

## **Fatal nosocomial Legionnaires' disease: relevance of contamination of hospital water supply by temperature-dependent buoyancy-driven flow from spur pipes**

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### SUMMARY

The investigation, epidemiology, and effectiveness of control procedures during an outbreak of Legionnaires' disease involving three immunosuppressed patients are described. The source of infection appeared to be a network of fire hydrant spurs connected directly to the incoming hospital mains water supply. Removal of these hydrants considerably reduced, but failed to eliminate, contamination of water storage facilities. As an emergency control procedure the incoming mains water was chlorinated continuously. Additional modifications to improve temperature regulation and reduce stagnation also failed to eliminate the legionellae.

A perspex test-rig was constructed to model the pre-existing hospital water supply and storage system. This showed that through the hydraulic mechanism known as 'temperature buoyancy', contaminated water could be efficiently and quickly exchanged between a stagnant spur pipe and its mains supply. Contamination of hospital storage tanks from such sources has not previously been considered a risk factor for Legionnaires' disease. We recommend that hospital water storage tanks are supplied by a dedicated mains pipe without spurs.

### INTRODUCTION

The prevention of hospital-acquired Legionnaires' disease (LD) remains an unresolved international problem. Legionellae can be found in most hospital water systems [1]. None of the methods presently used to suppress them can be guaranteed to be effective long term [2]. In addition, the increasing use of immunosuppressive therapy means that more patients will be susceptible to infection.

Guidelines on the control of legionellae in hospital premises, presently under review, were issued by the Department of Health and Social Services (DHSS) in

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1988 [3]. Recommendations were made on the installation, modification, and maintenance of hot and cold water storage and distribution systems. Neither routine testing to monitor legionellae, nor the use of continuous chlorination to control their growth, was recommended except in an outbreak.

In health care premises, water is normally supplied to wards and other clinical areas from storage tanks. It is rarely supplied directly from the incoming mains which is generally free of pathogenic micro-organisms. Provided storage tanks are clean and properly maintained, this arrangement has not previously been regarded as a hazard in the control of nosocomial infection.

In June 1990 LD was diagnosed in a patient receiving treatment in the Old Building of Glasgow Royal Infirmary. Following the recommendations of the Scottish Home and Health Department [4], the hospital Control of Infection Committee established a team to investigate and take necessary control measures. This paper describes the outcome of their investigations, the epidemiology of the subsequent outbreak and the lessons learned in attempting to maintain a water supply free of legionellae.

## METHODS

### *Case ascertainment*

After the first case, staff and patients in the ward, and patients in other wards with symptoms of hospital-acquired respiratory infection, were investigated for legionellosis [5]. Legionnaires' disease was confirmed if legionellae were isolated from a clinical specimen or if a fourfold or greater rise between acute and convalescent antibody titres was demonstrated in either the Indirect Fluorescent Antibody Test (IFAT) or Rapid Microagglutination Test (RMAT). A 'presumptive' diagnosis of Legionnaires' disease was made on the basis of a single high antibody titre (IFAT 128 for a single case, 64 in an outbreak; RMAT 32) or a positive urinary antigen test. The urinary antigen test was an enzyme-linked immunosorbent assay (ELISA) performed at Ruchill hospital [6, 7].

Nosocomial Legionnaires' disease was defined as a confirmed or presumptive case who spent all of the 10 days before the onset of symptoms in hospital. A 'probable' nosocomial case was defined as a confirmed or presumptive case who spent between 1 and 9 days before the onset of symptoms in hospital and where infection was associated with other nosocomial cases (definite or probable) in the same 6-month period.

### *Water sampling*

Five-litre water samples were collected from the mains water supply (street and hospital) and the storage and distribution system within the hospital. They were cultured for legionellae. After filtration of the sample through a 0.2  $\mu\text{m}$  nylon filter (Supor 200 142 mm membranes, product No. 60305), the filter was chopped and shaken in 50 ml of the original sample. Two 10 ml aliquots were then removed. One was treated with an acid buffer (HCl/KCl, pH 2.2) for 10 min, the other with heat (50 °C) for 30 min. Each aliquot was then cultured on four different buffered charcoal-yeast-extract (BCYE) agar plates supplemented with L-cysteine hydrochloride, ferric pyrophosphate and antibiotics [8]. These were incubated for 14

