

Communicable Disease Report

Management of asplenic patients in South Buckinghamshire: an audit of local practice

J MacInnes, D J Waghorn, E Haworth

Summary

People without spleens have an increased risk of pneumococcal and other infections. Immunisation is advised for this group of patients, but the role of prophylactic antibiotics remains unresolved.

Since 1992, general practitioners in South Buckinghamshire have been encouraged to immunise all asplenic patients against infections with *Streptococcus pneumoniae*, *Neisseria meningitidis*, and *Haemophilus influenzae* type b (Hib). In addition, an 'alert' card, similar in principle to a medical warning bracelet, has been produced for general practitioners to issue to asplenic patients.

General practitioners' clinical records of 293 asplenic patients were subsequently examined to evaluate this programme and assess the use of prophylactic antibiotics. Uptakes of 91%, 80%, and 79% were achieved for vaccines against pneumococcal, meningococcal, and Hib infections, respectively. Twenty-three per cent of patients had been advised immediately after splenectomy to take prophylactic antibiotics. Prophylaxis was advised for different periods of time, particularly in children. Thirty-four different antibiotic regimens had been recommended for adults. Clinical records suggested that 9% of patients were taking antibiotic prophylaxis at the time of the analysis. 'Alert' cards had been distributed to 88% of patients who were eligible.

It is likely that most districts within the United Kingdom could set up similar immunisation and 'alert' card programmes. The wide variation in recommendations for antibiotic prophylaxis highlights the need for further research and the development of national guidelines.

Introduction

People without spleens are at increased risk of severe, sometimes life threatening infection¹. Most serious infections in such people are caused by bacteria with a polysaccharide capsule, such as *Streptococcus pneumoniae*. The spleen clears opsonised bacteria from the blood, as well as storing B lymphocytes that respond to polysaccharide antigens. The loss of the spleen impairs the antibody response to antigenic challenge². Immunisation against common encapsulated bacteria reduces the risks of serious infection³.

The role of prophylactic antibiotics remains unresolved. They may lessen the risks in children, but scientific evidence for general use is limited⁴. Immunisation against pneumococcal infection for asplenic patients was recommended in the United States in 1984⁵, when a 23 valent pneumococcal polysaccharide vaccine (Pneumovax II), as well as a 14 valent vaccine (Pneumovax I), was available. The 23 valent vaccine superseded the 14 valent vaccine in 1989, and the Department of Health advised that patients over 2 years of age without functioning spleens should be immunised with Pneumovax II⁶. Eighteen months later the chief medical officer (CMO) advised that all asplenic patients should also be immunised against *Haemophilus influenzae* type b (Hib) and *Neisseria meningitidis*⁷. Unfortunately, no vaccine is available against type B meningococcus, the commonest serotype in the United Kingdom, but type C strains, covered by the vaccine, cause up to a quarter of serious infections⁸. The CMO also recommended that daily antibiotic prophylaxis, typically penicillin V twice daily, should be given to asplenic children up to the age of 16 years⁷.

Management of asplenic patients in South Buckinghamshire: an audit of local practice

J MacInnes
D J Waghorn
E Haworth

R173

A school and community outbreak of influenza A

C Brock
M Knowles
S Goh

R177

Legionnaires' disease surveillance: England and Wales 1994

C A Joseph, E J Hutchinson
D Dedman, R J Birtles
J M Watson, C L R Bartlett

R180

'Soundex' codes of surnames provide confidentiality and accuracy in a national HIV database

J Y Mortimer
J A Salathiel

R183

COVER /Körner 95-1 (April to June 1995)

J M White, M Rush
S Leon, M E B Ramsay

R186

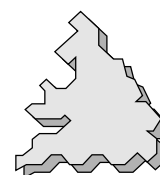


Figure 1 Splenectomy 'alert' card

Important	The following vaccines have been given:
This person	<u>DATE ADMINISTERED</u>
.....	Pneumovax (Pneumococcus):.....
has undergone removal of his/her spleen	Hib (H. influenzae type b):
on.....If taken ill and	Mengivac (Meningococcus A & C):
admitted to hospital, attending medical staff should be informed of
this.	Reason for splenectomy:
Signed
Medical Practitioner	

South Buckinghamshire Health District introduced a programme in 1992 that aimed to identify as many asplenic patients as possible, review their immunisation records, issue 'alert' cards, and optimise their management.

During the programme, it became apparent that recommendations differed for the use of antibiotics after splenectomy. Recommendations varied with the date of splenectomy and between individual doctors. An investigation was therefore carried out with four main aims: firstly, to assess what proportion of patients had been immunised; secondly, to review the recommendations for antibiotic prophylaxis; thirdly, to assess the proportion of patients taking antibiotic prophylaxis; and fourthly, to evaluate the distribution of 'alert' cards to patients. The protocol was submitted to and approved by the local medical ethics committee.

Method

In August 1992, the microbiologist for South Buckinghamshire began to compile a register of patients who had undergone splenectomy. Local general practitioners were asked to trawl their practice lists. Some practices displayed posters in their surgeries asking patients to inform the practice if they had had their spleen removed. General practitioners were asked to review the clinical records of these patients and to immunise those who had not already received pneumococcal, meningococcal, or Hib vaccines.

'Alert' cards were provided for issue to the patients, to carry the updated immunisation status and the date and reason for splenectomy (figure 1). Patients were recommended to carry the cards at all times in case of a medical emergency, to perform a similar function to a medical warning bracelet.

During the autumn of 1994, one of the authors examined the clinical records of general practitioners who gave their permission, to assess the uptake of immunisation and 'alert' cards, and to find out what advice about antibiotic prophylaxis these asplenic patients had received.

Results

From August 1992 to September 1994, 293 asplenic patients were added to the register. General practitioners had been unaware of some cases until the programme began. Since September 1994 the total number has continued to rise slowly. It was possible to calculate the age at which 255

cases underwent splenectomy and to place them into one of three categories according to the reason for splenectomy (table 1).

Immunisation audit

The immunisation records of 258 out of a possible 293 patients were available for review, and immunisation rates for the three recommended vaccines were calculated (table 2). Thirty-five records were unavailable; 27 because the patient had left the district, and five because access was denied.

A minority of cases had received pneumococcal immunisation before August 1992. After the programme for asplenic patients in South Buckinghamshire began in August 1992, immunisation uptake rose dramatically (figure 2). Five patients had received pneumococcal vaccines before 1984, when a 14 rather than a 23 valent polysaccharide antigen (Pneumovax I rather than II) was available. Before 1992, only three patients had been immunised against other encapsulated bacteria (Hib 1, meningococcal 1, Hib plus meningococcal 1).

Review of recommendations for antibiotic prophylaxis

Details of recommended antibiotic prophylaxis were available for 260 patients (table 3). None of the three children under 2 years of age had been recommended to take antibiotics after splenectomy. One patient, who had undergone splenectomy when aged between 2 and 15 years, had been advised to start taking antibiotics some 26

Table 1 Age at and reason for splenectomy

Reason	Age at splenectomy (years)					Total
	<2	2-15	16-34	35-64	≥65	
Haematological*	2	20	46	41	5	114
Traumatic†	—	23	43	14	1	81
Other‡	1	3	16	29	11	60
Total	3	46	105	84	17	255

* For example, lymphoma, myelofibrosis, idiopathic thrombocytopenia, hereditary red cell disorders.

† For example, road traffic accidents, sports injuries, assault.

‡ For example, non-haematological diseases affecting spleen, involved with tumour at time of surgery, accidentally damaged at time of surgery.

Table 2 Immunisation rates for the three recommended vaccines in 258 asplenic patients

Vaccine	Number of patients (%)
Pneumococcal	236 (91)
Hib	204 (79)
Meningococcal	206 (80)

years later. Among patients aged 16 years and over, one was recommended to take antibiotics after developing pneumococcal meningitis two years after splenectomy, and two others were advised to take prophylaxis 17 years after splenectomy.

There appeared to be little consistency in the time for which prophylaxis was suggested. For example, in nine patients aged 2 to 15 years the following durations had been advised and documented: 6 weeks, 8 weeks, 4 months, 1 year, until aged 8, until aged 14, until aged 18, until leaving school, until late adolescence. The remaining six patients had received no specific advice.

In patients of all ages, recommendations to take antibiotic prophylaxis after splenectomy were becoming more common. For those who had undergone splenectomy before 1977, there were no records of prophylaxis being advised. In the last ten years, an increasing proportion of patients had been recommended to take antibiotics (table 4).

In patients aged 16 years and over at splenectomy and advised to take prophylaxis, four different agents (penicillin, amoxycillin, erythromycin, co-trimoxazole) and combinations of these agents in different dosages had led to a total of 34 different regimens being recommended.

