Correspondence

Life event research

SIR,—Your leading article and the communication by Talley and Piper in *Gut*, 1986; **27**: 123–6, and 127–34, and their letter 223–224 summarise most of the methodological difficulties of life event research and its limitations as at present practised. Such studies have, as in this case, usually recorded little difference for recent life events *in propositi* compared to controls. Some people such as the Empire Rheumatism Council's Scientific Advisory Committee in 1950, have interpreted such negative findings¹ as proof that emotional stress was not a causative factor; in that instance the disease was rheumatoid arthritis. I wrote at the time² that this conclusion was not justified on the evidence and indicated that it ignored the fact that germination of a seed also depends on the quality of the soil.

Hambling,³ Wolf⁴ and I⁵ pointed out with supporting evidence that a physiological response to a stimulus can be misleading if it does not take account of the subject's negative or positive perception of the stimulus, and/or of the operator or questioner. The receipt of a police summons or a row with a spouse at breakfast may also affect the subject's responses that day, but are unlikely to do so some days later.

Talley and Piper say 'it is impossible using any valid scientific method to determine the importance of a life event to a particular individual'. I suggest that videotape subsequently scored by independent raters may permit this,⁶ and Talley and Piper's other objection that 'the initial interviewer may consciously or subconsciously alter the presentation' might be obviated by a standardised and/or pictorial presentation of relevant questions in the absence of an interviewer. Thirty five years ago I found pictorial stimuli useful while studying colonic motility.

When it comes to pitfalls in life event research perhaps the commonest is asking the wrong questions. What is stressfully provocative in one group of disorders is often not so in others in which relatively trivial situations rather than events are what to look for—for example, perfectionists having to meet deadlines in migraine^{7,8} and indecision and 'fence sitting' in the irritable bowel syndrome.^{9,10} However, while pathological mourning and loss are not especially relevant to migraine or irritable bowel syndrome they are relevant in the autoimmune disorders,¹¹ while threats of separation-engulfment (entrapment) are commonly provocative in multiple sclerosis¹² etc. Popper has claimed that scientific

advances result from the unexpected observation that falsifies an existing theory; some of the observations mentioned challenge current thinking on the life event theory, yet which suitably honed, could become a most valuable research tool.

Lastly, I plead for less use of the term 'anecdotal' as applied to observations based on substantial clinical studies, past or present. Too often the term is used as a cheap defence or to get a laugh, but its inappropriate use is at best unhelpful, and at worst destructive of progress.

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Dieulafoy vascular malformation

SIR,—The report by Van Zaten and his colleagues (*Gut* 1986; **27**: 213–22) emphasising the importance of the Dieulafoy vascular malformation as a cause of gastrointestinal bleeding is to be welcomed.

Upper gastrointestinal endoscopy in patients with severe bleeding may pose a particular problem in

assessment. The combination of a distressed restless patient and a stomach containing a large volume of blood may decrease the chances of accurately identifying the cause of bleeding. In these circumstances it is usually easier to assess the antrum and duodenal cap. If these are clear, attention should then be directed at the upper half of the stomach. A useful manoeuvre in these circumstances is to dilate the stomach with air when small lesions may become apparent. The diagnostic return more than balances the slight extra risk involved. Experience in Blackpool suggests that in haemorrhage associated with multiple gastric erosions which is recurrent or persistent, dilatation of the stomach may show that bleeding occurs from one minute lesion, usually high in the stomach and involving a submucosal artery. If endoscopic coagulation therapy is not available or not successful, early surgery is the best treatment.

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Biphasic hepatitis in HBV/HDV coinfectd parenteral drug abusers

SIR,—We would like to refer to a few of the issues raised by Dr Govindarajan and colleagues (*Gut* 1986; 27: 19–22). We agree with the authors that delta (HDV) infection is a cause of severe relapse of acute hepatitis B.^{1 2} To assess the magnitude of the problem, we have determined the prevalence of biphasic hepatitis in a large series of parenteral drug abusers admitted to our infectious diseases clinic between January 1979 and December 1983 for acute HBV/HDV coinfection or classical hepatitis B. Biphasic hepatitis was significantly more frequent in patients with delta coinfection (53/180; 29.4%) than in those with classical hepatitis B (13/98; 13.3%) ($p < 0.01$). In agreement with Dr Govindarajan, a significant proportion of delta infected patients (10/53; 18.9%) had severe relapses (prothrombin activity $< 50\%$ the level in normal controls) with a fulminant course (and death) in two of these patients. On the contrary, the relapses we have observed in patients with classical hepatitis B generally had a mild course. We would like to discuss two more problems. One regards the time of occurrence of relapses in HBV/HDV coinfectd patients. The authors report patients with a second peak of alanineaminotransferase within 30 days from the initial episode. We would emphasise that the relapse of acute HBsAg positive hepatitis serologically associated with HDV infection may be present in some patients (six in our series) during the second month, when the patients are still

HBsAg positive, but usually discharged from the hospital. One can argue these might be considered cases of HDV superinfection. The detection of serum δ Ag in four of these during the first acute episode, as well as the uneventful course of the disease in all the subjects, are against this hypothesis.^{3 4} Lack of follow up in parenteral drug abusers with acute HBV/HDV coinfection may produce an underestimate of the prevalence of relapses. Because of the possible asymptomatic course of the relapse, this error might be increased. We think that parenteral drug abusers with acute HBsAg positive hepatitis must be followed up with frequent clinical, biochemical, and serological tests until clearance of HBsAg.

The other problem regards the serological diagnosis of HDV infection in patients with acute (primary) hepatitis B virus infection. As anti- δ antibody in patients previously negative for serological delta markers is sometimes detectable only four to six weeks after the onset of the disease,³ the incidence of HDV infection, and thus the risk of severe hepatitis, might be underestimated if serial serum samples are not tested.

Regarding the question put by the authors about the possible development of chronic HBsAg positive hepatitis in patients with HBV/HDV coinfection with a biphasic course of the acute disease, our data show that, apart the occurrence of severe (and fulminant) hepatitis, these patients are not at risk of developing chronic hepatitis. All the patients who survived the acute episode (and the relapse) cleared HBsAg and seroconverted to anti-HBs.

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