Humidification in intensive care
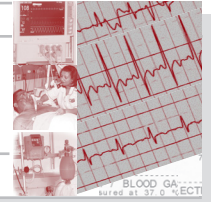
Department of Intensive Care Medicine, Middlemore Hospital, South Auckland Health, Otahuhu, Auckland, New Zealand
Tony Williams, BMedSc, MB ChB, FANZCA, FFICANZCA (FJFICM)

Humidification of inspired gases is an essential part of modern intensive care practice, but there is wide international variation in the application of humidification devices.[1] This review aims to briefly cover the reasons why humidification is important and the main methods of humidification used, outlining their different strengths and weaknesses.

Physics

In the range of pressures and temperatures we commonly encounter, water exists in three phases: solid, liquid and gas. It is useful to remind ourselves that water vapour (gas) is invisible and when we see clouds of white steam this is in fact an aerosol of droplets of liquid water. As a gas containing water vapour cools it becomes fully saturated and condensation begins to form; this temperature is called the dew point for that gas. Wherever there is an interface between gas and liquid water there will be water vapour in the gas. The equilibrium pressure of water vapour over liquid water is related to temperature and is termed saturated vapour pressure (SVP). As temperature increases the SVP increases, and when SVP is equal to or greater than the ambient pressure, water boils.

Humidity is the term used to describe the water vapour content of a gas. It can be expressed in several ways:

- Absolute humidity, which is the mass of water in a given volume of gas (mg/l, kg/m³). Absolute humidity varies with ambient pressure; at 37°C the absolute humidity of fully saturated gas is 44 mg/l.

- Relative humidity is the amount of water vapour present in a gas as a percentage of the saturated capacity. Relative humidity varies with temperature and pressure.

- The water vapour content of a gas can be expressed as a pressure according to Dalton's law of partial pressures (kPa, mmHg). At 37°C the SVP of water is 47 mmHg.

Energy is required for water to move from the liquid phase to gas phase. This energy is called the latent heat of vaporisation and results in cooling of the remaining liquid water in unheated humidification systems, limiting their efficiency.[2]

Physiology

The inspired gas is humidified and heated to body temperature during normal breathing by the upper and lower airways.[3] The nose is partially external to the body and contains turbinate bones which increase surface area. Expired gas is cooled below the body core temperature in the nose, and some of the water vapour it carries condenses. In humans the efficiency of this process is only moderate; it is much more advanced in desert mammals whose obligatory water loss is very low indeed.

Gas exchange in the lung occurs in microscopic air spaces in which the inspired gas has reached saturated humidity.[4] The water content of the alveolar gas is therefore determined only by the body temperature. At some point in the airways the inspired gas reaches body temperature and becomes fully saturated with water vapour; this point is termed the isothermic saturation boundary (ISB).[5] In quiet breathing the ISB is thought to be just below the carina[5] but the anatomical position of the ISB will move up and down the airway depending on the heat and water content of the inspired air, breath tidal volume, inspiratory flow rate and body temperature. To paraphrase Burton,[6] anyone who has performed vigorous exercise in very cold conditions will have noted the discomfort caused when the ISB is shifted distally by high inspiratory flow of air with low heat and water content. The effect of dry gas in the lower airway is thought to be the cause of exercise-induced asthma and exercise cough.[7,8]

At an epithelial level, humidification of the airway gases is achieved by evaporation of water from the thin layer of mucus which covers all respiratory surfaces. Respiratory mucus exists as two interacting layers.[9] The luminal gel layer contains mucin, which is produced by mucous cells, analogous to goblet cells in gut epithelium. The mucosal aqueous layer is produced by serous cells.[10] The mucus provides a liquid surface for humidification and allows particulate

filtering to occur. The mucus is moved by ciliary action to the pharynx where it is swallowed. All parts of the respiratory epithelium have this function, from the paranasal sinuses to the conducting airways.

