evidence on our present capabilities shows that most of us will fail when that moment arrives.

PETER J F BASKETT

Consultant Anaesthetist,
Frenchay Hospital,
Bristol BS16 1LE

1 Casey WF. Cardiopulmonary resuscitation: a survey among junior hospital doctors. *J R Soc Med* 1984;77:921-4.
2 Lowenstein SR, Hansbrough SF, Libby LS, Hill DM, Mountain RD, Scroggin OH. Cardiopulmonary resuscitation by medical and surgical house officers. *Lancet* 1981;ii:679-81.
3 Baskett PJF, Sowden GR, Robins DW. Ethics in cardiopulmonary resuscitation. *American Journal of Emergency Medicine* 1984;2:273-4.
4 Brown CG, Sanders AB, Gurley HT, Stair TO, Morkovin V, Jayne HA. Curriculum for undergraduate education in emergency medicine. *J Med Educ* 1984;59:427-9.
5 Anonymous. Evaluation of medical curricula [Editorial]. *Lancet* 1972;ii:1014-5.

Infective endocarditis: a preventable disease?

Sir Thomas Lewis described infective endocarditis as ". . . a progressive disease, ending fatally with so few exceptions that little or no hope of the patient surviving is to be entertained."[1] That was in the preantibiotic era, when the disease was causing about 1000 deaths a year, mostly in patients aged between 20 and 40.[2,3] The disease still carries a mortality of about 30% and causes around 200 deaths a year in England and Wales,[4] but most of the deaths now occur in patients aged 60 or more.[5] The decline in rheumatic fever, the increase in the elderly population, and the appearance of new groups at risk—particularly patients with prosthetic heart valves—have contributed to the changed pattern, while the prognosis has been improved by treatment with antibiotics and valve replacement.

Efforts to prevent the disease have failed—regrettably—as judged by the persisting frequency of streptococcal endocarditis.[6] The hope that much infective endocarditis should be preventable was based on the findings of workers in the preantibiotic era; they knew that the valves of normal hearts could be infected and destroyed in severe septicaemic illnesses but they recognised a much more common disease, subacute bacterial endocarditis, which appeared to occur only in patients with rheumatic or congenital heart disease and in which a common low grade oral pathogen, *Streptococcus viridans*, was almost invariably incriminated.[1] Subacute bacterial endocarditis after dental extraction had been reported in 1930,[7] and investigations in the mid-'30s showed that dental extractions commonly caused a bacteraemia.[8,9] The picture seemed clear: a structural abnormality of the heart was an essential risk factor and dental procedures were the most common cause of the bacteraemia which initiated the illness.

When antibiotics arrived a formula for preventing many cases of infective endocarditis seemed obvious—treat patients with valvular or congenital heart disease before dental procedures. The success of this formula was clearly going to depend on three factors: firstly, that the premises were correct; secondly, that all patients at risk could be identified; and, thirdly, that the selected prophylactic regimens were given correctly and were effective. Unfortunately, difficulties and doubts have emerged on each count. The importance of dental procedures is probably not as great as was originally thought,[10-12] infective endocarditis often occurs in patients with no known heart disease,[13] recommended prophylactic regimens have not been followed,[14] and, finally, no study has ever been performed to show that prophylaxis prevents infective endocarditis.

The current pattern of infective endocarditis and the part played by dental procedures in particular have been put in broad perspective by the results of a study (planned by the British Cardiac Society in association with the Medical Services Study Group of the Royal College of Physicians of London) in which cases of infective endocarditis during 1981 and 1982 were investigated retrospectively by means of a questionnaire.[15] Unfortunately, reporting was incomplete and, as judged by the mortality statistics from the Office of Population, Censuses, and Surveys and from the *Weekly Communicable Disease Reports*,[5,16] the cases notified to the investigators were less than one third of the total occurring during the inquiry. Nevertheless, 544 episodes of infective endocarditis were available for analysis making the study the most comprehensive ever undertaken in the British Isles.

