Journal of Hospital Infection (1999) **43**: 85–100

REVIEW

Mopping up hospital infection

S. J. Dancer

Department of Microbiology, Vale of Leven District General Hospital, Alexandria, Dunbartonshire, G83 OUA

Summary: Hospital cleaning is a neglected component of infection control. In the UK, financial constraints have forced managers to re-evaluate domestic services and general cleaning has been reduced to the bare minimum. Services have been contracted out in some hospitals, which has further lowered standards of hygiene. Control of infection personnel believe that cleaning is important in preventing hospital-acquired infections but they do not manage domestic budgets and have failed to stop their erosion. It is difficult to defend high levels of hygiene when there is little scientific evidence to support cleaning practices. This review examines the common micro-organisms associated with hospital-acquired infection and their ability to survive in the hospital environment. It also describes studies which suggest that comprehensive cleaning disrupts the chain of infection between these organisms and patients. It is likely that restoring hygienic standards in hospitals would be a cost-effective method of controlling hospital-acquired infection. Furthermore, good cleaning is achievable whereas the enforcement of hand washing and good antibiotic prescribing are not.

Keywords: Cleaning; domestic services; hospital-acquired infection.

Introduction

Cleaning staff blend into the background on a busy ward. Although they go about their duties generally unnoticed, their work is hardly unimportant. Keeping the hospital clean is a heavy responsibility. It is, in fact, likely to be a critical factor in infection control and in the continuing fight against hospital-acquired infections.

Hospitals provide a reservoir of micro-organisms, many of which are multiply-resistant to antibiotics. Patients and staff supply some of these organisms, but the hospital environment forms a substantial repository for others – if allowed. Thorough and conscientious cleaning significantly disturbs the ecological niches found by potentially pathogenic organisms. If there were no domestic staff, it seems likely that there would be far more cases of hospital-acquired infection. Apart from reducing reservoirs of micro-organisms, cleaning also has an aesthetic purpose.[1–4] The apparent level of cleanliness is important for patient confidence. Patients and their visitors make comments about hospitals they consider dirty and will associate this with a general lack of care.[1] Thus the environment should not only be microbiologically safe, it should be clean enough not to cause concern to patients.

Aesthetic and infection control considerations should provide enough impetus to support a comprehensive cleaning programme for all hospitals. This is not the case at present, because there appears to be an overall deterioration in

Correspondence to: S. J. Dancer, Department of Microbiology, Vale of Leven District General Hospital, Alexandria, Dunbartonshire, G83 OUA

0195–6701/99/100085 + 16 $12.00

hygiene in our hospitals.[1,4,5] Why is this? It could be hypothesized that current cost-cutting in the British National Health Service (NHS) has targeted cleaning services because a reduction in the domestic budget does not directly affect the patient waiting lists. Cleaning, by its very nature, is labour-intensive and staffing costs are the greatest single cost to the NHS. When financial savings are required, labour-intensive services are inevitably going to be scrutinized for potential savings.[1]

Contracting out hospital cleaning services has further contributed toward falling standards.[5,6] There is no one to champion the cleaner's cause except for infection control personnel. Hospital cleaning is hardly glamorous.[1] Infection control staff themselves are in short supply and sometimes not even based on-site.[7] They rarely have enough power to stop managers tinkering with the domestic budget and the whole issue is compounded by the fact that hospital infection control lacks a scientific basis.[1,8] There is little convincing evidence that the work of infection control practitioners has any effect on the incidence of hospital-acquired infections. Forced to economize, therefore, managers will not favour an extensive service for cleaning policies when there is a perceived low risk to patients from dirty hospitals.[3,9–11]

Is there any evidence with which to confront the business-minded? Florence Nightingale can be credited with one of the first accounts describing the effect of hospital cleaning on patient outcome. Taking on senior doctors and the War Office, she reduced the death rate from cholera, typhus and dysentery from 42% to only 2% in a Crimean hospital in the 1850s.[12] This she did by organizing a laundry service, cleaning equipment, structural repair of wards and hospital cleaning. She also imposed her own views about general and personal hygiene on her team of nurses. Her writings regarding such issues are as relevant today as they were over a century ago.[13] She fully realized (unlike many doctors at the time) that dirt could be fatal to health. With this in mind, she had the floors of the hospital scrubbed, '… for the first time anyone could remember'.[12]

Now, of course, it is known that floors provide a huge reservoir of bacteria which can be cultured, identified and assessed for pathogenic risk.[9] Florence Nightingale did not have the benefit of current knowledge regarding microbiology and control of infection. Indeed, at that time, the significance of micro-organisms was still unconfirmed and certainly no one had postulated a link between microscopic creatures and macroscopic infections. She utilized experience and common sense and a certain forcefulness of character. One wonders how she would have fared with today's Chief Executives? Sadly, one hundred and fifty years later, there is still little evidence regarding the link between cleaning and hospital-acquired infections. What impact does thorough cleaning actually have on infection control? If we are to examine this, we should first consider the organisms associated with hospital-acquired infection and their epidemiology.

Hospital-acquired micro-organisms

Many of the organisms associated with hospital-acquired infections display two particular features: firstly, they are pathogens of well-established medical importance and secondly, they can withstand the rigours of the hospital environment. It benefits them to survive outside temperate human tissues because an appropriate environmental niche will provide shelter until some timely mechanism facilitates their transfer back to patients. Not all of them demonstrate this capacity; some originate from the patients' own flora, especially those who are immunocompromized and others can survive only in human tissues and thus rely upon person-to-person spread in order to disseminate. In an article about generalized hospital cleaning, therefore, only the common pathogens of established environmental longevity will be considered.

Micro-organisms have a choice regarding their environmental habitats. These can be discrete, such as a piece of equipment, or general, such as the hospital floor. Some organisms may

reside on both. In an outbreak situation, it is always advisable to seek a discrete source because identification of such a reservoir is more likely to allow eradication of an organism and bring the outbreak to a prompt conclusion. Then, once a point source is established, local policies can be fashioned or adapted to reduce the risk of further outbreaks.

More general reservoirs are not so easily managed. It may be difficult to know how to target a general or widespread source of an 'outbreak' organism. Complex epidemiology (staff carriage, cross-infection, etc.) may further hamper control efforts. Such outbreaks are at risk of becoming endemic. Generalized cleaning is rarely seen as a primary solution at the present time.

Attributing individual cases of hospital-acquired infection to general deficiencies in routine cleaning is also rare, due to the difficulties in providing sufficient evidence. Whilst the specialized typing of organisms may suggest an outbreak source, such investigations are of little benefit for sporadic cases over long periods of time and are costly and time-consuming. It is therefore not surprising that hospital cleaning lacks a scientific basis.

