LETTERS TO THE EDITOR

Serum pepsinogens in blood transfusion donors

EDITOR,—We read with great interest the paper by Veenaadal et al.1 In this study the authors showed that blood donors positive for Campylobacter pylori antibody had higher serum pepsinogen A and C levels than their seronegative counterparts. In addition, the authors plotted the measured IgA and IgG absorbance index against age and showed a weak but significant correlation between the height of the absorbance index and rising age. The authors suggested that this increase could be caused by progression of chronic superficial gastritis.

We cannot confirm these findings and do not agree with this conclusion. In an earlier study we showed that the positive/negative (P/N) ratio (identical to Veenaadal’s absorbance index) in patients with non-ulcer dyspepsia (NUD) and proved active helicobacter associated gastritis was significantly higher than in healthy seropositive blood donors (mean 3-63 (1-24) range 1-06–6-45) and patients with non-ulcer dyspepsia (1-32–6-97) respectively. Although considerable overlap was present in the lower ranges, it was concluded that high P/N ratios occur with active inflammation and that lower P/N ratios can reflect a serological scar of past infection as well. It was also shown that inflammation with polymorphonuclear cells invading the mucosa causes a higher P/N ratio, hence antibody response, compared with a milder degree of inflammation.1 In the Table

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Mean P/N ratio (SD)</th>
<th>Number</th>
<th>Mean P/N ratio (SD)</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>21–30</td>
<td>3-34 (0-93)</td>
<td>7</td>
<td>2-14 (0-43)</td>
<td>10</td>
</tr>
<tr>
<td>31–40</td>
<td>3-57 (1-24)</td>
<td>8</td>
<td>2-32 (0-41)</td>
<td>35</td>
</tr>
<tr>
<td>41–50</td>
<td>3-75 (0-97)</td>
<td>55</td>
<td>2-35 (0-49)</td>
<td>55</td>
</tr>
<tr>
<td>51–60</td>
<td>3-71 (0-92)</td>
<td>36</td>
<td>2-46 (0-48)</td>
<td>36</td>
</tr>
<tr>
<td>61–70</td>
<td>4-37 (1-25)</td>
<td>7</td>
<td>2-23 (0-62)</td>
<td>7</td>
</tr>
</tbody>
</table>

and Figure the mean P/N ratio and the standard deviation of the healthy seropositive blood donors and the patients with NUD are plotted against age. It is clear that the findings of Veenaadal et al cannot be confirmed as no increase in P/N ratio with rising age is seen in the healthy blood donors. On the other hand, a slight increase in antibody response is seen among the patients with non-ulcer dyspepsia. Comparison of age cohorts 21–30 and 31–40 with age cohort 61–70 revealed a p value of 0-08 and 0-02 respectively (Student’s t test). The other cohorts showed no differences in the height of the P/N ratio. In our opinion, this does not indicate that the antibody titre in an individual patient increases with rising age. Longitudinal serological follow up has not been done to our knowledge. The only data reported in previous studies show a decrease in antibody titre after therapeutic intervention aimed at suppression or eradication of H pylori and an increase in the incidence of recrudescence or reinfection. From the plot of Veenaadal et al it cannot be concluded that the antibody titre from an infection with H pylori acquired at an early age rises when the patients get older.

The statement that chronic active gastritis becomes worse with rising age can be considered correct if the development of intestinal metaplasia and glandular atrophy are considered a part of the deterioration of gastric mucosal histology. As reported in previous studies, however, the role of H pylori during the course diminishes and it is more logical to expect that the antibody response in the individual patient does not rise during the course of helicobacter gastritis. Serological follow up in gastritis patients is necessary to learn more of the natural history of the antibody response.

R J L FLOFFLED
Department of Internal Medicine,
Ziekenhuis De Hert Zaanstad,
E H STOBBERINGH
Department of Microbiology
J W ARENDS
Department of Pathology,
University Hospital,
Maastricht,
The Netherlands


Faecal pH and colon cancer

EDITOR,—I would like to comment on the excellent letter1 from Johannesburg reporting on the slow faecal pH levels in African subjects and their possible connection with the low incidence of colorectal cancer.

My own experience in India supports the concept of a possible relation between faecal pH and the incidence of large bowel cancer. A group of 60 patients with colorectal cancer, principally south Indians, had a median faecal pH of 6–6–9–0, compared with a median pH of 6–1–6–5 in 120 matched healthy Indian subjects. The large differences in pH values between patients with colorectal cancer and the control group appear to be dependent on diet. The former ate non-masticatory meals of boiled refined rice, which were low in dietary fibre, and low in fermented milk products. The latter ate high fibre meals of thick whole wheat chapatties, vegetable and legume curries, and their diets were also high in fermented milk products such as yoghurt, yoghurt drink, white cheese, and ghee – made from fermented milk and rich in short chain fatty acids. Fibre fermenters in the gut lumen liberating large quantities of acetic acid (precursor of short chain fatty acids). This augments the H+ ion in the case of the controls.

S L MALHOTRA
27 East 37th Street, Apt 4
New York, NY 10016 USA

