others scrape along and one claims that it cannot afford to keep all its beds in use. Payments from the NHS to many units are unpredictable—and the prospects for the next few years look very gloomy, with ever increasing pressure on NHS budgets.

Much of this confusion was apparent last year when the BMA annual representative meeting debated the issue. Among the problems raised were the high cost of drugs in independent hospices—a familiar problem in all branches of non-NHS medicine—the lack of any planning in the distribution of hospices around the country, and the need for specialist training for the medical and nursing staff.

Now at least two groups are being formed from doctors and charitable workers to promote the financing and further development of the hospice movement. Several questions need to be answered at this stage if the era of well intentioned amateurism is to be succeeded by hard headed professionalism.

Firstly, should the care of the dying be part of the NHS? Or should it be part NHS, part private sector? If the private sector is to provide for much of the future expansion of terminal care, who will plan the siting of new units? Who will set standards of staffing and performance? If, on the other hand, the NHS is to take on the responsibility, will enough new money be found to provide both capital and revenue costs? Or will the sorry story of end stage renal failure be repeated, with never enough money to treat the patients in need?

Secondly, whether in NHS or private units, whose responsibility should be the training of staff? The National Society for Cancer Relief has helped organise an ad hoc education group which is planning two training courses for doctors and two for nurses, but very soon the policy making bodies will need to decide whether patients in hospices should be looked after by any doctor willing to do the work or by consultants and junior staff trained in the same way as in other medical specialties. Ideally at least some medical staff should be in undergraduate teaching units; and account needs also to be taken of the risk of "burnout" in staff working in this specialty; possibly career appointments should be part time or rotating with other branches of oncology.

What should not be allowed is a continuing drift with no planning, no long term financial provision for running costs, and no system of either training or audit. Many people in the hospice movement are practical, experienced, and knowledgeable; what is needed now is a meeting of the various charities and other parties with a view to achieving consensus.

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Pneumococcal infections

Streptococcus pneumoniae has been recognised as a pathogen for 100 years, yet we still do not know its full clinical importance. Recent diagnostic techniques such as counterimmunoelectrophoresis have helped reawaken interest in this organism and in the importance of serotyping the pathological strains. *S pneumoniae* is a main cause of pneumonia, but the severity of the disease varies both among patients and among serotypes. In Nottingham counterimmunoelectrophoresis and culture implicated *S pneumoniae* as a pathogen in three quarters of patients with community acquired pneumonia.[1] In patients with pneumonia but negative sputum cultures in Edinburgh, pneumococcal antigen was detected by counterimmunoelectrophoresis in 69% of sputum samples and from 37% of serum samples.[2] Despite intensive care and antibiotic treatment non-bacteraemic pneumococcal pneumonia still has a mortality of 6-19% in hospital patients[3]; in bacteraemic pneumococcal pneumonia this rises to 30-40%.[1 4–6] In patients with renal or hepatic insufficiency the mortality for bacteraemic pneumococcal pneumonia may be as high as 68%.[6] Increasing age appears to be an independent risk factor in bacteraemic disease,[6] though this effect may be attributed in part to atypical presentation and delayed treatment in the elderly.[7]

In both bacteraemic and non-bacteraemic pneumonia the type 3 pneumococcus has long had a sinister reputation.[4 8] In Nottingham 37% of patients with type 3 infection died as opposed to 17% with type 1 and 10% with type 7 infections.[1]

S pneumoniae meningitis may follow bacteraemia or be associated with an underlying mastoiditis, sinusitis, or skull fracture. In children under 10 it is the third most common cause of meningitis but may carry a higher mortality than infections with meningococci or *Haemophilus influenzae*.[9] Most cases occur in children under 2, and the risk of neurological sequelae was found to be 14% in Danish children.[10] In adults the highest incidence and mortality are seen in people over 60.

Occult pneumococcal bacteraemia in children deserves further interest and study.[11] If North American experience is representative, two or three per 100 young children presenting with fever—and often with signs of an upper respiratory tract infection only—will have *S pneumoniae* bacteraemia.[12] Of those who do not have an obvious focus of infection, some recover without antibiotic treatment. A substantial proportion will, however, develop pneumonia, meningitis, or otitis media, occasionally even when antibiotics have been given for the initial illness.[12] The incidence of occult pneumococcal bacteraemia in British children is unknown,[13 14] and its relation, if any, to pneumococcal carriage and concurrent viral infection has yet to be elucidated.[15]

