# NOSOCOMIAL PULMONARY INFECTIONS RELATED TO ANAESTHESIA

**A G Duse**

Caring for critically ill patients in special high-technology units is an essential component of modern medicine. Invasive diagnostic and therapeutic modalities are commonly used and, although they may play a vital role in patient management, life-support systems disrupt normal host defence mechanisms. Given the severity of the illnesses affecting patients in intensive care units (ICUs), it is not surprising that mortality rates often exceed 25%. If patients experience complications, the mortality rate can be in excess of 40%. Nosocomial infections (NIs) are among the most common medical complications affecting intensive care patients. In many South African ICUs the anaesthesiologist/intensivist is one of the key professionals involved in the care of critically ill patients. The purpose of this brief review is to discuss the role of the anaesthesiologist in preventing nosocomial pulmonary infections. Many of the principles outlined in this document can, however, readily be applied to the ICU setting and are therefore relevant to all intensivists.

According to data from the National Nosocomial Infection Surveillance (NNIS) survey in the USA, nosocomial pneumonia is, after urinary tract infections, the second commonest hospital-acquired infection, accounting for approximately 15% of the total. The mechanisms by which ventilator-associated pneumonia (VAP) is caused include the aspiration of endogenous oropharyngeal organisms and inhalation of exogenous micro-organisms which contaminate respiratory therapy equipment.

## INFECTIONS ASSOCIATED WITH INHALATION ANAESTHESIA

Endotracheal intubation bypasses the upper airway which is responsible for warming, humidifying and removing particles from inspired air. Several studies conducted in animals indicate that endotracheal tubes quickly disrupt the ciliated tracheal epithelium, resulting in defects in mucociliary transport and a local inflammatory response. In addition to disrupting the respiratory tract's defences, endotracheal (including nasotracheal) intubation can result in the transfer of bacteria from the patient's pharynx into the trachea, the innoculation of exogenous organisms directly into the bronchial tree, and transient bacteraemia. Furthermore, inhalational anaesthetic drugs, and high concentrations of oxygen, which may be administered during anaesthesia, may further impair mucociliary function. Mechanical ventilation also exposes the patient to fluid-filled devices such as in-line nebulisers and humidifiers that are a source of micro-organisms and associated with respiratory infection in these patients.

The role of anaesthetic respiratory equipment as a source of micro-organisms causing nosocomial pneumonia remains controversial. Proponents for the motion that respiratory equipment is a source of infection base their argument on the following evidence: (*i*) bacteria have been shown to contaminate all parts of the anaesthetic circuits, with the greatest number contaminating the parts that are closest to the patient; (*ii*) some micro-organisms can be carried by anaesthetic gases from the apparatus to the patient, and vice versa; (*iii*) soda lime filters bacteria imperfectly and, although it does kill many pathogens, *Mycobacterium tuberculosis* and *Bacillus* species survive prolonged exposure. As a consequence of these and other findings, some experts believe that breathing circuits should be sterilised between patients. Proponents for the motion that anaesthetic equipment is not an important source of infection base their opinion on evidence that anaesthetic machines, even when contaminated, do not transmit significant numbers of bacteria as microbes do not survive readily in this hostile environment. Organisms are desiccated by the flow of cold, dry (if inadequately humidified) anaesthetic gases; rubber and metal components of the apparatus are bacteriostatic; and the highly alkaline condensate at the bottom of the carbon dioxide absorber also inhibits the growth of bacteria.

