The Stafford outbreak of Legionnaires' disease

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SUMMARY

A large outbreak of Legionnaires' disease was associated with Stafford District General Hospital. A total of 68 confirmed cases was treated in hospital and 22 of these patients died. A further 35 patients, 14 of whom were treated at home, were suspected cases of Legionnaires' disease. All these patients had visited the hospital during April 1985. Epidemiological investigations demonstrated that there had been a high risk of acquiring the disease in the out patient department (OPD), but no risk in other parts of the hospital. The epidemic strain of *Legionella pneumophila*, serogroup 1, subgroup Pontiac 1a was isolated from the cooling water system of one of the air conditioning plants. This plant served several departments of the hospital including the OPD. The water in the cooling tower and a chiller unit which cooled the air entering the OPD were contaminated with legionellae. Bacteriological and engineering investigations showed how the chiller unit could have been contaminated and how an aerosol containing legionellae could have been generated in the U-trap below the chiller unit. These results, together with the epidemiological evidence, suggest that the chiller unit was most likely to have been the major source of the outbreak.

Nearly one third of hospital staff had legionella antibodies. These staff were likely to have worked in areas of the hospital ventilated by the contaminated air conditioning plant, but not necessarily the OPD. There was evidence that a small proportion of these staff had a mild legionellosis and that these 'influenza-like' illnesses had been spread over a 5-month period. A possible explanation of this finding is that small amounts of aerosol from cooling tower sources could have entered the air-intake and been distributed throughout the areas of the hospital served by this ventilation system. Legionellae, subsequently found to be of the

epidemic strain, had been found in the cooling tower pond in November 1984 and thus it is possible that staff were exposed to low doses of contaminated aerosol over several months.

Control measures are described, but it was later apparent that the outbreak had ended before these interventions were introduced. The investigations revealed faults in the design of the ventilation system.

INTRODUCTION

In April 1985 an outbreak of severe pneumonia occurred in Mid-Staffordshire. The public concern was heightened when the diagnosis was established as Legionnaires' disease and when early epidemiological investigations identified the new District General Hospital (DGH) in Stafford as the probable source. The outbreak was of a scale not previously encountered in the United Kingdom and the incident prompted the first Public Inquiry into an outbreak of Legionnaires' disease in this country [1]. This paper describes the history of the incident, the methods used in the investigation, and the principal epidemiological and environmental findings.

Background and early history of the outbreak

The outbreak became apparent during the weekend of 20 April 1985, when 12 patients (two of whom later died) were admitted to the DGH with pneumonia. The initial diagnosis was influenza, although legionnaires' disease was also considered. The illness was severe, with a high incidence of complications, including respiratory and renal failure. Reports from general practitioners and records of sickness absence in staff employed at the hospital and elsewhere suggested a widespread epidemic of respiratory illness in the district. On 30 April, with no sign of the outbreak abating and no conclusive microbiological diagnosis, the Public Health Laboratory Service Communicable Disease Surveillance Centre (CDSC) was invited to assist with the investigation. At this stage an estimated 136 patients had been admitted to the DGH with respiratory infections, few with any confirmed diagnosis. By 3 May two post-mortem lung specimens examined at Birmingham Public Health Laboratory (PHL) yielded organisms which were provisionally identified as *Legionella pneumophila*, serogroup 1. In addition, antibodies to *L. pneumophila* were reported in seven DGH patients by Manchester PHL.

A collaborative study began which involved the CDSC team, PHLS microbiologists, local clinicians, community physicians and engineers in an intensive epidemiological study of this outbreak.

The predominantly rural Mid-Staffordshire Health District had a population of 306000, centred mainly in three towns. Acute hospital services were based in the town of Stafford at the DGH, the Staffordshire General Infirmary (SGI) and the Kingsmead Hospital (KH). The DGH had 334 beds and was opened in June 1983. It had a combination of natural ventilation and air-conditioning for central areas supplied by four air conditioning plants. Each plant had a separate evaporative cooling tower situated on top of the building (Fig. 1).
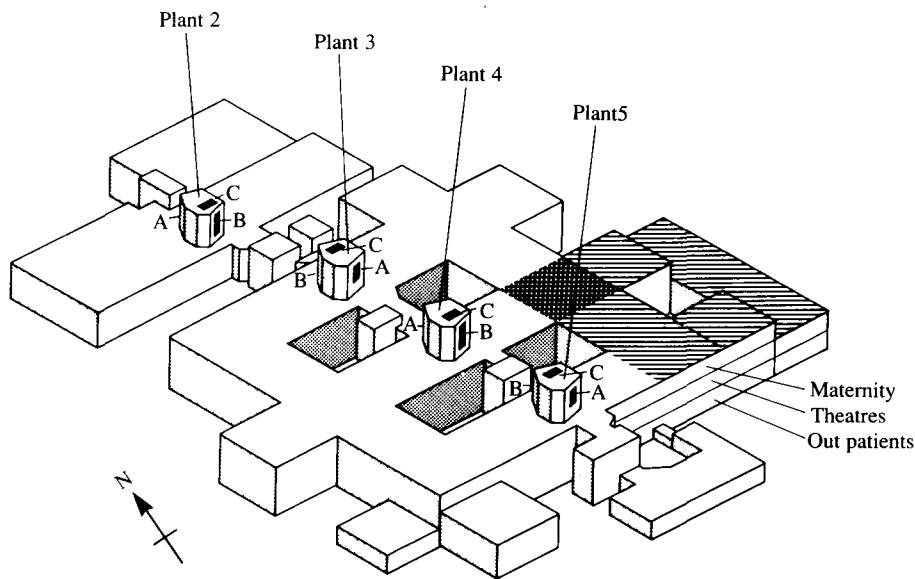
Fig. 1. Diagram of Stafford District General Hospital : The hatched area represents, at ground floor level, the outpatient department and the cross-hatched section is the main waiting area. The roof-top concrete towers of the four air-conditioning systems (known locally as plants 2–5) are labelled; these towers have ventilation exhausts A, ventilation intakes B and cooling tower exhausts C.

