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Bacterial Interactions between Anesthesiologists, Their Patients, and Equipment

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FIFTY MILLION DOLLARS are spent every year in anesthesiology for single-use devices which are designed to prevent the transmission of bacteria. Yet the evidence that contaminated anesthesia equipment is responsible for postoperative infection is at best inconclusive. Certainly, in view of the 20 million anesthetics administered each year in the United States, it is surprising that there is no adequately documented study that unquestionably incriminates the anesthesia machine as the reservoir. The concept of breathing system transmission of infection has been debated continually since the advent of the modern anesthesia machine. Recent increased interest in cross-infection has been due to the dramatic increase in nosocomial infections, particularly those caused by gram-negative bacilli.[1] The elegant studies by Sanford and Pierce and others correlating necrotizing pneumonia with modern respiratory therapy procedures, particularly nebulization, unquestionably incriminated equipment as a main cause of morbidity and mortality.[1] From this correlation an inappropriate extrapolation occurred to general anesthesia equipment. The potentiality for anesthesia equipment to cause infection was never substantiated by good clinical studies. To begin to understand why anesthesia equipment has not been implicated in infection one must closely examine the environment presented to microorganisms. This environment consists of the patient's respiratory tract, including the intubated or non-intubated trachea and the anesthesia machine, generally the circle system. Within this enclosed environment a number of different factors together contribute to the overall risk of contaminating a machine with bacterial pathogens.[2] We will examine the following: 1) liberation of organisms from the airway of an infected patient; 2) inoculum size needed to infect a subsequent patient; 3) effect of aerosols (including droplet size and evaporation) on the viability of microorganisms; 4) effect of relative humidity on microorganism viability; 5) effect of anesthesia and oxygen on microorganism viability; 6) effect of metallic ions on microorganism viability; 7) effect of plastics on microorganism viability; 8) clinical investigations—transmission of bacteria from infected patients to the anesthesia machine; and 9) problems associated with acid-fast bacillary infections.

Liberation of Organisms from the Airway of An Infected Patient

Between 0 and 250 bacteria-bearing droplets exit the airway during speaking of an average sentence. Higher numbers are released during forceful speech—a compelling argument for calm in the operating room.[3] During quiet breathing, bacteria cannot be detected leaving the airway. In contrast, coughing and natural or artificial sneezing release between 3,500 and 1 million bacteria-bearing droplets from healthy test subjects per sneeze or cough. Subjects vary greatly. Individuals with bacterial pneumonia were not studied in these experiments. During anesthesia a state of quiet breathing is the most frequently encountered state and it is apparent that during this period very small numbers of organisms appear to be liberated.

Organisms that are released from the airway during violent forms of expiration originate largely from the anterior portion of the oropharynx.[3] Rarely are organisms that have originated from the nose or pharynx expelled. Most respiratory pathogens colonize the nose and pharynx of their host. This observation was supported in a study on three groups of patients suffering from streptococcal infections, diphtheria, or tuberculosis.[4] Patients were asked to cough on either a culture plate or microscope slide held 3 inches from their mouths. Only 39 of 87 patients harboring hemolytic streptococci expelled organisms that were detectable with an average of two infected droplets in each cough. Ten of 50 patients with diphtheria released diphtheria bacilli during voluntary coughing. Half the patients with open pulmonary tuberculosis released acid-fast bacilli, however, only 36 of 410 droplets collected in 120 coughs contained these bacilli. Tubercle bacilli were the only organisms of the group studied that were found in secretions from the anterior half of the mouth.

Inoculum Size Needed to Infect a Subsequent Patient

Organisms to be somehow conveyed to a susceptible patient via a contaminated anesthesia circuit must survive

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in large enough numbers to establish colonization and infection. Not only must the infecting dose be sufficient to induce infection, but the patient must have sufficiently depressed host resistance to facilitate colonization and infection. In fact, however, airborne infection is a relatively difficult event to carry out experimentally. In animal studies comparing various routes of infection with gram-negative bacilli the respiratory tract requires more organisms to initiate infection than other routes including intraperitoneal, subcutaneous, intracerebral, intravenous, intracutaneous, and percutaneous.[5] In reality, patients with severely compromised lungs are more susceptible to infection because of their damaged host defense mechanisms, such as ciliary clearance and alveolar macrophages.[6] A smaller inoculum in these patients can lead to colonization and infection. If surgery and anesthesia can be postponed until the patient's host defenses have improved, the chance of infection from the anesthesia machine again becomes negligible.[6]

