Contribution of Water Condensation in Endotracheal Tubes to Contamination of the Lungs*

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We postulated that water condensate in endotracheal tubes (ETTs) transports bacteria in the ETTs into the lungs during mechanical ventilation. Thirty-two ETTs obtained from freshly extubated patients were studied under wet and dry conditions using a physiologic lung model. All bacteria expelled from the ETTs were collected on culture plates positioned beneath the ETT. The lung model was ventilated with saturated air at 37°C over two time periods (60 min each), one in which condensation formation was prevented and the second in which condensation formed within the ETT. A mean of 457.6 colony-forming units (CFU)/h were expelled with condensation compared to a mean of 2.4 CFU/h without condensation. We concluded that bacteria were continuously transported from the ETT into the lungs during mechanical ventilation in water droplets. Prevention of water condensation abolishes this constant bacterial inoculation in a lung model. (Chest 1993; 104:127-29)

Methods

Equipment

A lung model was developed mimicking human thermal and humidity conditions to study the effect of water condensation on contamination of the lower respiratory tract. Thirty-two ETTs were obtained from freshly extubated patients between September 1, 1990 and March 31, 1991 at the Ottawa General Hospital, Ottawa, Ontario (Canada).

Lung Model

The lung model consisted of a rigid cylinder mounted on a detachable base (Fig 1). The cylinder had two ports located on the top surface. One port received the ETT and the other was connected to an Anderson sampler. Culture plates were fixed 3.5 cm below the distal end of the ETT. These plates could be changed without moving the lung model or the ETT. The floor of the cylinder consisted of a rubber membrane that moved with ventilation. The lung model and most of the ETT, except for the proximal 3 cm that normally protrudes from the patient, were contained in an incubator (Isolatte Infant Incubator, model No. C-96, Air-Shields Inc, Hatboro, Pa) set at 37°C to approximate internal body temperature.

Anderson Sampler and Pump

The Anderson sampler† collects airborne particles in categories of decreasing particle size. It allows calculation of the particle size spectrum of bacterial aerosols. The outlet of the Anderson sampler was connected to a pump designed to draw air at a rate of 28.32 L/min. Humidified air was sampled from both the room and lung model simultaneously as the Anderson sampler sampled air at a greater rate than the ventilation of the lung model (28.32 L/min compared with 7.2 L/min). A second Anderson sampler sampled room air only to control for bacterial aggregates present in the room.

Ventilator and Humidifier

A respirator (Companion R800, Puritan-Bennett, Lenexa, Kan) and ventilator circuit (T11550, Puritan-Bennett), were used for the experiments. Filters inherent to this ventilator and circuit include both an air intake filter (T11337, Puritan Bennett) and a bacterial filter (Respigard-II, No. MQ-303-01, Marquest Medical Products Inc, Englewood, Colo, pore size 0.3 μm). Ventilating parameters were as follows: tidal volume, 600 ml and; respiratory rate, 12/min;
inspiratory flow rate, 50 L/min; and FIO2, 0.21. The inspired air was humidified with a humidifier (ConchaTherm III, No. 390-80, Respiratory Care Inc, Arlington, Ill).

Design

Following extubation, the exterior of the ETT and cuff were cleaned with 70 percent isopropyl alcohol, dried, wrapped in sterile towels, and transported to the laboratory. If the experiment could not be performed within 2 h of extubation, the ETTS were held at 4°C overnight. The ETTS were inserted through an opening in the incubator and then into the lung model. The cuff was inflated sealing the entry of the lung model.

The contribution of ETT colonization to contamination of the lungs was evaluated in two different experiments. In experiment A, culture plates were positioned beneath the tip of the ETT, whereas in experiment B, the presence of airborne bacterial aggregates within the lung model was evaluated with Anderson samplers.

Experiment A: The model was ventilated over two controlled time periods (60 min each) both with saturated air at 37°C. In the first time period, condensation formation was prevented by heating and insulating the exposed part of the ETT and dead space tubing of the ventilator with insulating tubing (Rubatex) and insulating tape (Wratpate). After measuring the exposed part of the ETT and deadspace tubing, a piece of insulating tubing was cut, sliced along its long axis to facilitate its insertion over the ETT and deadspace tubing. Insulating tape was wrapped around the junction between the ETT and the incubator, thus sealing the incubator opening. In the second time period, the insulation was removed permitting condensation to form within the ETT. To collect all the expelled bacteria from the ETT, culture plates were positioned 3.5 cm below the tip of the ETT and were changed every 10 min.