Within 24 h of the initial legionella reports, the number of patients with clinical and serological evidence of legionella infection had increased to 29. Interviews with 23 of these patients or their relatives pointed to the DGH as the most likely source of infection, in particular the outpatient department (OPD). After appropriate water and environmental samples were taken, control measures were introduced, taking into account all potential sources in the DGH. Measures comprised chlorinating the cooling tower water to 50 p.p.m. followed by continuous chlorination to 5 p.p.m. free residual chlorine, raising of the hot water temperature in the calorifiers to 60–63 °C to provide temperatures of 55–63 °C at the tap, and continuous chlorination of the cold water systems to 1–2 p.p.m. free residual chlorine. In addition, the air conditioning to the OPD and floors above was turned off and all spray attachments to wash basin taps were removed.

METHODS

Epidemiological investigations

An incident room was set up at the DGH and provided a base for the next 5 months for the following stages of the investigation: case-finding and case definition, a case control study of cases of Legionnaires' disease, a study of staff at the DGH and two other Stafford hospitals, a study of DGH outpatient attenders, and a serological survey of obstetric patients who had attended DGH antenatal clinics. These studies and their objectives are described below.

Case definition and case finding

A case definition was formulated so that the extent of the outbreak could reliably be described. Criteria included clinical findings and results of microbiological investigations. Cases analysed in this report are defined as patients who had either pneumonia or acute lower respiratory tract infection and where there was laboratory evidence of one of the following: (i) culture of *L. pneumophila* from post-mortem lung or (ii) a fourfold rise in titre of legionella antibodies to 64 or greater using the immunofluorescent antibody test [2] (IFAT) or (iii) a single, reproducible IFAT titre of 128 or (iv) an IFAT titre of 64 on two occasions or (v) the staining of legionella organisms by direct immunofluorescence microscopy on post-mortem lung.

A search was made for evidence of legionella infection in the 175 patients who were admitted to one of the three Stafford hospitals with acute lower respiratory tract infection during the outbreak period, which was defined as 18 March to 20 May, or who were admitted for other reasons and developed a chest infection during their stay. General practitioners in the health district and physicians in neighbouring health districts were asked to report patients with suspected Legionnaires' disease.

Case control study

The aim of this study was to investigate objectively the source of infection, and to confirm or refute the impression that the DGH was involved. This impression had been formed after interviews relating to the first 23 patients.

The study included 53 definite in-patient cases of Legionnaires' disease. At the time of the study some diagnoses were presumptive, but all 53 cases were found to fulfil the case definition when the microbiological results were complete. Four possible controls were selected for each case, using the local Family Practitioner Committee alphabetical lists of patients by practice. Each control was selected by taking the next person to the case on the list, matching for sex, age within 10 years and neighbourhood within two streets. In rural areas neighbourhood matching was within 1 mile. Interviewers were provided with the four names and addresses and instructed to obtain the control interviews from the first two names, the alternatives being acceptable only if there had been a change of address or prolonged absence from the district. The interviewers were community physicians and one research nurse.

Each interview was made using a standard questionnaire, previously piloted, which included questions on admissions or visits to the DGH, SGI or KH in March or April, as well as exposure to other possible sources of legionellae such as air-conditioned shopping centres, cooling towers and environmental water. Blood samples were requested from all controls for legionella serology which was done at Birmingham PHL.

Study of OPD attenders

In the case control study the interviews of patients with Legionnaires' disease and their matched controls had implicated the OPD as the source of infection.

Further study was needed to look for specific associations with time of day, date of visit, exposure to the water distribution system by use of toilets, taps or showers and exposure to the air-conditioning system. The aim of the OPD attenders study was to investigate these factors, to establish the period during which there had been risk of infection and to determine whether some OPD attenders developed milder forms of legionellosis such as Pontiac fever.

Relatively small numbers of patients were exposed at any one time, on any one day, to any one part of the OPD and therefore a large study was required. All patient appointments for clinics during the month of April and a 10% sample of patient attendances in March and May were included, totalling 10834 appointments. A questionnaire with a stamped, addressed envelope was sent to these patients requesting information on movements and activities in different areas of the OPD, including use of hand washing and toilet facilities. They were asked if they or accompanying friends or relatives had developed an 'influenza-like' illness during the 2 weeks after the visit. The symptoms listed were fever, shivering, sore throat, aching limbs, headache, blurred vision, and shortness of breath.

Serological sampling of OPD attenders in this study was not possible, except for 204 obstetric patients who had attended antenatal clinics during the time of the outbreak. Their serum was tested for the presence of legionella antibodies. The results were compared with any illness of the patient or harmful effects to their babies. As this was the only outpatient group for whom blood samples could be tested for legionella antibodies, a secondary objective was to seek evidence of seroconversion in outpatients in this group.

Study of hospital staff

The objectives of this epidemiological and serological study of hospital employees were: (*a*) to determine whether there was any association between development of a positive antibody response to *L. pneumophila* and exposure to the DGH as compared with the other two hospitals: (*b*) to investigate exposure to different areas within the DGH and to study positive antibody response and development of respiratory illness. There had been anecdotal reports of increased respiratory illness both at the time of the outbreak and in the preceding months. Questionnaires were sent to 1581 staff based at the DGH and to 747 staff at the other two hospitals, the SGI and KH, which shared some staff with the DGH. Questions included place or places of work, occupation, exposure to different areas of the DGH and illnesses from 1 December 1984 to 31 May 1985. Besides being asked to give their 'main work location' staff were also asked how many days in an average week they spent in each area of the DGH. They were asked to report if they had had 'influenza-like' illnesses and whether they remembered having any of the 15 listed symptoms: fever, sore throat, unusual tiredness, severe headache, dry cough, productive cough, shortness of breath, blurred vision, dizziness, muscle aches and pains, chest pain, abdominal pain, nausea, vomiting or diarrhoea. Staff were asked about any predisposing or chronic illness.

Staff were requested to give a blood sample. The serological survey was carried out by the Occupational Health Department and Birmingham PHL. Legionella antibody results were collated with the questionnaires.

