Leading article

Life events and gastrointestinal symptoms

There is a considerable amount of anecdotal evidence to support the notion that life events provoke physical illness.\(^1\) Research evidence does not, however, always support this view and many studies have yielded equivocal results.\(^2\)

The relationship between psychological stressors and physical illness can be studied in three ways. Firstly, animals and human volunteers can be exposed to experimental stressors. The classic experiments conducted by Stewart Wolf and his colleagues on subjects with gastrectomies require brief mention.\(^3\) In fistulous subjects Wolf found that fright, depression, and attitudes of being overwhelmed were associated with pallor of the mucosa and diminished gastric acid secretion and motor activity. Conversely, when feelings of resentment, anger and hostility were artificially induced in the laboratory there was a tendency toward increased acid secretion and gastric motility of the gastric mucosa.\(^4\) These studies provide fascinating insights into the relationship between psyche and soma, but have shortcomings because they are based on observations in only a few patients. It is not clear how these experimental situations relate to the spontaneous onset of physical illness.

A second approach involves examination of the impact on a population of specific and easily identified life events such as bereavement or natural disasters. In a recent example, Trichopoulos et al.\(^5\) reported an increase in death from cardiac events after the Athens earthquake in February 1981. The increase was significant during the third day after the earthquake and appeared mainly among people with atherosclerotic heart disease. It seems highly likely that the emotional disturbance induced by the earthquake led to the development of cardiac rhythm disturbances associated with intense vagal activity, or increased sympathetic drive.\(^6\) These disturbances of conduction were the possible trigger mechanisms of death.

The third method of studying the relationship between psychological stressors and physical illness is to investigate a population of people with the same illness of recent onset. This homogenous population can then be studied to determine the amount of recent life stress they have experienced. Such studies are best with methodological problems, which will now be briefly outlined.

Most retrospective studies that attempt to measure life events have used one of the various self-administered scales. Unfortunately these studies have been confounded by the phenomenon of ‘effort after meaning’,\(^7\) whereby ill people tend to seek a reason for their illness. Connolly\(^8\) has drawn attention to the fact that the investigator may also be involved in a similar process, colluding with the patient to ‘discover’ an increase in life events before the onset of a physical illness.
Exact dating of the onset of illness is also essential to ensure that events precede the onset of illness and do not result from it. Because it is impossible to determine exactly when a neoplasm begins, such illnesses as cancer cannot be studied in this way. As Creed\(^9\) has pointed out, the date of presentation can be documented, but this is quite different from the date of onset of the illness. Another contentious issue in research on life events concerns the length of time before the interview for which information is sought. There will obviously be a fall-off of reporting events with increasing time before interview because of poor recall. This is more likely to occur with pencil and paper questionnaires, where the definition of inclusion and quality of life events is left in the hands of the respondent, rather than the research worker.

The sociologist George Brown and his colleagues have designed an instrument which attempts to overcome some of these problems – the Life Events and Difficulties Schedule (LEDS).\(^{10}\) This is reliable and superior to other measures of life events, because each event has been previously and precisely defined to avoid distortion by the subjects' mental state. The interviewer goes through a list of events with the subject, giving examples and asking probing questions. If an event has occurred further questions are asked about its exact nature, firstly to determine whether it meets LEDS inclusion criteria and secondly so that various qualitative dimensions, such as a measure of severity, may be rated. Each event is then rated by members of a research team who are kept ignorant of the presence or absence of a psychiatric disorder, and of the subject's reported reaction to the event. Ratings are made by an investigator based on the belief that it is possible to assess in an approximate sense the meaning of a particular event for an individual in terms of how the average person would react, given the identical event and background circumstances. Because this approach ignores what the respondent says he or she actually felt in response to the event the danger of this bias is substantially reduced. This issue of personal meaning is very important in life event research and its complexity is illustrated in the following example. Childbirth for a single homeless mother who has not planned her pregnancy may not only be wholly undesirable, but experienced as a loss in terms of freedom and independence. Conversely, for a married woman a planned pregnancy may be extremely desirable and presage eagerly awaited new roles and commitments. Respondent based measures of life events take no account of these issues and for this reason the methodologically superior interview based measures are to be preferred. Craig\(^{11}\) recently reviewed the methodological shortcomings of checklist measures and Brown\(^{12}\) and Creed\(^9\) have both suggested that these instruments should no longer be used in life event research.

