LETTERS TO THE EDITOR

Staphylococcus lugdenensis and endocarditis

Further to the recent correspondence regarding *Staphylococcus lugdenensis* we report a further case of endocarditis due to this organism.

A 32 year old man presented with a history of fever, rigors for one week preceded by malaise, and weight loss for one month. He described a transient pain and weakness in the right forearm and hand (ulnar aspect) two days before admission. A toolmaker by profession, with no history of rheumatic fever or drug abuse, his only surgery (dental or otherwise) had been a vasectomy three months earlier.

Examination showed that his temperature was 38°C and blood pressure was 130/40 mmHg. Splinter haemorrhages were present on all fingers of both hands. A collapsing pulse was noted. On auscultation signs consistent with aortic regurgitation were found without obvious outflow obstruction, but no signs of cardiac failure. Clinical examination yielded otherwise normal results. Investigations included a white cell count of $12.5 \times 10^9/l$, haemoglobin concentration of 13.8 g/l, platelets $152 \times 10^9/l$, erythrocyte sedimentation of 70 mm/hour, and C-reactive protein of 19.6 mg/dl. Serum biochemistry and urine examination yielded normal results. An echocardiogram showed a tricuspid aortic valve with a single vegetation.

Staphylococci were isolated from a total of eight blood culture bottles. The slide coagulase test was positive with human plasma and negative with rabbit plasma. The tube coagulase test was negative with both plasmas.

Both Staphylase (Oxoid) and Staphaurex (Wellcome) tests were positive. The plate DNase test was negative after overnight incubation but positive at five days. API Staph (API Products Ltd, Basingstoke, Hampshire) identified the organism as *Staphylococcus hominis* biotype 1. A positive ornithine test identified it as *Staphylococcus lugdenensis*.² The organism was fully sensitive to penicillin, methicillin, aminoglycosides and vancomycin. It was non-phage typeable, using the standard set of phages.

The patient started intravenous flucloxacillin (2 g every four hours) plus gentamicin 120 mg twice daily, but within five days he developed early signs of cardiac failure, necessitating urgent valve replacement. At operation a grossly damaged and perforated aortic valve was replaced with a St Jude bileaflet mechanical prosthesis. Postoperative tidal concentrations were inadequate and the patient was changed to benzylpenicillin 1.2 g every four hours plus gentamicin 120 mg twice daily. After three weeks on this regimen he developed a severe allergic reaction to penicillin requiring a change to vancomycin 1 g twice daily plus netilmicin 80 mg twice daily for a further week. The patient made a steady recovery and remains well to date.

Coagulase negative staphylococci cause 5% of native valve endocarditis³ and of these 28% are on previously normal valves.⁴ At 32 years of age this is the youngest reported case of *Staphylococcus lugdenensis* native valve

endocarditis.⁵ It followed the same aggressive course as those described.^{1,5} The transient weakness in the patient's arm suggests an embolic phenomenon not previously reported in this condition and uncommon in other coagulase negative staphylococci endocarditis.⁴ The source of the organism remains unknown, although it is interesting to speculate that it may relate to his vasectomy. Further research is needed to establish the skin distribution and pathogenesis of endocarditis due to this organism.

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- 2 Freney J, Brun Y, Bes M, et al. *Staphylococcus lugdenensis* sp. nov. and *Staphylococcus schleiferi* sp. nov., two species from human clinical specimens. *Int J System Bacteriol* 1988;38:168-72.
- 3 Pelletier LL, Petendorf RG. Infective endocarditis. A review of 125 cases from the University of Washington Hospitals 1963-72. *Medicine* 1977;56:287-313.
- 4 Geraci JE, Hanson K, Giuliani ER. Endocarditis caused by coagulase negative staphylococci. *Mayo Clin Proc* 1968;43:420-34.
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Lack of in vitro activity of omeprazole against *Campylobacter pylori*

There is considerable evidence linking *Campylobacter pylori* infection with gastritis, peptic ulceration, and ulcer relapse.^{1,2} We investigated the presence of *C pylori* in 15 patients undergoing diagnostic upper gastrointestinal endoscopy and tested the antibacterial activity of omeprazole, a new anti-ulcer agent,³ against cultures of *C pylori* obtained from these patients.