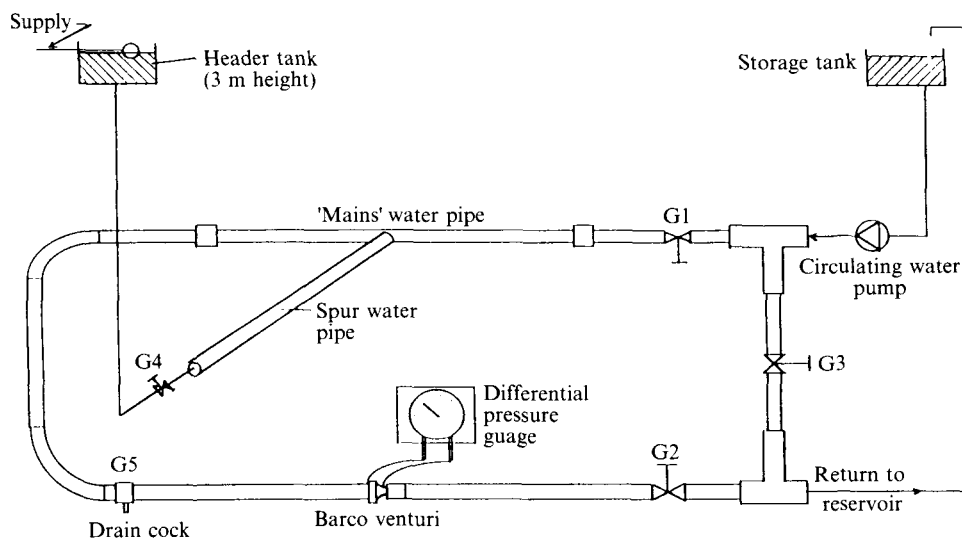


Fig. 1. Schematic of test facility.

days at 37 °C in 4% CO<sub>2</sub>. Direct cultures from the water sample and the 50 ml aliquot containing the filter were also performed on four agar plates as above. Colonies were initially recognized by their characteristic ground glass appearance. On exposure to Ultra Violet (u.v.) light, some species of *Legionella*, e.g. *L. anisa*, fluoresced blue/white. Colonies were verified as being *Legionella* spp. by failing to grow on subculture to non-supplemented BCYE agar (Oxoid CM655). At least one colony per plate was confirmed and serotyped using immunofluorescent sera supplied by Public Health Laboratory Service (PHLS), Colindale, London NW9. Selected isolates of *L. pneumophila* sg1 were sub-typed at the Department of Laboratory Medicine, Ruchill Hospital using monoclonal antibodies produced by Oxford PHLS [9]. The numerical count of legionellae per litre was recorded. The minimum value possible by the above method was 100 colony forming units per litre (c.f.u./l).

#### *Chlorination of mains water supply*

The aim was to chlorinate the incoming water to provide a free residual chlorine level of between 1 and 2 p.p.m. at all outlets. This required the installation of individual electrochlorination units (Portacel Cl<sub>2</sub> system) in three incoming mains supplies.

#### *Investigation of hospital mains water supply*

A test-rig was constructed of cast acrylic (perspex) pipe modelled on the hospital water system [10]. It allowed the mixing of water between a spur pipe and its mains water supply to be visualized (Fig. 1). A spur pipe 2.5 metres (internal diameter 78 mm) in length was taken from a ring water mains. Two water tanks were connected. The first was a header tank used to fill the system and flush the mains spur. The second was a storage tank used to supply the circulating mains. A fluorescein dye was dissolved in the circulating mains. This enabled the exchange of water between the 'mains circuit' and the 'spur pipe' to be monitored. Gate valves fitted to the header tank and the flow and return pipes in

the ring mains enabled the spur pipe to be flushed clear of dye and the water velocity in the mains circuit to be accurately controlled. Water temperature was measured by thermocouples accurate to within 0.1 °C in the range 0–40 °C.

Water velocity in the mains circuit was increased from 0.2 metres per second (m/s) to 1.0 m/s. The maximum turbulence depth in the spur pipe and the rate of water exchange between the spur pipe and mains circuit was recorded. Water temperature in the mains circuit (but not the spur pipe) was then raised and the experiment was repeated.

## RESULTS

### *Patients*

Between June and October 1990 three patients with Legionnaires' disease were diagnosed in the Old Building of the Glasgow Royal Infirmary.

The first patient, a 59-year-old female, was admitted on 17 May 1990 with an exacerbation of systemic lupus erythematosus. Following high dose immunosuppressive steroid therapy she was discharged on 8 June 1990 but was re-admitted 2 days later with a suspected myocardial infarction. The provisional diagnosis was community-acquired pneumonia. Legionnaires' disease was later diagnosed when antibody titres to *L. pneumophila* serogroup (sg) 1, requested on 21 June 1990 because X-ray changes were slow to resolve, were found to be raised (Table 1); previous sera collected for biochemical tests were available. The patient, who was treated with erythromycin and made a good recovery, was considered to have acquired the infection while in hospital.