The clinical importance of micro-organisms isolated from anaesthetic machines therefore remains hotly debated. Although these machines become contaminated during use, there is little evidence that they are primarily responsible for the transmission of micro-organisms to patients. Despite this, a 1995 report by the NSW Health Department (Australia) documenting 5 cases of nosocomial hepatitis C, allegedly caused by contaminated circuitry, has resulted in the revision of the anaesthetic aspects of an infection control policy in that country. There have been some criticisms of this investigation. Firstly, although the outbreak might have been transmitted through contaminated respiratory secretions, there is no conclusive evidence to prove that this was the case. Secondly, despite claims that all infection control measures were in place, adequate investigations to rule out other sources of infection were not performed owing to the retrospective nature of the analysis. Nevertheless, features of the revised anaesthetic policy include rigorous implementation of standard (universal) precautions; disposal or decontamination and high-level disinfection of instruments and apparatus that come into contact with patients or their blood and body fluids; and

*Division of Hospital Epidemiology and Infection Control, South African Institute for Medical Research and School of Pathology, University of the Witwatersrand, and Johannesburg Hospital, Johannesburg*

**A G Duse**, MB BCh, Nat Dip Med Tech, DTM&H

protection of the breathing circuit for every patient by either a disposable filter or high-level disinfection of all parts of the circuit that are not so protected. Given that the parts of the circuit closest to the patient are the most highly contaminated, it seems reasonable to follow the American Society for Anesthesiology (ASA) recommendation that breathing circuits and masks be cleaned and disinfected between cases. Furthermore, although filters effectively prevent the transfer of micro-organisms from the patient to the anaesthetic machine and vice versa, currently available data do not uniformly support routine filter use. Two clinical trials did not identify statistically significant differences in postoperative pneumonia rates between patients anaesthetised with either disposable corrugated plastic circuits containing filters (0.22 microns) or similar circuits without filters. In addition no difference was found between sterile disposable circuits versus clean reusable circuits. However, neither of these two studies was designed to detect clinically significant differences in postoperative pneumonia rates. Clinical trials comparing postoperative pneumonia rates involving patients anaesthetised with filtered or non-filtered circuits with sterile or cleaned circuits, and sterile or periodically cleaned anaesthesia ventilatory equipment, are warranted.

With the advancement in our knowledge of the epidemiology of serious blood-borne infections such as HIV and viral hepatitis B and C, and of multidrug-resistant tuberculosis, it is important to maintain and improve measures to minimise the risk of transmission of such infections in patients undergoing inhalation anaesthesia and long-term assisted ventilation. Although studies involving blood-borne viruses, or convincing reports of cross-infection due to blood-borne viruses, are lacking, there is a theoretical risk of transmission of these agents through saliva or respiratory secretions that contaminate respiratory apparatus. Similarly, although contamination of facemasks by viable tubercle bacilli has been documented, reports of transmission of tuberculosis via breathing apparatus are also lacking. Bacterial contamination of circuits used on long-term ventilated patients is predominantly due to non-pathogenic microbes. Outbreaks of respiratory tract infections due to contaminated anaesthetic circuits were mainly reported before routine implementation of infection control procedures in hospitals. There is convincing evidence to suggest that bacterial filters in anaesthetic breathing circuits may not be necessary if a strict regime of cleaning and disinfection is followed or clean disposable circuits are used. Parts of equipment that come into contact with mucous membranes (facemasks, endotracheal tubes) or become contaminated with respiratory secretions (Y-piece, inspiratory and expiratory tubing and attached sensors) should optimally be sterilised, or high-level disinfected. Routine sterilisation or disinfection of the internal machinery of a breathing system is not advocated, as it has not been shown to be easily susceptible to contamination in controlled studies.

Combined heat moisture exchange devices and bacterial filters not only optimise heat and moisture output, thereby reducing condensation in the tubing and minimising the risk of bacterial growth, but also have bacterial and viral retention properties. Although there are no good controlled studies showing the usefulness of microbial filters on breathing circuits in patients on long-term ventilation, it is reasonable to assume that the use of such filters can be recommended, especially if the frequency of change of breathing circuits, labour, and equipment costs are reduced as a cost-saving strategy.

## CONCLUSIONS

On the basis of currently available evidence it is reasonable to conclude that, until filters with high microbial retention properties are available at a reasonably low cost, the use of such filters, although optimal, is not justified in health care facilities with severe financial constraints. A careful cost-benefit analysis needs to be conducted in the South African context. Every hospital should have a clear policy relating to breathing circuits. Implementation of rational and meticulous infection control measures should considerably minimise rates of ventilator-associated pneumonia and other NIs associated with mechanical ventilation and anaesthesia.

