions the pins have been successfully passed without any operative intervention.

We write to point out to any other surgeons under whose care he may come that despite the rather worrying appearance of the pins on x-ray, he seems to have the capacity to pass them through the gastro-intestinal track without coming to any harm.—We are, etc.,

J. A. C. Neely.

St. Bartholomew's Hospital,
London E.C.I.

Alan Rhodes.

Walsgrave Hospital, Coventry.

SIR:—I must protest about the assumption that the hepatic damage attributable to iprindole administration is due to a hypersensitivity mechanism. The four letters published to date (7 February, p. 367; 25 April, p. 238; 7 November, p. 368; 21 November, p. 494) do not contain any evidence to suggest that the reported tissue damage is any more than a direct toxic effect of the drug. The finding which prompted Dr. P. H. J. Leesar's "feeling that the iprindole administration (7 February, p. 367) is insufficient to incriminate an immunological mechanism. Thus to continue using the term allergy is both misleading and unjustified.

I wonder for a moment what the word allergy implies. In the first instance the individual must come into contact with an antigen, which in the case of drugs is usually a macromolecular complex of drug and protein. Then, as Von Pirquet proposed, a state of altered reactivity exists within the individual with respect to the antigen. This "allergic state" is not manifest as a clinical entity. Upon further contact with the antigen a hypersensitivity reaction might be produced which results in some degree of tissue damage detectable at a clinical level.

With iprindole, therefore, it is imperative to demonstrate that the liver damage is due to either the interaction of a normal antibody with antigen or between specifically allergized cells and antigen. This has not yet been reported and until it has the term "allergy to iprindole" should be discontinued.—I am, etc.,

H. E. AMOS.

Department of Pathology.
University of Cambridge.

REFERENCES

1. Von Pirquet, C. E., Archives of Internal Medicine, 1911, 7, 259.

SIR:—The suggestion by Drs. D. F. Harrison and I. M. Stanley that the drug iprindole be withdrawn on the evidence presented of hepatotoxicity (7 November, p. 368) may be warranted by recent reports by J. C. Klein and M. D. Cashman (21 November, p. 494). The drug has been in regular use in this hospital for seven years with no evidence of any serious side effects. In fact, continued clinical use has fully confirmed the findings of our clinical trial that the drug has a particularly low incidence of side effects.

It is not disputed that the occasional hepatotoxic reaction can occur, but it should borne in mind that this possibility exists for other tricyclic antidepressants as well, and has been recorded. The tricyclic antidepressants are regarded as a particularly safe group of drugs. Greater danger exists in the possibility of some to