The mean age of patients was 52 years and men outnumbered women two to one, possibly reflecting the higher incidence of bicuspid aortic valves and calcific aortic stenosis in men. Two hundred and thirty patients (43%) either had normal hearts or a previously unrecognised cardiac abnormality before the onset of endocarditis. Seventy seven (14%) had prosthetic heart valves. Seventy four patients (14%) died (mean age 59 years), and there was a differential mortality according to the nature of the infecting organism—30% in staphylococcal infections, 14% in infections caused by bowel organisms, and 6% in other streptococcal infections. The most common infecting organism proved to be *Str viridans*, which was isolated in 262 (48%) of the reported cases. Dental procedures had been performed within the three months before the onset of illness in only 74 patients (14%), but within this group *Str viridans* was the infecting organism in 53 (72%). Half of the patients who developed endocarditis after a dental procedure were not in line for prophylaxis because they had no known cardiac abnormality, but in the 37 patients with a known cardiac defect a dental procedure was performed without prophylaxis in 30. Seven patients developed infective endocarditis after a dental procedure despite prophylaxis, and a *Str viridans* was isolated in four of these. A further 48 patients had not undergone a dental procedure but did have overt dental sepsis, and in 33 (69%) of these the infective agent was *Str viridans*. Thus in 122 (22%) of all reported cases a dental procedure or dental sepsis may have been implicated, but on clinical and bacteriological grounds a non-dental source of infection was considered more likely in 19 reducing the totals to 64 (12%) for dental procedures and 39 (7%) for dental sepsis. *Str viridans* was the infecting agent in 176 patients in whom no recent dental procedure had been performed and in whom no dental sepsis was apparent. On the grounds that the mouth and nasopharynx are the most likely sources of the most common organism, *Str viridans*, and that periodontal disease is present in more than one third of our adult population,[17] poor dental hygiene was thought to represent a greater risk than dental procedures.

In two further papers based on the study Bayliss and his colleagues have provided an overview of the microbiological and pathogenetic features of infective endocarditis in the reported cases.[18,19] Of the 544 episodes, 63% were caused by streptococci (48% viridans, 15% other streptococci), 19% by staphylococci, a wide variety of organisms accounted for a few cases, and 10% were culture negative. Bowel organisms accounted for 14% of the episodes. In 60% of all cases a portal of entry was not apparent; the probable origins of infection in the remainder were dental (19%), alimentary, genitourinary, respiratory, or skin (16%), and invasive procedures affecting the bloodstream (5%). The list of disorders and procedures

which probably resulted in a bacteraemia was comprehensive and included all common gastrointestinal and genitourinary operations, endoscopic procedures (including bronchoscopy), and common infections; cases also followed the insertion of a vaginal pessary, liver biopsy, acupuncture, parturition, blood donation, phlebography, haemodialysis, fractures, and cardiac catheterisation. There were also six drug addicts among the 541 patients.

How should the results of this survey influence future practice? Firstly, the authors call for better routine dental care. The dominance of *Str viridans* infection in cases of infective endocarditis even when no recent dental procedure has been performed and the reported high prevalence of periodontal disease in the community argue strongly in favour of this recommendation.[17] Would, however, the risk from the increased number of dental procedures required to achieve this goal of improved dental hygiene counterbalance the expected benefit? Secondly, though the risk of dental procedures (not merely extractions) does not appear so great as traditionally believed the investigators decided that prophylaxis must still be given to patients at risk. They supported the recommendations of the British Society for Antimicrobial Chemotherapy (amoxycillin for dental procedures; erythromycin for those allergic to penicillin; and a combination of amoxycillin and gentamicin against bowel organisms) and called for wider publicity for these recommendations.[20] Thirdly, the numbers of cases associated with infection from the alimentary, genitourinary, and respiratory tracts and skin indicate the need for antibiotic prophylaxis for patients with cardiac defects at the time of any surgical procedure or endoscopy.

These aims are prudent and are likely to be endorsed. There remains, however, the problem of a group of patients (43% in this survey) with normal or apparently normal hearts who develop infective endocarditis. Within this group elderly patients seem to be at particular risk, and Bayliss *et al* argue that antibiotic cover should be considered when any surgical, alimentary, or genitourinary procedure (including endoscopy) is performed in an elderly patient—whether or not a cardiac defect is present. Other groups at enhanced risk include diabetics, alcoholics, the immunosuppressed, drug addicts, and patients with malignant disease or any serious illness.

The provision of antibiotic cover at times of suspected risk to any group of patients with apparently normal hearts in an attempt to prevent infective endocarditis would be treading new ground. The number of surgical, investigatory, and dental procedures performed each year on patients aged 60 and over with apparently normal hearts must be enormous, and the advantages of prophylaxis for this group might accrue more to the manufacturers of antibiotics than to the patients. Indeed, such a policy may not be without risk. The difficulties of mounting a controlled trial to assess the benefits and risks of prophylaxis in such patients would be as great as the event rates would be small, but surely objective evidence must be obtained before this untested recommendation is adopted.

Finally, Bayliss *et al* point to the need for doctors and dentists to appreciate that the pattern of infective endocarditis has changed considerably in the past 50 years. There has been no shortage of publications on the subject, but perhaps we need a new method of maintaining awareness of the problem and a watch for further changes. This might be achieved by treating deaths attributed to infective endocarditis in the same way as maternal deaths and subjecting them to detailed and confidential inquiry. The annual number of deaths from the disease would not preclude this, and from the regular information provided we might get closer to the truth about the number of patients dying from infective endocarditis for want of prophylaxis—or despite it.

