Ventilation and Air Conditioning as Bacteriologic Engineering

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Historically, ventilation of operating rooms has been motivated by simple objectives. Odor control, heating and dilution of contagion are the oldest. Removal of accumulating anesthetic gases or scavenging of spilled volatile anesthetic agents to prevent explosion is a more recent and dominant goal. Air conditioning has assumed increasing importance to provide comfort for the staff and control of humidity to deter hazardous electrification. The realization that the physiologic effects of air conditioning benefit the patient is the most recent advance to be superimposed on a revitalized concept of controlling cross-infection. Thus, an optimum environment for the patient becomes the current goal. This involves control of temperature, humidity, particulate and microbiologic contamination throughout the hospital. A confusing array of concepts and techniques has been applied and studied in the operating room—too often in unoccupied rooms or with inappropriate methodology. The result is a chaos of uncritical data for the designer; disillusion and needless expense for the hospital. Failure to appreciate the role of the environment in contamination of the air with particulates and bacteria has been an important cause for disillusion.

Contrary to widespread belief, the atmosphere is not a reservoir for bacteria. It merely transports dispersed bacteria and particulates. Sterilized air is just as effective a vehicle as contaminated air. The sources of bacteria in the air of the operating room are well known, and are susceptible to control. The most dangerous and ubiquitous source of pathogenic organisms is the forcibly expired air of personnel, including that of the surgeon and the patient. A related source of potentially dangerous organisms is desquamating epithelium shed by personnel. A secondary source derived from the fallout of human activity is dust scuffed from the operating room floor, brushed from clothing, or whisked into the air from bedding or dressings. A third source of organisms is street soil carried on the shoes of personnel or dust from open windows. Pigeon droppings, which pollute most hospital window sills, are often concentrated pellets of clostridia. A fourth and often-overlooked source of bacteria is contaminated ventilating air.

The original work of Pasteur on fermentation focused so much attention on airborne bacteria that early efforts to eliminate postoperative suppuration were aimed at controlling airborne contagion. As the bacteriology of surgery was described it became obvious that other factors had greater priority, and efforts to control airborne bacteria gradually relaxed. During the last 30 years, airborne contamination has again been recognized as the cause of a residuum of postoperative infection which escapes control by other methods. The magnitude of this problem is grossly demonstrated by dust motes settling through the tyndall beam from a surgical light. In terms of bacteria, 30,000 to 60,000 viable organisms may settle on the surgical field per hour. The fact that still air in an unoccupied room is sterile emphasizes the role played by personnel in shedding from their nasopharynges and skins, and in stirring up organisms and dust by their movements.

The major source of dust in the properly-managed operating room is the lint from textile clothing, bedding, drapes and mops. In the first three the particulate contamination stems from the laundry and varies with the quantity of linters and paper products that are discarded with the soiled textile. These dis-

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integrate in the washer and are dispersed to adhere to the surfaces of textile. The only economic and effective control is to separate trash from soiled linen. The use of short-string mops decreases the quantity of fiber abraded during mopping. The short life of such mops precludes the rotting that disintegrates longer strings to linters.

The origin of pathogenic microorganisms in the operating room is people. Overt infection in patients and personnel is too obvious to require elaboration. Latent infection—the carrier state in apparently-healthy people—continues as the leading etiologic agent in surgical wound sepsis and postanesthesia pulmonary infections.

Efforts to control cross-infection customarily are restricted to a limited hospital function, such as surgery. Control measures are directed toward perfection of manual techniques for patient care and the adoption of disposable supplies. The use of antibiotics is limited and asepsis is intensified. Evanscent improvement usually frustrates and disillusiones successive campaigners. Cross-infection persists.

Several widely-ignored factors are responsible for the persistence of cross infection: 1) it is based broadly throughout the hospital and not confined to just one functional area; 2) the entire hospital environment is a bacteriologic backdrop for activities that disseminate bacteria; 3) patients and personnel reflect their exposure to this environment by becoming colonized by the accumulated bacterial debris from previous patients and carriers, thus extending cross-infection in time and place.

Members of the surgical team are the couriers of hospital-based infection to the aseptic field, for they, too, share the occupational hazard of the hospital environment (table 1). Personnel remain the chief source of exogenous bacteria even in the properly-managed operating room.

Surfaces, including skin, instruments, devices, clothing, bedding, equipment, and furniture, in direct contact with patients are obviously primary vehicles in cross-infection. Yet, when contact transmission is controlled by aseptic technique, cross-infection stubbornly persists—deployed in location or separated by intervals sufficient to exclude contact as the mode of transmission. In this situation, surfaces throughout the entire hospital succeed to a paramount role as secondary reservoirs of bacteria that contaminate the ventilation vortex in which the hospital population lives (fig. 1).