Initially this incident was thought to be sporadic. No other cases were identified within the hospital complex. However, 12 weeks later on 7 September 1990 it was confirmed that a 37-year-old female patient who had received a bone marrow transplant also had LD. This patient had been admitted with acute lymphoblastic leukaemia on 15 August 1990. In preparation for transplant she received cyclophosphamide 60 mg/kg on 18 August 1990 and 19 August 1990, and Total Body Irradiation at a neighbouring hospital between 20 August 1990 and 24 August 1990. On the evening of 24 August 1990 she returned to the Glasgow Royal Infirmary, entered Laminar Flow Isolation and received her marrow. On 31 August 1990 she became febrile, later developing a right upper lobe pneumonia. The causal organism, *L. pneumophila* sg1 subtype Pontiac, was isolated on 7 September 1990 from a bronchoscopy sample collected 4 days earlier. The patient died on 8 September 1990.

The third patient, a 75-year-old female, was admitted on 18 September 1990 with a suspected cerebro-vascular accident. She was treated with high dose steroid immunosuppressive therapy for temporal arteritis and was discharged on 8 October 1990. Four days later she was re-admitted to a neighbouring hospital with right lower lobe pneumonia. Presumptive LD was diagnosed on the basis of a positive urinary antigen test for *L. pneumophila* sg1 [6, 7]. The patient died on 16 October 1990.

On 11 October 1990 continuous chlorination of the mains cold water supply to the Old Building of the Glasgow Royal Infirmary was commenced. No further cases of LD believed to be nosocomial were diagnosed in the following 2 years.

Table 1. Antibodies to *L. pneumophila* sg1 in first case

Date of sample	RMAT*	IFAT†
12 April 1990	-ve	-ve
21 May 1990	-ve	-ve
1 June 1990	-ve	-ve
4 June 1990	-ve	-ve
11 June 1990	1/8	-ve
21 June 1990	1/1024	1/1024

\* Rapid microscopic agglutination test.

† Indirect fluorescent antibody test.

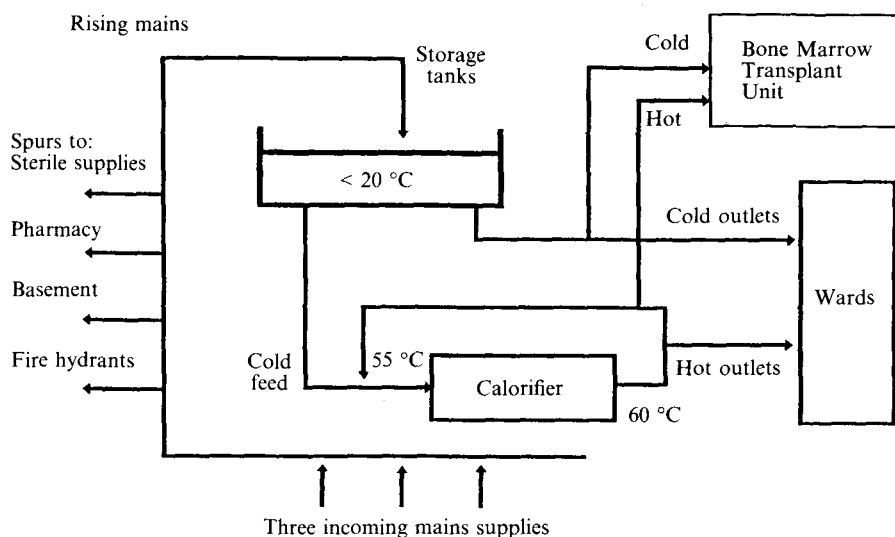


Fig. 2. Old Building, Glasgow Royal Infirmary original water storage and distribution system.

#### Water sampling

A simplified diagram of the water supply, storage and distribution system in the Old Building of the Glasgow Royal Infirmary at the start of this outbreak is presented in Fig. 2. The revised water system at the conclusion of the outbreak is presented in Fig. 3.

Three cold water mains supplies entered the hospital through water-meters in the sub-basement. Three-inch (75 mm) diameter mains pipes ran from each water-meter before rising to supply the hospital's roof storage tanks. A number of spurs (branch pipes) were taken from the mains pipes before they reached the storage tanks. These supplied water to a network of 14 hydrants for use by the fire brigade, areas of the sub-basement and basement, and pharmacy and central sterile supplies. Each fire hydrant outlet was served by an individual spur pipe which ran for approximately five metres in the warm sub-basement, then a further five metres buried in the external lightwells before emerging in the open and rising a further 4.5 metres to external carpark level. Although the pipework in the open air was insulated, it was undoubtedly subject to solar heat gain and large seasonal temperature variations.