What are the common organisms associated with hospital-acquired infections and where are they found? One of the most robust is *Clostridium difficile*, the anaerobic Gram-positive rod linked with antibiotic-associated colitis. This spore former can withstand temperature extremes, drying and the majority of hospital disinfectants.[14–16] It is found in the gastrointestinal tract of both symptomatic and asymptomatic patients, but the major vehicle of spread is patients with diarrhoea.[17–18] Sampling bed-clothes, commode and the floor under the bed of a patient with antibiotic-associated colitis often reveals heavy contamination with *C. difficile*.[18,19] The organism can also be isolated from sphygmomanometers, hands and underneath finger-nails.[17,20] Spores will survive for months if left undisturbed.[18,21] Visitors and staff consequently carry the spores away from the patient on hands, shoes and clothes and eventually the entire ward may become a reservoir.[9,17,21] Both outbreaks and sporadic cases spring from such fertile ground, especially in conjunction with heavy antibiotic prescribing.[17,22] In a recent outbreak attributed to *C. difficile*, twenty-four patients were affected over four months; the first cases identified in early November.[23] Throughout the outbreak there was rigorous attention to environmental cleaning, staff movement, hand washing and antibiotic usage – all factors known to be associated with *C. difficile*. Despite these infection control measures and two complete ward closures, the outbreak continued. It was eventually noticed that the radiators on the affected ward were surrounded by a 'cage' which was fixed to the wall so that removal was difficult. Cleaning behind the cages was not routine and thick dust and dried faecal material were found behind them. Sampling this material revealed *C. difficile*. The authors noted that the outbreak began when the radiators were turned on and suggested that thermal convection from the radiators may have played a part in disseminating spores to susceptible patients.[23]

Environmental contamination by *C. difficile* is now well accepted as a risk factor for the acquisition of *C. difficile*.[16–19,21,24] It is likely that the frequency of environmental spread, together with prolonged viability of spores, explain the difficulty in eliminating *C. difficile* once it has become established in hospitals.[25] Vacuuming up dust from floors is probably one of the best and simplest methods to remove spores from the environment.[25,26] Buffing and mopping may only serve to spread them around an even wider area.[27] Most authors lament the lack of a cheap non-toxic effective sporicide but reality in simple mechanical removal is all that is required.[25,27] Too much reliance is placed on miracle 'kill-all' disinfectants.[27] The cleaning of commodes used by infected patients, in particular, is a responsibility that should not be delegated to an uninformed member of staff.[1,19,25] The value of generalized cleaning is demonstrated by a seven-year study examining the incidence of hospital-acquired infections in an American hospital. There was a sustained decrease in nosocomial *C. difficile*, when

cleaning was included as a major part of an aggressive infection control programme.[28]

Bacillus is another spore-forming rod. This ubiquitous organism clouds the atmosphere and carpets the floors of homes and hospitals. Spores can persist in soil for years and are remarkably resistant to high temperatures.[29] The presence of *Bacillus* on agar plates in the laboratory is often dismissed as contamination, yet there are many species linked with significant human infections.[30] *B.cereus* is particularly associated with food poisoning, where precipitous vomiting occurs just hours after a take-away meal.[31] Outbreaks of non-gastrointestinal *B.cereus* infections have been recorded in maternity, surgical and intensive care units.[32–35] The main vehicles for transmission were contaminated nappies or linen, although patients in hospitals undergoing building work appear to be at increased risk of infection.[35] Last summer, extensive building work at one end of the maternity unit in this hospital resulted in 12 cases of *B.cereus* infection in neonates (Dancer SJ: unpublished observations). The hospital management had refused to move the ward elsewhere because it was deemed too expensive to transfer the surveillance cameras necessary for security purposes. The ward was noticeably dirty throughout the work. Conclusion of the building work brought a halt to the outbreak.

Investigation into a large outbreak in a London teaching hospital highlighted environmental contamination originating from a building site.[35] It was also found that the cleaning in the hospital laundry had been abolished in deference to financial constraints. Serological typing confirmed a link between building site, laundry and patients. It was concluded that the building works were responsible for increased numbers of *Bacillus* spores around the hospital, but the organism concentrated in the laundry from a reservoir on the floor. It was noted that clean linen frequently touched the floor whilst undergoing steam-pressing. At least forty-five cases were identified with two deaths.

Gram-negative bacteria associated with hospital-acquired infections do not form spores and are generally not quite so well accomplished at environmental longevity. Given the right conditions, however, they can attract infection control interest by virtue of isolation from compromized patients and occasional outbreaks. Although they often originate from patients themselves, they are adept at finding an appropriate environmental niche if allowed. They prefer equipment and areas which are damp and inadequately cleaned, disinfected or sterilized.[27,36] Florence Nightingale herself stated that, 'Dry dirt is comparatively safe, but (that) wet dirt becomes dangerous'.[13] Numerous reports describe infections from genera such as *Klebsiella, Enterobacter, Escherichia, Serratia* and *Pseudomonas* spp.. Their environmental sources are many and varied. *Klebsiella* and *Escherichia*, for example, are frequently found on mops and in buckets.[37,38]; *Serratia* has been described on a theatre shaving brush[39]; *Pseudomonas* can contaminate sink surrounds.[40,41] The latter organism can also colonize basins and respiratory equipment.[42,43] *Enterobacter* prefers parenteral feeds.[44] These organisms do not survive if equipment and surfaces are kept scrupulously clean and dry, although *Klebsiella*, in particular, is known to survive well on surfaces.[13,27,45,46] Once a reservoir is established, the organisms may be transferred to patients directly or indirectly, generally via hands.[11,47,48] *Acinetobacter* is another Gram-negative rod implicated in an increasing number of hospital-acquired infections and outbreaks.[49] Members of this genus appear to have a significant capacity for long-term survival on dry surfaces or dust particles compared to the Gram-negative bacteria already mentioned.[50] One species, *A. radioresistens*, exhibits a phenomenal ability to survive on dry surfaces as shown by a mean survival time of 157 days on glass coverslips.[51] It is likely that the persistence of these bacteria in the hospital environment increases the opportunity for transmission, colonization and ultimately infection.[50,51]

The Gram-negative organisms often generate infection control interest because of their propensity for multiple antibiotic resistance. Hospitals tend to select for resistant bacteria following heavy use of antibiotics.[52,53] Such

organisms become the dominant colonizing flora of patients as well as nosocomial pathogens in those who are more vulnerable.[53] One postulated mechanism is that an antibiotic resistant flora is endemic within the institution and is transferred to the patient during the course of hospitalization.[54] The constitution of that endemic reservoir, i.e., other patients, staff, environment, etc. and the importance of each of these in infection control, remains unclear.

Environmental sources are more likely to exist for non-Enterobacteriaceae (*Acinetobacter, Pseudomonas*) because they are not cultured from hypopharynx or rectum before their appearance in tracheal cultures.[55] This is in contrast to Enterobacteriaceae, which are.[55] This suggests that generalized cleaning could be useful in controlling *Pseudomonas* and *Acinetobacter*, although some of the environmental habitats for Enterobacteriaceae already mentioned could also fall within this remit.[36–38,40,41] Several authors stress the importance of meticulous environmental cleaning in controlling *Acinetobacter*.[56,57] One report describes an outbreak of *A. baumanii*, which was controlled by isolation, handwashing and cleaning after patients' rooms were found to be widely contaminated by the outbreak organism.[56] If an organism can find an appropriate niche in which to survive, it will do so, whatever the origin; careful and conscientious cleaning provides the opportunity to eliminate such niches, thus disrupting the chain of transmission. In contrast, therapy for patients infected with multiply-resistant Gram-negative bacteria is difficult, toxic and expensive.[58]

Viruses, too, can be included in the list of resilient hospital-acquired micro-organisms. Small round structured virus, or Norwalk agent, causes winter vomiting disease, a type of gastro-enteritis.[59] This condition generally affects older patients although staff and visitors of any age may be involved during an outbreak. The virus really does appear to fly through the air. If one person vomits in the middle of the ward, patients nearby are likely to exhibit symptoms within 48 h and so on, until the whole ward is affected.[60] It is presumed that infective droplets are projected into the air, probably during vomiting and remain suspended until they eventually fall to the ground.[59] Alternatively, they may be inhaled or ingested by others either directly from the air or indirectly from a contaminated environment. Survival is long enough to infect persons entering the ward hours after an index case displayed symptoms.[61,62] Thorough and vigorous cleaning with double strength chlorine-based detergents is advised to eradicate the environmental reservoir, especially toilets; cleaning is also required for curtains, carpets and other soft furnishings.[59,63,64] Most authors agree that thorough environmental cleaning is essential to contain an outbreak.[59,63,64] Patients themselves should be isolated or cohorted if the outbreak is to be confined to one ward or unit. Staff movements must also be restricted, as asymptomatic carriers can transmit the virus to other staff and patients elsewhere.[59] It is not unusual for this virus to close entire units during the winter and early spring – precisely the time when hospital admissions tend to be high anyway.