Statistical methods

The questionnaires were coded and checked prior to computer data entry at CDSC. Data were entered and then verified by a second data processor using 'Quip and Quote' software developed at St Thomas' Hospital computing department. Cross-tabulations were prepared and various statistical tests used [3]. These included comparisons made between proportions made using χ^2 or Fisher's exact test and in the case control study comparison was made between the two groups using a χ^2 test for multiple matched controls. The χ^2 test for linear trend was used to examine proportions of staff seropositive according to lengths of exposure in a particular area of the DGH. Where there were sufficient data the independent effects of multiple variables were studied using Cochran's test or the statistical software package GLIM (Generalized Linear Modelling).

Microbiological investigations

Two routes of infection were considered, the domestic hot and cold water services and the air conditioning systems. Approximately 500 water samples were collected, before and after the implementation of control measures, from water storage systems, calorifiers, taps and from various sites in the ventilation water system such as cooling tower ponds, drains and chiller units. These samples were concentrated by filtration or centrifugation and cultured on selective buffered charcoal yeast extract media (BCYE) [4]. Some of the pre-chlorination samples were also inoculated into guinea-pigs (being an alternative, sensitive isolation technique), which were subsequently examined for the presence of legionella organisms.

Soil samples were taken from the DGH grounds and air samples from the OPD. Cork-incorporated insulating material (mastic) was removed from the chiller units within air-conditioning plant 4, which served the OPD, and was examined for the presence of legionellae.

Isolates were identified and any *L. pneumophila*, serogroup 1, were typed with monoclonal antisera [5] at the PHLS Centre for Applied Microbiology Research (CAMR), Porton Down. Serum samples from all suspected inpatient cases were tested at both Manchester and Preston PHLs for legionella antibodies against antigens prepared from *L. pneumophila*, serogroup 1, using the IFAT polyvalent immunoglobulin conjugate [2]. Serum samples from staff and community controls were similarly tested at Birmingham PHL. Post-mortem lung specimens were examined at Birmingham PHL. Smears were prepared and examined by direct immunofluorescence, both untreated and heat treated (50 °C for 30 min) preparations were cultured onto BYCE media.

Environmental and engineering investigations

A thorough survey of the engineering system was undertaken by engineers at the DGH, an engineer from another health authority and by scientists from CAMR. Additional investigations were done on behalf of the public enquiry. On-site and laboratory experiments were also done to investigate the possibility of aerosol formation in the OPD chiller unit of the air conditioning system. On-site

experiments included replacing the chiller unit water seal (U-trap) with a glass U-trap [1]. Later, laboratory experiments were done using the original U-trap from the OPD chiller unit in which the U-trap was filled with a suspension of *Bacillus globigii* (*Bacillus subtilis* var. *niger*) spores. Air at between 25 and 100 mm water pressure was blown through the trap to produce bubbling and an aerosol.

RESULTS

Epidemiological results

The outbreak

Case-finding revealed 175 hospital inpatients with chest infections in the three Stafford hospitals between 18 March and 20 May. Of these, 59 met the case definition for Legionnaires' disease, including 3 patients who had developed the illness whilst in hospital. A further 9 cases were identified in hospitals outside Stafford by reports from attending physicians and from information obtained from the OPD attender study. Thus 68 cases, of whom 22 died, were identified amongst hospital inpatients. Records of the remaining inpatients in Stafford with chest infections revealed a further 21 patients, 6 of whom died, who had clinical evidence consistent with Legionnaires' disease but for whom there was insufficient microbiological evidence to satisfy the case definition. In addition, 14 patients who had not been admitted to hospital were identified from general practitioners' records. These patients had had lower respiratory tract infections and IFAT titres of $\geqslant 64$. Thus the total of microbiologically confirmed and clinically suspected cases in the outbreak was 103 and the total deaths were 28. In this report data on the 68 confirmed inpatient cases and their 22 associated deaths are presented.

All 68 patients had visited or had been treated in the DGH between 1 and 19 April and 64 between 9 and 19 April (Fig. 2). Two of the 68 patients had been admitted some time before the outbreak and the exact time of their exposure was unclear. The onset of illness in these 68 confirmed cases was from the 7 to 27 April (Fig. 2). No clear date of onset was recorded for 6 patients, 4 of whom died. Every case of confirmed Legionnaires' disease had been to the DGH within 12 days of the onset of their symptoms. For the 58 patients with a single recorded visit the median time to onset was 5 days (range 1–12 days).

Fifty-seven of the 68 had visited the OPD, 46 for an appointment and 11 accompanying relatives or friends. Eleven patients went elsewhere in the hospital, 7 as inpatients and 4 as visitors to wards. The available information suggests that at least 7 of these 11 people visited OPD for investigations. It is not known whether the remaining 4 passed through OPD, but it is likely that they went close to the area since the entrances to OPD were adjacent to both the main hospital entrances and to the corridor leading to lifts and staircases giving access to the wards. The duration of reported exposure for the 64 patients known to have been in the OPD varied from as little as 10 minutes to several hours.

The ages of the patients ranged from 36 to 88 years, with a mean age of 62 years. Forty-one were males who tended to be older than the 27 females; 34 of the 41 men were aged over 60 years compared with only 16 of the 27 women ($P = 0.04$). Seventeen of the 22 deaths were men.
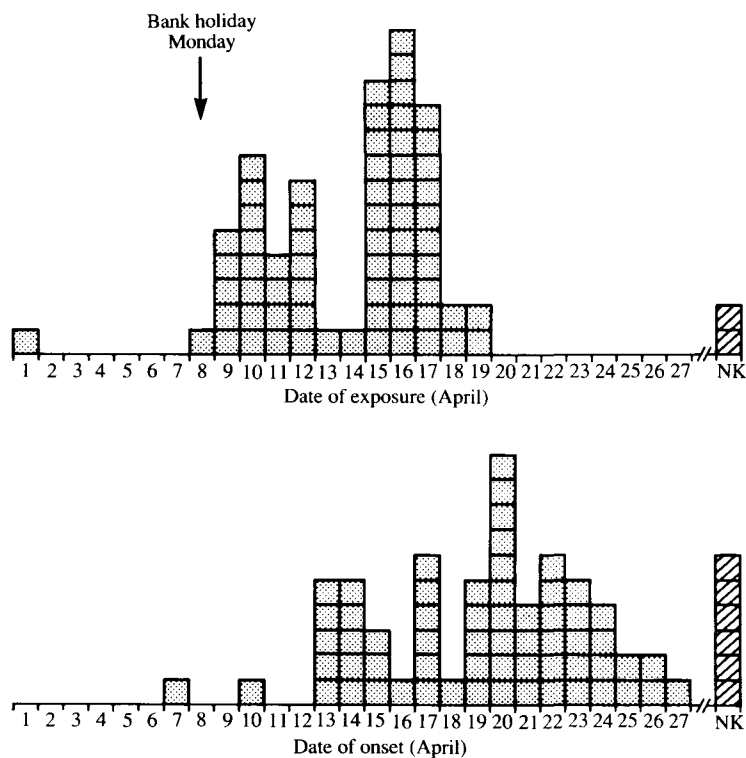
Fig. 2. Date of exposure to the DGH and date of onset of Legionnaires' disease for the
68 patients treated in hospital.