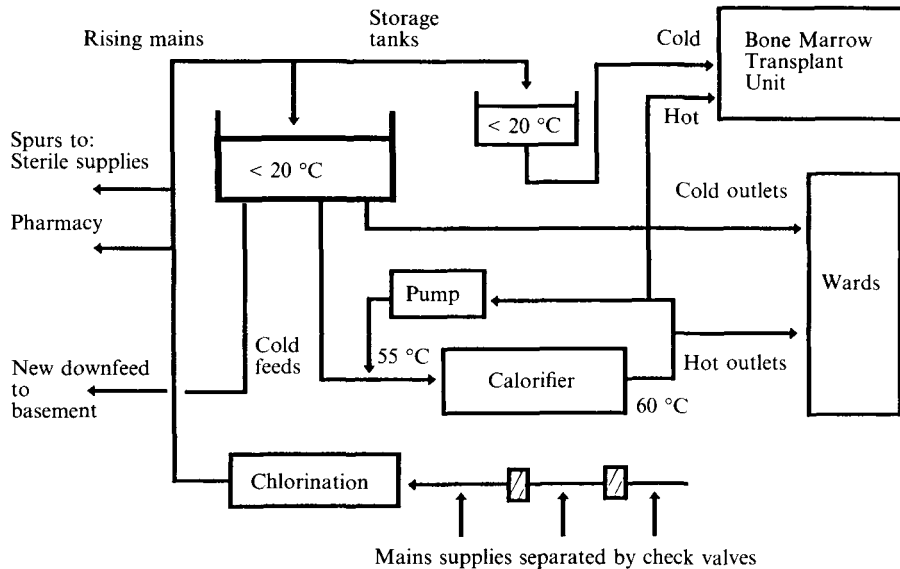


Fig. 3. Old Building, Glasgow Royal Infirmary revised water storage and distribution system.

Table 2. Results of water sampling before fire hydrant spurs were removed and mains water chlorinated\*

Samples		Positive cultures†		
		LP1	LA	Other
Street mains	9	0	0	
Street mains deadleg	3	0	0	LF
Water-meters	3	0	0	
Fire hydrant spurs	7	7	3	LS
Other mains spurs	6	6	5	
Rising mains	3	3	0	
Storage tanks	15	4	0	
Calorifiers	3	1	1	
Cold water at outlet	7	6	2	
Hot water at outlet	5	4	2	

\* Samples taken 26 June 1990 to 7 September 1990.

LP1, *L. pneumophila* sg1; LA, *L. anisa*; LS, *L. steigerwaltii*; LF, *L. feilii*.

Downfeeds from the storage tanks supplied cold water to wards (including the Bone Marrow Transplant Unit) and to steam-heated domestic hot water calorifiers in the sub-basement.

During initial sampling the causal organism, *L. pneumophila* sg1 (subtype Pontiac) was recovered from roof storage tanks and the hot and cold water distribution systems (Table 2). When cleaning and chlorination failed to eliminate contamination of the storage tanks, the supply to the tanks was tested and found to be contaminated with *L. pneumophila* sg1 (subtype Pontiac). The main source of this contamination was eventually traced to the network of spurs serving the fire hydrants from which were isolated *L. pneumophila* sg1 subtype Pontiac. *L.*

Table 3. Water supply to storage tanks: concentration of *L. pneumophila* sg1 per litre after fire hydrant spurs were removed and pre-chlorination\*

Date	Samples	Positive cultures LP1†	c.f.u./l‡
21 September 1990	8	4	> 20000 > 20000 2500 5000
2 October 1990	3	2	800 100
3 October 1990	3	2	100 100
5 October 1990	3	2	100 200
10 October 1990	4	1	100

\* Fire hydrant spurs removed 16 September 1990–19 September 1990 and chlorination commenced 11 October 1990.

† LP1, *L. pneumophila* sg1.

‡ c.f.u./l. Colony forming units per litre.

Table 4. Results of water sampling during continuous chlorination of water supply\*

	Samples	Positive cultures†		
		LP1	LA	Other
Rising mains	5	0	0	
Storage tanks	21	0	0	
Mains spurs	19	0	0	
Calorifiers	9	0	0	LS6
Hot water at outlet	12	0	0	
Cold water at outlet	16	0	1	
Bone Marrow Transplant Unit‡				
Cold water	4	0	0	
Hot water	1	0	0	

\* Chlorination commenced 11 October 1990. Samples taken 12 October 1990–1 July 1991.