In this issue Talley and Piper (p 127) report that the number of stressful life events was not significantly different in patients with a diagnosis of non-ulcer dyspepsia than in a matched community control group. How does their study stand up to examination in the light of the methodological issues already discussed? Firstly, by using a modified form of selfadministered inventory of life events, the authors did not elicit the meaning of each event for the individual. Secondly, the exact dating of the onset of illness is not clear. The authors state that the period under study was 12 months before diagnosis, but 46% developed their symptoms in the 12 months
before diagnosis and a further 11% had a history of continuous dyspepsia longer than one year. Because this study deals with the year before rather than the year after, it is really concerned with those factors that influenced consulting behaviour, rather than with those involved in initial symptom formation. Most previous studies have reported greatly increased rates of life stress before consultations, and so the absence of such an association in patients with non-ulcer dyspepsia in Talley and Piper's paper is rather unusual.

The study also lacks a measure of concurrent psychological distress, or mood disturbance. This would have furnished useful information concerning the association between life events, psychopathology and somatic symptoms. One such study was conducted by Creed, who used the LEDS to investigate the relationship between life events and appendicectomy. Creed found that a greater proportion of those undergoing appendicectomy (whether the appendix was inflamed or not) had experienced a life event before the operation, than a community comparison group. Events which carried a severe and lasting threat were related to the onset of abdominal pain only amongst those whose appendix turned out to be normal histologically. Such events have previously been shown to cause depression.

The abdominal pain in those subjects whose appendix was not inflamed may have been caused by the depression, which in all cases preceded the onset of pain. This finding was similar to the community survey of Murphy and Brown, in which severe life events were related to the onset of physical illness only when depression followed the event. Creed followed his patients for 12 months and found that the depression was associated with continuous complaints of abdominal pain. Thus it seems possible that in some patients a severe event may lead to the development of affective disorders which presents as 'somatic' symptoms.

In most patients without an inflamed appendix, however, the relationship between the severe life event and the pain seemed direct. The severe event in these patients may be related either to the onset of abdominal pain and/or to the decision to seek treatment. The processes by which disturbing life experience is expressed in the form of somatic complaints and/or treatment-seeking behaviour are complex and can occur in the absence of conspicuous psychiatric disturbance.

These studies highlight the need for aetiological investigations of gastrointestinal disorder to integrate many different types of factors simultaneously into their causal models. Talley and Piper have used a methodologically flawed instrument to measure only one of the many variables which mediate between the environment and physical illness. Conclusions based on the findings of such studies can only be tentative.

The underlying assumption in their study is that life events may be causally related to illness onset. That is to say psychological factors, especially negative emotions such as sadness and chronic resentment, can cause somatic disease, or derangements of function that are not accompanied by detectable organic abnormalities. As Lipowski has pointed out, this conception of causality is simplistic and obsolete. Psychological factors are believed to be involved, to a greater or lesser extent, in every disease and episode of illness. The multifactorial model of causation assumes that all disease is the result of a complex interplay of biological, psychological
and sociological variables. Each of these three classes of variables may play either a necessary or a contributory causal role. Thus we should not be asking whether life events are related to the onset of illness, but to what extent are life events related to the onset of this particular illness, in this particular person, at this particular time. This means that we should, in addition to life events, be measuring other variables that may have a bearing on the onset of illness. For example, what are the patients' personality attributes, coping resources and the extent and quality of his available social support? What are the effects of these mediating variables on the relation between life events and illness onset? Unfortunately none of these variables was quantified in this study, and for this reason we are no nearer to disentangling the contribution of life events to illness onset in patients with this kind of functional bowel disorder.

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References