Trypticase agar supplemented with 5 percent sheep blood media was used in all culture plates. The culture plates taken from the lung model were streaked for quantification and incubated in CO2 (7 percent) at 35°C overnight. Colonies were counted on all plates and expelled colony-forming units (CFUs) per hour were calculated.

Experiment B: Culture plates were removed and the lung model was again ventilated over identical periods, at first with condensation and then without. During the experiment, one Anderson sampler sampled air from both the lung model and the room and another one from the room alone. All Anderson sampler plates were incubated as above. Colonies were counted and species were identified.

Analysis

A paired t test was used to compare the rate of CFUs expelled during ventilation with and without condensation (experiment A). A Student’s t test was used to evaluate the contribution of air to the microbiologic growth obtained in the Anderson samplers (experiment B).

RESULTS

Experiment A

Thirty-two ETTS were analyzed. When condensation was allowed to form in the ETT, a mean of 457.6 CFU/h of ventilation were expelled (SD = 530.9) compared with a mean of 2.4 CFU/h (SD = 5.1) when condensation was prevented (p<0.0001).

Experiment B

Data using the Anderson sampler were compiled for analysis from experiments on 11 tubes. None of the bacteria isolated on Anderson sampler plates were respiratory tract pathogens. When condensation was allowed, a mean of 4.4 bacteria (SD = 5.7) were recovered from the room and lung model per hour of ventilation compared with a mean of 7.1 (SD = 7.1) when condensation was prevented (p=0.23). The
number of bacteria sampled from the room alone was 14.8 (SD = 17.7). The volume of air sampled during this time period was 1,699.2 L (28.32 L/min).

**DISCUSSION**

We consider that bacteria within the ETT may reach the airways by three different mechanisms: (1) high inspiratory flows may dislodge bacterial microaggregates from the ETT biofilm surface and into the airstream; (2) gravity and inspiratory flows may move bulky secretions caudally; and (3) water condensate, formed as warm saturated gases come in contact with the cooler ETT, may remove bacteria from the biofilm surface and carry them into the patient's lungs. Our study indicates that the contribution of the first two mechanisms is negligible.

Bacterial microaggregates suspended in the air are unlikely to contribute to the pathogenesis of pneumonia because the concentration of bacteria suspended in the air from both the room and lung model was similar to that of the air sampled from the room alone. In addition, the organisms recovered were typical of environmental contaminants. To ensure that airborne bacterial microaggregates from the lung model did not impact on tubing walls before reaching the Anderson sampler, the tubing connecting the sampler to the model was washed with saline solution after the experiments. This fluid was sterile.

Bulk movement of secretions from the ETT into the lung model did not occur in this experiment. Thus, we believe this to be an unlikely mechanism in the pathogenesis of pneumonia. In addition, bulky secretions can be easily coughed out or suctioned and are therefore unlikely to enter the lungs.

The data presented suggest that in ventilated patients, bacteria may be carried into the lungs in droplets that have formed in ETTs due to water condensation. Although this hypothesis has not been tested *in vivo*, a model was utilized that mimics both thermal and humidity conditions seen in humans. The model did not humidify expired air as occurs in patients; however, this would have increased only the volume of condensate and amplified the effect observed. The order of the cycles with and without condensation was not randomized because water droplets that accumulated in the ETT during a wet cycle could have been expelled during the following dry cycle. To avoid this bias, a dry cycle was always preceded by a wet one. Seven tubes were ventilated during two consecutive condensate-free cycles to control for the effect of time. There was no observed difference in the number of CFU recovered in the second hour of ventilation compared with the first.

The ongoing inoculation with bacteria-laden water droplets could overwhelm the airway and alveolar defenses causing pneumonia. The abolition of water condensation during ventilation offers a simple and possibly effective method of reducing the risk of nosocomial pneumonia. The significance of this finding will need to be determined in further clinical studies. Related practices such as the instillation of saline solution into the ETT prior to suctioning also merit investigation.

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