† LP1, *L. pneumophila* sg1; LS6, *L. pneumophila* sg6; LA, *L. anisa*.

‡ Water outlets in 'Sterile Room' fitted with Ultra Violet Disinfection Units.

*anisa* and *L. steigerwaltii*, all in high counts (20000 c.f.u./l). Mains water entering the hospital was free from legionellae but become contaminated after the first fire hydrant spur. Seventeen fire hydrant spurs were subsequently removed. This reduced the concentration of *L. pneumophila* sg1 in the water supplied to storage tanks from  $2 \times 10^4$  c.f.u./l to 100 c.f.u./l (Table 3).

#### Control measures

As a control procedure the hospital mains water supply was chlorinated continuously while major modifications to the water system, designed to prevent the supply of contaminated water to storage tanks, improve temperature regulation and reduce stagnation, were undertaken (Fig. 3). Mains spur pipes, excluding those supplying pharmacy and central sterile supplies which are the

Table 5. Results of water sampling in surgical block following engineering changes and when chlorination was stopped for evaluation\*

	Samples	Positive cultures†		
		LP1	LA	Other
Water-meters	1	0	0	
Storage tanks	16	1	1	
Mains spurs	2	1	0	
Calorifiers	7	0	0	
Hot water at outlet	8	0	0	
Cold water at outlet	31	1	10	LS6

\* Chlorination stopped 17 July 1991. Samples taken 22 July 1991-9 September 1991.  
 LP1, *L. pneumophila* sg1; LS6, *L. pneumophila* sg6; LA, *L. anisa*.

Table 6. Results of water sampling after chlorination was recommenced in surgical block\*

	Samples	Positive cultures†	
		LP1	LA
Water-meters	3	0	0
Storage tanks	3	0	0
Cold water at outlet	6	0	2
Bone Marrow Transplant Unit‡			
Hot water	4	0	1
Cold water	9	0	2

\* Chlorination recommenced 10 September 1991. Samples taken 11 September 1991-9 October 1991.

† LP1, *L. pneumophila* sg1; LA, *L. anisa*.

‡ Water outlets in 'Sterile Room' fitted with Ultra Violet Disinfection Units.

subject of water regulation [11], were replaced by storage tank downfeeds. All redundant (dead-leg) piping discovered during alterations was removed and the water storage capacity was reduced. The hot water system was changed from gravity to pumped feed.

During the modifications, *L. pneumophila* sg1 was not isolated from the water system (Table 4). However, when continuous chlorination was stopped in the surgical wing to evaluate the effectiveness of the control procedures, *L. pneumophila* sg1 was cultured from samples taken within five days (Table 5). In the interest of patient safety, chlorination was recommenced. *L. pneumophila* sg1 was again suppressed but *L. anisa* continued to be cultured on occasions from chlorinated water. This organism was also cultured from water samples taken from the 'sterile room' of the Bone Marrow Transplant Unit whose outlets were fitted with UV Disinfection Units (Table 6).

#### Test-rig experiments

Turbulence in the spur pipe when the mains water velocity was increased is illustrated in Fig. 4. This ranged from 0.1 m (velocity 0.2 m/s) to 0.475 m (velocity 1 m/s). However, when water temperature in the mains circuit was raised, the rate and depth of water exchange increased sharply [10] (Fig. 5). Within 5 min. two



