Clean air symposium—Part I

The role of airborne bacteria in theatre-acquired surgical wound infection

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A desire to minimize the risk of postoperative wound infection dictates many of the routine procedures of modern surgical practice. Nonetheless, an estimated 7.5% of all operations are followed by wound infection[1] despite traditional emphasis on aseptic technique, antiseptic practice, and careful tissue and wound management.

Recent technologic advances, primarily in the aerospace and electronics industries, have produced air filtration and delivery systems that are apparently far more efficient than traditional air-conditioning systems in reducing airborne particulate and microbial contamination.[2] These systems incorporate high efficiency particulate final air filters and high flow ventilation to achieve clean, minimally turbulent, unidirectional air flow. They have been variously described as laminar, linear, or unidirectional air flow (UAF) systems, but the last term appears most descriptive of their effects in hospital operating theatres.[3, 4] Unidirectional air flow, for the control of surgical wound infection, has received enthusiastic support in the medical literature.[2, 4, 5] As a result of these developments, we at the Center for Disease Control (CDC) have

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received numerous inquiries from hospitals requesting our advice regarding installation of these systems, and some hospitals have expressed concern that a failure to use these systems may result in excessive medical-legal liability.

Before endorsing use of UAF systems in hospital operating theatres, we must carefully evaluate their potential benefits. It is most important to remember that the primary goal in the use of UAF in hospitals is to decrease the incidence of patient disease, since there is little benefit in decreasing environmental contamination unless the risk of disease is also diminished. Therefore, two questions must be raised. First, what role does airborne contamination play in the acquisition of a surgical wound infection? Second, are UAF systems capable of decreasing the risk of surgical wound infection? No definitive answer to either question is available; however, a review of the epidemiology of surgical wound infections can begin to place these questions in perspective.

Epidemiology of surgical wound infections

Acquisition of an infection requires (1) a reservoir of a potentially pathogenic organism, (2) a suscepible host, and (3) an appropriate mode of transmission to link the two. Each is an important determinant of the risk of wound infection.

Nosocomial reservoirs responsible for surgical wound infections. Until recently, most epidemiologic studies of wound infections have focused primarily on coagulase-positive staphylococci (*Staphylococcus aureus*). The reasons for this are several. First, *S. aureus* is a major nosocomial pathogen and is responsible for a substantial portion of wound infections. Second, staphylococcal nursery and wound infection epidemics were widely reported in the 1950s and 1960s at a time of emerging penicillin resistance; these reports led to the widespread impression that nosocomial infections were, in large part, staphylococcal. Finally, phage typing of staphylococci has been a valuable epidemiologic tool for studying the transmission of specific strains within the hospital environment.

Because of this emphasis on *S. aureus,* other important nosocomial pathogens have often been slighted. *S. aureus* accounted for only 16% of organisms isolated from surgical wound infections reported to CDC's National Nosocomial Infections Study[6] in the first half of 1972; Group A beta-hemolytic streptococci accounted for 3%, while aerobic fermentative gram-negative rods accounted for over 45% of isolates.[7] A classic cooperative evaluation of the influence of ultraviolet (UV) light on wound infection sponsored by the National Research Council of the National Academy of Sciences found a similar, wide distribution of pathogens: *S. aureus* 31%, Group A streptococci 2%, and comparable gram-negative organisms 63%.[1] Staphylococci were noted more frequently in a recent prospective study of orthopaedic surgical wound infections at the Boston City Hospital, where 43% of isolates were *S. aureus* and only 23% were gram-negative.[8] In contrast, several recent reports of wound infection following total hip replacement have shown the predominant isolation of coagulase-negative staphylococci.[9, 10]

Epidemiologic studies of the reservoirs and acquisition of staphylococci are not necessarily applicable to the

other organisms responsible for wound infection.[11] Hospital personnel and patients are the major nosocomial reservoirs of *S. aureus;* up to 85% of normal individuals carry the organism on occasion, and fully 15% to 35% are persistent asymptomatic carriers.[12] While people are also the major source of *Escherichia coli,* fecal carriage is presumably a more important reservoir than skin. In contrast, the hospital's inanimate environment is most often implicated as the important reservoir of *Pseudomonas species* and some other gram-negative pathogens.