Rationale for humidification

In the past, when humidification technologies were poorly developed, tenacious secretions caused morbidity and mortality. Early papers describing methods for preventing mucociliary dysfunction gave descriptions of the severity of the problem.[6,11] The effect of ventilation with dry gas on mucociliary transport in dogs was published by Burton in 1962.[6] Ten years later, using cytology in humans, Chalon et al.[12] demonstrated that ventilation with dry gas caused time-dependent injury to respiratory epithelial cells. These changes were seen after periods of dry gas exposure as short as 1 hour. Early recommendations were that when the natural airway was bypassed, inspired gas should have 33 mg/l of water vapour to maintain normal mucociliary function.[13] With the use of adequate humidification, complete occlusion of the endotracheal tube by tenacious secretions is a rare event. However, reduction in lumen diameter does occur over time and may be minimised by using more effective humidification systems.[14,15]

In 1996 Williams et al.[16] proposed a model which related the degree of epithelial dysfunction or injury to the magnitude and duration of any disturbance to normal airway humidity and temperature. The authors suggested that there would be a pattern of worsening dysfunction beginning with abnormal mucus viscosity, proceeding to decreased mucus transit velocity, followed by complete interruption of mucus transit, cessation of cilia movement, and finally loss of normal epithelial cell architecture. By way of validation of the model, the authors examined the results of some 17 existing studies and produced a reasonably good fit of the data to their model. More recent models have been based on thermodynamic principles (the energy requirements of humidifying and heating inspired gases).[17,18] There is a lack of data concerning the effects of partial humidification or heating of inspired gas, but one in vitro study has demonstrated a time-dependent loss of cilial function with one-way flow of saturated gas at 34°C.[19] The rate of recovery from an epithelial injury caused by under-humidification of inspired gas is not known.

Methods of humidification

Various methods of increasing the water content of airway mucus have been published. These include direct methods such as fluid instillation[11] and administration of nebulised water,[20-22] various types of heated humidifiers[23,24] and passive heat and moisture exchangers.[25-27]

All the current humidification technologies are in evolution. Studies that evaluate one device are only strictly applicable to that device, and as with other immature technologies, equipment quickly becomes obsolete. These two factors, when taken together, make it difficult to interpret or apply the results of studies comparing different devices. Results from well-conducted studies may lead to changes in products or devices that address the problems raised by research; this is one of the mechanisms by which humidifier performance has improved over time. It is also important to remember that manufacturers will always present data derived from their devices operating under optimal conditions. The actual performance of devices when tested in clinical conditions may not be the same as quoted by the manufacturer.[28,29]

Passive humidification

The simplest method to achieve some humidification of the inspired gas is by using a condenser humidifier in the airway. These devices are known as heat and moisture exchangers (HME) and act by condensing some of the water vapour in the expired gas which in inspiration evaporates, humidifying the inspired gas.[30] This technology emulates the heat and water conservation of the natural airway.[31] HMEs are simple devices which offer partial humidification of inspired gas; they are portable and relatively inexpensive. These properties make them an important part of respiratory care for patients in whom the natural airway is bypassed.

The humidifying efficiency of HMEs tends to decrease with increasing tidal volume and inspiratory flow.[32] Some HMEs have the condensing surface impregnated with a hygroscopic substance, usually a metal chloride salt, which increases the amount of water condensed in expiration.[33] Other HMEs include bacterial and viral filtration of the expired gas.[34]

HMEs must be in the part of the breathing circuit that has tidal ventilation. They contribute to the resistance and dead space of the circuit. The resistance to flow is usually low but can affect respiratory parameters.[35,36] The HME's condensation surface may become wet with condensate, secretions, blood, oedema fluid or vomit, potentially causing a marked increase in resistance.[37-39] The extra dead space may be a factor in paediatrics or where minute ventilation is restricted.[36,40,41] HMEs are not generally considered appropriate for non-invasive ventilation[42,43] or for use in situations where not all the inspired gas returns to the HME, for example bronchopleural fistulas and tube leak at the larynx.

