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Transmission of Pathogenic Bacterial Organisms in the Anesthesia Work Area

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Background: **The current prevalence of hospital-acquired infections and evolving amplification of bacterial resistance are major public health concerns. A heightened awareness of intraoperative transmission of potentially pathogenic bacterial organisms may lead to implementation of effective preventative measures.**

Methods: **Sixty-one operative suites were randomly selected for analysis. Sterile intravenous stopcock sets and two sites on the anesthesia machine were decontaminated and cultured aseptically at baseline and at case completion. The primary outcome was the presence of a positive culture on the previously sterile patient stopcock set. Secondary outcomes were the number of colonies per surface area sampled on the anesthesia machine, species identification, and antibiotic susceptibility of isolated organisms.**

Results: **Bacterial contamination of the anesthesia work area increased significantly at the case conclusion, with a mean difference of 115 colonies per surface area sampled (95% confidence interval [CI], 62–169; $P < 0.001$). Transmission of bacterial organisms, including vancomycin-resistant enterococcus, to intravenous stopcock sets occurred in 32% (95% CI, 20.6–44.9%) of cases. Highly contaminated work areas increased the odds of stopcock contamination by 4.7 (95% CI, 1.42–15.42; $P = 0.011$). Contaminated intravenous tubing was associated with a trend toward increased nosocomial infection rates (odds ratio, 3.08; 95% CI, 0.56–17.5; $P = 0.11$) and with an increase in mortality (95% CI odds ratio, 1.11–∞; $P = 0.0395$).**

Conclusions: **Potentially pathogenic, multidrug-resistant bacterial organisms are transmitted during the practice of general anesthesia to both the anesthesia work area and intravenous stopcock sets. Implementation of infection control measures in this area may help to reduce both the evolving problem of increasing bacterial resistance and the development of life-threatening infectious complications.**

HOSPITAL-ACQUIRED (nosocomial) infections now affect 10% of patients admitted, and the amplification of bacterial resistance is an evolving problem worldwide.[1,2] As a result, community and hospital outbreaks of infections secondary to resistant organisms are occurring at increasing frequency.[3] In addition, while infections due to resistant bacterial strains were primarily associated with acute care settings, they are now occurring at increased rates within the community.[3] It is well known that intensive care units are epicenters of bacterial resistance, but little is known about the role of the anesthesia environment and anesthetic practice in this process.[1]

This article is featured in "This Month in Anesthesiology." Please see this issue of ANESTHESIOLOGY, page 9A.

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For several reasons, the intraoperative environment serves as a risk factor for the development of hospital-acquired infections.[4–10] This, combined with evidence that general anesthesia is associated with immune suppression, suggests that anesthetic practice as a whole may also be linked to the development of hospital-acquired infections.[11,12] Given the current increasing community awareness surrounding this issue and the impending arrival of pay for performance policies, there is further impetus to develop preventative measures. A better understanding of the underlying mechanisms by which bacterial transmission occurs and resistance is increasing may facilitate this process.

The intraoperative environment includes both aerosolized particles and healthcare tools used within the anesthesia work area. This is theoretically associated with the development of nosocomial infections, but there is a lack of objective evidence linking these factors with direct transmission of bacterial organisms to patients.[4,7,9,13]

The mechanism by which these factors generate nosocomial infections remains unclear. However, there is a high probability of patient contamination during the practice of anesthesia due to rapid patient care combined with frequent contact with potential sources of bacterial transmission. In addition, transmission of multidrug-resistant bacterial organisms likely occurs but has not yet been described.

This paucity of objective evidence has led to the belief that the anesthesia work area and anesthetic practice have no role in the development of hospital-acquired infections or amplification of bacterial resistance. As such, we currently have inadequate implementation of infection control measures in this area.[14–16] The aim of this study was to characterize the risk of bacterial transmission during the practice of general anesthesia to stimulate important measures for risk modification and reduction of hospital-acquired infections. We hypothesized that intraoperative bacterial contamination of the anesthesia work area was associated with contamination of peripheral intravenous stopcock sets, partially explaining the association of general anesthesia with the development of nosocomial infections.

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Materials and Methods

Patients

All work in this study was performed at Dartmouth-Hitchcock Medical Center, a tertiary care and level 1 trauma center for the state of New Hampshire with 400 inpatient beds and 28 operating suites. Approval was obtained from the institutional review board for the protection of human subjects following expedited review with a waiver for informed patient consent (Lebanon, New Hampshire). Over 6 days, 61 operating rooms were randomly selected by computer-generated analysis, and data were collected for the first person of the day cared for in that room.