A surgical wound may be infected by organisms acquired from endogenous or exogenous sources. Endogenous infection, or infection caused by organisms carried by the host as his own flora, may occur in several ways. Bacteremia at the time of operation can contaminate the operative wound.[13]

Transient bacteremia, occurring at other times, may seed and infect cardiac and skeletal prostheses. A patient's own cutaneous flora may cause autogenous wound infection, for most studies have demonstrated asymptomatic carriers of staphylococci are far more likely to develop staphylococcal surgical wound infections than are noncarriers.[14] Most wound infections caused by enteric organisms, especially infections following abdominal procedures, are probably acquired from the patient's own fecal flora.[15]

Exogenous infection is caused by organisms that are not carried by the affected patient, and these organisms may be acquired during or after operation. Few studies have adequately assessed both the operating theatre and the postoperative ward as potential sites of acquisition; the simple demonstration that a pathogenic organism is present in the operating theatre is insufficient evidence to implicate the theatre as the site of acquisition. For example, a surgeon demonstrated to carry staphylococci of the same phage type as that causing disease generally has multiple opportunities to transmit these organisms to his patients; he not only operates on them, exposing them to theatre-acquired infection, but he also treats them preoperatively and postoperatively, seeing them several times daily, changes wound dressings, and performs other manipulations that may serve to transmit the infecting organism.

Several investigators have attempted to differentiate the relative roles of theatre and ward in the acquisition of wound infections. Some favor a predominance of the theatre,[16] and others favor the wards,[8, 17] but most studies have concluded, as might be expected, that exogenous infections are acquired both in the operating theatre and on the wards.[8, 14, 18]

Host factors influencing risk of wound infection. Potentially pathogenic organisms may be isolated not only from the animate and inanimate hospital environment, but they may also be found frequently in surgical wounds at time of closure. In a thorough study of 50 clean surgical procedures, Burke[19] demonstrated bacterial wound contamination in each at closure. Forty-six of the 50 (92%) were contaminated with *S. aureus,* and an average of 5.8 different staphylococcal strains were found in each wound. The UV light study[1] has also confirmed the frequent contamination of wounds at the end of a procedure. Since only a small proportion of these contaminated wounds become clinically in-

Table 1. Incidence of surgical wound infections following selected clean surgical procedures[1]

Operation	Wounds	Infections	%
Herniorrhaphy	1,312	25	1.9
Partial mastectomy or local excision of breast lesion	827	18	2.2
Radical mastectomy	227	43	18.9

Table 2. Factors associated with an increased risk of surgical wound infection[1]

Increased age
Extreme obesity
Concurrent remote infection
Delayed closure of operative wound
Presence of surgical drain
Prolonged procedure
Prolonged hospitalization before operation
Use of prophylactic antimicrobials
Steroid therapy (possible)

fected, factors other than the mere presence of organisms in a surgical wound must influence the risk of developing disease.

Host resistance is a major determinant of the susceptibility to wound infection. In the UV light study, the incidence of wound infection increased in direct proportion to the relative bacterial contamination associated with the operation, but a wide variation in wound infection rates was noted even among procedures having the same relative amount of contamination (*Table 1*). Radical mastectomy was associated with a 10-fold greater risk of wound infection than was herniorrhaphy and substantially greater risk than simple mastectomy.[1] The UV light study also demonstrated other apparent predispositions to the development of wound infection (*Table 2*); but even when rates of infection were adjusted for all known variables, differences in infection rate between institutions were noted that could only be explained by differences in operative technique[1] or other unknown factors.