Patients taking antibiotic prophylaxis

Data on the prescription of antibiotic prophylaxis was available for 255 patients. At the time of the audit, nine patients were still aged 2 to 15 years and three of these were taking antibiotic prophylaxis. Two hundred and forty-six cases were aged 16 years and over, and only 21 (9%) were taking prophylaxis. It is possible that additional patients who were taking antibiotics may have been concealed by incomplete clinical records.

Twenty-eight patients had undergone splenectomy between August 1992 and September 1994 and were therefore within the period that carries the greatest risk of serious infection. One patient had died. Thirteen of the remaining 27 patients were taking antibiotic prophylaxis, although the records showed that 20 had been advised to do so. The agents recommended were penicillin (10), amoxycillin (6), penicillin then amoxycillin (3), and 'the usual' (1).

'Alert' card audit

By the beginning of the audit, 'alert' cards had been issued to general practitioners to complete for 219 patients. A number of patients, detected mainly in the weeks leading up to the audit, could not be evaluated as their general practitioners had not received the cards. Distribution to 34 of the 219 could not be assessed because they had left the

district. One hundred and sixty-three of the 185 cases reviewed (88%) had received cards. Four of the 22 patients without a card had not yet completed their immunisations. One person was suffering from dementia and so the issue of a card was considered inappropriate. General practitioners had not distributed the cards to a further 17 cases.

Discussion

This analysis of the management of asplenic patients in one district shows that doctors and patients need continuing education. It also shows how, with motivation, a continuing programme can support case finding and the maintenance of a population based register. Patients need to understand the health risks associated with splenectomy if they are to comply with advice or treatment⁹. We are currently studying the knowledge and concerns of asplenic patients.

At the time of the audit, 293 living patients who had undergone splenectomy had been entered on the register, for a resident population of about 270 000. Using the current rate of splenectomy in England of 2.9 per 100 000 population per year¹⁰, this represents, without adjustment for mortality, 40 years' incidence data for the district. The recorded uptake of pneumococcal immunisation, 91%, compares favourably with another reported programme¹¹ but could be improved. The uptake of 80% for Hib and meningococcal immunisation reflects the facts that their administration has been recommended more recently and, perhaps, that the evidence of their value after splenectomy is not as clear as for pneumococcal vaccine.

A study of immunisation intervals for the three vaccines was beyond the scope of this review, but this area warrants further investigation. The Department of Health currently suggests that a booster dose of pneumococcal vaccine should be given every five to 10 years⁶. For Hib and meningococcal vaccines, boosters have not yet been recommended. Most of the uncertainty arises from the fact that the protective efficacy of antibody levels in asplenic patients is not known. It seems likely that the response to immunisation will vary according to the reason for splenectomy. Danish children and adolescents who had

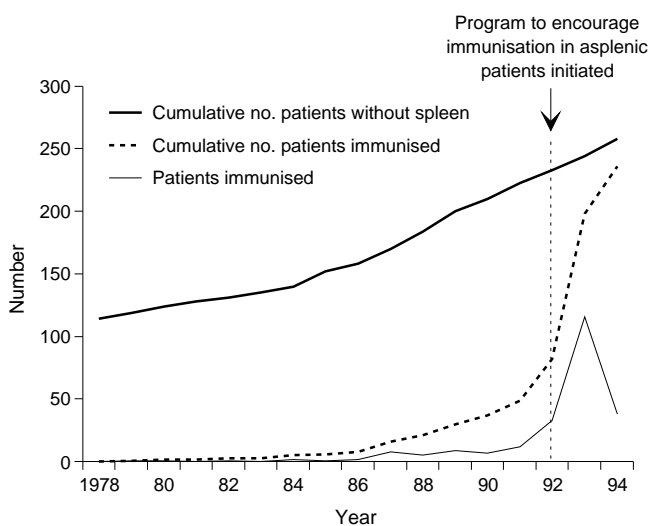
Figure 2 Number of patients immunised against pneumococcal infection

Table 3 Patients advised to take prophylactic antibiotics immediately after splenectomy

Age (years) at splenectomy	Number of patients	Number advised to take prophylaxis (%)
<2	3	– (–)
2-15	46	15 (33)
≥16	211	46 (22)
All	260	61 (23)

undergone splenectomy for various reasons, however, developed high levels of antibodies after receiving Hib vaccine¹². People who undergo splenectomy for trauma but who are otherwise well may mount a greater immunological response than those with underlying malignancy. Work is in progress to investigate antibody responses in our asplenic population.

This review illustrates the continuing confusion about antibiotic prophylaxis after splenectomy. Confusion persists because there is no clear evidence of its efficacy. Two studies have shown that penicillin protects children with sickle cell disease from serious pneumococcal infection^{13,14}. Sickle cell disease can result in functional asplenicism but extrapolation of conclusions from this group to patients who have undergone splenectomy may be inappropriate. The CMO has recommended penicillin after splenectomy for children up to 16 years of age⁷. Using this as the accepted guidance, only 31% of children in our group received the correct advice. Furthermore, although young children are recognised to be at the greatest risk of infection and make poor responses to polysaccharide vaccines, none of the three children under 2 years of age at splenectomy had been prescribed antibiotic prophylaxis.

Most cases of overwhelming post-splenectomy infection (OPSI) have been reported to occur within the first two to three years after surgery¹⁵. Several sources therefore recommend that patients who undergo splenectomy at any age should receive prophylaxis for at least this period¹⁵⁻¹⁷. We support this advice and in addition we currently suggest that prophylaxis should continue in all asplenic children until the age of 15 years. We also suggest indefinite antibiotic prophylaxis for patients with severe underlying disease – for example, malignancy. Concern about adequate absorption and the need for 12 hourly doses of penicillin V has made amoxicillin once daily a popular alternative¹⁸.

We found that antibiotic prophylaxis has been advised more commonly in recent years. This probably reflects heightened awareness of OPSI¹⁹ but the 34 different regimens we identified show that a consistent antibiotic policy has not been adopted. We are presently undertaking a national study of OPSI surveillance and this may shed further light on the use and efficacy of antibiotics, although small numbers of cases may limit interpretation²⁰.

The introduction of an 'alert' card system has been advocated by others^{15,21} and has now been endorsed by the Department of Health²². The card's purpose is not only to provide a patient held record of immunisations but also to 'alert' both the patient and any attending doctor, particularly in an emergency. A distribution rate of 88% is

Table 4 Patients recommended to take antibiotic prophylaxis according to date of splenectomy

Year of splenectomy	Number of patients (all ages)	Number recommended to take antibiotic prophylaxis (%)
1985-89	60	19 (32)
1990-92	32	20 (63)
1993-94	23	17 (74)

encouraging, but this does not necessarily mean that the patient carries the card every day. Delay in distributing the card to individuals limits its use in identifying those at increased risk of infection following splenectomy.

Every effort should be made to reduce the risk of OPSI and this study has shown what a locally managed programme can achieve. Further research is needed, however, particularly into immunisation response and antibiotic prophylaxis, so that guidelines can be agreed nationally, implemented, and assessed.

Acknowledgements

We thank Dr Dick Mayon-White for his help with this audit and its report.

References

1. McMullin M, Johnston G. Long term management of patients after splenectomy. *BMJ* 1993; **307**: 1372-3.
2. Mayon-White R. Protection for the asplenic patient. *Prescriber's Journal* 1994; **34**: 165-70.
3. Amman AJ, Addiego J, Wara DW, Lub B, Smith WB, Mentzer WC. Polyvalent pneumococcal polysaccharide immunisation of patients with sickle-cell anaemia and patients with splenectomy. *N Engl J Med* 1977; **297**: 897-900.
4. Read RC, Finch RG. Prophylaxis after splenectomy. *J Antimicrob Chemother* 1994; **33**: 4-6.
5. Immunization Practices Advisory Committee. Update: pneumococcal polysaccharide vaccine usage – United States. *MMWR* 1984; **33**: 273-81.
6. Department of Health, Welsh Office, Scottish Home and Health Department, Department of Health and Social Services Northern Ireland. *Immunisation against infectious disease*. London: HMSO, 1992: 100-3.
7. Department of Health. Asplenic patients and immunisation. *CMO's Update* 1994; **1**: 3.
8. Jones DM, Kaczmarek EB. Meningococcal infections in England and Wales: 1994. *Communicable Disease Report* 1995; **5**: R125-30.
9. Baddeley PG. Splenectomy and prevention of overwhelming infection. *Hospital Update* 1993; **19**: 365-7.
10. Department of Health. *Hospital episode statistics, England: financial year 1993-4*. Vol 1. London: Government Statistical Service, 1995.
11. Kinnersley P, Wilkinson CE, Srinivasan J. Pneumococcal vaccination after splenectomy: survey of hospital and primary care records. *BMJ* 1993; **307**: 1398-9.
12. Kristensen K. Antibody response to a Haemophilus influenzae type b polysaccharide tetanus toxoid