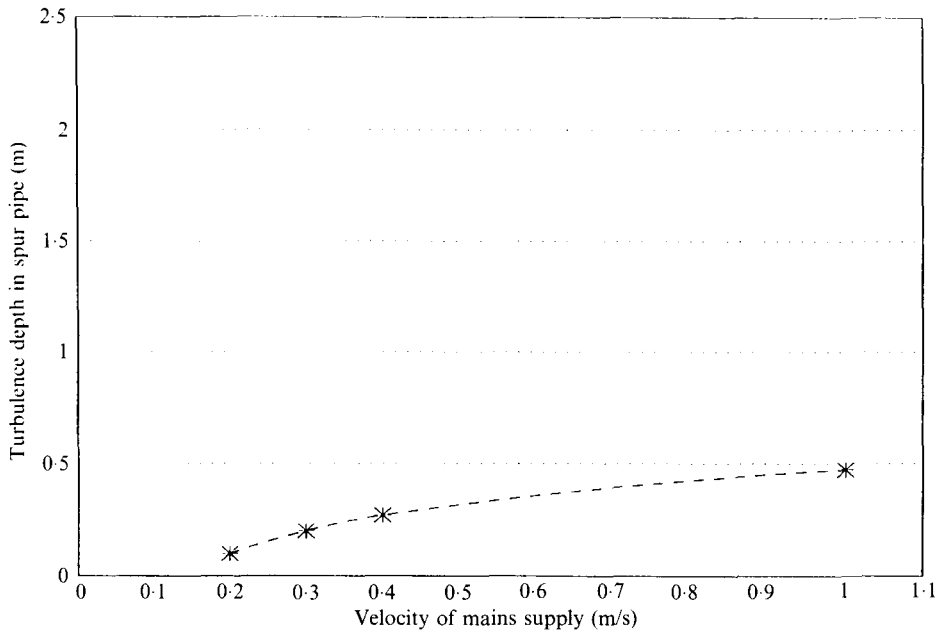


Fig. 4. Test rig experiment. Water exchange between spur pipe and mains supply of equal temperature caused by turbulent mixing.

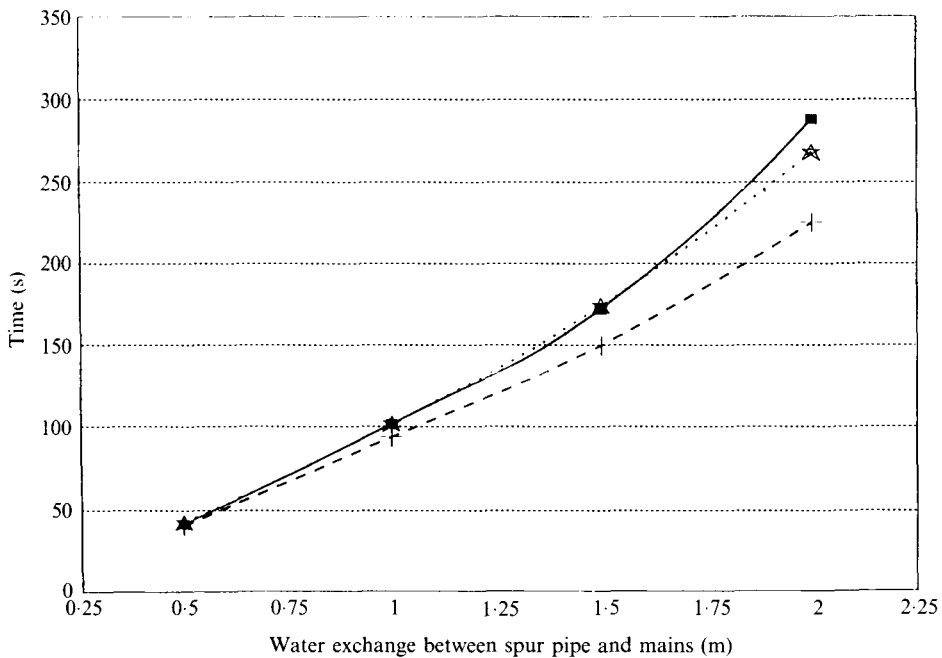


Fig. 5. Test rig experiment. Buoyancy driven water exchange between spur pipe and mains supply with temperature differential of 7.5 °C. Mains water velocity: +, 0.2 m/s; ☆, 0.3 m/s. ■, 1 m/s.

metres of water in the spur pipe had exchanged with the mains supply. Increasing water velocity in the mains circuit had little effect on this rate of exchange.

#### DISCUSSION

There are important lessons to be learned from the investigation of this outbreak. Legionnaires' disease is a severe pneumonic illness which may clinically be indistinguishable from other pneumonias. Cases may also be separated both in space and in time. During this outbreak, the first patient to contract LD was initially believed to have community-acquired pneumonia. The second and third patients contracted LD 12 and 18 weeks later in different clinical locations. All were immunocompromised, supporting the view that such patients with hospital-acquired pneumonia should be aggressively investigated for Legionella infection [12].

Legionnaires' disease, and non-pneumonic legionellosis (Pontiac fever [13]), are thought to be caused by the inhalation of aerosols containing legionellae [14]. Sources of infection include cooling towers [15–17], air conditioning [18] and water distribution systems [19, 20]. Person to person spread of infection has not been described.

The Old Building of Glasgow Royal Infirmary was not air conditioned and none of the three cases received inhalation therapy. However, the domestic water distribution system (hot and cold) was contaminated with *L. pneumophila* sg1 subtype Pontiac, though to be similar to the strain causing fatal infection. It is reasonable to conclude that all three patients were infected from this source.

