

July 2010

Suction Regulators: A Potential Vector for Hospital-Acquired Pathogens

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Abstract

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Infect Control Hosp Epidemiol 2010; 31(7):772-774

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Suction Regulators: A Potential Vector for Hospital-Acquired Pathogens

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Hospital-acquired infections (HAIs) are the leading cause of death in US noncardiac intensive care units (ICUs).¹ Additional efforts are warranted to prevent and control the spread of HAIs and infection with multidrug-resistant (MDR) pathogens in the ICU. Suction regulators, which are frequently used for intermittent automated nasogastric drainage, have not been studied previously, to our knowledge, as an infection source, and there are no regulations regarding the cleaning of these devices. Suction regulators might serve as a reservoir for nosocomial pathogens, including MDR bacteria, and might play a role in horizontal patient-to-patient transmission of pathogens. This study aims to challenge 3 hypotheses: (1) suction equipment does not get contaminated, because the system employs a waste canister and overflow protection; (2) aspirates will not be transmitted back to the patient, because suction flows only in one direction—toward the wall source—opposite of the patient; and (3) contaminants that are drawn into a suction regulator cannot be transmitted back to a patient.

METHODS

Four hundred seventy actively used suction regulators were sampled from the ICUs of 11 medical facilities located in 5 different states. Samples were collected by culturing the patient port of the regulator (CultureSwab Liquid Stuart; BD Diagnostic Systems). All culture specimens were streaked immediately onto tryptic soy agar plates (BD Diagnostic Systems), and each culture that yielded growth was sent to the North American Medical Science Associates (Northwood, OH) for identification using standard procedures.²

To address hypotheses 2 and 3, an experimental circuit was developed. Five types of suction regulators were set to 100

mm Hg in intermittent mode: (1) Amvex Corporation, Digital 0–300; (2) Boehringer Laboratories; (3) Chemetron, Allied Healthcare Products, Vacutron 0–300 Cont/Intermit; (4) Ohio Medical Corporation, PTS-ISU; and (5) Precision Medical, 0–200 Cont/Intermit. A referent strain of *Escherichia coli* ATCC 29425 was grown (Difco Nutrient Broth; BD Diagnostic Systems). One hundred milliliters of solution containing a 1×10^6 cfu/mL concentration of the referent strain was aspirated into each regulator. After aspiration, tests were conducted to confirm that the referent strain was in the device. To investigate the potential migration of organisms toward the patient, the contaminated regulator was connected to the simulated nasogastric circuit (Figure 1). A 6-foot (183-cm) piece of sterile Kendall Argyle, Nonconductive Connection Tubing (Covidien), connected the patient port of the regulator to the vacuum port in the wall canister, and another piece of sterile tubing (Covidien) connected the patient port of the canister to the simulated stomach. The simulated stomach was a 1,200-mL handmade Pyrex beaker with a polyethylene lid. The lid was equipped with a 0.001-inch (0.25 mm) orifice with a 0.22- μ m filter (Millex Filter Units) to allow a controlled amount of filtered air into the system. The entire simulated stomach was autoclaved before use. The simulated stomach was then filled with 1,000 mL of sterile nutrient broth media (BD Diagnostic Systems). The contaminated regulator was then set to 100 mm Hg and run in intermittent mode for 48 hours. Five-hundred-microliter samples were removed from the wall collection canister and the simulated stomach at intervals of 0.5, 1, 2, 4, 8, 24, 32, and 48 hours.

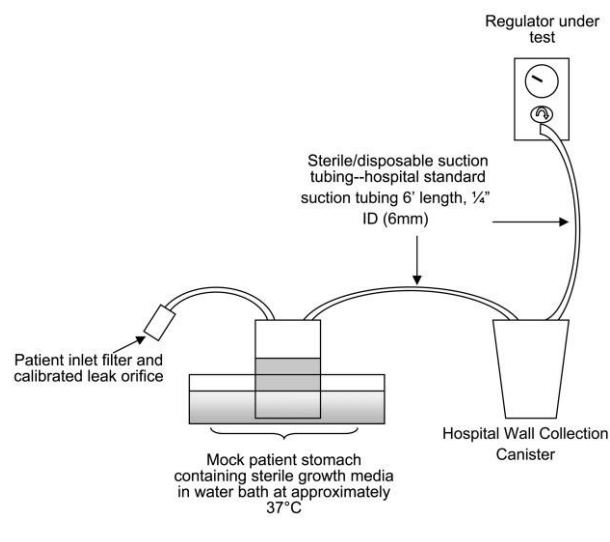


FIGURE 1. Diagram depicting the simulated nasogastric circuit that was used to evaluate the potential for a contaminated regulator to spread bacteria to the patient.