Case control study

This study was of the first 53 of the 68 inpatient cases to be recognized, and
included 29 men and 24 women and their 106 matched controls. All 53 patients
and 31 of the control group had visited the DGH during the period (March and
April), although many of these 31 had visited wards and not the OPD. There was
a significant association between visiting the OPD and developing legionnaires'
disease (Table 1). People who visited this department were 98 times (odds ratio)
more likely to have developed the disease than those who had not. All 49 of the
patients who had definitely been to the OPD had been there between 1 and 19
April, whereas, of the 21 people in the control group who had visited the OPD,
only 3 had been there during that period.

There was no significant association with visiting other parts of the hospital and
acquiring Legionnaires' disease (Table 1). Nor was there any association with
sitting by an open window (reported by only one case and one control), nor with
using the toilets and wash hand basins. There was no association with exposure to
sites other than the DGH, including other hospitals.

Two of the 53 patients were severely disabled and had not been outside their
homes for several weeks, except to visit the DGH. The case group included a
slightly higher proportion of people with underlying chronic illness, 32 (60%)

Table 1. *Results of the case control study: exposure to the District General Hospital and areas within it*

	Cases of Legionnaires' disease			Matched controls			
Location	Exposed to the location	Not exposed to the location	Not known	Exposed to the location	Not exposed to the location	Not known	Fisher's exact P
DGH	53	0	0	31	74	1	< 0·00001
Within DGH							
Outpatient department*	49	2	2	21	84	1	< 0·00001
Wards	9	39	5	15	88	3	0·3

* Odds ratio = 98 (odds ratio is an indirect measurement of relative risk, the ratio of the rate of disease among those exposed to the OPD and those not exposed).

compared with 54 (51 %) in the control group. A significantly greater proportion were cigarette smokers, 23 (43 %) of cases compared with 23 (22 %) of controls. Blood samples for serology were obtained from 98 of the 106 controls and positive IFAT titres at 16 were found in only 3 (3 %) and these had had no known contact with the DGH.

Study of OPD attenders

Of the 10 834 questionnaires sent out 9339 (86 %) were completed and returned. These included 7969 returns from patients who had attended clinics in April and the attack rate of Legionnaires' disease was 0·6 % (46 cases). The age group and date of attendance were unambiguously recorded for 9296 (99·5 %) of returned questionnaires and the age, sex and clinical summary for these are shown in Table 2. The highest attack rate of Legionnaires' disease was in those aged 65 or over and affected 3·9 % (28 of 710) people in this age group who had visited the OPD in the second and third weeks of April (Table 2). The corresponding rate for males was 5·8 % and for females 2·6 %.

There were 777 (8·4 %) reports of influenza-like illness following the 9296 attendances. For the 65 years and over age group the rate of reported influenza-like illness peaked in the second week of April, coinciding with the rise in cases of Legionnaires' disease. In younger age groups the 'influenza-like' illnesses tended to be more frequent later in the month and there was no apparent correlation with the pattern of Legionnaires' disease. Thus there appears to have been a high rate of reported respiratory illness in outpatient attenders which may possibly have included some legionellosis, especially in the older patients, but without serological evidence no firm conclusion can be drawn.

The questionnaires were analysed to study movement within the OPD and use of facilities. There was no association between reported influenza-like illness or developing Legionnaires' disease and visiting or waiting in any particular area within the OPD. Neither was there any significant association with time of day of visit nor with use of toilets or wash hand basins.

Table 2. *Study of outpatient attenders: attack rates of Legionnaires' disease and reported influenza-like illness by age and sex group for appointments in April and 10% samples of appointments in March and May*

Period of appointment		Age Group (years)			All males	All females	Total
		< 40	40–64	65 +			
March and 1st week of April	Total attendances	1158	758	562	999	1479	2478
	Cases of Legionnaires' disease	—	—	1 (0·2%)		1 (0·1%)	1 (0·04%)
	Reported influenza	69 (6·0%)	54 (7·1%)	21 (3·7%)	61 (6·1%)	83 (5·6%)	144 (5·8%)
2nd and 3rd weeks of April	Total attendances	1618	1048	710	1440	1936	3376
	Cases of Legionnaires' disease	—	17 (1·6%)	28 (3·9%)	26 (1·8%)	19 (1·0%)	45 (1·3%)
	Reported influenza	133 (8·2%)	110 (10·5%)	48 (6·8%)	116 (8·1%)	175 (9·0%)	291 (8·6%)
4th week of April and May	Total attendances	1693	1026	723	1387	2055	3442
	Cases of Legionnaires' disease	—	—	—	—	—	—
	Reported influenza	188 (11·1%)	120 (11·7%)	34 (4·7%)	131 (9·4%)	211 (10·3%)	342 (9·9%)

Table 3. *Study of District General Hospital staff: serological results and air conditioning plant serving main place of work*

Location	Serological result		Total staff tested
	+ ve*	− ve	
Plant 2 (kitchens)	6 (18%)	27	33
Plant 3 (A & E, physio, X-ray)	19 (25%)	57	76
Plant 4 (OPD, records, theatres, pharmacy)	107 (46%)	124	231
Other areas, exposure to multiple plants (Administration, dental, maintenance wards)	103 (23%)	339	442
Total	235 (30%)	547	782†

* IFAT ⩾ 16.
† Information on work location not recorded for eight staff.

