Letters to the Editor

Comparison of radiometric and gas capture systems for blood cultures

In their paper comparing radiometric and gas capture systems for blood cultures King *et al*¹ used a stock culture for *Gardnerella vaginalis*. We report a case of *G vaginalis* septicaemia detected by the gas capture system in a 60 year old man following transurethral resection of the prostate. On the third postoperative day the indwelling urinary catheter was removed and the patient developed rigors. A midstream specimen of urine and blood for culture in an Oxoid signal bottle were collected. The patient was successfully treated with gentamicin.

Examination of the urine showed scanty white and red blood cells on microscopy and no significant bacteriuria on culture on cystine lactose electrolyte deficient agar after 18 hours of aerobic incubation. The Oxoid signal bottle was gently mixed twice daily and showed a positive signal and haemolysis after 24 hours. A Gram film of the broth showed Gram variable bacilli. Culture on chocolate agar in 10% carbon dioxide and blood agar anaerobically yielded a pure growth of small Gram variable bacilli, which were shown to be oxidase and catalase negative, X and V factor independent, resistant to 5 µg but sensitive to 50 µg discs of metronidazole, and resistant to a 1000 µg disc of sulphamethoxazole, and sensitive to gentamicin. Use of API Strep gave the numerical profile 2052001, consistent with *G vaginalis*, an "excellent identification."

Asymptomatic carriage of *G vaginalis* was found in 11.4% men attending a clinic for sexually transmitted diseases.² Reports of *G vaginalis* septicaemia in men are rare, but include one following transurethral resection of the prostate (TURP), in which the blood cultures were positive after seven days of incubation,³ and one in which septicemic shock occurred, blood culture positive after two days.⁴ We believe this to be the first detection and isolation using the gas capture system.

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Identification of non-capsulate strains of *Streptococcus pneumoniae* by coagglutination

Dr Pease *et al* have shown the need for a reliable method to differentiate non-capsulate strains of *Streptococcus pneumoniae* from other non-pneumococcal α -haemolytic streptococci, isolated from eyes.¹ Although they found that non-capsulate pneumococci were susceptible to optochin, the results of confirmatory tests such as bile solubility and biochemical identification using API 20 Strep (API Laboratory Products) were variable. Shayegani *et al*² used immunodiffusion and counter-immunoelectrophoresis to determine the presence of common pneumococcal antigen on non-capsulate strains, and workers at the World Health Organization Laboratory for Reference and Research on pneumococci consider that antiserum to pneumococcal C-polysaccharide is an additional diagnostic aid for the differentiation of pneumococci

from other streptococci (Sorensen UBS, Henrichsen J, personal communication).

As part of a large study to determine the distribution of pneumococcal types in different kinds of clinical material from patients in Scotland, we recently examined 628 strains of *S pneumoniae* that had been isolated from eyes over 18 months. Most of these strains were referred to us for serotyping by other diagnostic bacteriology laboratories in Scotland. Seventy seven of the 628 strains (12%) did not react in the coagglutination test carried out with diagnostic pneumococcal antisera (Pools A to I and type specific antisera, Statens Serum-institut, Copenhagen). They all reacted, however, in tests with antiserum to pneumococcal C-polysaccharide (Statens Serum-institut, Copenhagen). Using the same criteria, we also found that 1.5% of 2254 strains of pneumococci from respiratory specimens and 1.3% of 151 strains from ear swabs were non-capsulate. By contrast, all of 449 strains of *S pneumoniae* isolated from invasive pneumococcal infection were capsulate.

Clearly, factors exist which select for non-capsulate variants of pneumococci in eyes.

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Book reviews

Inflammation. Basic Mechanisms, Tissue Injuring Principles, and Clinical Models. Ed P Venge, A Lindbom. (Pp 488; 353 Sek.) Almquist & Wiksell International. ISBN 91-22-00790-3.

This is the second in a series of volumes, intended to update important aspects of inflammation. It draws on a series of lec-

tures given at Uppsala University Hospital in November 1984. The book is multiauthor and comprises 34 chapters, each written by an authority on that subject. For convenience the volume is divided into three sections: basic mechanisms; tissue injury principles; clinical models. The first deals essentially with cells in inflammation, starting with the complement system, then microvascular endothelium which includes permeability and leucocyte adherence. Sadly, no mention is made here of the role of interleukin I, although the leucotrienes are

adequately covered (this could be a reflection of the 1984 timing when the lecture was prepared). It also indicates how rapidly the field changes and the need for such updating volumes as this. This first section proceeds through mast cells, eosinophils, neutrophils, phagocytosis, macrophages and lymphokines, finally discussing platelet derived growth factor. Many of us are poised on the edge of the pool of molecular biology knowledge, and this chapter certainly helps to get one's feet wet and was a refreshing experience.