Horizontal surfaces are the most significant factor in the ecology of supplicative disease, because settling bacteria accumulate under circumstances that prolong life and abet redistribution.2 The largest horizontal surface, the floor, is also the base for the human activity that sends bacteria into the air. Floor cultures are excellent indicators of the bacteriologic types of prevailing infection. These reflect qualitatively and quantitatively the bacteriology of the occupants of the area and act as an index of infectivity of the environment. Horizontal surfaces are the staging area for bacteria that become airborne as an essential but subtle epidemiologic step in attaining a portal of entry.

A review of the modes of airborne dissemination of bacteria is essential to gain the perspective that will give meaning to the interrelationship between environment and carrier in the spread of bacteria.

Airborne transmission of disease occurs by three vehicles; dust laden with bacteria, droplets transporting bacteria, and droplet nuclei harboring bacteria.
Bacteria-laden dust derives from the fragmentation of dried excrements, excretions, or discharges; from lint contaminated by these wastes; or from desquamating epithelium. Droplets that carry bacteria are expelled by tussic expirations and plosive speech sounds. Both dust and droplets settle out rapidly and hence have short trajectories. These bacteria-laden particles accumulate on horizontal surfaces where they add their increment of bacteria to these secondary reservoirs that persist for a period sufficient to provide continuity of hazard. Trivial motion or slight air currents resuspend this bacteria-laden dust.

Dust particles may settle onto burns, open wounds, or other susceptible tissue, such as that of a tracheostomy or unhealed umbilicus. Of greater epidemiologic import is the fact that they are inhaled and inoculate the mucous membranes of the upper respiratory tract. Colonization results, and the subsequent attenuated infection is known as the carrier state.
Disseminating carriers lend ubiquity and continuity to the spread of bacteria by expelling, wherever they go, droplets and droplet nuclei which may be sprayed directly into wounds or inhaled by others. Droplets settle on skin, clothing, and other surfaces, eventually giving rise to bacteria-laden lint and dust.

Control of airborne bacteria by ridding the environment of lint, desquamated epithelium, and particulate soil is best accomplished by vacuum cleaning. A well-designed vacuum cleaner removes dust quickly, effectively, and definitively. When vacuuming is combined with flooding of the floor with a germicidal detergent and wet pickup of the resulting slurry, the largest and most persistent secondary reservoir for bacteria—the floor—becomes epidemiologically innocuous. No substitute control measure of equal hygienic effectiveness has been demonstrated.

Droplet nuclei are expelled during forceful expiration such as coughing and sneezing. They remain airborne indefinitely, and may penetrate to the alveoli of the lungs to initiate pulmonary infection. Attenuation of droplet nuclei occurs by several means: natural death, settling out, or destruction or dilution in a moving stream of air. Relative humidity is an important factor in determining bacterial survival in air. At high humidity, multiplication of bacteria may occur; at low humidity, hardy desiccation-resistant bacteria in a population seem to survive. In occupied places settling is so slow (less than 0.1 foot/min) that droplet nuclei remain constantly airborne until inhaled or removed by attenuation, ventilation or disinfection.

Removal by ventilation is related to the rate of air change. At ventilation rates required to control odor (six changes per hour), the concentration of a single dispersion of droplet nuclei falls to about one per cent of its initial value in 45 min. Ventilation in the conventional sense thus contributes little to the control of continuously-emitted contagion. The reason is obvious—at low rates of flow, turbulence redistributes the droplet nuclei, and insufficient clean air is provided to dilute continuing cascades of airborne organisms beyond the threshold where new infections do not occur. Threshold sanitary ventilation requires a combination of dilution, destruction, and removal of bacteria equivalent to one change of air per minute.

In the context of this extended and expanded concept of asepsis, specific recommendations can be made to protect patients from bacterial contamination of nosocomial origin.

Endogenous sources of bacteria that cause wound sepsis must be suppressed. A basic weapon is prompt containment isolation of sepsis, however trivial, until bacteriologic monitoring demonstrates freedom from shedding into the environment. Equally important is prohibition of personnel with infection from working in the hospital. A complementary essential is removal of accumulated bacterial debris by effective housekeeping techniques and sufficient ventilation to rid the environment of mobile bacteria.

Carriers among patients usually reflect exposure to the hospital environment. They may become victims of their own latent infections when manipulation of their airways breaches the mucous membrane barrier. This is an unappreciated portal of entry for nosocomial infections.

Specific virulent strains of bacteria accumulate in the hospital environment and in carriers among its personnel. Current concepts in the epidemiology of gram-negative infections reemphasize the nosocomial nature of cross-infection. As is evident from reports on the role of the anesthetist in cross-infection, each component of patient care, however transient, must be appraised as a potential factor in cross-infection. Great emphasis must be placed on the hygiene of angiographic, cardiac catheterization, pulmonary function and other procedural laboratories. The recovery room and intensive care unit are obviously functional areas in which breaks in aseptic technique and environmental sepsis mutually fortify cross-infection. Here, too, special services such as inhalation therapy and migratory personnel such as the laboratory technician encroach on the bacteriologic integrity of the individual patient.

