of Mrs. X had the breath-holding spells. None of these people show any intellectual impairment. So a number of people in the same family had the complaint. Convulsions occurred when the attack lasted for more than half to one minute. The younger the child the greater is the possibility of the convulsion. The reason for this is not known. Apart from the precipitating factors mentioned in your leading article, there are many others to be elucidated. —I am, etc.,

Mangalore, India. M. S. NILAKANTA RAO.

Biliary and Anginal Pain

SIR,—Your annotation on "Biliary and Anginal Pain" (*Journal*, December 17, p. 1494) recalls to my mind a remark made to me by the late Lord Moynihan when watching him operate on a patient of mine some 30 years ago: "I cure more cases of so-called angina by removing their gall-bladders than physicians do by only concentrating on their cardiac symptoms." A generalized, genial, and typically good-humoured statement by this eminent surgeon, but holding in it much truth, as your annotation of 1955 confirms. The late Sir Henry Head's zone of hyperalgesia is invariably present in these cases if carefully mapped out.—I am, etc.,

Chipping Campden. C. B. HEALD.

Serum Amylase and Acute Abdominal Disease

SIR,—May I thank Mr. P. A. Lane Roberts (*Journal*, November 19, p. 1269) for pointing out an apparent anomaly in my letter (*Journal*, October 29, p. 1086)? I did not make it clear that conservative treatment was employed during the acute phase in those cases of pancreatitis admitted this year.

I quite agree with his observation that if there are signs of general peritonitis laparotomy is essential, but the incidence of acute pancreatitis together with a further condition, such as a perforated gall-bladder, giving rise to this cannot be high. It is in some of those patients presenting with severe abdominal pain and vomiting and having marked tenderness out of all proportion to the degree of rigidity of the abdominal wall, which may even be absent, that pancreatitis should be suspected rather than peritonitis. Anyway, there is no question of delaying operation to await estimation of the serum amylase, as this can be performed in less time than it takes to prepare the patient and theatre.

Turning to Mr. W. Burnett's letter in the same issue of the *Journal*, I would assure him that I am aware of many of the statements he has made, but limitation of space forbade me to enlarge on any particular aspect in my original letter, which was neither a treatise on pancreatitis nor a detailed evaluation of the causes of a raised serum amylase, so that I may have been more dogmatic than was my intention. Even so, although it is well known that small perforations may not reveal intraperitoneal gas, I cannot conceive that this would be absent in a case where the amount of peritoneal soiling is such as to cause a considerable rise in serum amylase. But one should mention another factor in this connexion: the extent of the rise is dependent also upon the functional state of the pancreas being greater in the case of perforation following within an hour or so of a meal when the pancreas is in its maximal secreting phase.

I did not think I gave the impression that I considered biliary tract disease to be the sole aetiological factor concerned in acute pancreatitis. I was seeking only to explain the possible cause of a rise in amylase in a patient with acute cholecystitis, citing the observation that these conditions have occurred simultaneously in the same patient. Mr. Burnett has seen this also, apparently, although he has made no mention of it in his article (*Journal*, September 24, p. 770). Therefore I fail to see that there can be any useful differential diagnostic significance in the degree of elevation of the amylase level. At which point is one going to put the dividing line? I attach no diagnostic importance to figures of less than twice the upper limits of normal, unless at a later stage the normal level is found to be very low. Often the patient with a less severe degree of cholecystitis and/or pancreatitis is not admitted until the second or third day of the illness when what might have been an initially raised serum amylase has fallen substantially. To sum up, one can only say that all the clinical features of the case, together with any ancillary aids, have to be carefully considered together in arriving at a diagnosis without attaching undue importance to any one item.

In conclusion, I might add that a further case, which I did not see personally, death having occurred shortly after admission, has come to my notice. Post-mortem examination revealed acute cholecystitis and acute pancreatitis.—I am, etc.,

London, S.E.10. R. F. BOLAM.

Perforation of Intestine by Foreign Body

SIR,—It was with interest that I read of the first two cases of intestinal perforation reported by Mr. George T. Mair (*Journal*, November 26, p. 1310). I would like to record a similar, but fatal, case.

On April 14, 1955, I performed a laparotomy on a 75-year-old lady and recovered a small serrated sliver of wood —1½ in. by ⅛ in. (3.8 cm. by 0.32 cm.)—which had perforated the ileum some four feet from the ileocaecal valve. There was a profuse general peritonitis. Her immediate post-operative condition was satisfactory, and when questioned she denied having swallowed any foreign body. Over the next few days she developed a suppurative parotitis which was drained by incision. On the ninth post-operative day she suddenly became comatose and died within 12 hours. At necropsy there was evidence of a terminal staphylococcal septicaemia, presumably arising from the parotid infection. The original peritonitis had completely resolved and the small deficiency in the ileum, which had been oversewn, was healed.

It is probable that the wood was embedded in some article of food which, in the absence of efficient mastication, was swallowed as a single large bolus. It is interesting to note that the foreign body in the first two cases presented by Mr. Mair was also a sliver of wood approximately 1½ in. (3.8 cm.) in length.—I am, etc.,

Cambridge. DESMOND COOPER.

Anticoagulants and Prothrombin Pattern

SIR,—Dr. T. B. Begg (*Journal*, November 19, 1955, p. 1268) has criticized the use, in our study (*Journal*, October 15, 1955, p. 947) of response over the 24 hours to anticoagulant therapy, of "prothrombin concentrations" rather than, as he would wish, "prothrombin times." We agree of course that Quick's test also measures factor VII activity, and to that extent both terms are ambiguous. Nevertheless, *faute de mieux*, prothrombin concentrations obtained by Quick's method are readily appreciated, and are of proved clinical value. Prothrombin times, on the other hand, are not so easily standardized, and can be misleading, particularly in published figures. We agree with Dr. Begg that the "prothrombin index," which is sometimes used as a form of standardized prothrombin time, has little to commend it, and adds only to the confusion of terminology by its retention.

Dr. Begg is, of course, quite right in presuming that our concentrations were obtained by means of a saline dilution curve for normal plasma. The dramatic fall in the ethyl biscoumacetate concentrations, to which he draws attention, might well be regarded askance if the curve of Fig. 1 referred to one patient only. It represents, however, the pattern of mean levels for a group of nine patients, and the fall he refers to is statistically significant. Fig. 1 is compounded of individual curves, several of which showed very much greater variation than their average. For the same reason, the experimental error is very much less than the figure Dr. Begg gives; the standard error of the mean is about 2%. Because of the hyperbolic relationship between clotting times and prothrombin concentration, a difference in concentrations at a low level represents a very much