G KEITH MORRIS

Senior Lecturer in Medicine,
University Hospital,
Nottingham NG7 2UH

1 Lewis T. *Diseases of the heart*. London: MacMillan and Co Ltd, 1943.
2 Hayward GW. Infective endocarditis: a changing disease. *Br Med J* 1973;ii:706-9.
3 Hayward GW. Infective endocarditis: a changing disease. *Br Med J* 1973;ii:764-6.
4 Oakley CM. Infective endocarditis. *Br J Hosp Med* 1980;**24**:232-43.
5 Office of Population Censuses and Surveys. *Mortality statistics: England and Wales*. London: HMSO, 1974-80.
6 Oakley CM, Somerville W. Prevention of infective endocarditis. *Br Heart J* 1981;**45**:233-5.
7 Rushton MA. Subacute bacterial endocarditis following the extraction of teeth. *Guy's Hospital Reports* 1930;**80**:39-44.
8 Okell CC, Elliott SD. Bacteraemia and oral sepsis with special reference to the aetiology of subacute endocarditis. *Lancet* 1935;ii:869-72.
9 Burket LW, Burn CG. Bacteremias following dental extraction. Demonstration of source of bacteria by means of a nonpathogen (Serratia marcescens). *J Dent Res* 1937;**16**:521-30.
10 Lowes JA, Hamer J, Williams G, *et al*. 10 years of infective endocarditis at St Bartholomew's Hospital: analysis of clinical features and treatment in relation to prognosis and mortality. *Lancet* 1980;i:133-6.
11 Schnurr LP, Ball AP, Geddes AM, Gray J, McGhie D. Bacterial endocarditis in England in the 1970s: a review of 70 patients. *Q J Med* 1977;**46**:499-512.
12 Moulsdale MT, Eykyn SJ, Phillips I. Infective endocarditis, 1970-79: a study of culture positive cases in St Thomas's Hospital. *Q J Med* 1980;**49**:315-28.
13 Weinstein L. Infective endocarditis: past, present and future. *J R Coll Physicians Lond* 1971;**6**:161-74.
14 Durack DT. Current practice in prevention of bacterial endocarditis. *Br Heart J* 1975;**37**:478-81.
15 Bayliss R, Clarke C, Oakley C, Somerville W, Whitfield AGW. The teeth and infective endocarditis. *Br Heart J* 1983;**50**:506-12.
16 Communicable Disease Surveillance Centre. *Weekly Communicable Disease Reports*. London: Public Health Laboratory Service, 1981-2.
17 Todd JE, Walker AM, Dodd P. *Adult dental health. United Kingdom*. Vol 2. London: HMSO, 1980. (OPCS.)
18 Bayliss R, Clarke C, Oakley CM, Somerville W, Whitfield AGW, Young SEJ. The microbiology and pathogenesis of infective endocarditis. *Br Heart J* 1983;**50**:513-9.
19 Bayliss R, Clarke C, Oakley CM, Somerville W, Whitfield AGW, Young SEJ. The bowel, the genitourinary tract and infective endocarditis. *Br Heart J* 1984;**51**:339-45.
20 Simmons NA, Cawson RA, Clarke C, *et al*. The antibiotic prophylaxis of infective endocarditis. Report of the working party of the British Society for Antimicrobial Chemotherapy. *Lancet* 1982;ii:1323-6.

Fixed drug eruptions

Fixed drug eruptions continue to puzzle dermatologists. A drug may be well tolerated by a patient for weeks, months, or even years, and then a further dose of the drug produces —usually within a few hours—the appearance of one or more raised, erythematous, sharply demarcated, round or oval plaques, often pruritic and sometimes bullous. The lesions may occur on any part of the skin; less commonly they may appear on mucous membranes. A lesion appearing on the lip simulating herpes labialis has recently been reported in the *BMJ*.[1]

The skin lesions heal with scaling, and finally all that is left is a sharply demarcated area of pigmentation. Further administration of the drug causes a recurrence of the lesion in the same area, though new lesions may develop elsewhere. There are usually no constitutional disturbances. The susceptible areas have no obvious characteristics to suggest why they become affected whenever the drug is taken while the surrounding skin continues to appear normal. Fixed drug eruptions must presumably be due to local abnormalities in the skin, which determine the sites of the lesions; and since their round or oval shape might correspond with the distribution of terminal vessels or nerves either or both of these may be implicated.

Several groups of workers have investigated the mechanism which determines the localisation of the skin