Pilot

We performed a pilot study to identify specific sites within defined areas of the anesthesia work space that were consistently contaminated by anesthesia providers during anesthesia. The agent flowmeter dial and the adjustable pressure-limiting valve complex on the Datex-Ohmeda (GE Healthcare, Piscataway, NJ) anesthesia machine (fig. 1A) consistently yielded the highest bacterial counts. An additional finding was that terminal intraoperative decontamination strategies used in the anesthesia work area proved to be ineffective in the majority of cases.[17] The terminal decontamination strategy used at our institution includes spraying anesthesia machine and monitor surfaces with a quaternary ammonium disinfectant (Dimension III; Butcher's, Sturtevant, WI). These surfaces are wiped down after a variable period of time, often less than the manufacturer's recommendation of 10 min.

Protocol

Sixty-one operative suites were randomly selected for analysis by a computer-generated list. One patient to receive general anesthesia according to usual practice was followed in each operative suite during the first case for the day. Each patient received a sterile set of intravenous tubing and stopcock set, 24 inch with three gang four-way and T-Connector (SetSource, San Clemente, CA), by nursing staff in the preoperative holding area. Alternatively, the sets were provided by anesthesia providers intraoperatively upon arrival from the inpatient or intensive care units. Culture of the intravenous stopcock set immediately upon removal from the packaging material was invariably negative.

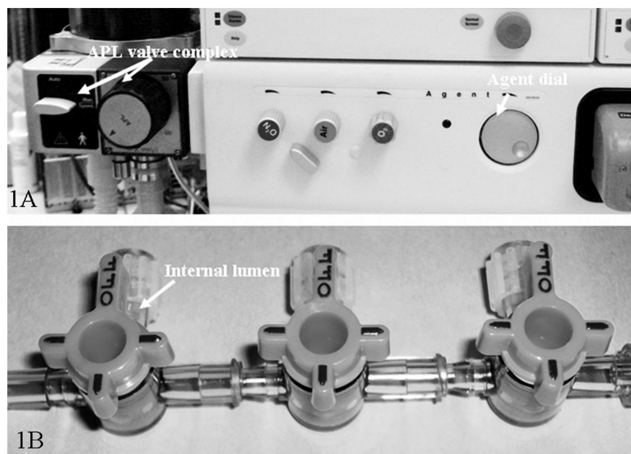

Fig. 1. (*A* and *B*) Sites where the anesthesia machine and the stopcock set were sampled. APL valve = adjustable pressure-limiting valve.

The adjustable pressure-limiting valve complex and agent dial were sanitized with the current intraoperative quaternary ammonium disinfectant (Dimension III) solution according to protocol. Baseline cultures were then obtained from these sites at time 0 and once again after completion of the case but before disinfection according to current protocol (time 1). In addition, the internal lumens of all three intravenous stopcock ports were cultured by completion of the surgical case (time 2).

The intravenous stopcock sets (fig. 1B) were shown initially to be consistently sterile. Time 0 cultures were considered to represent a baseline, such that any new pathogen cultured at the end of surgery was presumed to be acquired in the operating room. The primary outcomes were bacterial colonies per surface area sampled (CPSS) above baseline (time 1) and number of positive stopcock sets at time 2. A positive stopcock set was defined as greater than or equal to one colony per surface area sampled, consistent with previous study protocols.[18,19] The number of anesthesia providers, level of training, surgical procedure and duration, anesthesia type and duration, American Society of Anesthesiologists physical status, age, and sex of patients were also recorded. All patients with contamination of intravenous stopcocks were assessed retrospectively to evaluate for the presence of nosocomial infections documented by primary care providers and associated mortality.

Laboratory Investigations

Sampling of the Anesthesia Environment (Time 0 and Time 1). After decontamination of the adjustable pressure-limiting valve complex and agent dial with Dimension III disinfectant solution according to the manufacturer's recommendations, baseline cultures were obtained by using sterile polyester fiber–tipped applicator swabs moistened with sterile transport medium (BactiSwab; Remel, Lenexa, KS) to roll several times over the selected areas followed by culturing on sheep blood agar plates with a zigzag pattern and swab rotation to detect both gram-positive and gram-negative bacteria.[20]

Sampling of Peripheral Intravenous Tubing (Three-way Stopcocks [Time 2]). A sterile nasopharyngeal swab (BactiSwab) moistened with sterile transport medium was inserted into the internal surfaces of each port of the three-way stopcocks and rotated 360° ten times to culture. Each swab potentially containing bac-

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teria from any of the three lumens of the single stopcock set was then inoculated onto a sheep blood agar plate using a zigzag pattern and swab rotation.[20]

Microbial Culture Conditions. All blood agar plates were incubated at 35°C for 48 h, and microorganisms were quantified according to CPSS and identified according to standard laboratory methods as described below.