- conjugate vaccine in splenectomized children and adolescents. *Scand J Infect Dis* 1992; **24**: 629-32.
13. John AB, Ramlal A, Jackson H, Maude GH, Waight Sharma AW, Sergeant GR. Prevention of pneumococcal infection in children with homozygous sickle cell disease. *BMJ* 1984; **288**: 1567-70.
 14. Gaston MH, Verter JI, Woods G, Pegelow C, Kelleher J, Presbury G, et al. Prophylaxis with oral penicillin in children with sickle cell anaemia. *N Engl J Med* 1986; **314**: 1593-9.
 15. Chattopadhyay B. Splenectomy, pneumococcal vaccination and antibiotic prophylaxis. *Br J Hosp Med* 1989; **41**: 172-4.
 16. Ellison EC, Fabri PJ. Complications of splenectomy, etiology, prevention, and management. *Surg Clin North Am* 1983; **63**: 1313-30.
 17. Barnes JN, Deodhar HA, Marshall RJ. Long term management after splenectomy. *BMJ* 1994; **308**: 338.
 18. Reilly S, Prentice AG, Copplestone JA, Hamon MD, Sarangi J. Long term management after splenectomy. *BMJ* 1994; **308**: 131.
 19. Reid MM. Splenectomy, sepsis, immunisation, and guidelines. *Lancet* 1994; **344**: 970-1.
 20. CDSC. Surveillance of overwhelming infection following splenectomy. *Communicable Disease Report* 1994; **4**: 169.
 21. Evans DI. Postsplenectomy sepsis 10 years or more after operation. *J Clin Pathol* 1985; **38**: 309-11.
 22. Department of Health. Alert cards and information sheets for asplenic patients. *CMO's Update* 1995; **6**: 5.

J MacInnes MRCP
E Haworth MFPHM
 Department of Public Health
 Buckinghamshire Health Board
D J Waghorn MRCP
 Department of Microbiology
 Wycombe General Hospital

A school and community outbreak of influenza A

C Brock, M Knowles, S Goh

Summary

In May 1995 a department of public health medicine was informed of an outbreak of respiratory and gastrointestinal illness in a local school. Eighty-three pupils and staff were affected out of a total of 247 people – an attack rate of 34%. The outbreak was investigated, control measures were instigated, and the outbreak subsided. Pupils and staff were surveyed and faecal specimens were collected. Blood specimens from a sample of pupils were examined serologically. No organisms were isolated from faecal specimens. Nine of the 18 blood specimens taken showed raised antibody titres against influenza A. This labour intensive investigation revealed a community outbreak of influenza A. Investigations in schools can be useful in community surveillance.

Introduction

On 10 May 1995 a public health department was contacted by the headmistress of a primary school. The headmistress was concerned that over the previous three days, following local elections, about a quarter of the 231 pupils had been absent through sickness each day. Four members of staff were also ill.

Investigation and control

Two doctors from the public health department and an environmental health officer attended the school on 10 May 1995. Numbers of absentees had risen above the normal levels on Friday 5 May, after the school had been closed for local elections the previous day. By 10 May sickness absence was still high: 62 pupils (27%) were absent. The visitors were told that pupils had reported various

symptoms including sickness, diarrhoea, fever, cough, and headache. The illness appeared to last a few days, and some members of staff were also affected.

The immediate action taken was to give advice about hygiene and disinfection in the school. The head teacher asked parents to keep children at home until they were symptom free for 48 hours and similar advice was given to staff. Cleaning procedures were already satisfactory at the school. Cleaners were advised to use a 1/1000 hypochlorite solution and to clean toilets twice a day, with particular attention to toilet handles and door handles. Advice was also given to air the classrooms.

A questionnaire about onset dates and times, symptoms, and others ill at home was sent to parents and staff via the school, and was accompanied by an explanatory letter that reinforced the advice about remaining off school for 48 hours after recovery. Pots were left for faecal specimens for those with symptoms, particularly those with diarrhoea. A letter was also sent to general practitioners, informing them and asking them to help by advising parents about the 48 hour exclusion.

Questionnaire returns showed that upper respiratory tract symptoms were very common. Consent was sought from the parents of 25 children to take throat swabs for viral culture, and blood specimens.

The children chosen had all reported upper respiratory tract symptoms, some of which had been severe. Eighteen parents consented. A team from the local hospital and community trusts – consisting of a paediatric senior house officer, two school nurses, and a clerical officer – visited the school on 23 May. Blood specimens were taken from all 18 children and throat swabs from six children who were still complaining of upper respiratory tract symptoms.

The children were from several classes and the onset dates of their illnesses were representative of others,

Table 1 Attack rates by school class (age)

Class (age of children)	Number ill	Number in class	Attack rate (%)
Reception (5-6)	13	31	42
1 (6)	12	30	40
2 (7)	6	28	21
3 (8)	14	31	45
3/4* (8-9)	9	27	33
4 (9-10)	7	27	26
5 (10-11)	10	26	38
6 (10-11)	8	31	26

* Class 3/4 exists because of a large number of children aged 8 to 9 years.

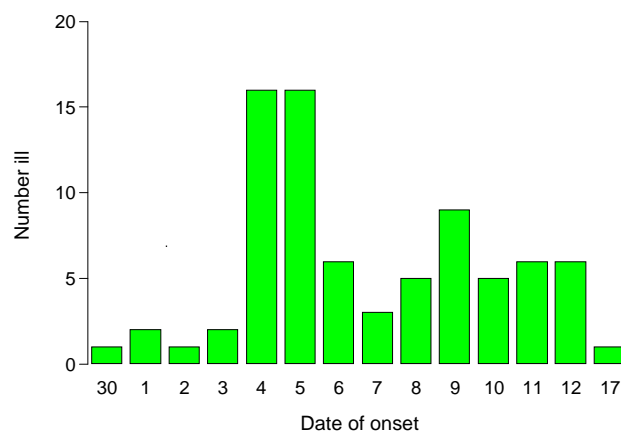
ranging from 4 to 12 May. No controls were approached as this was thought to be impractical: venepuncture is traumatic for children of this age group. Speed was essential to try and obtain positive results.

Blood was examined for titres of complement fixing antibodies against the following infections: influenza A, influenza B, respiratory syncytial virus, adenovirus, psittacosis, mycoplasma, mumps, leptospira, brucella, *Coxiella burnetti*, herpes simplex, and cytomegalovirus. The virologist at Carlisle Public Health Laboratory considered that it was too late in the outbreak to confirm the tests by viral cultures.

Results

Questionnaires were returned by the end of the week and, as stated, respiratory symptoms predominated; only a quarter of the children had diarrhoea. One hundred and forty-nine questionnaires were returned from a possible 247 pupils and staff – a response rate of 60%. According to the school registers, 85% of the children who had been off sick during the previous week returned a questionnaire. Analysis of completed questionnaires indicated an attack rate of 53% (79/149). Four of the 16 staff were ill – attack rate 25%. None of the affected staff worked in the kitchen. Attack rates in each class are shown in table 1.

The epidemic curve (figure 1) shows that the peak dates of onset were 4 and 5 May. Illness lasted from one to ten days (figure 2), but the modal period was two days. Many children consulted their general practitioner with symptoms, as shown in table 2, but none was admitted to hospital.

Figure 1 Epidemic curve for illness in the school**Table 2 Symptoms reported in pupils at the school**

Symptom	Number of pupils who reported particular symptoms (%)
Cough	66 (83)
Tiredness	61 (77)
Runny nose	57 (72)
Fever	56 (71)
Headache	54 (68)
Nausea	48 (61)
Vomiting	36 (45)
Stiff joints	21 (27)
Diarrhoea	20 (25)

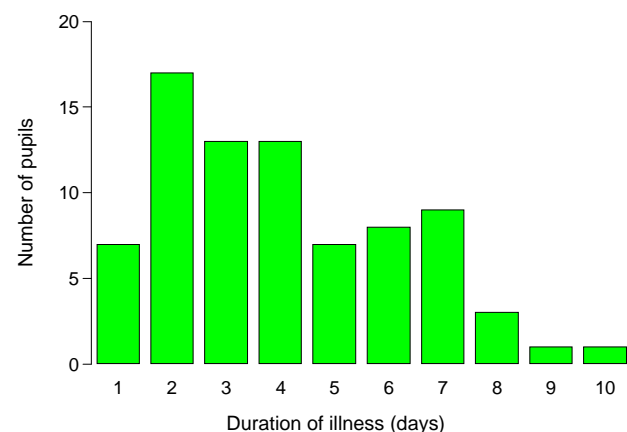
It seemed possible at this stage that two different organisms were involved, so the distribution of the respiratory and gastrointestinal symptoms were studied further. The onset dates of cough and diarrhoea are shown in figure 3. The two symptoms peaked at a similar time, although diarrhoea was not reported until the third day. The two symptoms were widely distributed among the classes. Sixteen of the 20 children with diarrhoea also had a cough. The two symptoms therefore seemed to be part of the same illness.