The epidemiological features of infection with *S pneumoniae* and carriage of the organism have been the subjects of many studies. Healthy people frequently carry *S pneumoniae* in their noses and throats, and most pneumococcal disease is probably contracted from carriers. In family studies children under 2 have the highest rates of carriage (38% in preschool children in one study) and carriage declines with age; in the absence of an epidemic, adults with children have a higher rate of carriage than those without (19% compared with 6%).[16] A new strain of pneumococcus may be acquired from another carrier or from a patient with pneumococcal disease, and families with one member with pneumococcal disease have a carriage rate of up to 74%.[17] Strains may be carried for months [18 19] though possibly most disease is caused by recently acquired strains.[18]

Studies of contracts of patients with disease caused by pneumococci resistant to antibiotics have helped our understanding of the spread of the pneumococcus in closed communities. In one incident starting with a patient with pneumococcal meningitis 27% of children from the same room at a day care centre carried the strain, as did 11% of older children and staff.[20] Families of the colonised children had a carriage rate of 33%, but no carriers were found in other day centres.

In Johannesburg in 1977, an outbreak of antibiotic multiresistant pneumococcal disease occurred in several hospitals.[21]

Most of the children admitted to a measles ward where a patient with pneumococcal disease had been nursed were later found to be carriers, and three developed pneumococcal bacteraemia. Attempts to eradicate carriage among the staff were complicated by the emergence of rifampicin resistance.

Epidemics of pneumococcal disease tend to occur in large closed communities.[22 23] Pneumococcal carriers have been firmly implicated in the spread of infection,[23] but there are well documented cases of transfer from patient to patient or patient to doctor.[24] More recently, simultaneous occult pneumococcal bacteraemia has been reported in twins,[25] and two sisters are reported to have developed pneumococcal disease within 48 hours of each other.[26] In large studies of pneumococcal bacteraemia several patients are usually found to have acquired their infection in hospital (p 1195).[4 6 21] If pneumococcal serotyping was carried out as frequently as in the 1930s more cases of cross infection would be reported.

Since man is the reservoir for *S pneumoniae*, and since pneumococcal carriage is so common, few measures to prevent cross infection, apart from sputum disposal, are justified outside an epidemic. In the past patients with pneumonia have sometimes been nursed in isolation,[27] but it is not common practice now, nor is it feasible in most hospitals.

Laboratory and clinical surveillance is needed, firstly, to detect any possible cases of cross infection and, secondly, to screen pneumococci for antibiotic resistance. Further measures such as serotyping, screening contacts, and isolation procedures should be instituted promptly as necessary. Antibiotic prophylaxis should be confined to serious epidemics. One hopeful prospect is that new developments in the pneumococcal vaccine may make it more immunogenic in children under 2—so more useful in curtailing potential epidemics.

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Wilson's disease

In 1911-2 S A K Wilson, a student of Babinski and of Pierre Marie, described a familial nervous disease associated with cirrhosis. It is found world wide, from Arabia to Japan, but is rare. Estimates of the incidence of Wilson's disease have, however, crept up steadily from five per million to 30 per million in the past decade—that is, from 250 to 1500 probable cases in Britain.[1-3] Of these, perhaps 1000 cases are unrecognised.[4] Scheinberg has estimated that the correct diagnosis is made in only a quarter of all patients with the disease.[5]

Untreated, Wilson's disease is always progressive and fatal. Treated, most patients have a normal life of normal length (unless terminally ill with liver or neurological damage when the diagnosis is made). This contrast is so dramatic, and the clinical picture so variable, that anyone between the ages of 5 and 50 with unexplained liver disease, enlarged liver and spleen, hypersplenism, or attacks of jaundice (the most frequent initial manifestations) should be considered to have Wilson's disease until otherwise proved. Similar suspicion should be aroused by signs of brain damage, tremor, clumsiness, ataxia, rigidity, failure at school, epilepsy, speech disorder, or dementia. Perhaps a fifth of patients present with a purely psychiatric illness,[1] and a few with renal or bone disease. All these are doomed to die in coma, bleeding, mute, immobile, or demented—unless the correct diagnosis is made and treatment given.

The diagnosis of Wilson's disease is exact, though several tests may be needed. The condition is due to copper poisoning from the body's failure to synthesise caeruloplasmin, or to an intrahepatic defect in copper metabolism. Studies of fibroblasts, which share the genetic defect with hepatocytes,[6] and also of Bedlington terriers, which have a recessively transmitted condition very like Wilson's disease,[7] are helping to explain how the autosomal recessive inheritance of two