Eleven of the 6634 people reported to have accompanied these patients in April also contracted Legionnaires' disease, giving an estimated attack rate for escorts of 0·2%.

Blood samples were obtained from 204 of 207 consecutive obstetric patients admitted to the DGH maternity unit between August and September 1985. These patients had attended antenatal clinics before May. Thirty-two women (16%) had *L. pneumophila* antibody titres of ⩾ 16, compared with only 3% found in the community. None developed Legionnaires' disease and their babies were normal.

Study of hospital staff

Altogether 1708 staff from the three hospitals returned questionnaires. These comprised 1135 (72%) of the 1581 staff based at the DGH and 573 (77%) of 747 staff based at the other two hospitals. Serological results were available for 1089 staff, 790 based at the DGH and 299 elsewhere. Of the 790 DGH staff 237 (30%) were seropositive with an IFAT titre of ⩾ 16. This was a significantly higher proportion than among the 299 staff based at the other two hospitals, where 31 (10%) were seropositive ($P < 0.00001$). Many of these 31 staff had been to the DGH during the 5 months studied, December to April, either in the course of their work or as visitors or patients. Of 187 who had not been to the DGH, 6 (3%) had antibodies to *L. pneumophila*, which is the same proportion found among controls in the case control study.

Table 3 shows staff based at the DGH according to their main work location within the hospital and the air conditioning plant serving this location. Staff whose main work location was in the areas served by plant 4, the OPD and the two upper floors of the same wing, were significantly more likely to be seropositive.

The questionnaires filled in by the DGH staff were then analysed with regard to the stated amounts of time spent in different parts of the DGH. There was a significant association with seropositivity and the amount of time spent on each

Table 4. *Study of District General Hospital staff: serological results for 166 staff based in the OPD according to time spent in the operating theatres area*

| | Visits to theatres | | | | | |
| | None | | | Some | | |
Time spent in OPD	+ ve*	− ve	Total	+ ve*	− ve	Total
Up to 2 days per week	13 (23%)	44	57	6 (55%)	5	11
3–5 days per week	41 (51%)	39	80	12 (67%)	6	18

* IFAT ≥ 16.

of the three floors of the OPD wing. For example, staff who spent 3 or more days per week working in the OPD were more likely to have antibodies than those who worked 1 or 2 days, and they, in turn, were more likely to have antibody than staff who never worked in the wing. The association of positive serology with working on these three floors was independent of staff movement between floors, that is staff who worked in the operating theatres on the middle floor of this wing, and who stated that they never visited the OPD, were more likely to have antibody than staff working in other wings. The same independent association was observed in staff working in the maternity unit on the top floor of this wing. There was evidence that effects of exposure could be cumulative. Table 4 shows the serological status of 166 staff who worked at least one day a week in the OPD according to the amount of time spent in the OPD and whether they visited the operating theatres on the middle floor of that wing. Staff who worked 3 or more days in OPD were significantly more likely to be seropositive and there was an additional association if they visited the theatres. The multivariate analysis did not demonstrate that working on any one of the three floors in this wing carried a higher risk of acquiring antibody than the other floors. However, the analysis could only have detected large differences in relative risks because patterns of movement of staff between floors did not follow those of an efficiently designed experiment. For example, few of the staff based in the theatres regularly visited the OPD or maternity department.

Of 80 DGH staff who reported never working in the OPD wing 17 (21%) were seropositive. Fifty-one of these 80 staff with no regular work exposure to the OPD wing had visited the area for other reasons, to see friends or to use the OPD as a route to other parts of the hospital.

In the 5 months, December 1984 to April 1985, 592 (75%) of the 790 DGH staff tested for legionella antibodies recalled having an 'influenza-like' illness. Of the 236 seropositive staff 193 (82%) reported an illness. A multivariate analysis was used to determine whether any of these 'influenza-like' illnesses might have been associated with acquiring antibody. This analysis was done by studying the combination of symptoms reported by seropositive staff who had been ill with those reported by seronegative staff who had been ill. Two symptoms were found to be independently associated with the presence of legionella antibodies. These were a dry cough which was positively associated and a sore throat which showed negative association. Table 5 shows that staff reporting influenza-like illnesses

Table 5. *Study of District General Hospital staff: serological results and reported
'influenza-like' illness*

	Serological results		
	+ve*	−ve	Total
Staff reporting an influenza-like illness symptoms including			
(a) Dry cough, no sore throat	23 (49%)	24	47
(b) Dry cough, sore throat	81 (39%)	129	210
(c) No dry cough, no sore throat	32 (32%)	67	99
(d) No dry cough, sore throat	45 (23%)	149	194
(e) Symptoms not recorded	12	30	42
Staff *not* reporting an illness	43 (22%)	150	193
Total	236 (30%)	549	785†

* IFAT ⩾ 16.
† Five staff did not answer the question on illness.

which included a dry cough or did not include a sore throat were significantly more likely to have legionella antibodies. Staff who had had influenza-like illnesses with a sore throat and no dry cough were no more likely to be seropositive than staff who had not been ill. Thus some, but not all, the influenza-like illnesses among staff may have been associated with legionella infection and this was more likely if the symptoms included a dry cough or no sore throat. Such illnesses had occurred throughout the 5-month period and were reported by significantly more of the staff working in the OPD wing than those based elsewhere in the hospital.

Bacteriological results

The epidemic strain *L. pneumophila* serogroup 1, subgroup Pontiac 1a, was isolated from 11 post-mortem lung specimens. In November 1984 *L. pneumophila* serogroup 1 had been isolated by the water treatment company's laboratory from the pond of the cooling tower of air conditioning plant 4. This isolate was subsequently typed as the epidemic strain. In January 1985 the pond of this tower was chlorinated, drained, refilled and rechlorinated. The organism was not isolated from a further sample taken by the water treatment company in March. Samples taken in response to the outbreak and before chlorination on 4 May did not yield the organism either from the cooling tower ponds nor from the hot and cold water systems. However, during the course of the Public Inquiry it emerged that a large amount (some 2 l) of undiluted biocide had been 'casually shot dosed' into the pond of cooling tower 4 on 30 April. It also became clear that the water treatment company had taken water samples on the 3 May, the day before chlorination, and that the epidemic legionella strain was isolated, albeit in 'minimal concentration'.