Forty-six children (55%) who were ill had eaten school dinners and 37 ill children (44%) took packed lunches. We asked about household contacts to try and gain an idea of the level of illness in the community. Fifty-four children (65%) had another ill person living in the same household and 26 (31%) had friends in other households who were ill.

Microbiological results

Twelve faecal specimens were collected. They were all negative for shigella, campylobacter, *Escherichia coli* O157, and cryptosporidium. No virus-like particles were visible on electron microscopy and no virus was cultured. All but two specimens were collected seven or more days after the onset of symptoms.

Throat swabs were taken from six children on 23 May and all were negative for viral culture. Nine of the 18 children who had blood tests for serology on 23 May had raised titres against influenza A: 2 children 1/1280; 2 children 1/640; 1 child 1/320; 3 children 1/160; 1 child 1/80. One of these children also had a raised titre of 1/160 to adenovirus. Eight of the remaining nine children had

Figure 2 Duration of illness

no raised titres and one had a marginally raised titre of 1/40 against influenza B.

Discussion

This was an outbreak of respiratory illness in a community in East Cumbria, which occurred over a few days from 4 to 12 May 1995. Children in one school in the district were affected. Attack rates were high – from 21% to 45% in each class. Upper respiratory tract symptoms predominated, but vomiting and diarrhoea were also reported.

The clinical and epidemiological picture suggested a viral illness. Diagnostic titres for influenza A in convalescent serum specimens from nine cases suggested that this agent was responsible for illness in the school. Convalescent specimens were used due to the unavoidable delay in producing questionnaires, distributing and collecting them, analysing the data, seeking consent for blood testing, and organising the community team to take blood. This took nine working days. The throat swabs were probably negative, because they were taken at least 11 days after the onset of illness although the children still had symptoms. Repeat convalescent blood specimens should, ideally, have been taken in some cases, but this was thought to be unacceptable in children.

The diagnosis of influenza A helped us to inform local doctors, including hospital paediatricians, about the prevalence of this virus in the community. Also, our investigation suggested that the levels of influenza A were higher than suspected at that time.

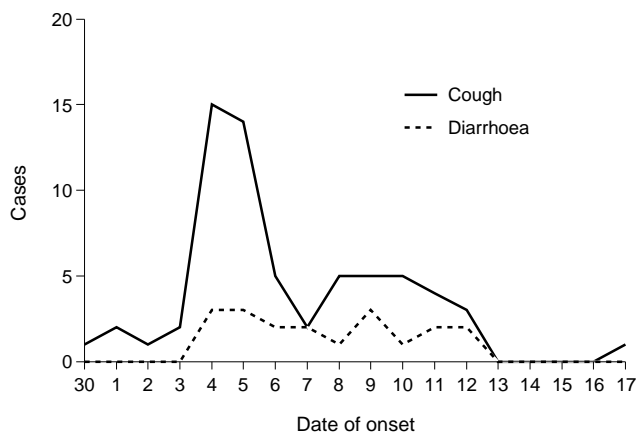
It is still possible that two or more different viral illnesses were circulating in the school. We know that one child had a borderline raised titre to influenza B and another to adenovirus. The diarrhoeal symptoms suggested the possibility of, for instance, small round structured virus. Gastrointestinal symptoms have, however, been reported in up to a quarter of children in school outbreaks of influenza A¹.

There was a high prevalence of illness in the community. Sixty-five percent of children had someone ill at home. Many of these people would have been siblings from school, but it seems likely that this was a community outbreak.

In the United States, amantidine and rimantidine are used to control outbreaks of infection with influenza A. To be effective they have to be given within 48 hours of the onset of symptoms, so this would not have been an option in this case. Also, although amantidine is licensed for use in children, there is limited evidence of its effectiveness in this context². The other option in an epidemic of influenza is vaccination. Unfortunately, antibody levels can take two weeks to rise after vaccination. This might still be considered in addition to chemoprophylaxis in an outbreak affecting groups 'at risk' as defined in the 'Green Book'³.

There are several benefits from having investigated this outbreak. Firstly, the school had an answer, and the picture of a wide community outbreak of a viral infection reassured them. Secondly, without the serology we would not have reached a diagnosis and learnt that the local prevalence of influenza A was high. One month later there was an outbreak of respiratory disease in a ward for elderly people. Our experience of this outbreak led us to suspect influenza A, which was rapidly confirmed. Thirdly, there is some evidence that outbreaks of influenza may be

Figure 3 Epidemic curve based on the onset of cough and diarrhoea



linked to meningococcal disease⁴. Seventeen cases of invasive meningococcal infection have been reported in North Cumbria so far this year compared with nine throughout 1994. Eight of the cases in 1995 arose in April and May. One case was a 2 month old baby who lived near the school where the outbreak of influenza occurred.

Success in performing this type of investigation depends on working relationships between public health services and the school, and the school and the community. The head of the school was very helpful. Parents cooperated in keeping ill children off school, and by consenting to have blood tests. The use of the community team, with a paediatric senior house officer to take blood, worked well. It enabled children to be seen in school rather than asking parents to make an appointment with a general practitioner.

The costs of performing an investigation of this type would include staff time of all concerned and laboratory costs. We were able to cover all the costs within existing contracts apart from a sessional fee for the doctor who took the blood.

Acknowledgements

The authors are grateful to Janet Blair of Carlisle Environmental Health department, Tony Tod of Carlisle Public Health Laboratory, and Ann Millican, Paul Phillips, and the team from Carlisle Hospitals and North Lakeland Healthcare, and to all the staff and pupils at the school.

References

1. Benenson A.S. *Control of communicable diseases in man*. Fifteenth edition. Washington DC: American Public Health Association, 1990.
2. Arden NH, Cox NJ, Schonberger LB. Prevention and control of influenza: part 2, antiviral agents. recommendations of the Advisory Committee on Immunization Practices. *MMWR* 1994; **43**: 1-10.
3. UK Departments of Health. *Immunisation against infectious disease*. London: HMSO, 1992: 95-8.
4. Cartwright KAV, Jones DN, Smith AJ, Stuart JM, Kaczmarek EB, Palmer SR. Influenza A and meningococcal disease. *Lancet* 1991; **338**: 554-7.

C Brock MRCGP, S Goh MFPHM
North Cumbria Health Authority
M Knowles FRCPATH
Carlisle Public Health Laboratory

Legionnaires' disease surveillance: England and Wales 1994

C A Joseph, E J Hutchinson, D Dedman, R J Birtles, JM Watson, C L R Bartlett

Summary

One hundred and sixty cases of legionnaires' disease in England and Wales were reported to the PHLS Communicable Disease Surveillance Centre in 1994, a rate of 3.1 cases per million population. Twenty-seven cases died. Eighty-nine cases (56%) were associated with travel, either in the United Kingdom (UK) or abroad, and six with a stay in hospital; the remaining cases were presumed to have acquired infection in the community. Seven outbreaks were detected in England and Wales: one was associated with a holiday centre, one with a hotel in London, two with industrial sites, and three occurred in the community. A further four clusters were associated with travel abroad: Spain, Ibiza, the Channel Islands, and a Mediterranean cruise. One hundred and twenty-eight of the 160 cases (79%) were sporadic – that is, not known to be associated with outbreaks – 43 of which (34%) were not associated with travel nor acquired in hospital.

Introduction

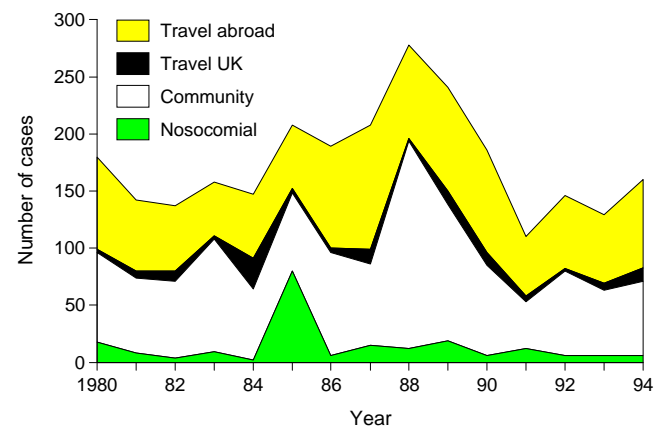
The National Surveillance Scheme for Legionnaires' Disease at the PHLS Communicable Disease Surveillance Centre (CDSC) has received reports of 160 cases of legionnaires' disease with onset of illness in 1994, the highest number since 1991 (figure). This increase is probably due to improved ascertainment, since few outbreaks involving large numbers of cases have been reported, but an increase has been observed in the number of sporadic cases associated with travel. Ten cases of Pontiac fever – the milder, non-pneumonic form of legionellosis – and 20 asymptomatic or suspected infections were also reported in 1994.