Surgical technique is a major determinant of the outcome of wound healing. Condie and Ferguson[20] compared the tissue reaction of experimental surgical wounds contaminated with 10^8 to 10^9 virulent staphylococci and closed by differing techniques; careful obliteration of dead space markedly diminished host susceptibility to wound infection. Further evidence of the importance of local tissue factors was demonstrated by Elek and Conen[21] who had to introduce between 2 and 8 million staphylococci to produce abscess formation when no foreign body was present; however, as few as 100 staphylococci produced disease if suture material were present in the wound.

Thus, contamination of a surgical wound is necessary but clearly not sufficient alone for the development of wound infection. Any technique that is offered as a measure to reduce the incidence of wound infection must be tested in a trial that will control, insofar as is possible, those host factors that are established as having a profound influence on the risk of wound infection.

Transmission of exogenously acquired wound infection. *Table 3* lists the potential modes of transmission for communicable diseases both at the time of operation and postoperatively. Seldom have epidemic surgical wound infections had the mode of transmission adequately defined. Many epidemic reports have not adequately described investigations of both the op-

Table 3. Modes of transmission of exogenously acquired surgical wound infections

Contact
Direct
Indirect
Droplets
Airborne
Common vehicle
Vector

erating theatre and the postoperative environment, have not differentiated between airborne and contact transmission, or have instituted control measures effective against both these modes of spread.[22-24]

Contact transmission may be direct, indirect, or by droplet spread. Hand contact and fecal-oral spread are examples of direct contact while contamination from fomites or instruments are examples of indirect contact transmission. A typical outbreak of contact transmission was reported by Penikett et al[25] who demonstrated direct contact transmission by suture material contaminated with staphylococci. Transmission by contaminated droplets, large particles usually derived from oral or pharyngeal secretions which are quite heavy and settle rapidly, is usually limited to a distance of approximately 1 meter from the source.[26] Although droplets pass through the air in settling, they contribute to contact rather than airborne transmission because of the limited area of spread and the ineffectiveness of alterations in ventilation in control.

True airborne spread involves organisms carried on small particles such as desiccated oral secretions (droplet nuclei) or dust. Such particles settle slowly and are wafted about in the ambient air by convection currents; these particles are most amenable to control by changes in ventilation.[26] An example of disease acquired in the theatre and transmitted by the airborne route was described by Shooter et al.[27] Air, highly contaminated with staphylococci from a nearby surgical ward, was drawn into a primitively ventilated operating theatre that was at negative pressure with respect to adjacent areas. For infection to be spread by the airborne route, the infectious agent must remain viable after severe desiccation. Staphylococci, *Mycobacterium tuberculosis,* various viruses, and bacterial spores are capable of airborne transmission, while many gram-negative organisms are rapidly killed by the harsh airborne environment.

Common vehicle transmission is also seen in hospitals and results from contamination of agents such as intravenous fluids or irrigating solutions. A recently reported outbreak of surgical wound infection caused by *Pseudomonas multivorans* resulted from common-vehicle transmission; a contaminated disinfectant solution was used in the operative and postoperative irrigation of wounds.[28] Vector-borne nosocomial infections are rarely seen in hospitals in the United States, but community-acquired illnesses such as Rocky Mountain spotted fever are spread by this route.

Several investigators have attempted to define the modes of spread of endemic or sporadic infections. Arguments both for and against the primacy of airborne transmission of sporadic, theatre-acquired wound infections have been made. Howe and Marston,[16] in an intensive investigation of 330 surgical patients found little evidence of airborne transmission

despite their attributing most wound infections to the operating theatre.

Bernard and Cole[29] could demonstrate no correlation between air contamination and the incidence of wound infection in clean surgical procedures, but they noted that documentation of minor changes in infection rate would require evaluation of many more patients than they had available in their 2-year study at a single large institution. On the other hand, Burke[19] considered air in the immediate area of the surgical wound to be the final common pathway for the high incidence of contamination that he demonstrated at the time of wound closure. In an operating room with ventilation that would apparently meet current Hill-Burton standards,[30] he showed by slit-sampler testing at the wound edge that 68% of organisms isolated from wounds were also isolated from the adjacent air.[19] He did not attempt to differentiate between airborne and indirect contact transmission.

