Correspondence

Specific food intolerance
sir—Farah et al (February issue) report a proven incidence of food allergy in 6% of patients in whom food intolerance was suspected as a cause of unexplained gastrointestinal symptoms and attribute symptoms in the remaining 94% to psychogenic causes.\(^1\) They suggest that the lower positive diagnostic yield recorded in their study than in comparable studies by other workers is due largely to inter-study variation in criteria for selection of patients. It is also possible, however, that the discrepancy is a result of basic flaws in the design of their trial protocol.

Initially all patients were placed on a ‘low allergenicity diet’. From our reading it would appear that all foods are potentially allergenic, and of the 19 foods permitted in the early stages of this trial we could find only four – salt and vinegar, prunes and apricot – which had not previously been observed to cause allergic responses in some subjects.\(^2\) \(^3\) A standardised exclusion diet is therefore impossible as one hallmark common to all previously published work in this field is the range and combinations of foods to which patients can be shown to respond.

In more rigorously controlled studies patients are given a severely restricted diet for two weeks consisting typically of one meat (lamb or chicken), one carbohydrate (potato or rice), one fruit (banana or apple), one vegetable (brassica), water and a vitamin supplement – the so called oligoantigenic diet. Patients who do not improve on this diet are offered a second such regime with no foods in common with the first diet.\(^2\)

The failure of Farah et al to offer an alternative diet to those patients who did not respond to the initial dietary screening (73%) casts doubt on the adequacy of their trial protocol and on their conclusion that most forms of adverse food reaction can be attributed to psychogenic causes. We note that the results of this trial have already appeared in print\(^4\) in abstract form and that after publication similar criticisms of the initial screening diet were made.\(^5\)

It is commonly assumed that the controlled clinical trial provides a careful and critical evaluation of the efficacy of new treatment regimes. However, inadequate trials, particularly in controversial areas of medicine, serve only to promote scepticism among clinicians and to lead patients to seek help from ‘alternative’ practitioners.

A P BURFORD-MASON and J M T WILLOUGHBY

References


Reply
sir,—In their comments on our paper on specific food intolerance, Drs Burford-Mason and Willoughby have misread our conclusions. Firstly, we did not claim an ‘incidence’ of specific food intolerance amongst our 49 patients and secondly, nowhere do we claim that symptoms were attributable to psychogenic cause in 94% of the patients. We specifically avoided reporting an incidence of specific food intolerance, as the study was not designed for that purpose. It would be wrong to draw any conclusions concerning the incidence of specific food intolerance for this reason.

In partial explanation of our low diagnostic yield (6%), our exclusion diet is criticised for being insufficiently rigorous. As most of the patients did not themselves suspect food intolerance at the outset, an acceptable and palatable exclusion diet was chosen (notwithstanding that we expected to ‘miss’ a few patients as a result). It was felt that compliance would suffer if the regimen was too rigorous, or too complicated.

It is certainly possible that an increased diagnostic yield would follow the use of a more rigorous dietary approach and our results should not deter those wishing to use such regimens. The only sure way, however, of excluding antigens from the diet is to use an elemental diet such as Vivonex, which is antigen-free. This may be regarded as too extreme to apply to a broad spectrum of patients, although it is an approach we have used clinically in selected cases. Any exclusion diet less rigorous than this will underestimate the incidence of specific food intolerance.

Finally in drawing attention to the greater number of placebo reactors than verified specific food intolerance patients, we did not intend the conclusion to be drawn that \textit{all} the remaining patients’
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Symptoms were attributable to psychogenic causes. We do feel, however, that there is sufficient evidence in our data to support the notion that symptoms may be attributable to psychogenic causes in appreciable numbers of patients—greater numbers than we were able to prove had specific food intolerance.

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Drug therapy and perforated peptic ulcer
Sir. — We read with interest the recent paper by Collier and Pain (Gut 1985; 26: 359–63) concerning drug therapy and perforated peptic ulcer. A recent survey in Oxford confirms that the incidence of perforation has changed little since the introduction of cimetidine. As in other studies perforation in patients currently receiving cimetidine was also observed.

Although Collier and Pain’s review provides confirmatory evidence of an association between non-steroidal anti-inflammatory drugs and peptic ulcer perforation, details of individual drugs which might have a particularly strong association with perforation were not given. Case controls were not available in the Oxford study but over half the 26% patients receiving non-steroidal anti-inflammatory drugs at the time of perforation were taking indomethacin. A report from Exeter utilising case controls has confirmed that indomethacin is associated with an increased risk of perforation of duodenal ulcers.

All retrospective studies are liable to underestimate the number of patients taking non-steroidal anti-inflammatory drugs. Jorgenson’s retrospective survey showed that less than 20% of patients suffering a perforation had taken drugs known to be associated with peptic ulceration while his limited prospective study showed the true incidence to be over 80%. Having established an association between non-steroidal anti-inflammatory drugs and perforated peptic ulcer further prospective studies with case controls are now essential to assess accurately the hazards associated with each agent. The availability of this information would enable the risks of individual drugs to be considered before they are prescribed.

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References

Reply
Sir. — We are pleased to be given the opportunity to reply to the letter of Watkins, Dennison, and Collin. The frequency of ingestion of non-steroidal anti-inflammatory drugs (NSAID) is given in the Table.

Table Frequency of ingestion of NSAID in patients with perforated peptic ulcers according to specified drugs

<table>
<thead>
<tr>
<th>NSAID</th>
<th>Perforated duodenal ulcers</th>
<th>Perforated gastric ulcers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indomethacin</td>
<td>31</td>
<td>10</td>
</tr>
<tr>
<td>Phenylbutzone</td>
<td>10</td>
<td>1</td>
</tr>
<tr>
<td>Aspirin</td>
<td>8</td>
<td>1</td>
</tr>
<tr>
<td>Piroxicam</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>Ketoprofen</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>Indomethacin suppos.</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Ibuprofen</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Benorylate</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Difunisal</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Mefenamic acid</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Others</td>
<td>9</td>
<td>4</td>
</tr>
</tbody>
</table>

Some patients were taking more than one NSAID. NSAID = non-steroidal anti-inflammatory drugs.

We were unable to obtain figures for the number of individual preparations prescribed within this regional health authority from the DHSS, for reasons of commercial secrecy, and therefore correlations for individual drugs were not assessed. Thus in our paper we compared the annual number of patients taking NSAID in specified age/sex groups and the annual number of prescriptions for all NSAIDs issued in this region, and showed a significant correlation in patients aged over 65, especially women. It is interesting, however, to note how frequently piroxicam was the NSAID being taken in view of the fact that it was only introduced during the latter three years of our 10 year study.

The late Morton Grossman wrote that ‘the gun must be loaded in order for an explosion to occur when salicylates pull the trigger’ it would appear that certain NSAIDs are more ‘trigger happy than others’; and furthermore that being over 65 and a woman makes it more likely that the gun is loaded.

D SU COLLIJR and J A PAIN