Initial recommendations were to change the HME daily. Several studies have evaluated the safety of less frequent changes and found a decrease in cost without an increase in adverse outcomes.[44-48]

It is possible to increase the efficiency of HMEs by combining a chemical reaction to generate heat and improve some aspects of performance, at the cost of increased resistance to flow and dead space.[49]

Some investigators have found that the use of HME devices incorporating a bacterial filter has reduced the rate of ventilator-associated pneumonia (VAP) compared with heated humidifiers.[50] They postulate that contamination of the circuit with secretions is part of the pathogenesis of VAP. Other investigators have found no change in rates of VAP with humidifier type.[51,52] A recent meta-analysis concluded there was a reduction in the incidence of VAP with HME;[53] however, the trials included in the meta-analysis used different brands of HME and heated humidifiers which have not been shown to be equivalent. For this reason the authors suggest that the conclusion should be interpreted with caution.

Active humidification

Active humidifiers use an external heat source to vaporise water in the circuit. They exist in three main types: pass-over water bath humidifiers, bubble-through type humidifiers, and those which use porous surfaces to humidify gas. Active humidifiers can deliver gas with absolute humidity close to that of alveolar gas, and some studies have demonstrated advantages of active humidification devices over HMEs.[54,55]

The gas temperature in the circuit must be maintained above its dew point to prevent condensation. Manufacturers and users of active humidifiers have been finding ways to prevent or control condensation since they were first described. These solutions have become gradually more and more effective. Most circuits use heated wire elements to prevent cooling of the inspired gas. The design and arrangement of these wires has evolved from simple wire loops to spiral wire coils manufactured with the circuit to provide even heating. These wires must be designed in such a way that they cannot overheat and burn the patient, damage the circuit, or be a fire risk in the oxygen-rich environment of the breathing circuit.[56-58] The temperature of the gas can be controlled by temperature sensors in the breathing circuit. The advent of turbine compressors has allowed ventilator manufacturers to produce devices that create fresh gas from oxygen and compressed room air. This fresh gas may be significantly warmer than ambient temperature, further increasing the complexity of active humidification.[59,60]

Expiratory limb condensation can be problematic for two reasons. Firstly, because water must be collected in traps, emptying these may add to nursing workload. Secondly, modern ventilators may malfunction if condensation forms in the expiratory mechanism where flow sensors are often positioned. Some modern breathing circuits have an expiratory limb created from material that is permeable to water vapour and are able to reduce the humidity of the expiratory gas before it enters the ventilator without significant condensation.

Pass-over humidifiers

Pass-over humidifiers rely on an element that heats water in a vapourising chamber. The humidifier raises the water content and temperature of fresh gas as it passes through the chamber, which is situated in the inspiratory limb of the breathing circuit. If condensation in the circuit can be controlled the pass-over humidifier has many desirable features. The bulky heater and controller are away from the patient, it has low resistance to flow,[61] and it does not contribute to dead space.[41,62] The volume of the humidification will form part of the compressible volume of the circuit. Under commonly experienced conditions inspired gas can be delivered to the patient with an absolute humidity close to that of alveolar gas.[54] The fresh gas is humidified before delivery to the patient, so these humidifiers can be used to humidify gas in breathing systems without to-and-fro ventilation such as mask continuous positive airway pressure (CPAP) devices and nasal cannulas.