The UV light study provided major support for the potential role of airborne transmission of infection in the operating theatre. Ultraviolet light is quite effective in sterilizing the ambient air, and controlled environmental sampling during this study demonstrated significant reductions in the numbers of organisms settling onto surfaces. Although no benefit was noted overall in patients exposed to UV light, a subsegment of the study population (patients undergoing refined-clean surgical procedures) showed a significant reduction in wound infection rate associated with UV exposure (*Table 4*).[1] These patients were perhaps least likely to be exposed to endogenous contamination, and a decrease in airborne contamination would most likely be reflected in a lower postoperative infection rate in this group.

Table 4. Incidence of surgical wound infections in refined-clean wounds, by exposure to UV light at time of operation[1]

Treatment category	Total cases	Definite infections (%)	Definite plus possible infections (%)
UV light	3,277	94 (2.9)	112 (3.4)
Control	3,379	128 (3.8)	154 (4.6)
		$P < 0.05$	$P < 0.05$

Effects of unidirectional air flow systems. Valid environmental testing of UAF systems requires sophisticated techniques and careful study design;[31] many environmental studies purporting to demonstrate reductions in airborne contamination with UAF provide little meaningful data because of invalid sampling techniques or test conditions that are not applicable to the operating theatre. Nonetheless, airborne microbial counts are probably reduced by these systems. However, current practices in operating theatres in the United States generally result in a very low level of airborne contamination. The benefits resulting from a further lowering of this contamination level by use of UAF must be measured by effects on patient disease; expensive or complicated alterations do not seem justified unless the incidence of infection is significantly decreased.

Charnley and associates pioneered the clinical use of UAF in total hip replacements. As they introduced progressively sophisticated measures to reduce airborne contamination, they noted a definite trend toward a reduction in surgical wound infections.[32] However, changes in operative technique, alterations in surgical skill,

variations in patient selection, and changes in ward care activities occurring concurrently were not evaluated and played an unmeasured role in these uncontrolled studies; furthermore, they also introduced measures to minimize direct contact transmission. Furthermore, although the overall incidence of wound infection decreased in Charnley's series, marked variations in incidence of infection, ranging from zero to four cases per hundred procedures, were noted in the period when minimal airborne contamination was demonstrable by environmental sampling. It is conceivable that alterations in infection rate were unrelated to alterations in airborne contamination. While other investigators have noted high infection rates following total hip replacement,[9, 10] others have apparently achieved infection rates comparable to Charnley's excellent results without resorting to unusual measures to decontaminate the air.[10, 33]

The few reported controlled studies of UAF surgical environments are difficult to interpret. In a recent study, Jones et al[34] showed a four-fold reduction in airborne contamination determined by settle plate and volumetric sampling in sham operations. Unfortunately, activity on the part of the surgeons was prohibited, and no patient was present on the operating table; since physical activity contributes substantially to airborne contamination evaluated by these techniques, this study is not comparable to an actual operative situation. Alpert et al[35] have briefly reported a three-fold reduction in surgical wound infections in an apparently controlled trial of a patient isolator, but no description of study design was offered to allow interpretation of these data.

Discussion

Despite many studies of surgical wound infections, the relative importance of airborne transmission in the operating theatre for the acquisition of wound infection remains unclear. Any assessment of that importance is, at the present time, a "product of judgment rather than arithmetic" as R. E. O. Williams has said.[14] The available studies imply that such transmission does contribute to wound infection. However, it appears that airborne transmission is not the major factor in the acquisition of these infections, and for many operative procedures it probably plays an insignificant role.