Cytology brushings were used to obtain the Gram negative, urease and catalase positive, highly motile bacteria which were cultured microaerophilically (7-8 days at 37°C) for up to five generations on Wilkins-Chalgren agar containing defibrinated horse blood (70 ml/l), amphotericin B (0.02 g/l), cycloheximide (0.5 g/l), trimethoprim (0.25 g/l), vancomycin (0.06 g/l) and nalidixic acid (1 g/l). Electron microscopic examination confirmed that the bacteria were *C pylori* (figure), and was performed on bacteria fixed for one hour in glutaraldehyde (2% in phosphate buffer, 0.1M, pH 7.4), then washed for 30 minutes in the buffer, resuspended in ammonium

Growth inhibitory activity of omeprazole and furazolidone against four isolates of *C pylori*

Drug concentration (M)	Width of the growth inhibitory zones (cm) Mean (SE)	
	Omeprazole	Furazolidone
10^{-7}	0.0	0.0
10^{-6}	0.0	0.05 (0.05)
10^{-5}	0.0	0.25 (0.15)
10^{-4}	0.0	2.85 (0.50)

molybdate (2% in distilled water), and then applied to formvar films coated with carbon and wetted with bacitracin.

C pylori were obtained, by culture, from five out of six gastric ulcer or gastritis samples, all four duodenal ulcer or duodenitis samples, and five out of eight oesophagitis samples. These findings correlated well with the histopathological inflammatory changes, assessed by haematoxylin and eosin staining (six out of six gastric ulcer or gastritis, two out of three duodenal ulcer or duodenitis, and seven out of seven oesophagitis samples), detected in biopsy samples taken at an adjacent site to the cytology brushings. Detection of the bacterium in the biopsy specimens using Giemsa staining, however, was much poorer (three out of six, none out of three, and none out of seven of the respectively grouped complaints). Twelve out of 15 patients were *C pylori* positive by the culture method; only three out of 13 (two not assessed) were positive by the Giemsa method. Histopathological changes were present in 13 out of 14 patients (one not assessed).

The antibacterial activity of omeprazole (synthesised by Fisons plc) was compared with that of furazolidone (Norwich Eaton Pharmaceuticals, New York, USA), a known inhibitor of the bacterium,⁴ against four isolates of *C pylori* using a surface inoculated agar-well technique. Whereas furazolidone inhibited growth of the bacterium, neither omeprazole nor the vehicle control (4% polyethylene glycol 400 in physiological saline) had any effect (table).

Like others,^{1,2,5} we found that the organism was present in a high proportion of patients with peptic ulcers or gastritis or duodenitis, as well as in those with reflux oesophagitis.

The superiority of microbiological culture in our hands compared with Giemsa staining for identifying the bacterium may have been related to the sampling techniques used. Given the patchy distribution of *C pylori*, it is more likely that the bacterium will be found by cytology brushing than by biopsy.

If *C pylori* is responsible for ulcer relapse² then it seems unlikely on this score that

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- 1 Marshall BJ, Royce H, Annear DI, *et al.* Original isolation of *Campylobacter pyloridis* from human gastric mucosa. *Microbios Lett* 1984;25:83-8.
- 2 Marshall BJ, Goodwin CS, Warren JR, *et al.* Prospective double-blind trial of duodenal ulcer relapse after eradication of *Campylobacter pylori*. *Lancet* 1988;ii:1437-42.
- 3 Anonymous. Omeprazole. [Editorial]. *Lancet* 1987;iii:1187-8.
- 4 Howden A, Boswell P, Tovey F. In-vitro sensitivity of *Campylobacter pyloridis* to furazolidone. *Lancet* 1986;ii:1035.
- 5 Borkent MV, Beker JA. Treatment of ulcerative reflux oesophagitis with colloidal bismuth subcitrate in combination with cimetidine. *Gut* 1988;29:385-9.

with donor plasma products. These disadvantages are overcome with extracorporeal antibody immunoabsorption in which extensive plasma treatment is feasible without recourse to the use of donor plasma. Patients with Christmas disease with inhibitors have been successfully treated in this way,⁴ but as yet there have been no reports in cases of haemophilia A.

Using venous access via the antecubital vein, over five days, a total of 35 litres of plasma was generated on a Hemonetics V-50 cell separator machine and immunoabsorbed on protein A sepharose columns using Citem 10 extracorporeal immunoabsorption treatment system (Excorim, Lund, Sweden). The figure shows that serum IgG concentrations and factor VIII inhibitor concentrations fell rapidly with each treatment. With about 30 machine cycles of 200 ml of plasma, the technique was very effective at reducing high concentrations of inhibitor but was less effective at lower concentrations. There were also slight overnight increases in IgG and VIII inhibitor concentrations which were probably explained by the equilibration of extravascular and intravascular IgG. On the fourth day the inhibitor concentrations had fallen to <0.3 IU/ml, but an infusion of human factor VIII calculated to raise the patient's factor VIII concentrations by 200 IU/dl only achieved an increase from 0 to 6 IU/dl. After a further two days of plasmapheresis and immunoabsorption a further infusion of factor VIII increased the

On the subsequent two days, similar infusions of factor VIII did not increase factor VIII concentrations and the inhibitor reappeared. To what extent this anamnestic response was modified by the factor VIII infusion on day 3 or the previous desensitisation regimen is uncertain. Factor VIII treatment was continued for a total of seven days and when reviewed three weeks later he had made a full recovery.

