Correspondence

The challenge and the reproach of infective endocarditis

Sir,

We have the following comments to make on Professor Goodwin's editorial (*Br Heart J* 1985; 54: 115-8).

(a) We know of no evidence that fungal endocarditis has increased, and certainly not as the result of "intensive and lengthy therapy for advanced bacterial infective endocarditis." We therefore do not agree with the recommendation for weekly prophylactic amphotericin. Nor do we know of any evidence that the immunocompromised are especially vulnerable to endocarditis.

(b) It is not known whether appropriate prophylaxis will prevent endocarditis although it is assumed that many potential cases are prevented by it.

(c) The recommendation of the British Society for Antimicrobial Chemotherapy Working Party is for a single oral dose of 3 g not 2 g of amoxycillin for dental prophylaxis.

(d) Not all "streptococci likely to have come from the mouth" can be satisfactorily killed *in vitro* with penicillin alone. Thus treatment with penicillin alone is inappropriate, as indeed is ampicillin alone for faecal streptococci.

(e) Potassium penicillin in large intravenous doses can cause hyperkalaemia, but it is no longer available for parenteral use.

(f) The recommendations for gentamicin blood concentrations in streptococcal endocarditis might result in toxicity. The working party recommended that gentamicin should be given in a low dose to enhance the activity of penicillin. Peak blood concentrations of 3-5 mg/l and trough blood concentrations <1 mg/l are sufficient for this purpose. Gentamicin should seldom be given for more than two weeks. Gentamicin can be used in combination with flucloxacillin to treat staphylococcal endocarditis and here the dose will need to be greater than that for streptococcal infection to achieve peak blood concentrations of 5-10 mg/l and trough blood concentrations of <2 mg/l.

(g) Early prosthetic endocarditis with *Staphylococcus aureus* is nearly always the result of sternal wound infection and resultant bacteraemia. The aetiology of late prosthetic endocarditis is quite different from early onset infection. It is caused by the same spectrum of organisms as those causing

natural valve infection.

(h) Antibiotic resistance of the infecting organism is seldom the reason for a fatal outcome in endocarditis. The mortality of this disease remains high because of late diagnosis, poor choice and incorrect dosage of antibiotics, and, more important, late referral to cardiologists and cardiac surgeons.

(i) The most important initial investigations in suspected endocarditis are blood cultures. An erythrocyte sedimentation rate and haemoglobin are not a substitute because they are non-specific. Where "viridans streptococci" are cultured from the blood the diagnosis is nearly always endocarditis and sometimes it may come as a surprise.

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This letter was shown to Professor Goodwin, who replies as follows:

Sir,

I welcome the opportunity of replying to Dr Eykyn's important comments as her letter gives me the chance to reinforce the message in my editorial that early diagnosis and early consultation with the cardiac surgeons are important for success in treatment and reduction of mortality.

(a) I know of no evidence that fungal infection has increased nor did I quote any. I suggested that "fungal infection may paradoxically have increased as a result of more intensive and lengthy chemotherapy for advanced bacterial infective endocarditis." This is a personal view based on personal experience, and I believe this view may be shared by others.¹

I pointed out that "patients who are immunologically compromised, such as those taking immunosuppressive drugs are especially vulnerable to infection of any sort." I do not think that any experienced clinician or microbiologist would seriously challenge this statement. I did not quote any