Bacterial Identification. Bacterial organisms found within the anesthesia work area but without associated stopcock contamination and/or hemolysis were presumptively identified by colony morphology, Gram stain, and simple rapid tests. All organisms associated with stopcock contamination and/or hemolysis underwent further identification as described below.

Gram-positive organisms were identified using the Dade Behring MicroScan (San Diego, CA) Positive Identification type 2 panel intended for identification of rapidly growing aerobic and facultative gram-positive cocci (some fastidious aerobic gram-positive cocci and *Listeria monocytogenes*). Organism identification was based on modified conventional and chromogenic tests using pH changes, substrate utilization, and growth in the presence of antimicrobial agents after 16–44 h of incubation at 35°C.

Recovered organisms were identified by standard clinical microbiology techniques supplemented by chromogenic panels (Dade Behring Microscan) and antimicrobial susceptibility by broth microdilution (Dade Behring Microscan) or Kirby-Bauer disk diffusion. Methicillin-resistant *Staphylococcus aureus* (MRSA) and vancomycin-resistant enterococcus (VRE) were confirmed by agar dilution minimal inhibitory concentration.[21]

Pulsed Field Gel Electrophoresis. The pulsed field gel electrophoresis protocol used was developed by the Orange County Public Health Laboratory (Santa Rosa, CA) and was based on the Centers for Disease Control and Prevention PulseNet Standardized Laboratory Protocol for Molecular Subtyping of *Escherichia coli* O157:H7 by Pulse Field Gel Electrophoresis (June 2004).

Retrospective Analysis of Nosocomial Infections. We reviewed all 61 patients with regard to the development of nosocomial infections and/or mortality over 30 postoperative days. We identified nosocomial infections based on the National Nosocomial Infection Surveillance System report.[1] We evaluated all patients for additional factors that could potentially explain the development of nosocomial infections, including temperature and glycemic control, inspired oxygen concentration, and prophylactic antibiotic therapy.[22]

Statistical Analysis

The primary outcome in this study was the presence of a positive culture from the peripheral intravenous tubing (stopcock). Secondary outcomes were the number of colonies on the anesthesia machine, bacterial speciation and resistance patterns, and morbidity and mortality related to stopcock contamination.

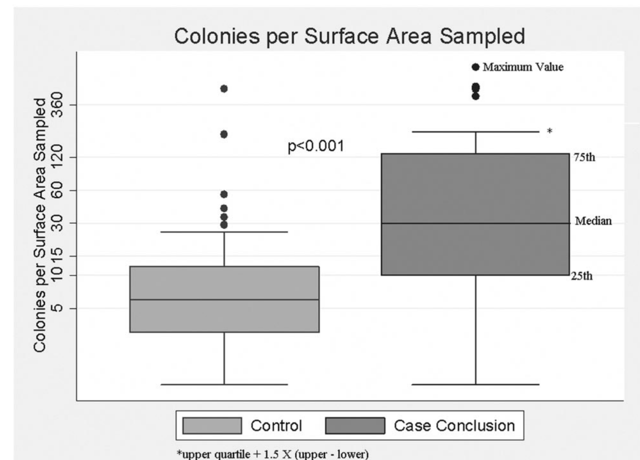

Fig. 2. Box plot illustrating increase in intraoperative contamination on the anesthesia machine as measured by colonies per surface area sampled. $P < 0.001$ for comparison of medians. Ninety-five percent confidence interval, −83.649 to 353.485.

Standard techniques for continuous variables and an unadjusted chi-square test for binary variables were used for the univariate analysis. We considered the number of colonies on either the stopcock or the two anesthesia sites as a Poisson process. Generalized linear models with either logistic link or log link were used to model binary and count data, respectively. Covariates used for adjustment included the duration of surgery; type of anesthesia (general or sedation); level of training of the provider; patient's age, sex, and health status (American Society of Anesthesiologists physical status); and the emergent nature of the procedure. A P value of 0.05 was taken to indicate statistical significance without adjustment for multiple comparisons. Ninety-five percent confidence intervals (CIs) are reported. Stata (StataCorp, College Station, TX) was used to conduct all statistical analyses.

We hypothesized a baseline rate of culture positive peripheral intravenous tubing (stopcock set) to be approximately 20% and considered the number of colonies recovered from the anesthesia work area as the primary predictor of a positive stopcock. Assuming an odds ratio of at least 2, approximately 50 sites would provide a power of 0.95 with a type I error rate of 0.05.