Another organism adept at closing wards is Methicillin-resistant *Staphylococcus aureus* (MRSA). MRSA has penetrated virtually every hospital in the country. A chronic endemic state remains in most, punctuated by episodes of cross-infection and an occasional flurry of outbreaks.[65] Although people are known to carry staphylococci and subsequently transmit them to others, there are plenty of reports citing environmental involvement. MRSA has been isolated from the knobs of television sets,[66] cushions,[67] computer keyboards[68] and even consultants' pens.[69] The staphylococcus can withstand desiccation and is thus a frequent component of hospital dust.[70] *S. aureus* is present on bedding, curtains and nurses uniforms.[71,72] It can survive for at least 175 days under dessicating conditions.[73] It can also be carried on doctors stethoscopes, clothing and hands.[74] Doctors are not renowned for washing their hands and many believe that their white coats and hands are bacteriorologically sterile.[75–77]

Some outbreak investigations have revealed a defined source of MRSA within the ward, e.g., ventilation grilles[78]; others have implicated

airborne spread, especially in burns and intensive care units.[79,80] Occasionally, members of staff or patients themselves are identified as initiating an outbreak.[81] Persistent carriers can recolonize themselves from a contaminated home environment, following apparently successful attempts at clearance.[82,83] In the event of a serious outbreak, the infection control team would be wise to consider the identification of carriers as well as attention to the ward environment, staff hand-hygiene and appropriate management of patients themselves. Complex staphylococcal epidemiology makes it mandatory to practise the whole package of infection control strategies, otherwise the outbreak tends to grumble on.[84] A recent outbreak at this hospital was almost certainly prolonged because ward cleaning did not include washing the curtains around patients' beds (Dancer SJ: unpublished observations).[71] Judging from the relentless rise in MRSA infections over the last few years, epidemic strains are spreading successfully throughout hospitals and community despite best efforts to contain them.[65,85]

Another organism set to devastate hospitals is vancomycin-resistant enterococcus (VRE). This appears to outclass the staphylococcus on several counts; it is more resistant to disinfectants and antibiotics, it survives longer in the hospital environment and there are no effective regimens available with which to clear human carriage.[86–88] It is also successfully disseminated into the environment by carriers.[89] It resides in both human and animal gastrointestinal tracts and once colonized, persists in its host long-term.[90] The origin remains a mystery. Europe has specified the use of the veterinary glycopeptide avoparcin, but this agent is little used in the USA, where there are also considerable problems with VRE.[91] Americans cite the increasing use of vancomycin and teicoplanin against *C. difficile* and MRSA as the main factors for VRE selection.[92] Certainly, there is now evidence to show that the veterinary use of antimicrobials in animals promotes antibiotic resistance in micro-organisms and that these organisms can be transferred to humans.[93] Thus, it is possible that ingesting VRE colonized chicken leads to gut carriage in the community; once admitted to hospital, such patients are then at risk of developing VRE sepsis, usually after receiving broad spectrum antibiotics. The organism is, however, less pathogenic than *S. aureus*, although when infections do occur, they are extremely difficult to treat.[90,94] VRE is also very difficult to eradicate from the environment.[89,95] A recent article describes the persistence of the organism following a blocked toilet and subsequent flooding of two isolation rooms.[96] The toilet was used by a patient who was actively isolated for VRE carriage. The toilet flooded after it was blocked with paper towels and water penetrated an adjacent side room. Both rooms were aggressively and thoroughly cleaned; this included washing down to clear the water and faeces and then disinfection with phenolic followed by bleach. The bleach treatment was repeated two days later when environmental cultures of the second room demonstrated persistence of VRE on the cleaned floor. Cultures taken after the repeated cleaning did not detect VRE in either of the rooms. Twelve days after the accident, another patient was admitted into the second room and subsequently became colonized with the organism. Molecular typing showed that the environmental strains, the strain from the first patient and the strain from the second patient were indistinguishable. There are other reports of the environmental persistence of VRE and published strategies on eradicating the organism.[88,95] As there is no effective protocol for clearing human gastrointestinal carriage, it is worth promoting tougher action on reducing environmental contamination by this organism. Despite its lesser pathogenicity, immunocompromized patients are at risk of untreatable infection and there is always the possibility that uncontrolled spread of VRE potentiates the transfer of multiple-antibiotic resistance genes to other organisms.[97] Glycopeptide-tolerant strains of *S. aureus* (GISA) have already been isolated in several countries, although there is no evidence that resistant enterococci have contributed towards the appearance of these strains.[98,99]

There are other organisms which are associated with hospital-acquired infections and are

capable of surviving in the hospital environment, e.g. *Legionella* spp. *Aspergillus* spp. and hepatitis B.[100–103] General cleaning could potentially play a role in controlling these, but their more specialized epidemiology make it inappropriate to include them in this article. Hospital kitchens and their cleaning also falls outside the scope of this article.[45,102] A mention should be made, however, of rather larger 'pests' which have implications for hospital infection. These are insects, birds and small mammals. There is a vicious circle involving litter, pests and a low standard of cleaning.[27] The presence of litter makes thorough cleaning impossible and provides hiding places for pests. If there is food or organic material such as soiled dressings or linen, then pests will feed on it. Droppings from the pests add to the litter and reduce the level of hygiene even further. The whole makes more work for the cleaners and destroys their incentive.[27] The most common pests include rats and mice, ants, cockroaches, pigeons and flies. All of these carry micro-organisms on their bodies and their droppings are also laden with microbes. Hospital hygiene is dependent upon eradicating such pests and ensuring that they do not return. Domestic cockroaches, in particular, have been shown to harbour a wide variety of pathogens in their hind gut and on their body surface, including enterobacteria, polio and hepatitis viruses and nematodes.[104–106] Salmonellae have been isolated from cockroaches infesting a children's ward where there was an extensive outbreak of gastroenteritis.[107] Ants also carry a variety of organisms.[108] Ants found in sterile packs make a mockery of sterilization methods.[109] Every hospital should have a mechanism in place for pest control and a member of staff responsible for such duties should regularly attend control of infection committee meetings.[27]

Impact of cleaning on infection control

Little is known about the effects of thorough cleaning in today's hospitals. Only scanty evidence supports a link between general domestic activities and hospital-acquired infection, other than outbreaks proven to originate from a contaminated point source – *Klebsiella* from mops, for example.[37] Hospital-acquired infection is itself complicated by staffing issues, airborne transmission, human carriage, person-to-person spread, disinfection and sterilization, hand washing practices and antibiotic policies. Simple environmental cleaning is usually placed at the end of the list and may not even be mentioned in discussions or publications on infection control. This is despite generalized agreement that a clean environment is necessary to provide both good standards of hygiene and maintain patient and staff confidence.[1–4,110]

There are some studies, however, which would appear to support a discernible role for domestic cleaning in the control of infection.[28,56,59,96,101] A letter in the Journal of Hospital Infection describes the environmental contamination of a new general surgical ward with MRSA.[111] Infection control personnel sampled the ward before and after the ward was opened. Within the first week, they found that 20% of sampled sites were positive for *S. aureus* and one of those isolates was MRSA, found on the top of a paper towel holder. The authors postulated that the presence of *S. aureus* in the first week reflected the transfer of dust and organisms on ward equipment and furniture from the old ward. They emphasised the importance of adequate cleaning of equipment, furniture and the ward environment.