The epidemic strain was isolated from the insulating material (mastic) used around the edges of the chiller unit (one of the 14 units in plant 4) which cooled air for the OPD.

Engineering investigations

The hospital had air conditioning systems which used four evaporative cooling towers on the roof of the main building. The cooling towers and associated
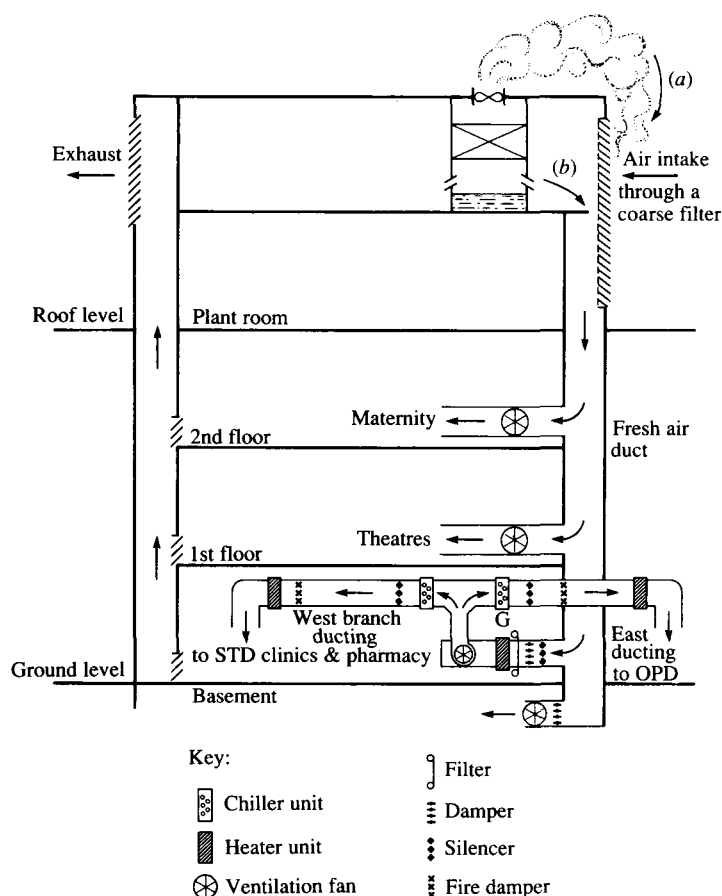
Fig. 3. Sectional diagram of part of the DGH illustrating relevant routes of airflow in air conditioning plant 4. (a) and (b) show the way bacteria would have had to go if spread took place from cooling tower drift and pond splash. G, indicates the chiller unit serving OPD and is also shown in Fig. 4.

engineering plants were housed in concrete towers and were an integral part of the building. Three of these provided air conditioning for internal and clinical areas of the hospital. The area supplied by plant 4 was the only one that required cooling during the winter and consequently, of the four plants, only the cooling tower of plant 4 was operating between November 1984 and April 1985. (The other three plants supplied uncooled ventilation with fans in operation.) Plant 4 supplied air to part of the OPD (including the main waiting area), the X-ray department, pharmacy, STD clinic and parts of the operating theatre suites and the maternity unit on the floors above (Fig. 3). Thorough investigations lasting many months disclosed a number of factors which may have promoted the outbreak or assisted its genesis.

The OPD chiller unit

There was a direct plumbing connection (Fig. 4) between the vertical pipe (diameter approximately 10 cm) which drained waste water from cooling tower 4
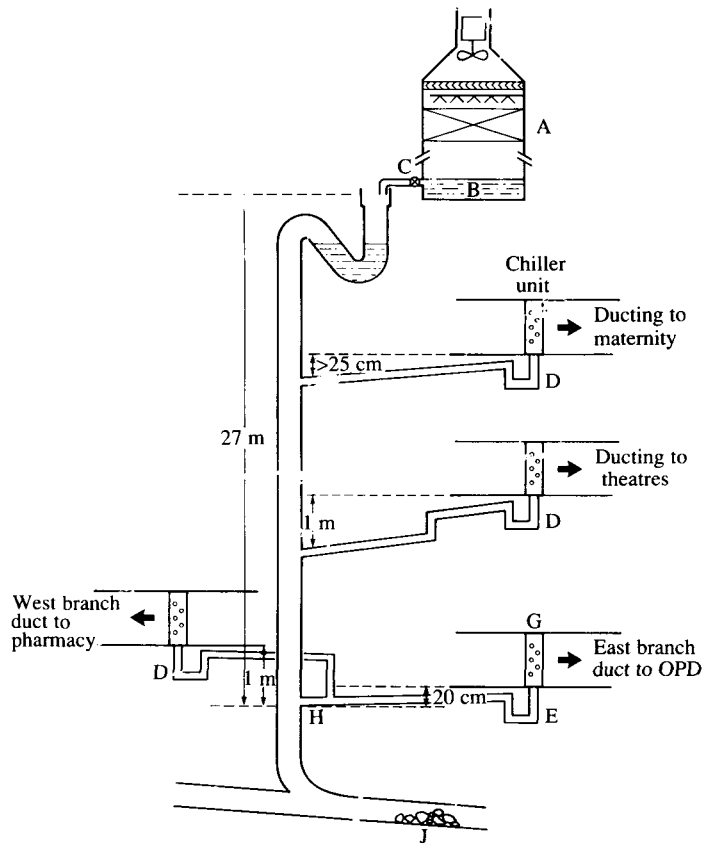
Fig. 4. Diagram of air conditioning plant 4 showing the cooling tower, its drainage stack and the relevant part of the tree of chiller units joined to it. In the diagram is shown the approximate vertical lift which any refluxed water from the tower drain would have to traverse to reach the base of each chiller unit. Labelled features are: A, cooling tower; B, pond from which *L. pneumophila* was isolated; C, blowdown (drain) valve; D, water traps found to be dry; E, water trap found to be wet; G, chiller unit from which *L. pneumophila* was isolated; H, right-angled (unswept) junction; J, builders' rubble.

pond and pipework connected to the chiller units. It was demonstrated that the water coming down from the drainage pipe from the cooling tower pond, some 27 m above, could enter the pipe draining condensate from the chiller unit in the air handling system serving the OPD. The configuration of pipework ran in an almost horizontal line from the drainage stack to the base of the chiller unit serving the OPD (only a 20 cm lift, Fig. 4). But, although there were similar direct plumbing connections to chiller units on the two upper floors and to a second unit on the ground floor, the configuration of pipework would not permit the backflow of water. The U-traps of all chiller units except that to OPD were dry on inspection.