Results

During the study period, a total of 61 patients undergoing anesthesia were enrolled. Patients underwent a variety of surgical procedures, including general (30%), pediatric (15%), orthopedic (13%), gynecologic (11%), cardiothoracic (10%), otolaryngologic (10%), neurosurgical (5%), urologic, (3%) and vascular (3%).

As shown in figure 2, the anesthesia work area, which was represented by sampling the adjustable pressure-limiting valve and agent dials intraoperatively, became

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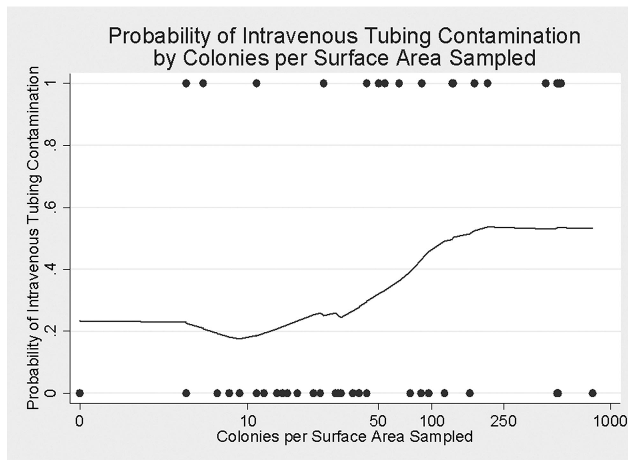

Fig. 3. Probability of obtaining bacterial growth in the stopcock as a function of the number of colonies per surface area sampled from the anesthesia machine.

contaminated at case conclusion above that of baseline controls, with a mean increase of 115 (median increase 24) CPSS ($P < 0.001$). Contamination occurred in cases lasting as little as 4 min. There was no apparent association between magnitude of contamination and duration of the surgical procedure based on a regression analysis of CPSS by time (results not shown).

Peripheral intravenous tubing (stopcock sets) became contaminated with potentially pathologic bacteria in 32% of cases (95 CI, 20.6–44.9%). As the bacterial burden as measured by CPSS increased, so did the probability of obtaining a positive stopcock. Figure 3 demonstrates the relation between CPSS in the work area and intravenous tubing contamination. For workspace contamination at approximately 10 CPSS, the probability of contamination is approximately 20%, but this increases to greater than 50% when the CPSS is more than 100. This occurred in 30% of cases.

Table 1 details the demographic differences between patients with and without a positive stopcock and demonstrates only a statistically significant difference with age. There were no significant differences in contamination based on sex, American Society of Anesthesiologists physical status, surgical procedure, urgency, patient location before the operation, anesthesia type, and case duration. There was no significant difference in contamination rates between anesthesia resident physicians, attending physicians, and non-M.D. anesthesia providers. Table 2 provides the result of the logistic regression adjusting for CPSS at baseline and the previously mentioned covariates. Even with adjustment for these factors, increased intraoperative contamination as per CPSS is associated with intravenous contamination (odds ratio, 1.67; 95% CI, 1.10–2.53; $P = 0.02$).

Table 3 characterizes the bacterial species found in the anesthesia work area and peripheral intravenous stopcocks. Of note are two cases where MRSA was transmitted to the anesthesia work area intraoperatively. Further, one case of VRE transmission occurred to both the anesthesia work area and the intravenous stopcock set. This relation was confirmed by DNA analysis using pulsed field gel electrophoresis (fig. 4). The additional organisms identified are described in table 3.

A retrospective chart review over 30 postoperative days revealed a nonsignificant increase in nosocomial infection rates (odds ratio, 3.08; 95% CI, 0.56–17.5; $P = 0.18$) and a significant increase in mortality (0 of 41 *vs.* 2 of 20; 95% CI, 1.11–∞; $P = 0.0395$) in those patients with contaminated intravenous stopcock sets. Five of 20 patients (25%) with contaminated stopcocks developed

Table 1. Comparison of Characteristics between Patients with Positive Stopcocks and Patients with Clean Stopcocks