The first patient enjoyed frequent showers and this may have been the vehicle of infection. Showering is important in the clinical care of patients, especially those undergoing surgical procedures. Deciding if, where, and on what basis to suspend it in the control of LD can be very difficult. During this outbreak, showering throughout the hospital was prohibited upon recognition of a second case. However, this failed to control the outbreak and 168 of 377 planned elective surgical admissions had to be suspended. Study of the case histories of the second and third patients indicated that both were probably infected by splashing associated with washing in baths and wash basins [21].

It is recognized that legionellae are common inhabitants of aquatic habitats [1, 22–24]. Their ecology in domestic water systems is complex and it has now been suggested that they should be regarded as normal commensal flora [25]. *L. pneumophila* sg1 is the strain most commonly associated with hospital-acquired infection [26]. Fortunately most hospitals whose domestic systems are contaminated have not reported cases of disease [27]. During this outbreak, three additional strains of legionellae, *L. pneumophila* sg6, *L. anisa*, and *L. steigerwaltii* were also isolated. Our experience indicates that suppressing and eradicating their growth in water which is to remain drinkable is very difficult.

Removal of the network of fire hydrant spurs considerably improved the quality of water supplied to storage tanks. However, it did not eliminate legionellae from the supply. Additional control measures including chlorination of the incoming mains supply and modifications to the distribution system also failed

to eradicate them. Similar difficulties in suppressing legionellae have been reported from other centres. In Nottingham, improved water temperature management failed to prevent two further cases of LD [28] and at Wadsworth Medical Centre, Los Angeles two further cases of LD occurred within a week of stopping continuous chlorination [29].

It is not currently appreciated that a cold water mains supply, free of pathogenic micro-organisms on entry to a hospital building, may become heavily contaminated en route to storage tanks or other points of use. The mechanism behind this contamination is now clear. We have evaluated a test rig of perspex piping and found that significant temperature-dependent buoyancy-driven water exchange occurs between a stagnant spur pipe (or dead leg) and its mains supply. The velocity of water in the mains pipe is relatively unimportant. The most significant factor is the difference in temperature between the spur pipe and the mains.

By the same premise, spur pipes taken from a tank supply may present a hazard if allowed to stagnate and become warmer than its downfeed. For example, spur pipes supplying out-patients clinics (which are unused during weekends and holiday periods), or spur pipes to vacant wards, may contaminate water outlets, hot or cold, downstream. At present, empty hospital wards should be subject to weekly flushing in accordance with the code of practice for Health Care Buildings [3]. However, flushing may not be carried out and a weekly basis may be insufficient. For this reason we recommend that water in empty hospital wards should either be run to waste on a daily basis, or the spur pipes supplying the area should be disconnected and drained.

There are many difficulties in the prevention of nosocomial Legionnaires' disease. However, a minimum standard should be to ensure that hospital domestic water storage tanks are supplied with water free of legionellae. We therefore recommend that hospital storage tanks are supplied via dedicated mains pipes without spurs. This is especially true for those hospitals treating immunosuppressed patients. Areas of the hospital presently supplied by mains spurs should be re-engineered from a tank supply, observing the well-established guidelines for preventing the growth of legionellae [3]. Mains spur pipes which cannot be replaced should be regarded as a hazard, especially if subject to any stagnation or temperature change.

In summary it has been found that mains spur pipes, not previously regarded as a hazard in the control of legionellae, may be responsible for sporadic outbreaks of disease. The warm ambient temperatures in health care premises, necessary for the care of patients, presents a challenge in the design of safe hot and cold water systems. Further work is required to prevent buoyancy-driven water exchange and the contamination of hospital water systems from this source.