A recent outbreak of MRSA in a surgical unit was attributed to sub-optimal cleaning (one cleaner for two hours daily) and deficiencies in the ward fabric. [112] The outbreak, involving fourteen patients, was halted by the institution of a major cleaning programme in all areas of the unit and improvements in the ward fabric. The problem of improving the routine cleaning was also addressed. The authors were quite sure that cleaning deficiencies were an important factor in this outbreak. They felt that the deterioration in the quality of regular cleaning services and the managerial loss of such services to outside organisations were also relevant.[112]

If hospital cleaning achieves low status, domestic activities at home achieve even less. People may or may not clean their homes and for most, this would have no bearing on hospital infection. Persistent carriage of MRSA, however, occasionally directs attention toward the home environment – especially if the carrier is a member of the hospital staff.[82,83] Patients with MRSA do not generally receive such attention on discharge from hospital. In any case, topical clearance of the organism is not regarded as high priority once the patient is in the community.[113] Unfortunately, patients tend to come back into hospital still carrying MRSA.[114,115] Infection Control teams neither have the time, nor indeed the jurisdiction, to attempt MRSA clearance from both body and home outside the hospital. Neither are they likely to offer cleaning advice to the family of a treated MRSA patient on discharge. The inexorable rise of MRSA is a problem in hospitals and the community.[116]

Two other reports highlight the home environment as a source of infection. The first describes an outbreak of *Nocardia farcinica* wound infections in surgical patients originating from a carrier anaesthetist.[117] The outbreak terminated only after extensive cleaning of his home. The second describes the effect of introducing a programme of education and environmental cleaning, including cleaning of toys, into a pre-school nursery for predominantly Down's Syndrome children. Cases of infection decreased significantly following implementation of this programme.[118]

Small round structured viruses ignore the divide between hospital and community. Outbreaks of viral gastroenteritis have been associated with schools, nurseries, camps, cruise liners, hotels and residential and nursing homes as well as hospitals.[63,64,119] An outbreak involving a hotel occurred in spring 1995 and proved difficult to control.[64] The hotel was large and traditionally offered weekend or midweek breaks, catering for up to 500, mainly older, people. The outbreak initially presented with several guests reporting ill, primarily with vomiting, approximately 48 h into their holiday. Control measures were introduced, including employing extra staff to clean the building. The numbers of affected guests continued to rise. During the third week, hotel management agreed to close the establishment for extensive cleaning. This had the desired effect and the outbreak was declared over after four weeks. In total, nearly 300 guests and 36 staff were affected.

Many patients in hospital are vulnerable to hospital-acquired infection, but none more so than burns patients.[79] High standards of environmental hygiene have been described in a number of burns units.[120] For example, a rigidly-controlled environment, including a laminar-flow air curtain, was described in a paediatric burns unit in America.[121] The use of this was associated with a large reduction in the number of bacteria settling on the floors and a subsequent reduction in mortality of children with 50% burns. Sophisticated air-filtering systems may augment the efforts made by cleaning staff, but it is noteworthy that high floor counts of bacteria were linked with the incidence of infection in these patients. Although hospital floors are accepted as a reservoir of potentially pathogenic micro-organisms, most authorities agree that the risk of acquiring infection from these surfaces, including operating theatres, is low for most patients.[3,9,11,45,122]

Other reports emphasise the importance of generalized hospital cleaning. The first describes the effect on sepsis rates of closing and cleaning hospital wards.[123] This was performed in order to control several outbreaks of MRSA in an old hospital with no side-rooms. There was an impressive reduction in staphylococcal infections following closure and cleaning. The authors suggested that besides the obvious benefits of thorough cleaning, the psychological effect of working in a clean ward may have caused nursing techniques to be carried out with increased care. The same authors published a further study two years later describing the reduction in hospital-acquired infections in the same district general hospital.[124] In the attempt to overcome the lack of isolation facilities, separate wards were closed to new admissions, emptied and cleaned. Concurrent activities also

included a disinfectant policy and a more rational use of chemical disinfectants. Substantial decreases in infections with staphylococci, *Streptococcus pyogenes* and *P. aeruginosa* occurred, prompting editorial comment in the Lancet that hospital infection can be controlled.[125] It was not possible to single out generalized cleaning as the sole reason for success, but domestic efforts provided a significant component in these studies.

More recently, a letter to the Lancet describes the effect of cleaning on a ward with a continuing problem with VRE.[126] A substantial increase in the number of newly colonized patients appeared to coincide with a high level of environmental contamination. The only intervention used was a very thorough and systematic cleaning of the ward, after which both the level of environmental contamination and the numbers of new patients with VRE decreased. The authors stated that cleaning of hospitals needs neither highly skilled personnel nor sophisticated equipment and is a cost effective method of infection control that should not be forgotten.[126,127]

Conclusion

Why is there so little written about cleaning? Perhaps most people prefer not to think about it. Housework has been a traditional female role for thousands of years and even now, approaching the 21st century and with far more women working, most domestic tasks are still performed by women. There are more men than women in the workplace and significantly more men in senior positions – such as hospital consultants, managers and infection control officers. These persons, including those who are women, probably have their housework done for them whilst they pursue their chosen career, i.e., those in the position to influence conscientious cleaning, may not be aware of its importance simply because they have never had to do it for themselves. On the wards, today's nurses perform specialized cleaning only, unlike the Nightingale nurses. Florence Nightingale emphasized that complete care of the patient included attention to their environment.[13] She said that, 'Very few people, be they of what class they may, have any idea of the exquisite cleanliness required in a sick room'! Nightingale made cleaning high priority, but that is not the case nowadays. Hospital cleaning is the 'Cinderella' of infection control. How often is it discussed in relation to this subject? Articles, editorials, original research and complete symposia on infection control rarely mention domestic duties. Even in the recent Department of Health publication, 'The Path of Least Resistance', examining the unremitting increase in antimicrobial resistance, there is only one paragraph headed 'Hygiene, Infection Control and Cross Infection'.[128] The paragraph includes the statement that infection control is outside the Terms of Reference of the subgroup and refers the reader to the Cooke report.[129] The remainder of the document deals mostly with antimicrobial prescribing. No one will ever be able to control antibiotic use world wide but good infection control, including cleanliness and hygiene, is eminently achievable.[112] As the report itself states, 'in some cases, the solutions are well known; it is implementation that is deficient'.[128]

Another recent publication addressing the problem of antimicrobial resistance comes from the House of Lords Select Committee on Science and Technology.[130] The Infection Control Nurses Association (ICNA) reported to the committee that their survey had revealed contractor's cleaning cloths and mops being unwashed from day to day and adequate cleaning and training requirements being omitted from contracts because of cost-cutting. The report also stated that, "Putting more resources into hospital cleaning might result in longer waiting times for treatments". It is not surprising, then, that the domestic budget is an easy target for NHS managers. If it is slashed, levels of cleanliness will almost certainly deteriorate. Over-stretched domestic staff will not have time to wipe over door handles, television controls or computer keyboards. One wonders how much of the current MRSA epidemic is

directly attributable to decaying standards of hygiene.