When sepsis becomes endemic, a search must be made for disseminating carriers among the personnel, including doctors and transient specialists of all divisions of labor. This is the
primary step in the control of exogenous wound infection. A record of periodic nasopharyngeal and skin cultures of personnel is of great assistance in spotting suspects as wound infections occur. This applies equally to gram-negative and gram-positive infections.

Aseptic technique must be expanded to include elimination of airborne bacteria. The most important single step is the exclusion of the disseminating carrier. Those with dermatitis must be included in this category. They not only shed bacteria, but when hands are involved they present the uncontrollable hazard of contact transmission.

Proper masking must be enforced. The most efficient filter type of mask is ineffective unless its edge is molded to the contours of the face to force expired air through the filter; loosely fitted masks are valueless. Masks must be discarded promptly upon removal from the face. Bacteria, accumulated in the mask, dry and are shed freely to the environment while the mask is dangling from the neck or perched on the forehead. When such a mask is reapplied to the face, contamination of the air can be prodigious.

Threshold sanitary ventilation must be achieved in order to rid the air of droplet nuclei. Ultraviolet irradiation is the simplest technique to bolster ventilation and decrease wound contamination, a perpetual threat because of the proximity of the surgeon's face to the surgical wound. To be effective, the lamps must be cleaned periodically to remove the film of dirt that filters the 2537 A germicidal rays. An energy level of 20 μw/sq cm at the level of the operating field is desirable. Because relative humidity exceeding 60 per cent renders ultraviolet radiation ineffective, this factor must be controlled. The air must be free from dust as demonstrated in the tyndall beam.

Hygienic air for ventilation is produced most effectively by filtration through properly installed and expertly maintained high-efficiency filter systems. The removal of airborne bacteria in the ventilated space depends upon the pattern of distribution of air in the room and the rate of air exchange.

Usually, air is introduced through diffusers in the ceiling or high in the walls that establish a turbulent pattern of air flow to exhaust points near the floor. Thirty or more changes of air per hour are required to purge bacteria dispersed from a localized source such as a carrier or a carelessly-handled dressing.

Attempts have been made to displace contaminated air downwards by diffusing the air at or through the ceiling and exhausting it symmetrically along the four baseboards. Less air is required to remove bacteria than with the turbulent system, but the downward piston effect is difficult to maintain during cooling phases of ventilation when doors are opened or while there is much activity.

The concept of laminar flow ventilation has been borrowed from industry. The air is introduced through the entire face of one wall or the ceiling and exhausted directly opposite through the entire face of the wall or floor. The rate of air exchange is secondary to the speed of the air in the field that is to be washed free of particulates and bacteria. Most laminar flow applications use air speeds higher than 60 feet/min. Speeds of 100–600 feet/min are used in industry. Recirculation of air through filter banks is customary to avoid the expense of cleaning and conditioning the huge mass of air that is moved. Because of the laminar flow pattern of fast streams of air about any obstruction, areas of negative pressure are created downstream of surgical lights, heads, hands or bodies. Bacteria are sucked into the eddy. Hence, laminar flow does not protect the surgical wound from bacteria shed from a carrier on the team.

Often attempts are made to isolate an individual operating room or the entire suite by maintaining positive pressure in the restricted area. These schemes fail because of the inversion convection that occurs each time a door is opened. The cold heavy air in the colder room displaces the air in the warmer room. The exchange between an operating room and the corridor may amount to ten air changes per hour. An ultraviolet barrier with an intensity of 25 μw/sq cm at the floor can be installed to isolate individual rooms or operating room corridor from the hospital.

Fear of accumulating an explosive atmosphere of flammable anesthetizing agents and recirculating pathogenic bacteria has led to
the dictum that 100 per cent outside air must be used in the operating room. Both assumptions are false. With proper filtration and humidification, air can be recirculated safely. The attendant economy permits high rates of air exchange that dilute and remove airborne bacteria. In an operating suite where wet vacuum cleaning is used and lint on textile is controlled, maintenance of filter banks is not expensive. Where dust is not suppressed, a series of roughing filters must be used to clean the air prior to its final polishing through high-efficiency filters. The rate of collection of bacteria on such filters equals the rate of the die-away except under conditions of high humidity and particulate contamination. When protected from dusty air, the pressure gradient across high-efficiency filters remains insignificant indefinitely and the flow of air is not impeded.