	Positive Stopcock (n = 20)		Clean Stopcock (n = 41)		Comparison	
	Percent Mean	n or SD	Percent Mean	n or SD	OR Difference	95% CI
Location						
ICU	15.0%	3	9.8%	4	1.63	(0.21 to 10.76)
Inpatient unit	10.0%	2	9.8%	4	1.03	(0.09 to 7.97)
Same day	75.0%	15	80.5%	33	0.73	(0.17 to 3.34)
ASA physical status						
I	15.0%	3	29.3%	12	0.43	(0.07 to 1.93)
II	25.0%	5	39.0%	16	0.52	(0.12 to 1.92)
III	40.0%	8	24.4%	10	2.07	(0.56 to 7.46)
IV	20.0%	4	7.3%	3	3.17	(0.47 to 23.65)
Sex (male)	50.0%	10	56.1%	23	0.78	(0.23 to 2.62)
Emergent status						
Elective	75.0%	15	85.4%	35	0.51	(0.11 to 2.51)
Urgent	25.0%	5	14.6%	6	1.94	(0.40 to 8.94)
Training (resident)	50.0%	10	73.2%	30	0.37	(0.10 to 1.30)
Age, yr	59.2	15.2	47.0	26.4	12.2*	(1.5 to 22.9)
Duration, h	2.1	1.8	2.5	2.5	−0.4	(−1.6 to 0.7)
Colonies per surface area sampled	39.5	116.8	9.7	12.7	29.8	(−25.0 to 84.6)
Stopcock (propofol or blood)	45.0%	9	26.8%	11	2.23	(0.62 to 7.84)

ASA = American Society of Anesthesiologists; CI = confidence interval; ICU = intensive care unit; OR = odds ratio.

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Table 2. Logistic Regression to Predict Probability of a Positive Stopcock

Variable	Odds Ratio	95% CI	*P* Value
Unadjusted			
Colonies per surface area sampled (intraoperative)	1.49*	(1.06–2.09)	0.02
Adjusted			
Colonies per surface area sampled (intraoperative)	1.67*	(1.10–2.53)	0.02
Colonies per surface area sampled (baseline)	1.02	(0.62–1.67)	0.94
Location of patient			
ICU	Reference		
Inpatient unit	3.50	(0.06–210.32)	0.55
Same day	8.07	(0.12–530.73)	0.33
ASA physical status			
I	Baseline		
II	1.08	(0.14–8.12)	0.94
III	5.32	(0.47–60.38)	0.18
IV	11.58	(0.21–630.06)	0.23
Duration, h	0.77	(0.52–1.15)	0.2
Age, yr	1.02	(0.98–1.06)	0.41
Sex (male)	0.86	(0.22–3.28)	0.82
Level of training*	0.38	(0.08–1.88)	0.24

* Level of training: resident. Comparison group is certified registered nurse anesthetists and attendings.

ASA = American Society of Anesthesiologists; CI = confidence interval; ICU = intensive care unit.

nosocomial infections, including ventilator-associated pneumonia (2), wound (2), and bloodstream (1) infections. Two patients in this group ultimately died after a prolonged stay in the intensive care unit secondary to bloodstream (1) and respiratory (1) infections. Five of 41 patients (12%) without stopcock contamination developed nosocomial infections, including urinary tract (1), bloodstream (1), and superficial wound (2) infections. There were no patient deaths in this group. Two of 3 patients (66%) associated with transmission of multidrug-resistant organisms to the anesthesia work area and/or peripheral intravenous tubing died after intensive care unit stays.

There were no significant differences in intraoperative temperature, glucose control, or prophylactic antibiotic administration in those patients with nosocomial infections as compared with those without. As compared with patients without nosocomial infections, patients with nosocomial infections received a higher concentration of inspired oxygen intraoperatively, with a mean of 89% *versus* 75%, respectively, with a mean difference of 14% (95% CI, 2–26; $P \leq 0.02$).

Discussion

Infection due to hospital-acquired resistant bacterial strains is an evolving problem worldwide, and horizontal transmission of bacterial organisms continues to maintain a high nosocomial infection rate in acute care settings, impacting 10% of patients admitted. Outbreaks are occurring with increasing frequency and have increased morbidity and mortality through infectious complications such as pneumonia, surgical wound, and vascular access–related bacteremia. Intensive care units are now becoming epicenters of antibiotic resistance, with VRE and MRSA the pathogens of greatest concern.[23–29]

Although infections due to resistant bacterial organisms were once localized to hospital settings, they are now occurring within the community at alarming rates, and this is now a major public health concern.[3]

Amplification of bacterial resistance occurs primarily through the combination of environmental contamination and poor aseptic practice. This has led to increased prevalence of antimicrobial-resistant organisms in surgical site and central venous catheter–related bloodstream infections. Surgical site infections are the most common adverse event (3%) occurring in surgical patients within a 30-day postoperative period, whereas the prevalence of catheter-related bloodstream infections ranges from 3% to 7%.[5,23,30–33] This problem is not limited to multidrug-resistant or common pathogens. Environmental organisms such as *Acinetobacter* species have been associated with clinically relevant catheter-related bloodstream infections in immunocompromised patients and are associated with significant morbidity and mortality, substantial cost, and consumption of limited resources.[34,35] Mortality associated with catheter-related bloodstream infections is reported at 15%, with an estimated cost of $9,000 per episode.[36–39]