A partial blockage caused by builders' debris was found at the base of the cooling tower drain. This would have increased the chance of backflow of water as it descended the drain. It was seen during on-site trials that, during a period of

backflow, water did in fact reflux into the OPD chiller unit itself. If the water contained viable legionella organisms this could both contaminate the U-trap with infected water and release an infectious aerosol into the air stream.

There had been large quantities of water draining down from the cooling tower pond due to a fault in a conductivity meter which controlled the drainage (blowdown) valve (Fig. 4) of the tower pond, causing it to remain permanently open. It was estimated that of the order of 4000 l of water per day drained from the pond over an unknown period of time. This provided ample opportunity for water to enter the pipework of the chiller unit and also raise the air pressure in the pipe. On-site experiments estimated the pressure to be raised by about 100 mm of water and bubbling through the U-trap was observed. Laboratory experiments at CAMR, using the original OPD chiller unit U-trap and pipework showed that a pressure difference of $\geqslant 25$ mm of water produced bubbling. Use of bacterial spores (*B. globigii*) demonstrated that similar bubbling would produce a bacteria-containing aerosol which would then have been carried into the air stream and distributed via the air conditioning system.

Drift from cooling tower 4

The air intake from the ventilation and air conditioning systems was adjacent to the exhaust from the cooling tower (Fig. 1). It was possible for drift from cooling tower 4 to enter its own air intake in two ways. Firstly, the prevailing wind was said to be north-westerly and experiments with sulphhexofluoride gas showed that 0·1 % of this gas released into the cooling tower plume could be detected in the plant 4 air intake (Fig. 3). Secondly, when the cooling tower fans were switched off water drained from the pack in the tower for a few minutes and then continued to dribble into the pond at about 20 l/min, creating splashing and thus an aerosol. The tracer gas studies also showed that about 10 % of the aerosol produced might enter the air intake without passing outside, because of a gap in the plant room floor. These experiments were conducted by scientists assisting the investigations of the Public Inquiry.

Volumes of aerosols generated in the drift and in the chiller unit

Calculations indicated that the proportion of the aerosol originating in the cooling tower drift that remained infectious and was likely to have been delivered into the OPD was very small. The maximum water loss, as drift, occurred when the fans were on and when water was flowing over the pack. The loss was estimated to be 3·4 l/min. If it is assumed that the drift was all in the form of droplets then only the equivalent of 0·1 % (3·4 ml/min) would have reached the air intake. Only droplets of $\leqslant 50$ μm diameter would have a chance of drying down to the critical size of $\leqslant 5$ μm before reaching OPD. Information received during the Inquiry showed that only about 5 % (170 μl) of the drift would be expected to be in this range. Even if all the particles dried down to this size, many would have been removed by the filter (Fig. 3). The filter, if clean, would remove 20–80 % of particles $\leqslant 5$ μm diameter, but would remove a higher percentage if dirty. Thus estimates suggest that, at most, only the equivalent of 34–136 μl/min from the original 3·4 l/min of drift would enter the main ventilation ducting. This aerosol would then have been divided between the three floors and basement of the OPD

wing and could have been further reduced by passage through ventilation ductings, and impaction on the chiller, heater, silencers and dampers (Fig. 4). In addition, before entering the air intake, the aerosolised legionellae would have been exposed to some ultraviolet light to which they are sensitive, and also to 'open air factor' [6].

Legionellae aerosolized from the U-trap in the chiller unit serving the OPD would not have been subjected to any of these reductions. Thus bubbling in the U-trap need only to have aerosolized a few tens of microlitres per minute to be equivalent to the drift reaching the OPD from the cooling tower. This volume was easily achieved in laboratory experiments under conditions similar to those thought to have occurred in the hospital. Seventy percent of the particles carrying bacteria were $< 6\ \mu$m diameter and 40% were $< 3\ \mu$m.

DISCUSSION

In 1985 this was the largest known outbreak of Legionnaires' disease in the UK [7]. The outbreak was severe, 22 (32%) of the 68 patients treated in hospital died, and it took place over a relatively short period of time. The source of infection was a hospital about 2 years old and one of its air-conditioning plants was implicated. Nearly one third of hospital staff had acquired antibody to *L. pneumophila*, as compared with only 6 and 7% in previous hospital outbreaks in the UK [8, 9].

Investigation of the outbreak required a multidisciplinary team and a variety of studies were undertaken in response to initial observations and, later, as a result of findings from earlier studies. The epidemiological case control study showed that visiting the Stafford District General Hospital was the only significant risk factor for acquiring Legionnaires' disease. Other possible sources such as industrial cooling towers, air-conditioned shopping centres and travel abroad were ruled out.

The cases of Legionnaires' disease were probably all infected between 1 and 19 April. Within the DGH it was the Out-Patient Department which was associated with risk of contracting Legionnaires' disease and no other part of the hospital was implicated. A large number of people in the age-matched control group had visited wards in the hospital, implying that many elderly people must have been to the hospital wards during April. Also the wards and theatres must have been used for susceptible patients and yet the excess of Legionnaires' disease cases was found only among people who had been to the OPD. The study of OPD attenders did not demonstrate any risk associated with use of the hot or cold water outlets in toilets or basins. Attention was concentrated on the air conditioning systems, especially plant 4 which served the OPD wing.

It transpired during the Inquiry that the water treatment company, contracted to maintain the air conditioning plants, had isolated the epidemic strain, *L. pneumophila* serogroup 1, subgroup pontiac 1a, from water in the cooling tower pond of ventilation plant 4 in November 1984 and again in May 1985. During the outbreak investigation we found this same legionella type in damp mastic sealing compound which lay in the end drainage tray of the chiller unit which had its drain connected to the same drain as the ventilation ducting leading directly to the OPD (Fig. 4).