Methods

The National Surveillance Scheme for Legionnaires' Disease in residents of England and Wales was established at CDSC in 1979. Cases are reported to CDSC by PHLS or hospital microbiologists, consultants in communicable disease control (CCDCs)¹, and other health care personnel. On learning of each new case, a questionnaire is sent to the reporting doctor for epidemiological and microbiological information about the case. On receipt of this information the case is entered into the national data set, which is searched for any cases linked in time or place. If clustering of cases is detected, the relevant CCDC is informed to facilitate local follow up¹.

Cases related to travel abroad are reported to the European Surveillance Scheme for Travel Associated Legionnaires' Disease, for which CDSC is the coordinating centre. After entry into the European database the data set is searched for other cases linked to the same place of accommodation and a date of onset within the same six month period. If no other linked cases are found, CDSC informs the country of infection about the case. If a cluster of cases is recognised, CDSC alerts all the European collaborators, and asks the World Health Organisation

Figure Legionnaires' disease in residents of England and Wales, 1980 to 1994



(WHO) to inform the ministry of health in the country concerned.

Results

One hundred and twenty-one of the cases of legionnaires' disease with onset in 1994 (76%) were male, and ranged in age from 21 to 84 years; 39 cases (24%) were female with an age range of 24 to 71 years. The median age was 52 years for both males and females. Twenty-seven of the cases (17%) were reported to have died.

Diagnosis of cases

The commonest methods of diagnosis are by demonstration of the presence of antibody to legionella in serum; culture of the organism from sputum, lung tissue, pleural fluid, or blood; and detection of antigen in a clinical specimen such as urine. Specimens are tested at public health or hospital laboratories and confirmation of the diagnosis, or further characterisation of the legionella strain, is available from the PHLS Legionella Reference Unit at the Central Public Health Laboratory in London.

Isolates were obtained from 26 cases of legionnaires' disease (16%), 24 of which were identified as *L. pneumophila*, one as *L. bozemanii* serogroup 1, and one was not assigned to a species. Twenty-one of the *L. pneumophila* isolates were serogroup 1, one was serogroup 8, and the serogroups of two were not given. Sixty-eight cases were confirmed by seroconversion (fourfold or greater rise in antibody titre), 46 were presumptively diagnosed by single high antibody titre, 18 by urinary enzyme linked immunosorbent assay, and two by direct immunofluorescence.

Travel associated infection

Eighty-nine cases (56%) gave a history of travel away from home (with overnight stays) in the ten days before the onset of symptoms of legionnaires' disease and were associated with travel to over 30 different countries worldwide. Eighteen cases were linked to Spain or the Spanish islands, 12 to the UK, eight to France, eight to Greece or the Greek islands, seven to Turkey, 19 to other European countries, five to the United States, and 12 to other countries outside Europe (table). Fifteen cases associated with travel died.

Table Cases of legionnaires' disease associated with travel: by country

Country	Number of cases
Spain and Spanish Islands	18
England and Wales	12
France	8
Greece and Greek Islands	8
Cyprus	3
Guernsey	2
Italy	2
Malta	2
Other European countries	4
Cruise ships and ferries	3
Turkey	7
Tunisia	3
Jordan	1
United States	5
Far East	3
South America	3
Australia	1
Multiple countries	4

Six clusters were identified and involved 12 cases, eight of which were linked to travel abroad. In the first cluster abroad, one case was associated with each of two consecutive Mediterranean cruises on the same ship. The ship's water supply and air conditioning units were investigated. Problems revealed were related to the collection of water in the condensate trays of the air handling units where, because of negative pressure, a potential existed for the development of an aerosol that could be drawn into the air conditioning ducts. *L. pneumophila* serogroup 3 was obtained from a water sample from one of the fan rooms containing the air handling units. This was a different serogroup from the *L. pneumophila* serogroup 1 infections diagnosed in both clinical cases, although no clinical isolates were obtained for comparison.

A cluster of two cases was linked to a hotel in Ibiza. Another was linked to a hotel in Spain, where a husband and wife who shared a hotel room both became ill with legionella infection on the same day. The fourth cluster outside the UK involved two cases of legionnaires' disease in people who stayed at a hotel in the Channel Islands.

In the UK, an elderly man and woman became ill while staying at a large holiday centre in the south west of England in March, for a celebration of D-Day attended by about 700 second world war veterans. A number of respiratory illnesses were reported from this group, but no other cases of legionnaires' disease were identified. No source of infection was found, despite extensive environmental investigations at the centre. The final cluster occurred in London: a man who stayed overnight in a hotel, and another man who worked at the hotel for three weeks cleaning the hotel's ventilation ducts, both developed legionnaires' disease. *L. pneumophila* serogroup 1 was isolated from several samples taken from the domestic water system at the hotel, including the room in which the travel case stayed, but no clinical isolates were available for comparison.

European surveillance of travel associated legionnaires' disease

In July 1993 CDSC, funded by the European Community, became the coordinating centre for European surveillance of travel associated legionnaires' disease, on behalf of the European Working Group for Legionella Infections (EWGLI)². Twenty-seven collaborators in 24 countries contribute to the surveillance scheme and 153 cases of travel associated disease with onset in 1994 were reported. Sixteen clusters were recognised and involved 34 cases. Eight other cases were linked to hotels that had been associated with cases in previous years. Altogether, cases were associated with visits to 35 countries.

Nosocomial infection

Six cases of nosocomial legionellosis were reported in 1994, four of whom died. All were diagnosed by culture of the organism and all occurred in hospitals where environmental investigations showed that the hospital water supply was contaminated. Four of the cases were from hospitals where other nosocomial cases had occurred in previous years. Two cases were patients with underlying malignancies in one hospital in May and June 1994, and one patient died. Isolates from one of these cases was positive for *L. bozemanii* serogroup 1 and the other for *L. pneumophila* serogroup 1. The hospital has reported several cases of *L. bozemanii* and *L. pneumophila* infection since 1987^{3,4}.

The third nosocomial case in 1994 was a man who underwent a heart transplant, and died after contracting legionnaires' disease in the cardiothoracic unit. An isolate obtained from the patient, and isolates from numerous hot and cold water samples from the hospital unit, were identified as *L. pneumophila* serogroup 1 and were indistinguishable by monoclonal antibody sub-grouping and restriction fragment length polymorphism (RFLP) subtyping. Two earlier cases of nosocomial legionnaires' disease were associated with this hospital, one in 1981 and one in 1985, although no source of infection was found on these occasions.

The fourth nosocomial case in 1994 was in a woman who died after admission to a hospital's renal unit. The patient underwent inhalation therapy using hot and cold tap water before becoming ill with legionella infection. Environmental investigations showed that the hospital's hot and cold water supply was contaminated with *L. pneumophila* serogroup 8, which was indistinguishable from a clinical isolate obtained from the patient.

The fifth case of nosocomial legionella infection occurred in an elderly woman with a history of Wegener's granulomatosis. Three days after admission she was transferred to another hospital where she developed symptoms of legionnaires' disease. Isolates of *L. pneumophila* serogroup 1 from water samples at the first hospital matched those of an isolate from the patient, and were indistinguishable on monoclonal antibody sub-grouping and RFLP subtyping. The patient had been admitted to a ward in the first hospital, where an isolate of *L. pneumophila* serogroup 1 was obtained from the hot water supply in 1985 following an outbreak of two cases in elderly men⁴.

The sixth case in 1994 was in an elderly woman who was admitted to hospital for investigation of cerebro-

vascular disease. She died after developing infection due to *L. pneumophila* serogroup 1, which was indistinguishable by monoclonal antibody subtyping and RFLP analysis from an isolate obtained from the hospital's water supply.

Community acquired infection

Two outbreaks at industrial sites and three community outbreaks were reported in 1994. Two cases of legionnaires' disease, one suspected case, and two cases of Pontiac fever occurred in June in five men who worked at a recycling plant for industrial solvents in the south of England. Operational problems were identified with a cooling tower on the site, from which *L. pneumophila* serogroup 1 was grown, but no clinical isolates were available for comparison.

