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SUPPLEMENT

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CORRESPONDENCE

Owing to the Government's restrictions on the use of power in industry, including printing, we have been obliged to defer publication of some letters and print others in shortened form. We regret this inconvenience. Correspondents are asked to help by keeping their letters as short as possible.

Presentation of Myocardial Infarction

SIR,—Angina pectoris and electrocardiographic abnormalities are unreliable predictors of myocardial infarction[1 2] and it is pleasing to read mention of alternative warnings in your leading article (5 January, p. 1). Kinlen's questioning of the relatives of those who died suddenly revealed patterns of changing behaviour[3] such as we have encountered among survivors.[4-6] The abnormal tiredness which can overcome the will of an active person and modify his habits or make him struggle to maintain his usual ways, the jettisoning of inessential activities, and the "accelerated" ageing are probably caused by the heart's losing the ability to meet all the demands of the anxious individual in the year before infarction.[6]

Other warnings[6] are probably caused by the steep rise of left heart pressure which occurs in coronary disease, but not in health, when the heart is challenged towards its limits—uncomfortable dyspnoea, rhythm disorders, and giddiness are common and early to appear. A rapidly growing distaste for physical challenges is usually attributed to ageing. Angina may not appear until after the infarction. It is important to interview relatives because most patients present a "paradoxical and blatently illogical" denial of obvious disability.[7 8]

The increasing effort needed to keep up customary activities may cause the symptoms of strain which typically precede a breakdown in health[9] and also the reversible metabolic consequences of increased catecholamine secretion, such as abnormal blood cholesterol and lipid patterns, raised serum uric acid and blood sugar levels, and raised blood pressure.[10]

Abnormal tiredness has many causes and the heart must be demonstrated to be abnormal before it can be incriminated. We believe that the physical signs of disease can be elicited a year or more before the E.C.G. changes, provided that the patient is examined in the left lateral position.[11] The area of the left ventricular apical pulsation can be seen and felt to be abnormally great and the left atrial contraction is morbidly enlarged, creating a palpable and audible pathological presystolic triple rhythm in place of the normal double rhythm.[12]—We are, etc.,

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Ulcerative Colitis and Acute Salmonella Infection

SIR,—I read with great interest the article by Dr. M. W. Dronfield and others (19 January, p. 99) on the difficulties of recognition and management of coincident salmonella infection and ulcerative colitis. This is a problem not infrequently encountered in our unit, with its large case-load of acute diarrhoeal syndromes of all kinds, infectious or otherwise. They recommend that the diagnosis of ulcerative colitis should be considered promptly if apparent salmonella diarrhoea is profusely bloody, and presumably confirmation be sought from endoscopic and histological features. It is not generally realized, however, that severe colonic inflammation can occur in salmonella infection alone. Though it is customary to regard the small intestine as the predominant site of affection in salmonellosis, our knowledge of the pathogenesis of salmonella diarrhoea in humans is rather limited, and most of the available information has been obtained from animal experiments. However, features such as frequent small-volume bloody motions, tenesmus, and tenderness over the sigmoid colon indicating large bowel involvement are not uncommon in salmonella bowel infections. To ascertain the frequency of such involvement, I have now sigmoidoscoped a series of patients during the acute stage of their illness, and a detailed account of my findings will be submitted for publication later this year.

The study so far has revealed that the incidence of colonic involvement can be quite high in salmonella infection, and in some cases the appearance of the colonic mucosa and the histological changes, including typical crypt abscess formation, may bear a striking resemblance to those of ulcerative colitis. Thus the presence of severe colonic involvement does not necessarily imply the diagnosis of ulcerative colitis, which after all is a disorder of non-specific inflammation with no pathognomonic endoscopic or histological features. That typical endoscopic and radiological pictures showing pseudopolyposis and total colonic involvement can occur in the acute dysentery syndrome was strikingly illustrated in a recent patient with severe shigella dysentery.

Thus, in the *early* stages of salmonella infections a confident diagnosis of a coincident *first* attack of ulcerative colitis can be very difficult indeed, as the endoscopic and histological features would have fairly limited discriminatory value. Persistent bloody diarrhoea for more than two to three weeks would be very uncommon, however, in salmonella colitis, and it has been my practice to consider corticosteroid therapy if there is persistence of colitic features after a short intensive course of chloramphenicol therapy, or at an earlier stage if there are associated systemic abnormalities such as severe anaemia, high erythrocyte sedimentation rate, or hypoproteinaemia.—I am, etc.,

B. MANDAL

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