Negative outcomes occurring secondary to bacterial transmission impact all healthcare providers.[40] Epidural catheters used for short-term postoperative analgesia have been found to be contaminated after insertion, the most common organism being coagulase-negative staphylococcus. Similar to central line catheters, the most common route of epidural catheter colonization is bacterial migration along the epidural catheter track from the skin insertion site. Therefore, maintenance of skin sterility around the insertion site through strict adherence to aseptic practice is necessary to prevent serious, life-threatening infectious complications.[41] Bacterial meningitis occurring secondary to poor compliance with preventative measures during epidural catheter placement has been described.[42]

Overall, hospitalized patients are becoming more vulnerable to these infections because of aging and more aggressive medical and surgical interventions. Evidence suggests that more emphasis should be placed on identification of modifiable risk factors and implementation of strategies designed to reduce transmission of infectious organisms hospital-wide.[5,40] Three main techniques are important to prevent infection transmission from the provider to the patient. These include aseptic practice, proper hand hygiene, and appropriate barrier techniques as recommended by the Centers for Disease Control and Prevention.[40]

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Table 3. Identified Bacterial Sample Aggregates in Patients with Growth from Stopcocks

Patient Sample	Operative Suite Type	Sample Time and Location	Organism(s) and Biotype
2A	Cardiac	T1APL valve	Alpha *Streptococcus* species
B	Cardiac	Stopcock set	Coagulase-negative *Staphylococcus* species
C	Cardiac	T1 agent dial	*Enterococcus* species
4A	General	T1 APL valve	*Micrococcus* species
B	General	Stopcock set	*Micrococcus* species
C	General	T1 agent dial	*Micrococcus* specie
5A	General	Stopcock set	Coagulase-negative *Staphylococcus* species
			Bacillus species
B	Orthopedics	T1 APL valve	*Micrococcus* species
6A	General	T1 APL valve	Alpha *Streptococcus* species
B	General	T1 agent dial	Alpha *Streptococcus* species
C	General	T0 agent dial	Alpha *Streptococcus* species
7A	Vascular	T1 APL valve	*Micrococcus* species
B	Vascular	Stopcock set	Coagulase-negative *Staphylococcus* species
9B	General	T1 APL valve	*Micrococcus* species
			Corynebacterium species
C	General	T0 APL valve	*Micrococcus* species
			Corynebacterium species
12A	Vascular	T1 APL valve	*Micrococcus* species
B	Vascular	Stopcock set	*Micrococcus* species
14A	Thoracic	T1 APL valve	*Micrococcus* species
			Alpha *Streptococcus* species
			Bacillus species
B	Thoracic	Stopcock set	*Micrococcus* species
			Alpha *Streptococcus* species
			Bacillus species
C	General	T1 agent dial	*Micrococcus* species
			Alpha *Streptococcus* species
			Bacillus species
17A	General	Stopcock set	Alpha *Streptococcus* species
			Micrococcus species
			Enterococcus species
B	General	T1 agent dial	Alpha *Streptococcus* species
			Micrococcus species
			Enterococcus species
C	General	T1 APL valve	Alpha *Streptococcus* species
			Micrococcus species
			Enterococcus species
1A	Vascular	T1 APL valve	*Staphylococcus epidermidis* (316164)
B	Vascular	Stopcock set	*Staphylococcus epidermidis* (207064)
C	Vascular	T1 agent dial	*Staphylococcus epidermidis* (707164)
3A	Cardiac	T1 APL valve	*Staphylococcus capitis* (306111)
B	Cardiac	Stopcock set	Coagulase-negative *Staphylococcus* (712226)
			57% *Staphylococcus lugdunensis*
			35% *Staphylococcus hominis*
			8% *Staphylococcus haemolyticus*
C	General	T1 agent dial	*Staphylococcus capitis* (306111)
8A	General	Stopcock set	*Staphylococcus haemolyticus* (322312)
			Staphylococcus epidermidis (307064)
B	General	T1 APL valve	*Staphylococcus epidermidis* (207064)
9D	Neurology	T0 agent dial	*Staphylococcus hominis* (342064)
10A	General	Stopcock set	*Staphylococcus epidermidis* (217064)
B	General	T1 APL valve	*Staphylococcus epidermidis* (307166)
			Methicillin-resistant *Staphylococcus aureus*
11B	Orthopedics	Stopcock set	*Staphylococcus epidermidis* (203124)
C	Orthopedics	T1 agent dial	*Staphylococcu s epidermidis* (316164)
13A	General	T1 agent dial	*Staphylococcus epidermidis* (717164)
			Enterococcus *faecium* (216757144), vancomycin resistant
B	General	T1 APL valve	*Staphylococcu s epidermidis* (307064)
			Enterococcus faecium (216757144), vancomycin resistant
C	General	Stopcock set	*Staphylococcus epidermidis* (317064)
			Enterococcus faecium (216757144), vancomycin resistant
16A	General	T1 agent dial	*Staphylococcus epidermidis* (317164)
B	General	T1 APL valve	*Staphylococcus epidermidis* (317164)
18A	General	Stopcock set	*Enterobacter cloacae* (77103162)
B	General	T1 APL valve	Methicillin-resistant *Staphylococcus aureus*
19A	General	Stopcock set	*Staphylococcus epidermidis* (307064)
B	General	T1 APL valve	*Staphylococcus epidermidis* (307064)

