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.

B WALSH

JP MOUNSEY

Departments of Bacteriology and Cardiology,
John Radcliffe Hospital,
Headington,
Oxford, OX3 9DU.

- 1 Smyth EG, Wright ED, Marples RP. New type of staphylococcal endocarditis. *J Clin Pathol* 1988;41:809-14.
- 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.
- 5 Etienne J, Pignon B, Leport C, et al. *Staphylococcus lugdenensis* endocarditis. *Lancet* 1989;i:390.

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

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.

A BUSUTTIL

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

ATB '90

**6th European Edition of the
Oak Ridge Conference on
Advanced Technology for the
Clinical Laboratory and
Biotechnology**

MILAN FAIR
28-30 November 1990

Further details from:

Conference Secretariat:
Via C. Farini, 70-20159 Milan, Italy
Telephone: (02) 6070074
Fax: (02) 680064

Association of Clinical Pathologists

Model Training Programmes

Histopathology/Cytopathology/Morbid
Anatomy
Medical Microbiology
Haematology and Blood Transfusion
Chemical Pathology
Immunology

The Second revised version of these training programmes has now been published in a single booklet. It offers detailed guidance on training in each of the laboratory medical disciplines with recommended reading lists and learning objectives set for those aspiring to consultant status. The booklet should also be of interest to medical graduates who wish to find out what a career in each of these disciplines entails.

Copies are available from the General Secretary, Association of Clinical Pathologists, School of Biological Sciences, Falmer, Brighton BN1 9QG, UK.

Price £7.50 (inclusive of postage and packaging). Cheques/sterling drafts should be made payable to the Association of Clinical Pathologists.

ACP Locum Bureau

The Association of Clinical Pathologists runs a locum bureau for consultant pathologists.

Applicants with the MRC Path who would like to do locums and anyone requiring a locum should contact Dr David Melcher, Histopathology Department, Sussex County Hospital, Eastern Road, Brighton BN2 5BE.