The absence of data concerning the relative importance of airborne contamination in the genesis of wound infection makes a decision regarding the potential value of UAF systems quite difficult. Again, I wish to stress that the purpose of these systems is to minimize disease in patients; measured against this goal, I am aware of no adequately controlled study demonstrating that they are effective. Although there are numerous theoretical arguments and artificial *in vitro* tests supporting the potential efficacy of UAF systems for selected surgical procedures, I do not believe that we can rely upon these arguments to determine whether these systems are to be adopted. Neither can we yet accept the contention that it is unethical to withhold these systems from patients. Ethical practice demands that such systems be demonstrated to have a high probability of effectiveness before they are widely adopted. Medical history is replete with examples of our embracing techniques, procedures, or medications that are theoretically attractive or

anecdotally effective but that, when subjected to carefully controlled study, are shown to be ineffective or potentially dangerous.

Adequate studies will be neither simple nor inexpensive. They must control for the multifaceted etiology of surgical wound infections. To achieve the large number of cases required to reach statistical significance, they will almost certainly have to be cooperative studies. Investigators must carefully (1) define what is accepted as a wound infection, (2) consider both the operating theatre and the ward as potential points of acquisition, (3) evaluate all potential pathogens, and (4) focus initially on those patients considered to be at greatest risk for the development of airborne-transmitted infection. Furthermore, I believe that firms now marketing UAF systems have a responsibility to underwrite, at least in part, these controlled studies; the resources directed towards market development, funding uncontrolled clinical trials, or underwriting studies simply evaluating environmental contamination should instead be utilized to test the potential value of UAF in decreasing *patient disease.*

Ultimately, the expense and effort involved in such studies are clearly indicated if we are to avoid having hospitals uniformly adopt an expensive but ineffective system, or fail to adopt an effective measure. Until these studies are conducted, the installation of unidirectional air flow systems does not appear justified.

Summary

Airborne microbial contamination in operating theatres may contribute to the risk of surgical wound infection, but endogenous infection, infection acquired postoperatively, and infection transmitted by contact or common vehicle are of vastly greater importance. Unidirectional air flow systems in operating theatres may potentially reduce the risk of wound infection in selected circumstances, but there are no controlled studies to support their efficacy thus far. Hospitals should not be encouraged to install these systems until such efficacy is documented by clinical trials demonstrating a significant reduction in patient disease, and until the population groups that benefit from these systems are defined.

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References

1. National Academy of Sciences—National Research Council: Postoperative wound infections; the influence of ultraviolet irradiation of the operating room and of various other factors. Ann Surg **160** (Suppl): 1–192, 1964.
2. Fox DG: A study of the application of laminar flow ventilation to operating rooms. Washington, D. C., U. S. GPO, Public Health Monograph no. 78, 1–50, 1969.
3. Charnley J: Operating-theatre ventilation (letter to the editor). Lancet **1**: 1053–1054, 1970.
4. Scott CC: Laminar/linear flow system of ventilation: its application to medicine and surgery. Lancet **1**: 989–993, 1970.
5. Whitcomb JG, Whitfield W, King J, et al: Ultra-clean operating rooms. Lovelace Clin Rev **2**: 65–69, 1965.
6. Bennett JV, Scheckler WE, Maki DG, et al: Current national patterns: United States, *In* Proceedings of the International Conference on Nosocomial Infections. Chicago, The American Hospital Association, pp 42–49, 1971.
7. National Nosocomial Infections Study, Center for Disease Control, unpublished data.