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## REFERENCES

1. Public Health Laboratory Service Collaborative Study of Legionella Species in Water Systems. HAC 1985.
2. Muraca P, Stout JE, Yu VL. Comparative assessment of chlorine, heat, ozone, and UV light for killing *Legionella pneumophila*. *Appl Environ Microbiol* 1987; **53**: 447–53.
3. The control of legionellae in health care premises. A code of practice. London: HMSO, 1988.
4. The hospital infection manual. Including a code of practice for the Scottish health service. Scottish Home and Health Department, 1988.
5. Surveillance definitions for Legionnaires' disease. Respiratory Diseases Section, Communicable Diseases Surveillance Centre, September 1992.
6. Kohler RB, Winn WC, Wheat LJ. Onset and duration of urinary antigen excretion in Legionnaires' disease. *J Clin Microbiol* 1984; **20**: 605–7.
7. Mitchell K. Rapid diagnosis of legionella infections and identification of *Legionella* species isolated from man and the environment [PhD thesis]. Glasgow University, 1991.
8. Edelstein PH. Improved semi-selected medium for isolation of *Legionella pneumophila* from contaminated clinical and environmental specimens. *J Clin Microbiol* 1981; **14**: 298–303.
9. Watkins ID, Tobin JO'H, Dennis PJ, Brawn W, Newnham R, Kurtz JB. *L. pneumophila* sg1 subgrouping by monoclonal antibodies: an epidemiological tool. *J Hyg* 1985; **95**: 211–16.
10. The building services research and information association. Investigation of the extent of water exchange between pipe and spur. Report 66850/1, 1991.
11. DHSS and Welsh Office. Accommodation for pharmaceutical services. Health Building Note 1988; No 29: Para 8.44.
12. Goetz A, Victor L, Yu MD. Screening for nosocomial legionellosis by cultures of the water supply and targeting of high-risk patients for specialized laboratory testing. *Am J Infect Control* 1991; **19**: 63–6.
13. Glick TH, Gregg MB, Berman B, Mallison G, Rhodes WW, Kassanoff I. Pontiac fever. An epidemic of unknown aetiology in a health department: clinical and epidemiological aspects. *Am J Epidemiol* 1978; **107**: 149–60.
14. Baskerville A, Fitzgeorge RB, Broster M, Hambleton P, Dennis PJ. Experimental transmission of Legionnaires' disease by exposure to aerosols of *Legionella pneumophila*. *Lancet* 1981; ii: 1389–90.
15. O'Mahony MC, Stanwell-Smith RE, Tillett HE, et al. The Stafford outbreak of Legionnaires' disease. *Epidemiol Infect* 1990; **104**: 361–80.
16. Outbreak of legionellosis in a community. Report of an Ad-Hoc committee. *Lancet* 1986; ii: 380–3.
17. Bhopal RS, Barr G. Maintenance of cooling towers following two outbreaks of Legionnaires' disease in a city. *Epidemiol Infect* 1990; **104**: 29–38.
18. Timbury MC, Donaldson FR, McCartney AC, et al. Outbreak of Legionnaires' disease in Glasgow Royal Infirmary: microbiological aspects. *J Hyg* 1986; **97**: 393–403.
19. Tobin JO'H, Bartlett CLR, Waitkins SA, et al. Legionnaires' disease: further evidence to implicate water storage and distribution systems as sources. *BMJ* 1981; **282**: 573.
20. Best M, Yu VL, Stout J, Goetz A, Muder RR, Taylor F. Legionellaceae in the hospital water supply. Epidemiological link with disease and evaluation of a method for control of nosocomial Legionnaires' disease and Pittsburgh pneumonia. *Lancet* 1983; ii: 307–10.
21. Woo AH, Yu VL, Goetz A. Potential in-hospital modes of transmission of *Legionella pneumophila*. *Am J Med* 1986; **80**: 567–83.
22. Harf C, Monteil H. Pathogenic microorganisms in environmental waters: a potential risk for human health. *Water Internat* 1989; **14**: 75–9.
23. Arnow PM, Weil K, Para MF. Prevalence and significance of *Legionella pneumophila* contamination of residential hot-tap water systems. *J Infect Dis* 1985; **152**: 145–51.

24. Stout JE, Yu VL, Yee YC, Vaccarello S, Diven W, Lee TC. *Legionella pneumophila* in residential water supplies: environmental surveillance with clinical assessment for Legionnaires' disease. *Epidemiol Infect* 1992; **109**: 49–57.
25. Stout JE, Yu VL, Best MG. Ecology of *Legionella pneumophila* within water distribution systems. *Appl Environ Microbiol* 1985; **49**: 221–8.
26. Legionnaires' disease surveillance: England and Wales 1991. *Communicable Disease Report* 1992; **2**: No 11.
27. Tobin JO'H, Swann RA, Bartlett CLR. Isolation of *Legionella pneumophila* from water systems: methods and preliminary results. *BMJ* 1981; **282**: 515–17.
28. Colville A, Crowley J, Dearden D, Slack RCB, Lee JV. Outbreak of Legionnaires' disease at University Hospital Nottingham. *Epidemiology, microbiology and control. Epidemiol Infect* 1993; **110**: 105–16.
29. Edelstein PH. Control of legionella in hospitals. *J Hosp Infect* 1986; **8**: 109–15.