Effect of Aerosols (Including Droplet Size and Evaporation) on the Viability of Microorganisms

In the 1930s it was observed that droplets ejected into the air evaporated while they were falling. The solid residue of the evaporated droplets was termed "droplet nuclei."[7] Bacteria carried in this way can be transmitted considerable distances and constitute the greatest risk of infection. The size of droplets produced by the airways will vary depending upon the velocity of the gas, surface tension of the fluid comprising the droplet, and the relative humidity. An increase in velocity brings about a decrease in droplet size. In a sneeze, for example, air velocity approaches 300 m/s.[8] While a great range in droplet sizes is produced, the mean droplet diameter approximates 10μ. Normal breathing produces a small number of very large diameter droplets, not containing bacteria, which tend to fall rapidly out of the gas stream, depending upon the humidity of the surrounding environment.[9,10] *In vitro* studies of evaporation have relied upon pure water droplets as the vehicle of transmission. In reality, saliva and respiratory secretions contain a solute (1 per cent) which retards evaporation.[10] Temperature and humidity conditions commonly found within the anesthesia circuit further slow down the process. Production of droplet nuclei in this situation is rare. The considerable accumulation of moisture on the expiratory side of the anesthesia circuit results from collection of large diameter droplets. Gravitational pooling of these large droplets is seen farther down the expiratory side. Evaporation that does take place cools any bacteria severely depressing their metabolic function.[11,12]

Effects of Relative Humidity on Microorganism Viability

Airborne bacteria released into the circle system are exposed to extreme shifts in humidity. During anesthesia microorganisms are subjected to relative humidities ranging from 70 to 100 per cent depending on their location within the circle system. When the source of humidity is removed after the patient has been disconnected from the anesthesia machine, the internal humidity of the machine reverts to that of the ambient environment (typically 50 per cent in the operating room). Shifts in relative humidity are usually accompanied by shifts in temperature. Bacteria, particularly the gram-negative organisms, are sensitive to both these changes. Shifts in temperature and relative humidity are probably responsible for the greatest percentage of bacterial killing within the anesthesia circuit.[13-17] The lethality caused by humidity variations was confirmed by measurement of viability in cultures of *E. coli* maintained in different media and sprayed into the atmosphere as aerosols.[18,19] As the relative humidity increased from 60 per cent, viability decreased. For example, at a relative humidity of 86 per cent only 1.3 per cent of the original inoculum was still viable. Less than 1.0 per cent of the original inoculum was alive at a relative humidity of 96 per cent. Viability of *E. coli* never exceeded 35 per cent when the relative humidity was 76 per cent or higher. Killing is magnified in the presence of increasing concentrations of oxygen.[20] Most aerosols of *E. coli* are killed within the first half second after aerosolization.[21] The mechanism of rapid killing by increasing relative humidity is postulated to involve the sudden displacement of essential hydrogen bonding between water molecules and membrane bound proteins. This displacement by added water markedly affects bacterial viability. It is important to stress that the physical parameters of temperature, relative humidity, evaporation, and the abrupt changes in these factors that normally occur within the anesthetic circuit act in concert to affect bacterial viability.

Effects of Anesthesia and Oxygen on Microorganism Viability

Most studies have shown that general anesthetics have little influence on the survival of bacteria.[22-25] Representative of these findings is one showing no effects upon the viability of *Staphylococcus aureus, E. coli,* and *Pseudomonas aeruginosa* of clinical concentrations of halothane, methoxyflurane, diethylether, and trichloroethylene.[22] Bacteria exposed to halothane in clinical concentrations show similar effects although a probably unimportant dose-dependent depression of growth was observed upon exposure to concentrations ranging from 1 to 10 per cent[24] The effect of anesthetics on bacteria also has been studied when these organisms were deposited on simulated anesthesia equipment surfaces. Test strains of *Streptococcus pneumoniae* and *Hemophilus influenzae* were deposited upon cellulose acetate filters prior to exposure to anesthetic gases. Halothane, methoxyfluorane, and trichloroethylene in concentrations

varying from 1 to 3 per cent were tested. After exposure for 2, 3, and 4 hours killing was measured by counting colony-forming units appearing on the filter surfaces. Between 25–60 per cent of the original inoculum of *S. pneumoniae* were killed, depending in small part upon the concentration and type of anesthetic. Twenty to 60 per cent of *H. influenzae* bacilli were similarly killed.[25]

Halothane is detectable within the anesthesia circuit for many hours, even after removal of soda lime and rubber tubing.[26] This implies that organisms deposited by a patient into the anesthetic circuitry would still be exposed to the slightly deleterious effects of halothane.