With this background, it is possible to assess the role of ventilation in the control of airborne wound infection. Ventilation in most operating rooms is too poor to be significant in diluting the bacterial population. With fewer than ten to 12 air changes per hour, emphasis must be placed on measures to control airborne contamination. The primary ones are hygiene practices to suppress dust, wet vacuuming to remove dust, effective gowns and masking to contain shedding from personnel, elimination of disseminating carriers, and the use of ultraviolet over the wound to complement the ventilation.

Recirculation of air makes it economically feasible to increase the rate of ventilation to a bacteriologically-effective level. This requires a minimum of 30 air changes per hour in rooms with the usual 4- to 5-man team. More crowded quarters require increased rates. Even with such ventilation the sources of bacteria must be suppressed to achieve acceptable bacterial counts in the air during activity such as draping or shifting position of the patient.

Threshold sanitary ventilation—dilution of airborne bacteria to the point where airborne infection does not occur—requires 60 changes of air per hour or its equivalent. This can be attained by complementing ventilation by internal recirculation through high-efficiency filters or by ultraviolet irradiation of the upper

<table>
<thead>
<tr>
<th>Type of Culture</th>
<th>Total Count</th>
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<tbody>
<tr>
<td>Settling plates on aseptic field</td>
<td>3.3 (±1.9) organisms/sq ft/min</td>
</tr>
<tr>
<td>Settling plates on floor</td>
<td>4.3 (±1.9) organisms/sq ft/min</td>
</tr>
<tr>
<td>Volume air samples</td>
<td>3.7 (±2.7) organisms/cu ft</td>
</tr>
<tr>
<td>Floor cultures</td>
<td>2.6 (±2.0)* organisms/sq cm</td>
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* Geometric mean.

air to destroy airborne bacteria in a turbulent distribution pattern.

One air change per minute may seem excessive, but it must be evaluated in the context that 15–20 air changes per hour are required to remove heat and control humidity when rigid criteria for comfort from draft and desiccation are met. One air change per minute is trivial compared with the 50 air changes per minute required to provide laminar airflow at speeds of 100 feet per minute, a common specification.

Rapid air flow may be noisy and uncomfortable. Perceptive design of diffusers and exhaust systems can obviate both objections. Recirculation also helps because tempering requirements and humidity adjustment are minimal. Hence, temperature gradients and evaporative cooling are minimal and the sensation of draftiness is avoided.

Of the several techniques for cleaning, disinfecting, tempering and humidifying air, one of special interest for the operating room depends upon the hydrosopic properties of a hypertonic solution of lithium chloride. The air is scrubbed by passing through a shower of the solution, as it is tempered by a series of heating or cooling coils. The vapor pressure of the solution determines the relative humidity of the air it contacts. Because the osmolarity is high, microbial growth is checked and contamination of the air by entrainment of contaminated condensate that forms on conventional refrigerating coils is avoided.
Bacteriologic standards for the operating room have evolved over recent years. Values in table 2 are readily attainable. The settling plates reveal the fallout of large particles; volume air samples give total count, including droplet nuclei.

It is argued that a ventilation system must be designed so that maintenance is not a critical factor in the bacterial safety of the air. There is no such system. Carelessness or ignorance defeat the purpose of any system designed to handle the volumes of air described in this presentation. Enforcement of practices that ensure hygienic air is a matter of administrative understanding and conviction. It is simple compared with development of aseptic or housekeeping techniques. Air can be recirculated safely through a sealed distribution system inexpensively enough to permit threshold sanitary ventilation for patients and personnel alike.

Conclusion

The control of airborne contamination consists primarily in the suppression of dust and bacteria shed from people. Where the source of bacteria is near the wound, such as a disseminating carrier on the team, irradiation of the surgical field with ultraviolet provides the maximal protection.

Well-designed, properly installed and maintained air conditioning can dilute and purge the residual bacteria from the operating room.

References


Obstetrics and Pediatrics

OBSTETRIC SEDATION In many obstetric centers, large doses of intravenous barbiturates are used as premedication in labor, the prime consideration being to insure that the patient is asleep and will not remember her labor. The actual sedative effect on the mother and the depressant effect on the newborn with this type of premedication were studied in a group of 203 parturients. The results showed that the patients were not well sedated in labor, and even when the dose of barbiturate was reduced markedly and a phenothiazine compound (propiomazine) added, there was little improvement in the quality of sedation. Comparing Apgar scores and times of sustained respirations of the newborn infants with a comparable group whose mothers had been premedicated with small doses of narcotic (meperidine) and phenothiazine (propiomazine) revealed a much more marked depressant effect in the barbiturate group. The price for amnesia in the mother is paid by the depressed newborn infant. (Batt, B.: Are Large Doses of Intravenous Barbiturates Justified for Use as Premedication in Labor? Amer. J. Obstet. Gynec. 102: 591 (Oct.) 1968.)