Bubble-through humidifiers

Bubble-through humidifiers have a system that passes inspired gas through the water in the humidification chamber, thus providing a large gas/liquid surface for evaporation. One undesired effect of bubble-through systems is the possible production of an aerosol of water from the chamber, providing a means for bacteria to leave the chamber should it become colonised.[63,64] This mechanism of humidification results in increased resistance to flow in the inspiratory limb of the breathing circuit.[61]

Other heated humidifiers

New methods of humidification have recently been marketed. One system uses a heated porous chamber. This system has been designed for the humidification of gases in one-way circuits, for example nasal cannulas.[65] Another device incorporating features of the HME with active heating and water addition has been described and seems to be effective in preliminary tests.[66,67]

Conclusion

The critically ill patient often has many interventions before admission to the intensive care unit (ICU). Provision of adequate humidification may not be seen as an important priority in this pre-ICU period. However, it is reasonable to conclude that some such patients will already have sustained injury to their respiratory tract epithelium. The rate at which epithelial function improves after such an injury is not known. The most practical solution in non-critical care environments will usually be passive humidification, because it is cheap and portable.

After admission to the ICU, maintenance of patients' respiratory function requires adequate humidification.

The minimum humidification requirements to maintain normal function have not yet been clearly delineated. The method of humidification chosen will depend on the philosophy of care and the funding of the ICU.

Some units will choose to provide humidification simply and economically, keeping in mind that there are data suggesting that outcomes are not altered by humidification method.[68,69] We lack solid evidence of the optimal level of humidification; however, under-humidification is known to cause time-dependent dysfunction and injury. Many intensive care practitioners choose to provide the best possible humidification to eliminate any possibility that under-humidification could prolong length of stay or worsen outcome.[16,55]

31

1. Ricard JD, Cook D, Griffith L, Brochard L, Dreyfuss D. Physicians' attitude to use heat and moisture exchangers or heated humidifiers: a Franco-Canadian survey. *Intensive Care Med* 2002; **28:** 719-725.

2. Helmholz HF. 'Humidification' of inhaled gases. *Anesthesiology* 1964; **25:** 207-209.

3. Negus VE. Humidification of the air passages. *Thorax* 1952; **7:** 148-151.

4. Dery R. Humidity in anaesthesiology. IV. Determination of the alveolar humidity and temperature in the dog. *Can Anaesth Soc J* 1971; **18:** 145-151.

5. Dery R, Pelletier J, Jacques A, Clavet M, Houde JJ. Humidity in anaesthesiology. 3. Heat and moisture patterns in the respiratory tract during anaesthesia with the semi-closed system. *Can Anaesth Soc J* 1967; **14:** 287-298.

6. Burton JD. Effects of dry anaesthetic gases on the respiratory mucous membrane. *Lancet* 1962; **1:** 235-238.

7. Banner AS, Green J, O'Connor M. Relation of respiratory water loss to coughing after exercise. *N Engl J Med* 1984; **311:** 883-886.

8. Rundell KW, Jenkinson DM. Exercise-induced bronchospasm in the elite athlete. *Sports Med* 2002; **32:** 583-600.

9. King M, Agarwal M, Shukla JB. A planar model for mucociliary transport: effect of mucus viscoelasticity. *Biorheology* 1993; **30**(1): 49-61.

10. Ballard ST, Inglis SK. Liquid secretion properties of airway submucosal glands. *J Physiol* 2004; **556**(Pt 1): 1-10.

11. Bryant LR. A technique for adequate humidification with mechanical respirators. *J Thorac Cardiovasc Surg* 1963; **46:** 404-407.

12. Chalon J, Loew DA, Malebranche J. Effects of dry anesthetic gases on tracheobronchial ciliated epithelium. *Anesthesiology* 1972; **37:** 338-343.

13. Forbes AR. Humidification and mucus flow in the intubated trachea. *Br J Anaesth* 1973; **45:** 874-878.

14. Villafane MC, Cinnella G, Lofaso F, et al. Gradual reduction of endotracheal tube diameter during mechanical ventilation via different humidification devices. *Anesthesiology* 1996; **85:** 1341-1349.

15. Jaber S, Pigeot J, Fodil R, et al. Long-term effects of different humidification systems on endotracheal tube patency: evaluation by the acoustic reflection method. *Anesthesiology* 2004; **100:** 782-788.