APL valve = adjustable pressure-limiting valve; T0 = time 0; T1 = time 1.

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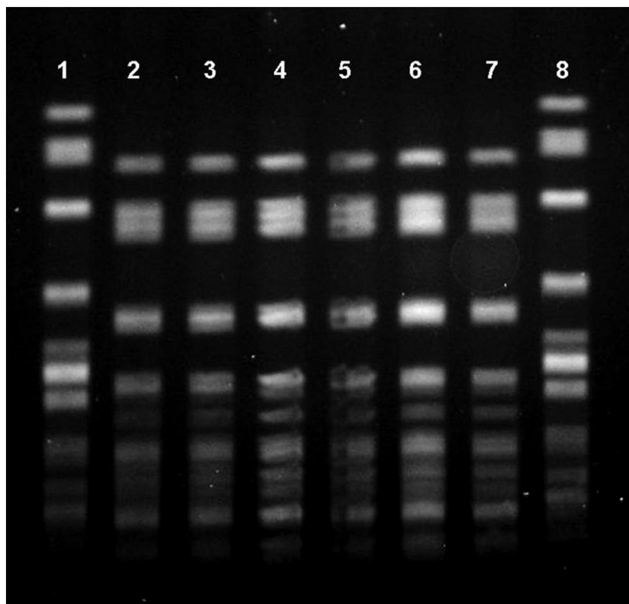

Fig. 4. Pulsed field gel electrophoresis of DNA isolated from bacteria recovered from the anesthesia machine and the stopcocks from patient 13. *Lanes 1* and *8*, *Enterococcus faecalis* (standard) American Type Culture Collection 2912; *lanes 2* and *3*, isolate 13A-2 (time 1 agent dial); *lanes 4* and *5*, isolate 13B-2 (time 1 adjustable pressure-limiting valve); *lanes 6* and 7, isolate 13C-2 (stopcock set).

The complex intraoperative environment has been theoretically associated with the development of nosocomial infections, but previous studies have been unable to provide evidence of direct microbial transmission to patients undergoing general anesthesia.[4,7,9,13,43] The mechanism by which this environment leads to such infections is therefore unclear. In addition, there are no studies to date evaluating the risk of transmission of resistant bacterial strains to patients receiving general anesthesia. As a result, there is currently inadequate implementation of infection control measures in this area.[11,14,44]

We have characterized bacterial contamination of the anesthesia work area intraoperatively and have demonstrated that the anesthesia work area becomes significantly contaminated with potential pathogens in as little as 4 min. Early transmission likely occurs secondary to contamination of provider hands after induction of anesthesia.[45] In 32% of cases, the intravenous tubing (stopcock) was affected. In addition to that previously described, we have detected patient transmission of pathogenic, multidrug-resistant organisms, including VRE.[10]

At our institution, there were 4,381 patient days in the intensive care unit in 2006. In that year, there were a total of 29 episodes of MRSA (22 infections and 7 colonizations) and a total of 13 VRE episodes, which were all *Enterococcus faecium* (10 infections and 3 colonizations). Even with the known limitations of the relatively insensitive culturing technique that we used, we detected 2 cases of MRSA and 1 case of VRE intraoperative transmission (8% and 7% of the intensive care unit burden, respectively) in 61 operative cases. This suggests an alarmingly high overall intraoperative transmission rate of pathogenic organisms.

