

disease remains unclear. *C. pylori* is strongly correlated with the presence of chronic, non-autoimmune gastritis, but its pathogenic role in peptic ulceration is uncertain.

A recent report from Ireland describes a study in which patients with gastric and duodenal ulcers, gastritis and oesophagitis were entered into a randomized trial of colloidal bismuth subcitrate against cimetidine. Bismuth is known to have antibacterial action and to heal ulcers. This trial, like many others, extrapolates data from a therapeutic study to try to explain the role of *C. pylori* in peptic ulceration. About three-quarters of all the patients were *C. pylori* positive at the outset and although colloidal bismuth subcitrate

cleared *C. pylori* in most patients, healing was similar in the bismuth and cimetidine groups, despite the fact that *C. pylori* persisted in the patients treated with cimetidine. Indeed, several patients whose lesions healed on cimetidine were colonized with *C. pylori* during treatment. Growth of the organism is said to be favoured by a less acid environment.

Studies like this, which conclude by hinting darkly that the answer to peptic ulcer disease is at hand, seem to contain a number of internal inconsistencies. Although ulcer relapse rates are rather lower in patients treated with bismuth than in those treated with H<sub>2</sub>-receptor blocking drugs, acid suppression undoubtedly cures and prevents many ulcers

recurring. If rendering the gastric environment relatively alkaline favours the growth of *C. pylori*, it is difficult to understand how a disease-initiating or even disease-maintaining role for the organism can be argued. Indeed, contrary to its claims, this study more than most seems to present evidence which supports the idea that *C. pylori* is merely an epiphenomenon in the natural history of peptic ulcer disease.

(R.J.)

Source: Humphreys H, Bourke S, Dooley C, *et al.* Effect of treatment on *Campylobacter pylori* in peptic disease: a randomised prospective trial. *Gut* 1988; 29: 279-283.

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## INFECTIOUS DISEASES UPDATE

### The winter's viral infections

Up to the time of writing there has been no major influenza outbreak this winter. However, influenza is normally a late winter/early spring problem and spotter practices and virus laboratories have shown some 'flu activity in recent weeks. Parainfluenza virus (a cause of croup) and the respiratory syncytial virus (the cause of bronchiolitis) have also been conspicuous in causing much smaller seasonal outbreaks than is usual.

### Hepatitis B

Throughout the UK over the last two to three years there has been a marked decrease in the numbers of reported cases of acute hepatitis B. Monthly reports are now back to the level they were around 10 years ago. There is evidence that this fall has occurred in both of the high risk groups; drug abusers and homosexual men. This could mean that the infection has now 'saturated' these population groups but it seems more likely that the major factor is a reduction in needle sharing and a change in sexual practices. Those with acute hepatitis B normally become non-infectious three to six months after their initial illness. This is not the case, however, with human immunodeficiency virus (HIV) infection, which mimics hepatitis B in its risk factors. This makes it easier for HIV infection to spread heterosexually from these risk groups. There is no room for complacency.

### Enteric fever

A recent outbreak of paratyphoid among guests at a reception in the Midlands drew attention to this infection which is usually imported from Asia and Africa. *Salmonella typhi* (not to be confused with typhimurium) is the most usual pathogen

involved and is typically spread through contaminated food or water supplies where these are not protected. There are a number of known long term carriers throughout the UK but in the fit faecally continent patient who is sensible about toilet hygiene the risks of spread to others is minimal. The resulting illness is typically a septicaemia, not as is sometimes supposed a diarrhoeal illness, although this can occur, especially in children.

### The 'MRSA'

Staphylococci are renowned for developing resistance to antibiotics. Penicillin resistance is now generally presumed to be present with hospital infections and is found frequently in general practice. Flucloxacillin has been useful for coping with penicillin resistant staphylococci although recently resistance to this drug has been appearing. Flucloxacillin sensitivity is tested for in the laboratory using a very similar compound, methicillin. Hence the term methicillin resistant *Staphylococcus aureus* (MRSA). Just to confuse us, MRSA is sometimes used to mean 'multiply' resistant *S. aureus* which means the organism is resistant to methicillin but often to other antimicrobials such as erythromycin as well. These resistant strains of staphylococci do not appear to be more virulent or pathogenic than the sensitive strains but warrant respect when they infect wounds, burns or bed sores since extra care with hygiene and sometimes isolation may be necessary to avoid contaminating compromised patients such as those with diabetes mellitus, cystic fibrosis or indwelling venous or arterial catheters. Treatment of staphylococcal pneumonia or septicaemia can then be that much more difficult.

### Immunization schedules

When several immunizations are required, concern can arise as to which vaccines may be administered simultaneously and how much flexibility is acceptable in recommended schedules. When separate injections are required for each vaccine there may be a limit to the tolerance of the patient in terms of discomfort from the injections and reactions. There is little evidence however, especially with killed vaccines, that the desired immune response will be diminished by administering immunizations simultaneously; diphtheria, tetanus and pertussis have been given together successfully for many years. However, caution has been advised when more than one live viral vaccine is administered because there is a theoretical possibility that interference may reduce the response to a second vaccination if given too soon after the first. It is doubtful whether this matters in practice but it is usual for live viral vaccines to be given either on the same day or else about three weeks apart. When immunoglobulin is administered, for example to protect against hepatitis A, small amounts of antibody could also theoretically diminish the immune response to active vaccines given at around the same time. However, immunoglobulin is sometimes given intentionally with measles vaccines to diminish side effects in those predisposed and good immunity is still normally achieved. Ideally then immunoglobulin should be given separately from active vaccines but if this is impossible little is probably lost.

Suggestions for topics to include in future updates are welcomed and should be passed to the contributor, Dr E. Walker, Communicable Diseases (Scotland) Unit, Ruchill Hospital, Glasgow G20 9NB (041-946-7120), from whom further information about the current topics can be obtained.