Managers generally regard infection control measures as more disruptive than effective, especially when following such procedures as isolating a potential MRSA patient from another hospital. Perhaps they should consider the cost of an outbreak of MRSA which ultimately closes a ward, unit or even the hospital.[65,131] Hospital-acquired infection costs money.[95,132–134] In an average sized district general hospital, 100 cases of *C. difficile* infection can be expected each year with an extra annual cost of £400,000 and 2100 lost bed-days.[135] How much does it cost to employ an extra cleaner? Most infection control personnel are in agreement regarding the importance of hospital cleaning, but they have yet to convince managers that cutting the domestic budget is a false economy.[1,6]

The Dutch have already noted that hospital economizing has led to the 'streamlining' of cleaning practices, resulting in lower standards of cleanliness.[136] A letter, detailing the in-vitro survival of an epidemic MRSA strain in the Netherlands, comments, '… the number of staphylococci hiding in dust can be substantially greater than on apparently clean dust-free surfaces.' The Netherlands currently have a much lower rate of MRSA than most other countries, following an active 'seek and destroy' policy instituted toward the end of the 1980's.[137]

In local Control of Infection (COI) Committees, the new NHS managerializm has had a deleterious effect. Liquid soap, as opposed to bar, and types of paper towel, engender squabbles over whose budget is accountable. This is regardless of the fact that hand washing is the single most important practise in the control of hospital-acquired infection.[138] If these committees cannot organize soap supplies, they are hardly likely to be able to reinstate comprehensive cleaning schedules.

Although there are few studies examining the effect of generalized cleaning on hospital-acquired infections, there is a flourishing number of reports describing outbreaks from a presumed or proven discrete point source. The sad procession of such outbreaks is a reminder that lessons are not rapidly learnt from the experience of others.[27] Mops, ice-machines, breast-milk pumps, ventilation grilles, scrubbing machines, toilets, curtains, nurses uniforms, sinks, ventilators, shaving brushes, etc., have all been implicated and doubtless many more will be. If an outbreak occurs from such a discrete source, cleaning disinfecting or sterilizing practices for that item should be reviewed. It is only fair to say that general cleaning of the surrounding environment should also be reviewed. Bad or misguided practice in one area may reflect bad or misguided practice in another.

A hospital which attends to cleanliness and hygiene is likely to attend to patient care. Sloppy wards suggest sloppy treatment.[1] Public perception is all important – if a hospital cannot be clean, what can be? Patient morale may be significantly damaged by a dirty hospital, whether or not the patient acquires an infection.[1–4] This could be a problem in today's litigious and quality-conscious society.[45,84]

The solution to stem the seemingly inevitable tide of multiply-resistant micro-organisms, should offer a whole package of practices, which includes hospital cleaning. The whole must be applied to achieve any discernible effect upon the spread of antibiotic resistant organisms.[84] Regarding MRSA, attention remains focused on the role of human carriers and point sources as identifiable and therefore eradicable origins of infection. Few are interested in highlighting the potential benefits of frequent vacuuming to remove skin scales (and adherent staphylococci), laundering of curtains or wiping over door handles (which everyone touches) etc. How often are the covers and cushions on hospital furniture cleaned, for example? Yet these are likely vehicles for the transmission of MRSA, especially in Care of the Elderly units.[67] Even national guidelines on the control of MRSA fail to deliver the correct emphasis on cleaning.[113] Like antibiotic prescribing, no-one will ever be able to ensure total compliance with hand washing, but a high standard of cleaning is achievable.[112]

There is no doubt that there is a need for national standards for hospital cleaning.[6,112] This has already been proposed and the Infection Control Nurses Association (ICNA) is currently preparing a draft following a pilot study.[5] Published guidelines will prompt additional comments and perhaps some research; this would be welcome, since at present practical, applied microbiological research is not considered sufficiently academically respectable and thus does not win funds.[6]

In the meantime, cleaning practises will continue to deteriorate unless the infection control team champion their retention. Extra support is required from the nurses. If nurses believe in the original Nightingale definitions of nursing, they should take a lively interest in the cleanliness of their patients' environment and campaign accordingly.[13] Cleaning practises should not be left to the managers. Significant improvements could be made if more nurses would take responsibility for the cleanliness and hygienic standards on their own wards.[1] Table I shows some aids to cleaner hospitals.[27]

Standards of hygiene are ultimately determined by people. There are no miracle cleaning materials or equipment. Effective cleaning is usually through hard work.[27] Contamination is important in a hospital, and cleaning is a valuable way of dealing with it; perhaps a new standard for hospital cleaning will show when the former matters and how the latter may best be performed.[139] Florence Nightingale would have approved.

Table I *Aids to cleaner hospitals*[27]

Some hospitals are clean; in others, cleaning standards are miserably low. There is no simple solution for the problem of a dirty hospital. Aids to cleanliness are:

I	Good relationships between those who direct cleaning and those who do it
II	Maintenance of hospital fabric to allow adequate cleaning
III	Prompt removal of rubbish
IV	Adequate lighting and ventilation for cleaners
V	Suitable equipment: easy to handle and easy to clean
VI	Facilities for washing, disinfecting, sterilizing and storing cleaning equipment
VII	Suitable cleaning agents
VIII	Supervision of equipment and cleaners
IX	Clean water; no surface can be cleaned with dirty water
X	Appropriate frequency of cleaning in different areas of the hospital
XI	Audit of standards and staff training

(Adapted from Maurer, *I.M. Hospital Hygiene*; Edward Arnold, London, 1974)

Acknowledgement

Thanks are due to Mrs Su Byme for typing the manuscript and Sisters Anne Crawford and Pauline Bell for excellent advice. I would also like to acknowledge the help given by Mrs Janette Healey.

References

1. Hempshall P, Thomson M. Dirt Alert. *Nursing Times* 1998; **94**: 63–64.
2. Maurer IM. *Hospital Hygiene*, 3rd edition. Wright PSG, Bristol, 1985.
3. Collins BJ. The hospital environment: how clean should a hospital be? *Journal of Hospital Infection* 1988; **11** (Suppl A): 53–56.
4. Gaze H. Sweeping Change. *Nursing Times* 1990; **86**: 27–28.
5. Hempshall P, Thomson M. Grime Watch. *Nursing Times* 1998; **94**: 66–69.
6. Rennie M. Germ Warfare. Editorial, *British Journal of Intensive Care* 1998; **8**: 77.
7. Hawker JI. A survey of the implementation of health service guidelines on arrangements for infection control in health trusts in the West Midlands. *Communicable Disease and Public Health* 1999; **2**: 54–58.
8. Worsley MA. Guidance for hospital personnel. In, *Control of Hospital Acquired Infections* Ed W C Noble. Update Books, London, 1983.
9. Ayliffe GAJ, Collins BJ, Lowbury EJL. Ward floors and other surfaces as reservoirs of hospital infection. *Journal of Hygiene* 1967; **65**: 515–536.
10. American Hospital Association. Statement on microbiologic sampling in the hospital. *Hospitals* 1974; **48**: 125–6.
11. Maki DG, Alvarado CJ, Hassemer CA, Zilz MA. Relation of the inanimate environment to