Between June and August an outbreak of eight cases, with one death, occurred in Birmingham. The cases were clustered within a two mile radius and were linked to the area by residence, travel, or employment. Altogether, 125 environmental samples were examined as part of the outbreak investigation, with 83 of these samples derived from cooling towers. Nine cooling towers on seven industrial estates yielded one or more species of legionella. Eight of these towers yielded *L. pneumophila* and three of the isolates were serogroup 1; the remaining positive results were either different legionella species or different serogroups of *L. pneumophila*. Epidemiological investigations showed that five of the cases were associated with one industrial plant, where a positive isolate from a cooling tower was indistinguishable by monoclonal antibody subgrouping and RFLP subtyping from a positive culture of *L. pneumophila* serogroup 1 from one of the patients. The other three patients in the outbreak were clustered in the north of the city and no source of infection could account for their illness as they had not been exposed to the three contaminated cooling towers. Responsibility for the maintenance of two of these cooling towers had been contracted out to the same water treatment company. At one of the industrial sites the cooling tower was poorly managed and demolition work at the site could have contributed to its contamination.

In Surrey, an outbreak of five cases occurred between May and September 1994 in people who lived or worked in two adjacent districts. All the cases were men, two of whom died. There was no clear evidence to link more than two of the cases to one possible source and no environmental source was found, despite extensive local investigations.

Two other similar small community outbreaks with inconclusive investigations also occurred in 1994. In East Anglia three cases in men, two of whom lived in the same residential district, were reported with onset of illness close together in August and September. Local investigations identified a cooling tower in the vicinity of the cases from which an isolate of *L. pneumophila* serogroup 1 was obtained, but no clinical isolates were available for comparison. In east London three cases occurred between October and December in male residents, one of whom died. There was no common occupational exposure, but two cooling tower systems geographically associated with these cases were investigated and, although no legionellas were isolated, both systems were judged by the local environmental health department to be poorly maintained.

Sporadic community acquired cases

Forty-three single cases of legionnaires' disease, which were assumed to be community acquired, could not be linked to any recognised source of infection.

Discussion

The rise in the number of reported cases of legionnaires' disease in 1994 was mainly related to an increase in the overall proportion of cases associated with travel. At 56% of the total cases, this proportion is the second highest since 1980 and almost matches the 58% of travel cases recorded in 1987, except that nearly a third of the travel cases then were part of recognised clusters or outbreaks abroad, in contrast to the 1994 data set which comprises mostly single cases. The European data set indicates that only four of the UK travel cases in 1994 were part of international outbreaks or were linked to hotels associated with cases in previous years. Better ascertainment of cases through national and international surveillance is likely to account for the rise in reports of travel associated infection.

The number of cases reported after travel to a particular country should always be considered in terms of the number of people who visit that country. Spain receives a large number of tourists and accounts for a large number of the annual cases associated with travel, but has seen a continuing decline in the annual rate of infection - to about three cases per million travellers from the UK in 1993 and 1994. In contrast, about 12 cases per million UK travellers were associated with Turkey in 1993 and 1994. This may be due to the rapid growth in tourism there and the problems of providing and maintaining hotel water and air conditioning systems.

The nosocomial cases of legionella infection highlight the difficulty in eradicating legionellas from complex water systems, despite regular maintenance and monitoring, and also the greater vulnerability of some hospital patients to this infection. In the hospital that reported two nosocomial cases in 1994, three cases of asymptomatic legionella infection were also recognised; one was due to *L. bozemanii* and two to *L. pneumophila* serogroup 1 infection. It is believed that these three culture positive 'cases', two of whom died and all of whom were immunocompromised, acquired legionella infection while in hospital, but symptoms specific to the infection could not be distinguished from their other clinical conditions.

Seven small outbreaks were detected in England in 1994 and accounted for 25 cases and four deaths. In only four of these outbreaks was a suspected source of infection found, although extensive investigations took place on each occasion. Faults in the maintenance of suspected cooling towers were identified and unregistered cooling towers were discovered during two of the outbreak investigations. The statutory notification of cooling towers has led to more effective monitoring of their maintenance records by environmental health departments or the Health and Safety Executive, but unknown unregistered towers may contribute to delay in the investigation and control of outbreaks. Rapid implementation of effective control measures once an outbreak has been recognised and investigated⁵⁻⁸ remains paramount.

The 43 community acquired single cases indicate the continuing need for research into the sources of sporadic legionellosis. A case control study began in 1994 to

determine whether sporadic cases in the community are linked to domestic water systems, and is being undertaken by the Building Research Establishment, in collaboration with CDSC and the PHLS Water and Environmental Unit⁹. Guidelines for the investigation of single cases of legionnaires' disease have recently been published¹.

Acknowledgements

We would like to thank Dunja Car for her invaluable administrative support, and all the microbiologists and public health professionals who have contributed data to the national surveillance scheme.

References

1. Saunders CJP, Joseph CA, Watson JM. Investigating a single case of legionnaires' disease: guidance for consultants in communicable disease control. *Communicable Disease Report* 1994; 4: R112-14.
2. Joseph CA, Dedman D, Birtles R, Watson JM, Bartlett CLR. Legionnaires' disease surveillance: England and Wales, 1993. *Communicable Disease Report* 1994;4: R109-11.
3. Swinburn CR, Gould FK, Corris PA, Hooper TL, Odom NJ, Freeman R, et al. Opportunist pulmonary infection with *Legionella bozemanii*. *Thorax* 1989; 44: 434-5.
4. Joseph CA, Watson JM, Harrison TG, Bartlett CLR.

- Nosocomial legionnaires' disease in England and Wales, 1980-92. *Epidemiol Infect* 1994; 112: 329-5.
5. Health and Safety Executive. *The control of legionellosis including legionnaires' disease*. London: HMSO, 1993.
6. Health and Safety Commission. *The prevention or control of legionellosis (including legionnaires' disease): approved code of practice*. London: HMSO, 1991;
7. Health and Safety Commission. *The Notification of Cooling Towers and Evaporative Condensers Regulations 1992. Statutory Instrument, 1992 No 2225, Health and Safety*. London: HMSO, 1992.
8. NHS Estates. *Health Technical Memorandum 2040: the control of legionellae in healthcare premises – a code of practice. Volume 1 Management policy: Volume 2 Design considerations: Volume 3 Validation and verification: Volume 4 Operational management: Volume 5 Good practice guide*. London: HMSO, 1993.
9. CDSC. Legionellosis and domestic water systems. *Communicable Disease Report* 1994; 4: 217.

C A Joseph MSc, E J Hutchinson MSc, D Dedman MPhil
J Watson FFPHM, C L R Bartlett FFPHM
PHLS Communicable Disease Surveillance Centre
R J Birtles PhD
Legionella Reference Unit
PHLS Central Public Health Laboratory

'Soundex' codes of surnames provide confidentiality and accuracy in a national HIV database

J Y Mortimer, J A Salathiel

Summary

Clinicians and microbiologists will participate in voluntary national reporting of HIV infections and AIDS only if they have confidence in the scheme's confidentiality. At the same time, if the data are to be accurate, it must be possible to recognise reports that refer to the same individual. The use of surname 'soundex' code in combination with date of birth meets both requirements. We describe its use in the database of reported HIV infections held at the PHLS AIDS Centre.

By the end of 1994 over 93% of the 20 407 reports on the database were soundex coded, and 70% of AIDS reports were linked to independent reports of HIV infection from microbiologists. In 1994, 22% of the reports of HIV infection were recognised as duplicating earlier reports of infection. Coding surnames using soundex is an acceptable and practical tool in surveillance of an infection for which confidentiality is a prime concern.

Introduction

The PHLS Communicable Disease Surveillance Centre (CDSC) began the surveillance of AIDS in the United Kingdom in 1982. It was realised that the system for reporting needed to allay fears that patients could be

identified by any one other than the reporter, but allow duplicate reports of the same case from different sources to be recognised. The 'soundex' system for coding surnames was developed more than 70 years ago¹. Soundex code represents any name by four characters: its initial letter followed by three digits. Simple rules transform a maximum of three sounded consonants after the initial letter to numbers from one to six. Codes for long names are limited to three digits and short ones are extended by adding zeros. Vowels are not retained apart from initial letters (box). No soundex code can be unique to a single surname, because non-initial vowels and the second of double consonants are ignored and several consonants are usually coded to a single digit. Taken in conjunction with the date of birth, however, the soundex code has been shown to have a high probability of identifying reports of the same person².

Laboratory diagnoses of HIV infections have been reported to CDSC since HIV antibody testing was introduced late in 1984. The same needs of recognising duplicate reports and ensuring patient confidentiality apply to these reports as to reports of AIDS cases. Both surveillance systems have now used soundex codes for over 10 years. The combination of date of birth and soundex code is used not only for recognising duplicates within each system but also for recognising when the same patient has been reported under both systems.