Oxygen is the one gas employed in anesthesia practice that might be thought not to be deleterious to the survival of the facultative anaerobic organisms such as the gram-negative bacilli. Sufficient work has been done to show that exposure to concentrations of oxygen as low as 1 per cent are lethal to bacteria suspended in aerosols. Obligately anaerobic bacteria, as one might expect, are killed quickly and present no problem to the patient.[20,27,28] When organisms are airborne they are particularly susceptible to the effects of the inspired oxygen concentration given with the general anesthetic. These concentrations of oxygen, since they are deleterious to bacteria, provide a protective mechanism which partly counterbalances the depression of alveolar macrophage function caused by inspired oxygen concentrations above 21 per cent.[29]

Effect of Metallic Ions on Microorganism Viability

Bacteria entering the anesthetic circle find a significant portion composed of metal surfaces. Metals most commonly found within circuits include chromium, zinc, and copper; the latter two forming the alloy, brass. Ionic forms of these three metals have oligodynamic properties that by definition prove toxic in extremely small concentrations to vegetative bacterial cells.[30–32]

In order to transport iron, for example, bacteria require a class of iron-binding proteins known collectively as siderophores. In the presence of trivalent chromium ions this function meets interference, resulting in the precipitation of iron. This action prevents the growth of bacteria.[33] Zinc will be bound by heterocyclic cell constituents such as the purine bases or riboflavins, and this combination will interfere with cell function.[31] Copper provides the most dramatic example of a metal capable of poisoning by chelation, the actively metabolizing bacterial cell.[31] The high affinities of these heavy metals for essential amino acids cause the formation of structures which effectively inactivate the amino acids. One example is the reaction of the cupric ion with glycine. Histidine and cysteine also react with copper in much the same way. Phosphoric acid derivatives, including the acids of the tricarboxylic acid cycle, can also be bound, and thus inactivated by metals.

A practical use has been found for the oligodynamic properties of copper in the protection of patients from infection from contaminated respiratory therapy equipment. Inserting copper mesh or sponges into the expiratory limb of the ventilator prevents levels of bacteria from increasing to worrisome numbers. A number of studies have demonstrated the effectiveness of this use.[34–36]

Effect of Plastics on Microorganism Viability

The use of plastic and other petroleum-derived synthetic materials has become almost universal in anesthetic practice. The interaction of microorganisms with these compounds has received little attention by anesthesiologists, despite studies by the plastics industry.[37–39] Most anesthesia-related plastics are composed of either polyvinyl chloride (PVC) or high density polyethylene (>5 million MW). Phthalate ester plasticizers, stabilizers, and mold release compounds are commonly used in the production of PVC materials. The materials act as substrates for bacterial growth.[37–40] In one study *Serratia marcescens* was observed to degrade diethylhexyl phthalate.[37] Microbial degradation of plasticizers and stabilizers leads to a decrease in flexibility (modulus) and loss of product quality in PVC materials.[38] These materials are not used in the production of polyethylene-containing devices. Materials manufactured with polyethylene and polypropylene have also been found to be biodegradable by bacterial and fungal species only when exposed to previous photodegradation.[39]

Clinical Investigations—Transmission of Bacteria from Infected Patients to the Anesthesia Machine

We determined whether the anesthesia machine and circle system could be a significant source of bacterial contamination in cross-infection in anesthetized patients. We also investigated how best to interrupt this mode of transmission.[40] Fifteen patients were placed into two groups: six colonized with gram-negative bacilli and nine not colonized. Colonization was defined as isolation of 10 or more colony forming units of gram-negative bacilli from material from the throat or sputum on two or more consecutive days. Before each case, cultures were taken from various locations on each anesthesia machine. After each anesthesic procedure cultures were again taken of the same locations. In addition, corrugated tubing was divided into 6.4-cm segments and cultured semi-quantitatively. The level of bacterial contamination for all segments was between 1–9 colony-forming units per segment with the species most commonly isolated being gram-positive environmental saprophytes. Low numbers of gram-negative bacilli, specifically *Acinetobacter calcoaceticus*, Flavobacterium species, and *P. aeruginosa*, were found in corrugated tubing immediately after each procedure on two patients previously colonized with these organisms. Gram-negative bacilli were recovered from the corrugated tubing used on three previously uncolon-

ized patients; *Acinetobacter calcoaceticus* was isolated in low numbers from the expiratory tubes used on two patients, and one colony of *Pseudomonas maltophilia* was recovered from a single segment of expiratory tubing used on a third patient.