**Recommended reading**


Alexopoulos C, Jansson B, Lindholm CE. Mucus transport and surface damage after endotracheal intubation and tracheostomy. An experimental study in pigs. *Acta Anaesth Scand* 1984; **28:** 68-76.

American Society of Anesthesiologists. *Recommendations for Infection Control for the Practice of Anesthesiology.* Park Ridge, Ill.: American Society of Anesthesiology, 1992.

Berry AJ, Nolte FS. An alternative strategy for infection control of anesthesia breathing circuits: a laboratory assessment of the Pall HEM filter. *Anesth Analg* 1991; **72:** 651-655.

Berry FA, Blakenbaker WL, Ball CG. A comparison of bacteremia occuring with nasotracheal and orotracheal intubation. *Anesth Analg* 1973; **52:** 873-876.

Das I, Fraise AP. How useful are microbial filters in respiratory apparatus? *J Hosp Infect* 1997; **37:** 263-272.

Dominiquez de Villota E, Avello F, Grandos MA, Arcas M, Moles B. Early postsurgical bacterial contamination of the airways: a study on 28 open-heart patients. *Acta Anaesth Scand* 1978; **22:** 227-233.

Feeley TW, Hamilton WK, Xavier B, Moyers J, Eger EI. Sterile anesthesia breathing circuits do not prevent postoperative pulmonary infection. *Anesthesiology* 1981; **34:** 369-372.

Garibaldi RA, Britt MR, Webster C, Pace NL. Failure of bacterial filters to reduce the incidence of pneumonia after inhalation anesthesia. *Anesthesiology* 1981; **54:** 364-368.

Herwaldt LA, Rottinger J, Coffin SA. Nosocomial infections associated with anaesthesia. In: Mayfield CG, ed. *Hospital Epidemiology and Infection Control.* Baltimore, Williams & Wilkins, 1996: 658 (655-675).

Hilding AC, Hilding JA. Tolerance of the respiratory mucous membrane to trauma: surgical swabs and intratracheal tubes. *Ann Otol Rhinol Laryngol* 1962; **71:** 455-479.

Klainer AS, Turndrof H, Wu WH, Maewal H, Allender P. Surface alterations due to endotracheal intubation. *Am J Med* 1975; **58:** 674-683.

Knoblanche GK. Revision of the anaesthetic aspects of an infection control policy following reporting of hepatitis C nosocomial infection. *Anaesth Intens Care* 1996; **24:** 169-172.

Leijten DTM, Reiger VS, Mouton RP. Bacterial contamination and the effect of filters in anaesthetic circuits in a simulated patient model. *J Hosp Infect* 1992; **21:** 51-60.

Lichtiger M, Landa JF, Hirsch JA. Velocity of tracheal mucus in anesthetized women undergoing gynecologic surgery. *Anesthesiology* 1975; **42:** 753-756.

Nair P, Jani K, Sanderson PJ. Transfer of oropharyngeal bacteria into the trachea during endotracheal intubation. *J Hosp Infect* 1986; **8:** 96-103.

Sanada Y, Kojima Y, Fonkalsrud EW, Injury of cilia induced by tracheal tube cuffs. *Surg Gynecol Obstet* 1982; **154:** 648-652.

Schmidt WA, Schaap RN, Mortensen JD. Immediate mucosal effects of short-term, soft cuff, endotracheal intubation. *Arch Pathol Lab Med* 1979; **103:** 516-521.

Shiotani GM, Nicholes P, Ballinger CM, Shaw L. Prevention of contamination of the circle system and ventilators with a new disposable filter. *Anesth Analg* 1971; **50:** 844-855.

Smith JR, Howland WS. Endotracheal tube as source of infection. *JAMA* 1959; **169:** 343-345.

Way WL, Sooy FA. Histologic changes produced by endotracheal intubation. *Ann Otol Rhinol Laryngol* 1965; **74:** 799-812.