Samples were plated onto nutrient agar plates (Difco Nutrient Agar; BD Diagnostic Systems) and were incubated at 37°C for 24 hours. Resulting colonies were quantified and were identified by North American Medical Science Associates to ensure that the reference strain was the one isolated.² A control experiment was conducted in which a clean intermittent regulator was connected to the nasogastric circuit, with samples taken at the same intervals.

RESULTS

Of the 470 hospital regulators swabbed, 173 (37%) produced growth, including growth of well-established nosocomial pathogens such as *Pseudomonas aeruginosa*, *Staphylococcus aureus*, coagulase-negative staphylococci, *Enterococcus faecium*, and *Bacillus* and *Micrococcus* species; 13.2% of specimens were contaminated with one these pathogens. In the experimental circuits, all 5 intermittent regulators infected the canister with the referent strain during the 24-hour incubation period. Three brands (brands C–E) contaminated the canister in less than 30 minutes. Another regulator (brand A) did not contaminate the canister until a full 24 hours had elapsed. Figure 2A illustrates the growth of the referent strain

in the nasogastric circuit canister in the 5 different models. The mock stomach of the patient became colonized in 4 regulator models (brands B–E) within 24 hours. Only one model (brand A) did not demonstrate any detectable colonization within 48 hours in 5 separate runs. Figure 2B depicts the overall growth of the referent *E. coli* strain in the mock stomach for this experiment. To verify that the bacteria found in the wall canister and the patient stomach came from the regulator and not from an outside source, a control experiment was run. In this experiment, a new regulator was run with a sterile nasogastric circuit. In 3 separate runs, both the wall canister and patient stomach tested negative for bacterial growth after running for 48 hours.

DISCUSSION

HAIs are prevalent in ICUs, where highly resistant pathogens are frequently endemic. Gastric colonization with bacterial pathogens can increase a patient's risk of acquiring ventilator-associated pneumonia (VAP), one of the most common and serious types of HAI.³ HAIs and VAP can occur after encounters with contaminated devices, which have sometimes been used on multiple patients, therefore serving as a vector

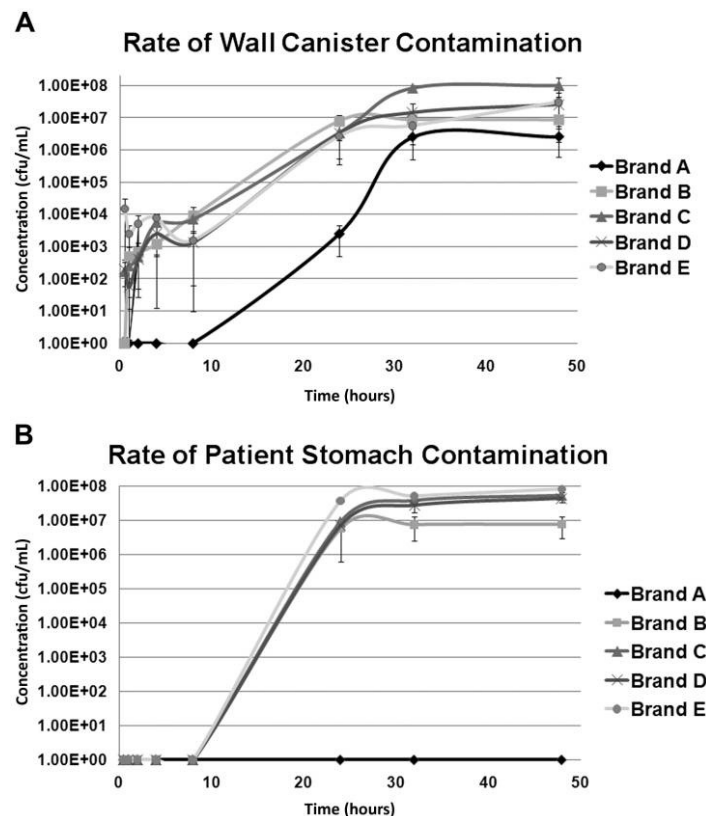


FIGURE 2. Growth of bacteria in the nasogastric circuit canister after connection to a regulator contaminated with *Escherichia coli* (A) and the rate of contamination of the mock stomach with the referent *E. coli* strain (B). All data are presented as the mean \pm standard deviation for 5 different runs.