Box**How to soundex code a name⁵**

1. The first letter of the name is retained.
2. A, E, I, O, U, and Y are not assigned a code number, but influence the consonants to be coded by separating groups of consonants.
3. W and H are ignored entirely except as initial letters.
4. Other consonants are given number codes as shown:

B, P, F, V	1
C, G, J, K, Q, S, X, Z	2
D, T	3
L	4
M, N	5
R	6
5. a) if an initial consonant is followed by one or more consonants from the same letter group, without a vowel in between, the consonants that follow are ignored.
 b) any consonants that follow another from the same letter group without a vowel in between are ignored.
6. The soundex code is limited to three digits. Any further consonants are ignored. Zeros are used as fillers for shorter names.

Information about soundex coding and a free program (written by Dr J Q Nash) are available from the PHLS AIDS Centre at CDSC, 61 Colindale Avenue, London NW9 5EQ.

The coding process is quite simple, and most people who use it regularly memorise the rules quickly. As an alternative, the authors can supply a computer program suitable for most microcomputers (box).

Method

The PHLS AIDS Centre aims to code all HIV infections diagnosed in patients aged 15 years and over reported by microbiologists in England, Wales, and Northern Ireland. The centre receives relatively few reports of HIV infection in children under 15 and such reports are followed up in collaboration with the Institute of Child Health without the collection of soundex codes or surnames.

If a new report of a patient aged 15 years or over at diagnosis has no soundex code the reporting microbiologist is contacted to see if the code can be obtained. Sometimes it is simplest for the PHLS AIDS Centre, with the microbiologist's approval, to contact the clinician concerned and ask for the soundex code. Since September 1992, in addition to collecting soundex codes for as many newly reported patients as possible, we have attempted to improve the data in many earlier reports. We have

contacted the reporting centres with a backlog of 20 or more reports lacking important information, such as soundex codes, and information has been sought from the clinicians who requested the test.

Reports that match on both soundex code and date of birth are checked to see whether any of the data available from them suggest that they refer to different individuals. If doubt remains, the reporter or the clinician may be contacted to see if further information is available to resolve the issue – for instance, forename initial or details of the history of exposure to HIV infection.

We recognise that coding and transcription errors and unclear handwriting may lead to incorrect soundex codes being recorded on the database. For this reason searches for reports that may refer to the same person are conducted on partial matches also. These rely on matching four out of five elements created from the code and the date of birth: the surname initial, the numeric segment of the code, and day, month, or year of birth. Such matches have a much higher 'false positive' rate and take time to resolve, but they increase the sensitivity of the search considerably.

We present here the extent to which follow up of incomplete reports has affected the provision of soundex codes for the database of HIV infections. We also illustrate the frequency with which identical soundex codes have occurred.

Results

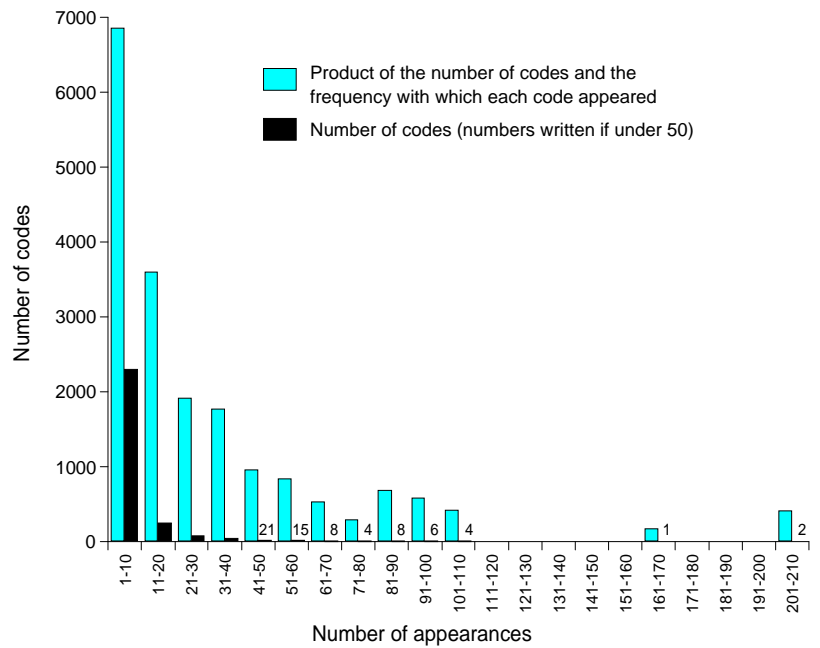
At the end of September 1992 there were 16 323 HIV diagnoses for people aged 15 or over on the AIDS Centre database. Three thousand four hundred and seventy-two of these reports (21%) had no soundex code. By the end of 1994 fewer than 7% of the reports lacked a soundex code (1382/20 407). In the same interval the proportion of reports characterised by both soundex code and date of birth rose from 71% to 89%. This increase in the proportion of well characterised reports has helped us to detect new reports that refer to an individual already on the database: 22% (635/2904) of the reports received in 1994 were recognised as referring to individuals already on the database. The high proportion of reports with both soundex code and date of birth has also allowed most of the AIDS cases reported to the centre to be associated with the laboratory report of their HIV infection. By the end of December 1994, a related laboratory report of HIV infection had been identified for 70% (6395/9078) of AIDS cases for whom a linked report was expected.

Nineteen thousand and twenty-five soundex codes were recorded on the HIV database at the end of 1994 (table; figure). There were 2750 different codes in the data set, 882 of which (32%) had appeared only once. At the other extreme, the codes that would include people whose surname was Brown, Jones, or Smith appeared 170, 203, and 209 times respectively. Thirty-six per cent of the soundexes in the data set (6852/19 025) appeared up to 10 times, and 35% (6656/19025) appeared over 30 times.

Using soundex code together with date of birth provides a marker of individuality unusual in a data set of this size. Among the 18 246 reports for which both were available, 32 pairs were found that matched on both soundex code and date of birth, but further investigation has shown that they relate to different people.

Table/Figure Surname codes observed in 19 025 soundexed reports of HIV infection

Number of appearances	Number of different codes	Number of appearances x number of different codes
1 to 10	2300	6852
11 to 20	253	3600
21 to 30	78	1917
31 to 40	50	1769
41 to 50	21	958
51 to 60	15	838
61 to 70	8	526
71 to 80	4	296
81 to 90	8	685
91 to 100	6	579
101 to 110	—	—
111 to 120	—	—
121 to 130	—	—
131 to 140	—	—
141 to 150	—	—
151 to 160	1	170
161 to 170	—	—
171 to 180	—	—
181 to 190	—	—
191 to 200	—	—
201 to 210	2	412



attempting to match reports of the same individual from different sources. In an experiment to investigate the performance of two coding schemes in linking records, it was found that the difficulty of entering the same surname consistently on separate occasions made the two coding schemes more efficient alternatives². An inaccurately recorded name, such as Davis for Davies, generally maps onto a code identical with that of the correct version.

The soundex system of coding has drawbacks. The part of a person's full name recorded as the surname may vary with place and time: what is chosen as a surname may be arbitrary in some cultures, the reporter may not know which is the forename and which is the surname if the names are unfamiliar, and double barrelled surnames may not always be recognised as such. The phonetic basis of the system means that although no code relates to a single surname the number of names that conform to a single code is very variable. The soundex system could provide 6734 different codes, but 3983 (59%) were not represented in the data set of over 19000 codes discussed in this paper. Of those that were used about a third were only recorded once, but another third were used at least 30 times. The retention of the initial letter makes it possible to speculate on surnames that could be associated with a particular code.

More efficient coding systems can be designed to meet particular needs. One adaptation has been developed with special reference to the names current in the ethnically distinctive population in Alberta, Canada⁴. Such systems may, however, be best suited to the computer coding of names already in a data set; they may be too complex for individuals to use to code names before reporting. It is impossible, in any case, to change the coding system once a surveillance system is established.

Soundex coding is simple, but people who do not code names regularly may not always be accurate – for example,

Discussion

The information required about the subjects of surveillance systems varies with the purpose and constraints of the system. The information collected for unlinked anonymous surveillance is designed to prevent the identification of any single individual either directly or indirectly. If surveillance includes a need to trace back through laboratories to clinicians and patients with, for example, a rare or important infection, the laboratory number is the most appropriate datum to store in the patient record. In contrast, genitourinary medicine clinics use locally unique patient numbers to conceal identity, but allow the follow up of individuals over time. The surveillance of HIV infection and AIDS requires the linkage of records from different sources over long periods. The system needs to be able to identify reports about the same patient while protecting that patient's confidentiality. Date of birth is valuable, but its use alone would be inadequate for all but small databases. In a group of as few as 23 people the chance that at least two share a birthday is more than 50%³. Thus, if a surveillance system contains more than 23 reports of people born in the same year there is more than a 50% chance that at least two of those people will share the same full date of birth.