8. O'Riordan C, Adler JL, Banks HH, et al: Wound infections on an orthopedic service. Am J Epidemiol **95:** 442–450, 1972.
9. Patterson FP, Brown CS: The McKee-Farrar total hip replacement: preliminary results and complications of 368 operations performed in five general hospitals. J Bone Joint Surg **54A:** 257–275, 1972.
10. Wilson PD Jr, Amstutz HC, Czerniecki A, et al: Total hip replacement with fixation by acrylic cement. J Bone Joint Surg **54A:** 207–236, 1972.
11. Williams REO: Epidemiology of airborne staphylococcal infection. Bacteriol Rev **30:** 660–672, 1966.
12. Nahmias AJ, Eickhoff TC: Staphylococcal infections in hospitals: recent developments in epidemiologic and laboratory investigation. N Engl J Med **265:** 120–128, 1961.
13. Howe CW: Experimental wound sepsis from transient *Escherichia coli* bacteremia. Surgery **66:** 570–574, 1969.
14. Williams REO, Blowers R, Garrod LP, et al: Hospital Infection: Causes and Prevention. London, Lloyd-Luke Ltd, 1969.
15. Thomsen VF, Larsen SO, Jepsen OB: Postoperative wound sepsis in general surgery. IV. Sources and routes of infection. Acta Chir Scand **136:** 251–260, 1970.
16. Howe CW, Marston AT: A study on sources of postoperative staphylococcal infection. Surg Gynecol Obstet **115:** 266–275, 1962.
17. Lindbom G: Studies of the epidemiology of Staphylococcal infections. II. Staphylococcal infections in a thoracic surgery unit. Acta Chir Scand **128:** 421–434, 1964.
18. Mitchell AAB, Timbury MC, Pettigrew JB, et al: An epidemic of postoperative wound infection by type-80 staphylococci. Lancet **2:** 503–505, 1959.
19. Burke JF: Identification of the sources of staphylococci contaminating the surgical wound during operation. Ann Surg **158:** 898–904, 1963.
20. Condie JD, Ferguson DJ: Experimental wound infections: contamination versus surgical technique. Surgery **50:** 367–371, 1961.
21. Elek SD, Conen PE: The virulence of *Staphylococcus pyogenes* for man; a study of the problems of wound infection. Br J Exp Pathol **38:** 573–586, 1957.
22. Ayliffe GAJ, Collins BJ: Wound infections acquired from a disperser of an unusual strain of *Staphylococcus aureus*. J Clin Pathol **20:** 195–198, 1967.
23. Browne AF, Ryan EA, Glassow FJ, et al: Staphylococcic wound infections: study of wound infections in several thousand hernia cases. JAMA **170:** 1274–1283, 1959.
24. Walter CW, Knudsen RB, Brubaker MM: The incidence of airborne wound infection during operation. JAMA **186:** 908–913, 1963.
25. Penikett EJK, Knox R, Liddell J: An outbreak of postoperative sepsis. Br Med J **1:** 812–814, 1958.
26. Langmuir AD: Epidemiology of airborne infection, *In,* Conference on Airborne Infection, Edited by W McDermott. Bacteriol Rev **25:** 173–181, 1961.
27. Shooter RA, Taylor GW, Ellis G, et al: Postoperative wound infection. Surg Gynecol Obstet **103:** 257–262, 1956.
28. Bassett DCJ, Stokes KJ, Thomas WRG: Wound infection with *Pseudomonas multivorans:* a water-borne contaminant of disinfectant solutions. Lancet **1:** 1188–1191, 1970.
29. Bernard HR, Cole WR: Bacterial air contamination and its relation to postoperative sepsis. Ann Surg **156:** 12–18, 1962.
30. Health Facilities Planning and Construction Service: General Standards of Construction and Equipment for Hospital and Medical Facilities. Washington, D. C., U. S. GPO, Public Health Service Publication, no. 930-A-7, 1969.
31. Kethley TW: Air quality specifications (microbial): the sampling problem. Svensk Farmaceutisk Tidskrift **73:** 854–866, 1969.
32. Charnley J, Eftekhar N: Postoperative infection in total prosthetic replacement arthroplasty of the hip-joint, with special reference to the bacterial content of the air of the operating room. Br J Surg **56:** 641–649, 1969.
33. Stinchfield FE, White ES: Total hip replacement. Ann Surg **174:** 655–662, 1971.
34. Jones MA, Johnson JC, French MLV, et al: Unidirectional air flow and surgical face mask exhaust system in the prevention of airborne surgical infection. Am J Surg **124:** 49–51, 1972.
35. Alpert S, Salzman T, Sullivan C, et al: Wound bacteriology and infection in patients operated upon by the surgical isolator technique or in a conventional environment. Surg Forum **22:** 65–66, 1971.