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Factor VIII inhibitors (—), serum IgG concentrations (-----), and factor VIII concentrations before and after factor VIII infusions in a patient treated with protein A immunoadsorption.

- 1 Rizza CR, Jones P. Management of patients with inherited coagulation disorders. In: Bloom AL, Thomas DP, *Haemostasis and thrombosis*. London: Churchill Livingstone, 1987:465-93.
- 2 Rizza CR, Mathews JM. Effect of frequent factor VIII replacement on the level of factor VIII antibodies in haemophiliacs. *Br J Haematol* 1982;37:53-66.
- 3 Cobcroft R, Tangini G, Dormandy KM. Serial plasmapheresis in a haemophilic with antibodies to factor VIII. *J Clin Pathol* 1977;30:763-5.
- 4 Nilsson IM, Jonsson S, Sundquist SV, Alberg A, Bergentz SE. A procedure for removing high titre antibodies by extracorporeal Protein A. Sepharose adsorption in haemophilia: Substitution therapy and surgery in a patient with Haemophilia B and antibodies. *Blood* 1981;58:38-44.
- 5 Palmer A, Welsh K, Gjorstrup P, Taube D, Berwick M, Thick M. Removal of anti HLA antibodies by extracorporeal immunoadsorption to enable renal transplantation. *Lancet* 1989;i:10-12.

Lecture Notes on Forensic Medicine. 5th ed. DJ Gee, AA Watson. (Pp 229; paperback £12.95.) Blackwells. 1989. ISBN 0-632-02595-6.

This paperback makes a very welcome return in its fifth edition and is mostly unscathed with only some loss of width, a gain in height, and the acquisition of a blushing Aesculapian serpent on its front cover: there is a decrease in the number of its chapters (30), pages (220) and in the breadth of the subjects covered. In this edition, regius Professor Alan Watson has joined emeritus Professor David Gee as a coauthor adding a modicum of Scottish law at the appropriate places and further enhancing the book's appeal to the countries on both sides of Hadrian's Wall. Presumably the reason why such subjects as criminal abortion are still covered at length also stems from the necessity to retain a transcontinental clientele.

The "Lecture Notes" series is specifically

intended for undergraduates and recently fledged doctors and there can be no doubt that this role is fulfilled most admirably in the specialty of forensic medicine through the commendable conciseness and clarity of text, the subdivision of the chapters by numerous subheadings, and the informative inclusion of simple illustrative line drawings—all at a price (slightly inflated from the last edition) easily accessible to a student's pocket.

The authors seem to have placed great faith on their previous reviewers and I therefore tentatively venture to suggest some changes in emphasis for the forthcoming edition: breath alcohol—assaying machines are here to stay—and child non-accidental injury and sexual abuse will persist in the limelight. The nuances of (Scottish) precognitions and fatal accident inquiries could be highlighted even further. I also hope that the pious hope expressed that the Procurator Fiscal "will always require an autopsy to be carried out" comes to pass by then.

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ERRATA

Errata 1

In the indexed letter, "Lack of in vitro activity of omeprazole against *Campylobacter pylori*," A M Gheleni *et al* (1990;43:171). The figure legend to the figure reproduced below was inadvertently omitted. We apologise for this error.

Electron micrograph showing *C pylori* negatively stained with ammonium molybdate. Although only two intact flagella are seen here, the basal portions of two other flagella are visible (arrowed). A terminal paddle is just visible on one of the two intact flagella. A terminal paddle from another specimen is shown enlarged in the inset.

Errata 2

Part of the Appendix in "Guidelines on oral anticoagulation: second edition" by the British Society for Haematology (1990;43:177-84) was incorrectly transcribed. The correct version is printed below. We apologise for this error.

Infections:

Aminoglycosides:	Griseofulvin
Amikacin	Rifampicin
Gentamicin	
Kanamycin	
Neomycin	
Streptomycin	
Tobramycin	
Co-trimoxazole	
Cephalosporins:	
Cephaloridine	
Cephazolin	
Cephalexin	
Latamoxef	

NOTICES

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