The use of a code based on surnames in place of surnames themselves has a fundamental benefit when

the rule that consecutive consonants represented by the same number should not be coded unless separated by a vowel is often overlooked. Repeats may not be detected if complete matches in code are relied upon to highlight duplicate reports. It must also be recognised that some people choose to be tested under an assumed name, and that surnames – for women in particular – may change. Despite its drawbacks, however, the use of surname soundex codes has allowed the development of a database of HIV infections from which many duplicate reports have been excluded. It also allows the completeness of reporting to be checked through comparison with data sets held elsewhere, and enables information from other sources – such as databases of CD4 counts – to be linked without compromising confidentiality.

The PHLS AIDS Centre at CDSC is planning to establish, in collaboration with the Scottish Centre for Infection and Environmental Health and the Institute of Child Health, a "person based" data set for the United Kingdom. The data set will link matching records of HIV infection, the development of AIDS, and of death with or without AIDS. The work of establishing the soundex code for as many of the HIV infection reports as possible, and of searching for the related AIDS and death reports is labour intensive, but it is vital to the establishment of a unified system.

Acknowledgements

We are indebted to the reporters and others who contribute to the surveillance of HIV infection and AIDS in England, Wales and Northern Ireland, and in particular to those who have sought out and coded surnames for reporting to the PHLS AIDS Centre. We are particularly grateful to Dr JQ Nash of Ashford PHL for making his soundexing program freely available.

References

1. Knuth DE: Searching. In: Varga RS, Harrison MA, editors. *The art of computer programming Volume 3: sorting and searching*. Addison-Wesley, 1973: 391-2.
2. Greenfield RH. An experiment to measure the performance of phonetic key compression retrieval schemes. *Methods Inf Med* 1977; **16**: 230-3.
3. Feller WF. *An introduction to probability theory and its applications*. 3rd edition. Wiley, 1967; **1**: 33
4. Fenna D: Phonetic reduction of names. *Computer Programs in Biomedicine* 1984; **19**: 31-6.
5. HB Newcombe *Handbook of record linkage*. Oxford: Oxford Medical Publications, 1988: 183-4.

J Y Mortimer MA, J A Salathiel BSc
PHLS AIDS Centre
Communicable Disease Surveillance Centre

COVER/Körner 95-1 (April to June 1995)

vaccination coverage statistics for children up to 2 years old in the United Kingdom

The COVER (cover of vaccination evaluated rapidly) programme, a scheme for the rapid evaluation of vaccine coverage, started in January 1987. Until May 1995, coverage data for sentinel antigens were collected quarterly for consecutive cohorts of children who had recently reached the target age of vaccination. The programme included districts in England and Wales and health boards in Northern Ireland. The COVER scheme has now been modified to be compatible with Körner coverage data previously collected by the Departments of Health in each country. This report on the first quarter of the merged COVER/Körner programme provides coverage data on all antigens for children who had completed primary vaccination by either their first or second birthday in districts and health boards in the United Kingdom (UK).

Methods

Modified versions of computer programs used for the annual Körner returns (KC51) were used to collect data from computerised child health information systems in each locality. Regional and district boundary definitions used were those as at 1 April 1995. Data were submitted during August and September 1995 for children resident in particular districts/health boards on 30 June 1995 and who reached their first or second birthday during the

evaluation quarter (1 April to 30 June 1995). The number of children completing a primary course for each antigen (three doses of diphtheria (D3), tetanus (T3), pertussis (P3), polio (Pol 3), and *Haemophilus influenzae* type b (Hib 3) vaccines; and one dose of measles, mumps, and rubella (MMR) vaccines) at any time up to their first or second birthday was requested.

Results

One hundred and twenty-two districts and health boards participated from eight English regions, Wales, and Scotland. Data were not available from the four health boards in Northern Ireland and from five English districts. In 11 districts, data were only provided for part of the district. The mean coverage (table) at 12 months was 93.1% for D3, T3, and Pol 3 (district/health board range 82.4% - 98.0%), 91.2% for P3 (78.0% - 96.7%) and 92.6% for Hib 3 (76.9% - 97.8%). Mean coverage at 24 months was 95.8% for D3, T3, and Pol 3 (84.7% - 100.0%), 93.5% for P3 (83.8% - 98.3%), 94.9% for Hib 3 (82.4% - 99.3%), and 92.5% for MMR (district range 77.6% - 98.2%).

Comment

Using the district and health board configurations as at 1 April 1995, we report COVER/Körner data from 122 of the

131 districts and health boards (92%) in the UK. Among the districts and health boards that participated in the last quarter of the COVER programme, fewer submitted data to the new programme for this quarter. The commonest reason for non-participation was lack of the appropriate computer software needed to generate the COVER/Körner data. This problem has now been or will shortly be resolved and data for these districts and health boards will be submitted retrospectively.

As expected, when compared with the last COVER evaluation¹, a fall of about 1% was observed in national coverage for all antigens at 12 months of age using the new programme. This is because the analysis of COVER/Körner data uses the vaccination status of children in the cohort on their first birthday, whereas COVER data were analysed using the vaccination status of children in the cohort up to three months after their first birthday. National coverage at 24 months of age, however, was the highest ever recorded² and the target of 95% coverage set out in *Health of the Nation*³ has been achieved for all antigens except third dose pertussis and MMR. Thirty-four districts in England and Wales achieved the 95% target for D3 (33%), 12 for P3 (12%), and 29 for Hib3 (28%) at 12 months of age. At 24 months of age, 78 districts achieved 95% coverage for D3 (76%), 31 for P3 (30%), and 69 for Hib3 (67%). Although coverage of MMR vaccine at 24 months was similar to that observed in the COVER scheme¹, only 28 (27%) of districts in England and Wales had achieved 95% coverage for MMR at 24 months, compared with 51% of districts that achieved this target in the last COVER evaluation¹. One reason for the fall in the proportion of districts reaching the target may be the change in the district boundaries employed, and the reduction in the total number of districts. The merger of districts tends to shift each district's coverage towards the mean and

therefore a smaller proportion of the larger districts achieved the 95% target.

The introduction of the modified COVER/Körner scheme is an appropriate time to readdress the original objectives of the COVER programme and to formulate future strategy. To facilitate this a user survey of vaccination coverage data has recently been undertaken by the PHLS Communicable Disease Surveillance Centre. Questionnaires were sent to district immunisation coordinators, consultants in communicable disease control, district and regional directors of public health, and medical advisors to family health service authorities, who were encouraged to copy the questionnaire to other colleagues to elicit the opinions of both purchasers and providers of immunisation services. A report on the findings of the survey should be available early in 1996.

References

1. White JM, Leon S, Ramsay MEB. COVER (Cover of vaccination evaluated rapidly):34. *Communicable Disease Report* 1995; 5: R105-6.
2. Government Statistical Service. *Vaccination and Immunisation 1993-94: summary information from forms KC50 and KC51, England*. London: Department of Health, 1995.
3. Department of Health. *The health of the nation*. London: HMSO, 1991.

J M White BSc,

M Rush,

S Leon,

M E B Ramsay MFPHM

Immunisation Division

PHLS Communicable Disease Surveillance Centre.

Table Completed primary vaccinations (all antigens) by 12 months and 24 months: April to June 1995

Region/country	Number of participating districts (total)	% coverage at 12 months			% coverage at 24 months			
		DTPol3	P:3	Hib3	DTPol3	P3	Hib3	MMR
England:								
Northern and Yorkshire ¹	14 (14)	93.1	91.0	92.8	95.8	93.5	95.7	93.4
Trent	8 (10)	92.2	91.1	91.9	95.7	93.9	94.7	92.4
Anglia and Oxford ¹	10 (10)	94.7	93.1	94.2	95.5	93.5	94.6	92.9
North Thames ¹	13 (15)	91.4	89.6	90.3	94.6	92.3	93.2	89.9
South Thames ¹	12 (12)	91.4	90.0	90.6	93.8	92.1	92.5	89.7
South and West ¹	12 (12)	95.4	93.8	95.1	97.4	95.3	96.5	95.1
West Midlands	14 (15)	92.6	90.3	92.2	96.1	93.8	94.5	92.8
North West ¹	16 (16)	91.4	89.2	90.9	95.4	92.8	94.3	91.7
Wales	8 (8)	94.2	90.5	93.5	96.9	92.2	96.0	93.0
Northern Ireland	– (4)	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Scotland	15 (15)	94.5	93.3	94.1	97.2	95.3	96.7	94.3
United Kingdom (District range)	122 (131)	93.1 (82.4-98.0)	91.2 (78.0-96.7)	92.6 (76.9-97.8)	95.8 (84.7-100)	93.5 (83.8-98.3)	94.9 (82.4-99.3)	92.5 (77.6-98.2)

1. One or more districts provided data for only part of district.

N/A not available.

The views expressed in the *Communicable Disease Report* are those of individual contributors and not, necessarily, those of the PHLS.

© PHLS 1995.