endemic nosocomial infections. *New England Journal of Medicine* 1982; **307**: 1562–1566.
12. Watkins D. Cleanliness in the Crimea *Hospital Equipment & Supplies* March 1997: 27.
13. Nightingale F. (1859) Notes on Nursing, Revised Edition 1952. Gerald Duckworth and Co. Ltd.
14. Struelens MJ, Maas A, Nonhoff C, *et al*. Control of nosocomial transmission of *Clostridium difficile* based on sporadic case surveillance. *American Journal of Medicine* 1991; **91** (Suppl. 3b): 1385–1445.
15. Rutala WA, Gergen MF, Weber DJ. Inactivation of *Clostridium difficile* spores by disinfectants. *Infection Control and Hospital Epidemiology* 1993; **14**: 36–39.
16. Fekety R, Kim KH, Brown D, Batts DH, Cudmore M, Silva J. Epidemiology of antibiotic – associated colitis: isolation of *Clostridium difficile* from the hospital environment. *American Journal of Medicine* 1981; **70**: 906–908.
17. McFarland LV, Mulligan ME, Kwok RYY, Stamm WE. Nosocomial acquisition of *Clostridium difficile* infection. *New England Journal of Medicine* 1989; **320**: 204–210.
18. Mulligan ME, George WL, Rolfe RD, Finegold SM. Epidemiological aspects of *Clostridium difficile*-induced diarrhoea and colitis. *American Journal of Clinical Nutrition* 1980; **33** (Suppl 11): 2533–2538.
19. Savage AM, Alford RH. Nosocomial spread of *Clostridium difficile*. *Infection Control* 1983; **4**: 31–33.
20. Manian FA, Meyer L, Jenne J. *Clostridium difficile* contamination of blood pressure cuffs: a call for a closer look at gloving practices in the era of universal precautions. *Infection Control and Hospital Epidemiology* 1996; **17**: 180–182.
21. Kim KH, Fekety R, Batts DH *et al*. Isolation of *Clostridium difficile* from the environment and contacts of patients with antibiotic-associated colitis. *Journal of Infectious Diseases* 1981; **143**: 42–50.
22. Worsley, MA. A major outbreak of antibiotic-associated diarrhoea. *PHLS Microbiology Digest* 1993; **10**: 97–99.
23. Teare EL, Corless D, Peacock A. *Clostridium difficile* in district general hospitals. *Journal of Hospital Infection* 1998; **39**: 241–242.
24. Hoffman PN. *Clostridium difficile* and the hospital environment. *PHLS Microbiology Digest* 1993; **10**: 91–92.
25. Worsley MA. Infection control and prevention of *Clostridium difficile* infection. *Journal of Antimicrobial Chemotherapy* 1998; **41** (Suppl. C): 59–66.
26. Wilson J. *Infection Control in Clinical Practice*. London, Balliere Tindall, 1995.
27. Maurer IM. *Hospital Hygiene* Edward Arnold, London, 1974.
28. Zafar AB, Gaydos LA, Furlong WB, Nguyen MH, Mennonna PA. Effectiveness of infection control program in controlling nosocomial *Clostridium difficile*. *American Journal of Infection Control* 1998; **26**: 588–593.
29. Wilson G. Bacillus: The aerobic spore-bearing bacilli. In Parker MT, Ed. *Topley and Wilson's Principles of Bacteriology, Virology and Immunity*. Baltimore. Williams and Williams, 1983.
30. Weber DJ, Rutala WA. *Bacillus* species. In Stratton CW, Ed. Topics in Clinical Microbiology. *Infection Control and Hospital Epidemiology* 1998; **9**: 368–373.
31. Johnson KM. *Bacillus cereus* food-borne illness – an update. *Journal of Food Protection* 1984; **47**: 145–153.
32. World Health Organisation. *Bacillus cereus* in a maternity unit. *Weekly Epidemiological Record* 1981; **12**: 93.
33. Birch BR, Perera BS, Hyde WA *et al*. *Bacillus cereus* cross-infection in a maternity unit. *Journal of Hospital Infection* 1981; **2**: 349–354.
34. Barrie D, Hoffman PN, Wilson JA, Kramer JM. Contamination of hospital linen by *Bacillus cereus*. *Epidemiology and Infection* 1994; **113**: 297–306.
35. Dancer SJ. An outbreak of Bacillus. MSc Thesis, University of London, August 1992.
36. Koerner RJ, Morgan S, Ford M, *et al*. Outbreak of Gram-negative septicaemia caused by contaminated continuous infusions prepared in a non-clinical area. *Journal of Hospital Infection* 1997; **36**: 285–289.
37. Aucken HM, Kaufmann ME, Pitt TL. The spread of a multi-resistant epidemic *Klebsiella* strain in London and beyond. Abstract P3·3·6·, 4th International Conference of the Hospital Infection Society, 13–17 Sept. 1998.
38. Forder AA. Buckets and mops in operating theatres. *Lancet* 1973; **1**: 1325.
39. Whitby JL, Blair JN, Rampling A. Cross-infection with *Serratia marcescens* in an intensive therapy unit. *Lancet* 1972; **2**: 127–129.

40. Levin MH, Olsen B, Nathan C, Kabin SA, Weinstein RA. *Pseudomonas* in the sinks in an intensive care unit: relation to patients. *Journal of Clinical Pathology* 1984; **37**: 424–427.
41. Heard S, Lawrence S, Holmes B, Costas M. A pseudo-outbreak of *Pseudomonas* on a special care baby unit. *Journal of Hospital Infection* 1990; **16**: 59–65.
42. Joynson DHM. Bowls and bacteria. *Journal of Hygiene* 1978; **80**: 423–424.
43. Phillips I, Spencer G. *Pseudomonas aeruginosa* cross-infection due to contamination of respiratory apparatus. *Lancet* 1965; **ii**: 1325–27.
44. Casewell MW, Cooper JE, Webster M. Enteral feeds contaminated with *Enterobacter cloacae* as a source of septicaemia. *British Medical Journal* 1981; **282**: 973.
45. Ayliffe GAJ, Lowbury EJL, Geddes A, Williams JD. *Control of Hospital Infection – A Practical Handbook*. Chapman and Hall Medical, London, 1992.
46. Cooke EM, Brayson JC, Edmonson AS, Hall D. An investigation into the incidence and source of Klebsiella infections in hospital patients. *Journal of Hygiene* 1979; **82**: 473–480.
47. Casewell M, Phillips I. Hands as route of transmission for Klebsiella species. *British Medical Journal* 1977; **2**: 1315–1317.
48. Sanderson PJ, Weissler S. Recovery of coliforms from the hands of nurses and patients: activities leading to contamination. *Journal of Hospital Infection* 1992; **21**: 85–93.
49. Bergogne-Bèrèzin E, Towner KJ. *Acinetobacter* spp. as nosocomial pathogens: microbiological, clinical and epidemiological features. *Clinical Microbiology Review* 1996; **9**: 148–165.
50. Musa EK, Desai N, Casewell MW. The survival of *Acinetobacter calcoaceticus* inoculated on fingertips and on formica. *Journal of Hospital Infection* 1990; **15**: 219–227.
51. Jawad A, Snelling AM, Heritage J, Hawkey PM. Exceptional dessication tolerance of *Acinetobacter radioresistens*. *Journal of Hospital Infection* 1998; **39**: 235–240.
52. McGowan JE. Antimicrobial resistance in hospital organisms and its relation to antibiotic use. *Reviews of Infectious Diseases* 1983; **5**: 1033–1048.
53. Roberts NJ, Douglas RG. Gentamicin use and *Pseudomonas* and *Serratia* resistance: Effect of a surgical prophylaxis regimen. *Antimicrobial Agents and Chemotherapy* 1978; **13**: 214–220.
54. Archer GL, Armstrong BC. Alteration of staphylococcal flora in cardiac patients receiving antibiotic prophylaxis. *Journal of Infectious Diseases* 1983; **147**: 642–649.
55. Schwartz SN, Dowling JN, Benkovic C *et al*. Sources of Gram-negative bacilli colonising the trachea of intubated patients. *Journal of Infectious Diseases* 1978; **138**: 227–231.
56. Pina P, Guezenec P, Grosbuis S, Guyot L, Ghnassia JC, Allouch PY. An *Acinetobacter baumanii* outbreak at Versailles Hospital Centre. *Pathol-Biol-Paris* 1998; **46**: 385–94.
57. Joly Guillou ML. Bacteria of the *Acinetobacter* genus. *Pathol-Biol-Paris* 1998; **46**: 245–52.
58. French GL, Phillips I. Resistance. In, O'Grady F, Lambert HP, Finch RG, Greenwood D, eds. *Antibiotic and Chemotherapy* Churchill Livingstone, 1997.
59. Caul EO. Small round structured viruses: airborne transmission and hospital control. *Lancet* 1994; **343**: 1240–42.
60. Russo PL, Spelman DW, Harrington GA *et al*. Hospital outbreak of Norwalk-like virus. *Infection Control and Hospital Epidermology* 1997; **18**: 576–579.
61. Doultree JC, Druce JD, Birch CJ, Bowden DS, Marshall JA. Inactivation of feline calicivirus, a Norwalk virus surrogate. *Journal of Hospital Infection* 1999; **41**: 51–57.
62. Cheesborough JS, Barkess-Jones L, Brown DW. Possible prolonged environmental survival of small round structured viruses. *Journal of Hospital Infection* 1997; **35**: 325–26.
63. Caul EO. Chairman: Viral gastro-enteritis sub-committee of the PHLS Virology committee. Outbreaks of gastro-enteritis associated with SRSVs. *PHLS Microbiology Digest* 1993; **10**: 2–8.
65. Cox RA, Conquest C, Mallaghan C, Marples RR. A major outbreak of methicillin-resistance *Staphylococcus aureus* caused by a new phage type (EMRSA-16). *Journal of Hospital Infection* 1995; **29**: 87–106.
66. Stacey A, Burden P, Croton C, Jones E. Contamination of television sets by methicillin-resistant *Staphylococcus aureus (MRSA)*. Letter, *Journal of Hospital Infection* 1998; **39**: 243–244.
68. Al-ansari N, Brown N, Foweraker J, Ludlam H. The Pre-Millennium Bug? Proceedings of the Pathological Society of Great Britain and