for patient-to-patient transmission of MDR pathogens.^{4,5} Although the role of other devices in HAI has been well described, the role of suction regulators has, to our knowledge, not been investigated. In this study, more than one-third of suction regulators from hospitals around the country were contaminated, often by notable pathogens, including *P. aeruginosa* and *S. aureus*, the most common causes of VAP.⁴⁻⁷

In addition to identifying suction regulators as potential reservoirs for nosocomial pathogens, this study demonstrated that contaminants can spread from a suction regulator to the wall-side canister within 30 minutes and can also spread back to a simulated patient stomach within 24 hours. Thus, suction regulators might be contaminated by one patient and then transmit pathogens to the stomach of a subsequent patient. Only 1 brand of regulator did not transmit pathogens back through the patient side of the circuit during the 48-hour study period. Gastric colonization is a clinically relevant phenomenon in ICU patients, who often receive antacids for peptic ulcer prophylaxis, often leading to gastric overgrowth and respiratory and systemic infections.³

One common misconception is that after “cleaning” a regulator after patient use, the regulator is free of contamination. Current manufacturers’ protocols state that back-flushing a regulator with disinfectants can remove contamination; however, there exist no data to support the efficacy of this practice. In addition, the internal flow paths in suction regulators can be convoluted, and bacteria can become trapped and can be aerosolized back during the venting cycle. The most effective method to ensure that a contaminated regulator does not contain pathogens is to sterilize it, which is costly and is not the presently recommended practice.⁸ Most brands cannot be safely sterilized. Identifying the suction regulator as a potential source of infection is noteworthy, and additional investigation is needed to clarify the risk that contaminated regulators pose to patients and to indicate optimal methods and protocols for disinfection.

ACKNOWLEDGMENTS

Potential conflicts of interest. All authors report no conflicts of interest relevant to this article.

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Received September 13, 2009; accepted January 17, 2010; electronically published May 20, 2010.

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REFERENCES

1. Vincent JL, Rello J, Marshall J, et al. International study of the prevalence and outcomes of infection in intensive care units. *JAMA* 2009;302(21):2323–2329.
2. Clinical and Laboratory Standards Institute (CLSI). *Performance Standards for Antimicrobial Susceptibility Testing: Sixteenth Informational Supplement*. Wayne, PA: CLSI, 2006. CLSI document M100-S16.
3. Heyland D, Mandell LA. Gastric colonization by gram-negative bacilli and nosocomial pneumonia in the intensive care unit patient: evidence for causation. *Chest* 1992;101(1):187–193.
4. Edwards JR, Peterson KD, Andrus ML, Dudeck MA, Pollock DA, Horan TC. National Healthcare Safety Network (NHSN) report, data summary for 2006 through 2007, issued November 2008. *Am J Infect Control* 2008;36(9):609–626.
5. Weber DJ, Raasch R, Rutala WA. Nosocomial infections in the ICU: the growing importance of antibiotic-resistant pathogens. *Chest* 1999;115(suppl 3):34S–41S.
6. Hidron AI, Edwards JR, Patel J, et al. NHSN annual update: antimicrobial-resistant pathogens associated with healthcare-associated infections: annual summary of data reported to the National Healthcare Safety Network at the Centers for Disease Control and Prevention, 2006–2007. *Infect Control Hosp Epidemiol* 2008;29(11):996–1011.
7. Craven DE, Steger KA, Barat LM, Duncan RA. Nosocomial pneumonia: epidemiology and infection control. *Intensive Care Med* 1992;18(suppl 1):S3–S9.
8. Rutala WA, Weber DJ. *Guidelines for Disinfection and Sterilization in Healthcare Facilities*, 2008. Atlanta, GA: Centers for Disease Control and Prevention, 2008.