Based on the National Nosocomial Infection Surveillance System reports, our institution is at less than the 25th percentile for MRSA episodes and at the 50th percentile for VRE episodes.[1] Given this information, we conclude that our institution is equivalent to or better than the majority at infection control. With the reasonable assumption that the aseptic practice by anesthesia providers at our institution reflects practice elsewhere, we expect the magnitude of contamination by anesthesia providers at other institutions to correlate with respective National Nosocomial Infection Surveillance System quartile ranges.

Our results demonstrate that bacterial contamination of the anesthesia work area occurs early and is unrelated to factors of case duration, urgency, or patient American Society of Anesthesiologists physical status. This strongly suggests that bacterial transfer to patients is associated with the variable aseptic practice of anesthesia personnel.

In multiple cases, we were able to confirm the transmission of the same pathogenic bacteria to both the anesthesia work area and the stopcock set. Although contamination of intravascular devices has been documented previously, it has not been shown to occur at such magnitude over such a short time frame. Previous studies looked at colonization at 72 or more hours.[18,19,33,46] In addition, this is the first demonstration of intraoperative contamination of peripheral intravascular devices with resistant bacterial strains.

In one case, biotyping and pulse field gel electrophoresis typing confirmed transmission of VRE from a patient to the anesthesia work area and back to the intravenous stopcock set. The patient was known to be colonized with VRE, suggesting that the likely mode of transmission was the hands of the provider. It has been shown that interaction by healthcare providers with patients known to be colonized with VRE is associated with a 41% chance of provider hand contamination with the organism.[47] This patient subsequently developed blood cultures positive for growth with VRE during the same hospital stay. Therefore, in contrast to those reported previously, our results support that the anesthesia machine is likely to play a role in microbial contamination of patients.[14]

We found a significant association between increasing magnitude of anesthesia work area contamination and contamination of peripheral intravenous tubing, likely due to the common denominator of variable aseptic practice of anesthesia providers. In the setting of unreliable operative decontamination strategies as we have demonstrated, this may lead to horizontal transmission of multidrug-resistant organisms and ultimately to increased hospital-wide nosocomial infection rates with associated increases in morbidity, mortality, and health-

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care costs. Further work is indicated to develop more effective decontamination strategies.

We followed all patients retrospectively over 30 postoperative days to observe outcomes related to hospital-acquired infections and overall morbidity or mortality. Although it was not a primary outcome, we observed an increase in mortality that may be attributable to stopcock contamination secondary to poor aseptic practice. There seems to be a trend toward increased nosocomial infection rates for those patients with intraoperative contamination of intravenous tubing as compared with those without such contamination (25% *vs.* 9.8%, respectively). A nosocomial infection rate of 9.8% in those without contaminated stopcocks is consistent with that reported in the literature.[1]

Other intraoperative variables known to be associated with the development of surgical site infections, such as glycemic control, hypothermia, and prophylactic antibiotics, were no different between those patients with nosocomial infections and those without.[23] Patients with nosocomial infections actually had statistically higher concentrations of inspired oxygen, but this difference is not clinically relevant with both concentrations (0.89 and 0.75) relatively high. This is not meant to suggest that increased intraoperative inspired oxygen concentrations increase the risk of postoperative nosocomial infection development, but simply to suggest that nosocomial infections are not explained by lower intraoperative concentrations of inspired oxygen.

The primary aim of this study was to evaluate bacterial transmission between the anesthesia environment and the patient. Within this framework, we intentionally excluded the evaluation of other parameters such as colonization of the oropharynx, nasopharynx, or other areas within the operative suite. Additional studies are indicated to further address the origin of the pathogenic organisms.

There are additional limitations to this study. We were unable to demonstrate statistically significant relations for variables other than the CPSS on the probability of a positive stopcock. This should not be interpreted as indicating that these factors are unimportant, because a more likely explanation is the correlated nature of these variables and the small sample size of our study. However, the purpose of the multivariate analysis was simply to understand whether these other variables taken together modified the impact of CPSS, and they did not. The relation of positive stopcock to mortality should also be interpreted with caution because it was not the primary outcome of this study. It should be considered exploratory and needs to be verified in other and larger studies. Finally, we were unable to directly link the bacteria in contaminated stopcocks to the causative organisms associated with subsequent hospital-acquired infections. This requires additional study.

In summary, we have demonstrated that variable aseptic practice of anesthesia providers leads to contamination of both peripheral intravenous tubing and the anesthesia work area with potentially pathogenic bacterial organisms. We have shown that such contamination is associated with a trend toward increased nosocomial infection rates and a significant increase in postoperative mortality. This provides further insight into a potential mechanism by which anesthetic practice leads to the development of nosocomial infections. More importantly, this may contribute to the emerging pattern of increasing bacterial resistance in both community and hospital-wide settings given the high throughput of today's operating rooms.

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