No positive culture was recovered from any site within the anesthesia machines used on the six colonized patients. Following administration of anesthesia to the uncolonized group only three positive cultures were obtained from six anesthesia machines: the first from the condensate at the bottom of the CO_2 absorber (*Staphylococcus epidermidis*), the second from a ventilator connection port (*S. epidermidis*), and the third from the expiratory port (unidentified gram-negative bacillus). Statistical analysis of all data indicated that the contamination pattern was consistant with a totally random distribution along the corrugated tubing. There was no indication that patients colonized with gram-negative bacteria expired these organisms into the circuitry with any regularity or in any quantity.

The introduction of bacterial filters and disposable circuits by a number of manufacturers has added a substantial cost to the patient. Other than those studies published by the designers of bacterial filters, there have been two clinical studies which assess the incidence of postoperative pneumonia in patients using filtering systems or disposable tubing—those of Garibaldi *et al.*[41] and Feeley *et al.*[42] In Garibaldi's study 520 patients were randomized prior to surgery. One group was anesthetized with circuits interposed with two 0.22-μ low-resistance filters at both inspiratory and expiratory ports of the absorber canister. The control group was anesthetized on circuits with no filters. The investigators compared postoperative pneumonia, postoperative fever, the presence of abnormal chest x-rays, and sputum production in both groups. There were no statistically significant differences found in any comparison.

In the Feeley study 293 patients scheduled for major surgical procedures were studied in two randomized groups. One group received anesthesia through a sterile disposable circuit, the other through a clean, unsterile reusable circuit. There was no significant difference in the postoperative development of respiratory infection.

Case reports have twice implicated bacterial filters in incidents of hypoxia. Schwartz describes a case of hypoxia during anesthesia on a patient undergoing open heart surgery.[43] During cardiopulmonary bypass the patient became increasingly cyanotic. Rapid removal of the filters from the circuit corrected the problem. It was later determined that cracks in the filter housing allowed gas to follow the path of least resistance and escape to the environment. A second report describes obstruction of the bacterial filter by edema fluid, a situation that also required the rapid removal of the filter.[44]

Municipal water supplies can deliver pathogenic organisms. We have recently described outbreaks of colonization and infection of surgical patients in which tap water contamination played a role.[45–48] Anesthesia circuits can be contaminated by tap water.

Problems Associated with Acid-fast Bacillary Infections

There has been no documented transmission of tuberculosis from a contaminated anesthesia machine to a patient. Due to the growth characteristics of acid-fast bacilli this type of documentation would be difficult to establish. Nevertheless, of all bacterial forms, the acid-fast bacilli are the most adaptable to adverse environments and have a particular resistance to dessication. Not only is *M. tuberculosis* of concern, but recently, increased incidences of the non-tuberculous mycobacteria have been reported. Diseases due to these organisms are insidious and difficult to treat with current antimycobacterial drugs. Most require surgical resection. In addition, transmission of these organisms occurs by environmental contact and not by person to person contact. Tap water has been implicated as the prime vehicle of transmission. We have documented nosocomial transmission of these organisms to patients via the hospital water supply, particularly during the summer months when numbers of these chlorine-resistant organisms are high. The anesthesiologist, when hand-washing, invariably comes in contact with these ubiquitous organisms and can presumably transmit them to patients. Since 1972 there has been a fourfold increase in pulmonary infection in adults and lymphadenitis in children due to the *M. avium-intracellulare* bacilli in Massachusetts.[49] Because of the increasing incidence of these infections[50] the Centers of Disease Control recently have begun to assemble epidemiologic data regarding these organisms.[51]

A greater degree of vigilance should be observed by anesthesiologists in dealing with patients with pulmonary disease conceivably due to acid-fast organisms.[52] This vigilance should include: 1) Initial sterilization or disinfection with appropriate chemicals, for example, activated glutaraldehyde, of nondisposable equipment after use. Equipment must subsequently be washed thoroughly. One must keep in mind that washing with tap water will recontaminate this equipment with waterborne organisms which may include acid-fast bacilli. Finally, wrapping, labeling, and terminal sterilization will follow. 2) Use of a disposable circle system and absorber may be helpful. 3) All operating room staff should be aware that the case is "contaminated." 4) Whenever possible, the patient should wear a mask. 5) All gowns, gloves, shoe covers, and masks should be appropriately discarded upon leaving the operating theatre. 6) Traffic and unnecessary equipment within the operating room should be kept to a minimum. 7) The anesthesiologist should be gloved and keep intraoral manipulation to a minimum.

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