Ireland. *Journal of Medical Microbiology* 1999; **48**: 610.
69. French GL, Rayner D, Branson M. Walsh M. Contamination of doctors and nurses pens with nosocomial pathogens. *Lancet* 1998; **1**: 213.
70. Rountree PM. The effect of dessication on the viability of *Staphylococcus aureus*. *Journal of Hygiene* 1963; **61**: 265–272.
71. Rountree PM, Beard M A, Loewenthal J, May J, Renwick S B. Staphylococcal sepsis in a new surgical ward. *British Medical Journal* 1967; **1**: 132–137.
72. Speers R, Shooter RA, Gaya H, Patel N. Contamination of nurses uniforms with *Staphylococcus aureus*. *Lancet* 1969; **2**: 233–235.
73. Wagenvoort JHT, Penders RJR. Long-term in-vitro survival of an epidemic MRSA phage-group III-29 strain. Letter, *Journal of Hospital Infection* 1997; **35**: 322–325.
74. Marinella MA, Pierson C, Chenoweth C. The stethoscope: a potential source of nosocomical infection? *Archives of Internal Medicine* 1997; **157**: 786–790.
75. Wilson P. Biological Warfare. *Hospital Doctor* 4 July 1996: 26.
76. Bauer TM, Ofner E, Just HM, Just H, Daschner FD. An epidemiological study assessing the relative importance of airborne and direct contact transmission of micro-organisms in a medical intensive care unit. *Journal of Hospital Infection* 1990; **15**: 301–309.
77. Kaplan LM, McGuckin M. Increasing hand washing compliance with more accessible sinks. *Infection Control* 1968; **7**: 408–413.
78. Kumari DNP, Haji TC, Keer V, Hawkey PM, Duncanson V, Flower E. Ventilation grilles as a potential source of methicillin-resistant *Staphylococcus aureus* causing an outbreak in an orthopaedic ward at a district general hospital. *Journal of Hospital Infection* 1998; **39**: 127–133.
79. Farrington M, Ling J, Ling T, French GL. Outbreaks of infection with methicillin-resistant *Staphylococcus aureus* on neonatal and burns units of a new hospital. *Epidemiology and Infection* 1990; **105**: 215–228.
80. Cotterill S, Evans R, Fraise AP. An unusual source for an outbreak of methicillin-resistant *Staphylococcus aureus* on an intensive therapy unit. *Journal of Hospital Infection* 1996; **32**: 207–216.
81. Boyce JM, Opal SM, Potter-Bynoe G, Medeiros AA. Spread of methicillin-resistant *Staphylococcus aureus* in a hospital after exposure to a health-care worker with chronic sinusitis. *Clinical Infectious Disease* 1993; **17**: 496–504.
82. Allen KD, Anson JJ, Parsons LA, Frost NG. Staff carriage of methicillin-resistant *Staphylococcus aureus* (EMRSA 15) and the home environment: a case report. *Journal of Hospital Infection* 1997; **35**: 307–311.
83. Masterton RG, Coia JE, Notman AW, Kempton-Smith L, Cookson BD. Refractory methicillin-resistant *Staphylococcus aureus* carriage associated with contamination of the home environment. *Journal of Hospital Infection* 1995; **29**: 318–319.
84. Dancer SJ. Swinging back the MRSA pendulum. Letter, *Journal of Hospital Infection* 1999; **42**: 69–71.
85. Anonymous Methicillin resistance in *Staphylococcus aureus* isolated from blood in England and Wales: *Communicable Disease Report*. 1994–1998. 1999; **9**: 65.
86. Frieden TR, Munsiff SS, Low DE, *et al*. Emergence of vancomycin-resistant enterococci in New York City. *Lancet* 1993; **342**: 76–79.
87. Kearns AM, Freeman R, Lightfoot NF. Nosocomial enterococci: resistance to heat and sodium hypochlorite. *Journal of Hospital Infection* 1995; **30**: 193–199.
88. Hospital Infection Control Practises Advisory Committee. Recommendations for preventing the spread of vancomycin resistance. *Infection Control Hospital Epidemiology* 1995; **16**: 105–113.
89. Smith TL, Iwen PC, Olson SB, Rupp ME. Environmental contamination with vancomycin-resistance enterococci in an outpatient setting. *Infection Control Hospital Epidemiology* 1998; **19**: 515–8.
90. Gray JW, Pedler SJ. Antibiotic-resistant enterococci. *Journal of Hospital Infection* 1992; **21**: 1–14.
91. Witte W, Klare I. Glycopeptide-resistant *Enterococcus faecium* outside hospitals; a commentary. *Microbial Drug Resistance* 1995; **1**: 259–263.
92. Van den Bogaard AE, Mertens P, London NH, Stobberingh EE. Letter, High prevalence of colonisation with vancomycin – and pristi-

