The measures to be taken following accidental contamination of the fingers or other parts of the body are difficult to define. There are good reasons for believing that while human immunoglobulin can attenuate infectious hepatitis it has almost consistently failed to influence the course of post-transfusion hepatitis.[1] Therefore the dictum that prevention is better than cure is perhaps the best advice that can be offered at this stage, and the degree of caution to be exercised in laboratories and post-mortem rooms cannot be exaggerated.—I am, etc.,

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Serum Hepatitis in a Haemophiliac

SIR,—Serum hepatitis, as pointed out by Drs. J. A. Whittaker and M. J. Brown (6 September, p. 597), is a rare but important hazard following the use of cryoprecipitated antihaemophilic globulin. We would like to report another non-fatal case.

A 51-year-old haemophiliac with proved chronic duodenal ulcer was admitted on 10 December 1967 because of melaena with haemarthrosis of his left ankle and left elbow. He was given a total of 72 units of cryoprecipitate before being discharged on 22 December 1967. No other blood product was given. He was readmitted again on 17 February 1968, following a fall at home, with extensive periorbital and facial haemorrhage. For this he required another 52 units of cryoprecipitate, the last being given on 23 February. On 17 April 1968 he felt nauseated, his skin began to itch, and he noticed he was jaundiced. His urine became dark and his stools were pale. No other members of his family were jaundiced and no cases of jaundice were reported from his immediate neighbourhood. When seen by us on 5 May he was still jaundiced and his liver was enlarged 1 in. (2·5 cm.) below the right costal margin. Serum bilirubin was 2·2 mg./100 ml., of which 1·8 mg./100 ml. was direct reacting.

Other laboratory data were as follows: thymol turbidity 10 units (normal 1–4), alkaline phosphatase 20 units (3–13), aspartate transaminase 95 units (5–40), alanine transaminase 165 units (5–40), pseudocholinesterase 113 units (130–310). Protein electrophoresis showed a polyclonal rise in gammaglobulin with immunoglobulin levels of IgG 750 mg./100 ml., IgM 468 mg./100 ml., and IgA 300 mg./100 ml. The urine showed increase in bilirubin but no increase in urobilinogen. Serological tests for leptospirosis were negative. No L.E. cells were found and L.E. latex test was negative. The prothrombin time was prolonged at 17 seconds (control 13 seconds). His jaundice gradually subsided and when seen on 5 June, apart from a bilirubin level of 1·5 mg./100 ml., his liver function tests were normal. The prothrombin time was normal. The IgM level was still raised at 480 mg./100 ml. Since then he has remained in good health.

The clinical and biochemical findings together with the history of exposure to a potentially infected plasma product are highly suggestive of serum hepatitis. Careful questioning of donors will exclude those having had clinical jaundice, but until a reliable serological test for viral hepatitis is available the donor with anicteric hepatitis will go undetected. Cryoprecipitate will remain a potential source for the transmission of hepatitis virus until previous attacks of this form of hepatitis can be reliably diagnosed or an effective means of sterilization without altering the factor VIII content is produced.—We are, etc.,

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C. C. KENNEDY.

City Hospital, Belfast.

Hiatus Hernia and Reflux Oesophagitis

SIR,—Your leading article (4 October, p. 1) states: "11% had angina-like pain with electrocardiographic evidence to exclude the diagnosis of coronary insufficiency."

If you believe that the diagnosis of coronary insufficiency can be excluded by the electrocardiograph you can believe anything.—I am, etc.,

JOHN W. TODD.

Farnham, Surrey.

Diet and Duodenal Ulcer

SIR,—Your leading article (27 September, p. 727), comes to the same conclusion as a similar one of 15 years ago,[1] and once again points out that the value of dietary restriction in the treatment of duodenal ulcer remains unproved. In the vast literature of peptic ulcer too little attention is paid to the effects of treatment, if any, on the natural history of the disease.[2] The symptoms of any active ulcer can usually be relieved by alkalis, milk, rest, and sedation, but no treatment can be considered satisfactory if it does not heal the ulcer and prevent further relapse. The natural history of duodenal ulcer is such that symptoms wax and wane without treatment, or in spite of treatment, and the disappearance of symptoms does not necessarily mean that an ulcer has healed.

During the last war we studied a group of men with proved peptic ulcers who were in the Royal Navy and whose services were of particular value on account of their special knowledge and training. They were treated in hospital until they were symptom free, and then transferred to a special unit where they were maintained on a strict dietetic regimen, with stable living and working conditions, and regular medical supervision for a period of over twelve months.[3,4] At the end of the observation period half of the men still had radiological signs of active ulceration, although three-quarters were symptom free. A similar proportion suffered from a relapse of symptoms necessitating time off duty. A follow-up study five years later[5] showed that less than 8% had remained completely free from symptoms. Forty per cent. still followed a regular regimen of diet and alkalis; 40% took alkalis, combined with diet in about half the cases, only when symptoms recurred. The remaining 20% had abandoned all forms of treatment.

The results led us to conclude that, in this particular series, the natural history of the disease was not materially influenced by dieting or prolonged medical treatment. Martin and Lewis[6] reached a similar conclusion. Our war-time experience also suggested[7] that prolonged dieting might make subsequent management more difficult, as in some cases a conditioned reflex may have been established, which, even in the absence of an ulcer, made it difficult for them to eat ordinary food without getting pain.

The answer to your question, "Is there a case for dieting patients with active duodenal ulcers?" would still, after 25 years, appear to be "no." We should therefore allow our ulcer patients to eat what they like, so long as they like what they eat. They probably do this anyway.—We are, etc.,

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Bradford.

R. S. ALLISON.

Belfast.

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SIR,—Your leading article (27 September, p. 727) on the failure of medical dietetic treatments in the case of duodenal ulcer comes as no surprise. Indeed, most such treatments would appear bound to fail because the foods have often been grossly changed from their natural state, and the patient is commonly made to eat foods he does not want and at times when he does not wish to eat at all.

It is not difficult to show[1] that the incidence of peptic ulcer throughout the world accurately parallels the taking of refined (carbohydrate) foods, the refining processes having removed in part (as in the case of flour), or in whole (as in the case of sugar), the protein component which alone neutralizes the gastric acid. There are many examples of this evidence. The ulcer belt of India is one of them, where either highly milled rice is consumed or else manioc (tapioca). The eaters of the latter show one of the highest rates of ulcer-incidence known. The second world war provided another but opposite example, peptic ulcer not being seen when refined foods were replaced by their coarse natural analogues. Finally, the enormous rise in ulcer-incidence since the turn of the century has accompanied an eightfold rise in sugar consumption over the last 150 years. Yet, as your leading article shows, ulcer-dieting usually involves the giving of just these ulcerogenic refined foods. No wonder it fails.

It can likewise be demonstrated[2] that the behaviour of the stomach is very intimately related to the desire accompanying the eating of food, both as regards the nature of the food and the times at which it is consumed. Yet, as previously stated, the patients' natural eating pattern is very commonly thwarted, whether by inducing him to eat food he does not seek, or to eat when he does not want to eat (frequent meals, to prevent the stomach from becoming empty, etc.).

In my opinion there is little to choose between letting the patient eat the wrong foods in accordance with his own desires and getting him to eat the right foods not in accordance with his desires; both fail. It is only by combining the two, in the principle of eating natural (unrefined) foods, naturally desired, that there is considered to be a real chance of success—and this needs far more attention to the individual patient than most