The finding of legionellae in the cooling waters of air conditioning systems is not

14

unusual. A problem arises only if an aerosol can be generated from them and if the aerosol is then inhaled. Engineering investigations at the DGH showed that aerosol generation was possible by two main routes. First, the air intake vent for the air conditioning system of plant 4 was sited close to the cooling tower. It was unfortunate that the position was such that the prevailing wind (from approximately 320° N) would carry part of the spray (drift) from cooling tower 4 towards its own intake vent (Fig. 1) and also towards that of plant 5, the ventilation of which was switched on though the air was not cooled. Experiments showed that only a very small volume of inhalable water droplets might have reached the three floors of the OPD wing. There was no reason to expect that a higher concentration of aerosol would reach the ground floor, and thus the OPD, as compared with the two higher floors. Maternity and theatres would have received an aerosol challenge equal to or greater than OPD, as ventilation was continuous to these units but was switched off overnight and at weekends to OPD. This route seems unlikely to explain why the risk of contracting Legionnaires' disease was demonstrated to be significant only for the OPD.

Second, an aerosol could be generated in the OPD chiller unit. We conclude that water, which contained the epidemic strain, coming down the vertical drain pipe from the cooling tower pond had refluxed up the drain pipe from this chiller unit: this was possible because the pipe had no air break. In addition the connecting pipe was almost horizontal (Fig. 4), being joined to the vertical stack at right angles, and so there was little impediment to prevent water travelling as far as the chiller unit. The epidemic strain of legionella was isolated from material (mastic) surrounding the unit. The U-trap in the drain pipe from the chiller unit contained water when examined, whereas all other traps in the same system were found to be dry. Furthermore air conditioning to the OPD was switched off overnight, allowing multiplication of legionellae in the warm stagnant trap water. Experiments showed that air bubbling through this U-trap – which was likely to have occurred because water falling down the vertical drain from the cooling tower pond will have raised the air pressure – was efficient in creating an aerosol of water droplets of appropriate size. Thus a mode of conveying legionella organisms directly into the air of the OPD was discovered. Because this coincides with the epidemiological evidence, which found the OPD to be the only area within the hospital where there was a significant risk of acquiring Legionnaires' disease, this team concludes that an aerosol from the contaminated chiller unit was probably the major factor causing the outbreak. This differs from some of conclusions given by the Badenoch Committee following the Public Inquiry' [1], where drift from the cooling tower was thought to have played the major role.

However it is not disputed that drift from the cooling tower may have played some part in contagion of the DGH atmosphere. The survey questioning hospital staff about movements and illness, linked with their serological tests, showed a different pattern of results from the study of cases of Legionnaires' disease. Thirty percent of staff based at the DGH had antibody to *L. pneumophila*. Staff who worked in the OPD wing were more likely to have antibody than those who worked elsewhere in the hospital. But high proportions of staff who worked on the two upper floors of that wing were seropositive, even if they never visited the OPD on the ground floor. Thus exposure to the organism appeared to have been higher

in all parts of the hospital which were served by air conditioning plant 4 and not just the OPD. Exposure leading to Legionnaires' disease was associated with the OPD and occurred only during the month of April. Exposure leading to seropositivity in staff may have taken place over many months. In a small proportion of staff the acquisition of antibody was associated with an influenza-like illness, with onset of illnesses over a 5-month period.

Unfortunately it was not possible to study staff who had left the DGH before April to find out whether or not they had developed legionella antibody before the epidemic began. The cooling tower pond was known to have contained legionellae in November and it seems possible that the intake of aerosol from the drift, albeit in small amounts, may have been a major factor in exposing staff to antigenic stimulus. The doses of legionella experienced this way may have been too small to have caused Legionnaires' disease even in vulnerable patients and elderly visitors to the hospital. In the 1976 Philadelphia outbreak [10] it was noted that hotel staff appeared to be immune from illness. Low level, intermittent exposure for 2 years before the outbreak was suspected.

There were no records of anyone suffering from Pontiac fever during the incident in Stafford. High levels of respiratory illnesses in staff and other members of the community were observed during the period. Therefore the epidemiological investigations included study of self-reported 'influenza-like' illness in hospital staff and in OPD attenders. Among staff, where there was serological evidence to complement the questionnaires, a small proportion of the large number of respiratory illnesses recalled over the winter period were associated with the presence of legionella antibody. These influenza-like illnesses were characterised by a dry cough or the absence of a sore throat amongst other symptoms. Approximately 16% of staff who had antibodies may have had such an illness but the majority had no associated illness. Eight percent of outpatient attenders reported an influenza-like illness with onset during the fortnight after their appointment. However the time pattern of these illnesses was unlike that of the cases of Legionnaires' disease, except among 65+ year olds. The pattern of reported symptoms was studied to see whether there was any similarity with the significant illnesses reported by seropositive staff. Unfortunately the questionnaire for outpatients did not distinguish dry and productive coughs. However, among outpatients who reported illnesses there were a higher proportion whose symptoms did not include a sore throat among the over 65-year-olds and following visits to the OPD during the first 2 weeks of April. This suggests that some mild legionellosis was occurring among older outpatients but, in the absence of serology, this conclusion remains speculative. Most of the influenza-like illness must have been unrelated to the outbreak.

It can be argued that this large outbreak of Legionnaires' disease might not have occurred had not the design of the plant pipework, deficient in airbreaks, permitted water contaminated with legionellae to gain access to the OPD ventilation trunking; then people visiting that department would not have contracted Legionnaires' disease. A second source of aerosol containing legionellae was drift from the cooling tower, which entered poorly sited air intake vents. This source may have been responsible for long-term, low-level atmospheric contamination leading to seroconversion in staff.

14-2

The combination of events which triggered the start of the outbreak is not known, nor are the reasons for its cessation. Timings coincided with unseasonally warm weather and so more demands were being made of the air conditioning system. But such demands must have been made on other occasions. Exposure to conditions leading to new cases had ceased before the outbreak was even recognized.

The extensive investigations and the Public Inquiry led to many recommendations for better design and maintenance of hospital ventilation systems [1].

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