namycin-resistant enterococci in healthy humans and pigs in The Netherlands: is the addition of antibiotics to animal feeds to blame? *Journal of Antimicrobial Chemotherapy* 1997; **40**: 454–456.
93. Threlfall EJ, Rowe B, Ferguson JL, Ward LR. Characterisation of plasmids conferring resistance to gentamicin and apramycin in strains of *Salmonella typhimurium* phage type 204C isolated in Britain. *Journal of Hygiene* 1986; **97**: 419–426.
94. Spera RV, Farber BF. Multidrug resistant *Enterococcus faecium*. An untreatable nosocomial pathogen. *Drugs* 1994; **48**: 678–688.
95. Lai KK, Kelley AL, Melvin ZS, Belliveau PP, Fontecchio SA. Failure to eradicate vancomycin-resistant enterococci in a university hospital and the cost of barrier precautions. *Infection Control Hospital Epidemiology* 1998; **19**: 647–652.
96. Noble MA, Isaac-Renton JL, Bryce DL *et al*. The toilet as a transmission vector of vancomycin-resistant enterococci. *Journal of Hospital Infection* 1998; **40**: 237–241.
97. Noble WC, Virani Z. Co-transfer of vancomycin and other resistance genes from *Enterococcus faecalis* NCTC 12201 to *Staphylococcus aureus*. *FEMS Microbiology letters* 1992; **93**: 195–198.
98. Hiramatsu K, Hanaki H, Ino T, Yabuta K, Oguri T, Tenover FC. Letter, Methicillin-resistant *Staphylococcus aureus* clinical strain with reduced vancomycin susceptibility. *Journal of Antimicrobial Chemotherapy* 1997; **40**: 135–136.
99. Hanaki H, Kuwahara-Arai K, Boyle-Varia S, Daum RS, Labischinski H, Hiramatsu K. Activated cell-wall synthesis is associated with vancomycin resistance in methicillin-resistance *Staphylococcus aureus* clinical strains Mu3 and Mu50. *Journal of Antimicrobial Chemotherapy* 1998; **42**: 199–209.
100. Mermel LA, Josephson SL, Giorgio CH, Dempsey J, Parenteau S. Association of Legionnaires' Disease with construction: contamination of potable water? *Infection Control Hospital Epidemiology* 1995; **16**: 76–81.
101. Loo VG, Bertrand C, Dixon C *et al*. Control of construction-associated nosocomial aspergillosis in an antiquated haematology unit. *Infection Control Hospital Epidemiology* 1996; **17**: 360–364.
102. Loudon KW, Coke AP, Burnie JP, Shaw AJ, Oppenheim BA, Morris CQ. Kitchens as a source of Aspergillus niger infection. *Journal of Hospital Infection* 1996; **32**: 191–198.
103. Edmunds LM, Rawlinson A. The effect of cleaning on blood contamination in the dental surgery following periodontal procedures. *Australian Dentist Journal* 1998; **43**: 349–353.
104. Burgess NRH. Hospital design and cockroach control. *Transactions of the Royal Society of Tropical Medicine and Hygiene* 1984; **78**: 293–294.
105. Bennett G. Cockroaches as carriers of bacteria. *Lancet* 1993; **1**: 732.
106. Fotedar R, Shrinivas U, Banerjee U, *et al*. Nosocomial infections: cockroaches as possible vectors of drug resistant *Klebsiella*. *Journal of Hospital Infection* 1991; **18**: 155–159.
107. Graffar M, Mertens S. Le role des blattes dans la transmission des salmonelloses. *Annale de Institut Pasteur* 1950; **79**: 654–660.
108. Beatson SH. Pharoah's ants as pathogen vectors in hospitals. *Lancet* 1972; **1**: 425–427.
109. Beatson SH. Pharoah's ants enter giving-sets. *Lancet* 1973; **1**: 606.
110. Ward K. All that Glistens. Nursing Times 1990; **86**: 32–34.
111. Marshall B, Sen RA, Chadwick PR, Keaney MGL. Environmental contamination of a new general Surgical Ward. Letter, *Journal of Hospital Infection* 1998; **39**: 242–243.
112. Corcoran GD, Kirkwood EM. Revised guidelines for the control of methicillin – resistant *Staphylococcus aureus* infection in hospitals. Letter, *Journal of Hospital Infection* 1999; **41**: 72–74.
113. Revised guidelines for the control of methicillin-resistant *Staphylococcus aureus* infection in hospitals. Report of a combined working party of the British Society for Antimicrobial Chemotherapy, Hospital Infection Society and Infection Control Nurses Association. *Journal of Hospital Infection* 1998; **39**: 253–290.
114. Decker MD, Schaffner W. The relationship between the hospital and the community. Bennett JV, Brachman PS, Eds. *Hospital infections* Boston: Little, Brown 1992: 221–230.
115. Hicks NR, Moore EP, Williams EW. Carriage and community treatment of methicillin-resistant *Staphylococcus aureus*: What happens to colonised patients after discharge? *Journal of Hospital Infection* 1991; **91**: 17–24.

116. Dancer SJ. MRSA in the community; who is responsible? *SCIEH Weekly Report* 1997; 97/01: 2–4.
117. Wenger PN, Brown JM, McNeil MM, Jarvis WR. *Nocardia farcinica* sternotomy site infections in patients following open heart surgery. *Journal of Infectious Diseases* 1998; **178**: 1539–1543.
118. Krilov LR, Barone SR, Mandel FS, Cusack TM, Gaber DJ, Rubino JR. Impact of an infection control program in a specialised preschool. *American Journal of Infection Control* 1996; **24**: 167–73.
119. Ho M, Glass RI, Monroe SS *et al*. Viral gastroenteritis aboard a cruise ship. *Lancet* 1989; **2**: 961–964.
120. Ayliffe GAJ, Lowbury EJL. Airborne infection in hospital. *Journal of Hospital Infection* 1982; **3**: 217–240.
121. Hummell RP, Macmillan BG, Maley M, Altemeier WA. Comparison of complete barrier isolation and unidirectional air flow isolation in the treatment of burn wounds. *Annals of Surgery* 1972; **176**: 742–747.
122. Ayliffe GAJ. Role of the environment of the operating suite in surgical wound infection. *Reviews of Infectious Diseases*. 1991; **13**: 5800–5804.
123. Noone P, Griffiths RJ. The effect on sepsis rates of closing and cleaning hospital wards *Journal of Clinical Pathology* 1971; **24**: 721–725.
124. Noone P, Shafi MS. Controlling infection in a district general hospital. *Journal of Clinical Pathology* 1973; **26**: 140–145.
125. Annotation. Infection Controlled. *Lancet* 1973; **1**: 981–982.
126. Chadwick C, Oppenheim BA. Letter, Cleaning as a cost-effective method of infection control. *Lancet* 1996; **347**: 1776.
127. Kelsey JC. Personal view. *British Medical Journal* 1973; **4**: 104.
128. Department of Health. The Path of Least Resistance. Standing Medical Advisory Committee Sub-group on Antimicrobial Resistance 1998.
129. Department of Health and Social Security. Guidance on the control of infection in hospitals prepared by the joint DHSS/PHLS Hospital Infection Working Group (The Cooke Report), London: DHSS, 1988.
130. Resistance to antibiotics and other antimicrobial agents. London: HMSO Stationery Office 1998.
131. Mehtar S, Drabu VJ, Mayet F. Expenses incurred during a 5-week epidemic of methicillin-resistant *Staphylococcus aureus*. *Journal of Hospital Infection* 1989; **13**: 199–220.
132. Mehtar S. How to cost and fund an infection control programme. *Journal of Hospital Infection* 1993; **25**: 57–69.
133. French GL, Cheng FB. Measurement of the costs of hospital infection by prevalence surveys. *Journal of Hospital Infection* 1991; **18** (Suppl.A): 65–72.
134. Spencer RC. Clinical impact and associated costs of *Clostridium difficile*-associated disease. *Journal of Antimicrobial Chemotherapy* 1998; **41** (Suppl. C): 5–12.
135. Wilcox MH, Cunniffe JG, Trundle C, Redpath C. Financial burden of hospital-acquired infection. *Journal of Hospital Infection* 1996; **34**: 23–30.
136. Anonymous. Schoomakers: Nederland vervuilt (Cleaners: The Netherlands are becoming dirty!) NRC Handelsblad 1993, April 27.
137. Vendenbroucke-Grauls CM. Control of methicillin-resistant *Staphylococcus aureus* in the Netherlands. In, Coello R, Casewell MW, Eds. *Methicillin-resistant Staphylococcus aureus*. Kent, UK: Wells Medical Limited 1993: 75–78.
138. Handwashing Liaison Group. Hand washing. *British Medical Journal* 1999; **318**: 686.
139. Annotation. Contamination, cleaning and common-sense. *Lancet* 1972; **1**: 1108–1109.