16. Williams R, Rankin N, Smith T, Galler D, Seakins P. Relationship between the humidity and temperature of inspired gas and the function of the airway mucosa. *Crit Care Med* 1996; **24:** 1920-1929.

17. Ryan SN, Rankin N, Meyer E, Williams R. Energy balance in the intubated human airway is an indicator of optimal gas conditioning. *Crit Care Med* 2002; **30:** 355-361.

18. Drew T, Vardy A, Tarnow-Mordi W, Lerski R. A numerical model of an intensive care ventilator-humidifier system. *Med Eng Phys* 1996; **18:** 251-261.

19. Kilgour E, Rankin N, Ryan S, Pack R. Mucociliary function deteriorates in the clinical range of inspired air temperature and humidity. *Intensive Care Med* 2004; **30:** 1491-1494.

20. Cushing IE, Miller WF. Nebulization therapy. *Clin Anesth* 1965; **1:** 169-218.

21. Sara C, Currie T. Humidification by nebulization. *Med J Aust* 1965; **191:** 174-179.

22. Herzog P, Norlander OP, Engstrom CG. Ultrasonic generation of aerosol for the humidification of inspired gas during volume-controlled ventilation. *Acta Anaesthesiol Scand* 1964; **12:** 79-95.

23. Spence M, Melville AW. A new humidifier. *Anesthesiology* 1972; **36**(1): 89-93.

24. Shanks CA, Gibbs JM. A comparison of two heated water-bath humidifiers. *Anaesth Intensive Care* 1975; **3**(1): 41-47.

25. Weeks DB. Humidification of anesthetic gases with an inexpensive condenser-humidifier in the semiclosed circle. *Anesthesiology* 1974; **41:** 601-604.

26. Weeks DB. Evaluation of a disposable humidifier. *Anesthesiology* 1974; **40:** 511-515.

27. Steward DJ. A disposable condenser humidifier for use during anaesthesia. *Can Anaesth Soc J* 1976; **23:** 191-195.

28. Lemmens HJ, Brock-Utne JG. Heat and moisture exchange devices: are they doing what they are supposed to do? *Anesth Analg* 2004; **98:** 382-385, table of contents.

29. Thiery G, Boyer A, Pigne E, et al. Heat and moisture exchangers in mechanically ventilated intensive care unit patients: a plea for an independent assessment of their performance. *Crit Care Med* 2003; **31:** 699-704.

30. Hedley RM, Allt-Graham J. Heat and moisture exchangers and breathing filters. *Br J Anaesth* 1994; **73:** 227-236.

31. Weeks DB, Ramsey FM. Laboratory investigation of six artificial noses for use during endotracheal anesthesia. *Anesth Analg* 1983; **62:** 758-763.

32. Unal N, Kanhai JK, Buijk SL, et al. A novel method of evaluation of three heat-moisture exchangers in six different ventilator settings. *Intensive Care Med* 1998; **24:** 138-146.

33. Gedeon A, Mebius C. The Hygroscopic Condenser Humidifier. A new device for general use in anaesthesia and intensive care. *Anaesthesia* 1979; **34:** 1043-1047.

34. Hedley RM, Allt-Graham J. A comparison of the filtration properties of heat and moisture exchangers. *Anaesthesia* 1992; **47:** 414-420.

35. Manthous CA, Schmidt GA. Resistive pressure of a condenser humidifier in mechanically ventilated patients. *Crit Care Med* 1994; **22:** 1792-1795.

36. Iotti GA, Olivei MC, Palo A, et al. Unfavorable mechanical effects of heat and moisture exchangers in ventilated patients. *Intensive Care Med* 1997; **23:** 399-405.

37. Turnbull D, Fisher PC, Mills GH, Morgan-Hughes NJ. Performance of breathing filters under wet conditions: a laboratory evaluation. *Br J Anaesth* 2005; **94:** 675-682.

38. Warmington A, Peck D. HME plus heated humidifier danger. *Anaesth Intensive Care* 1995; **23**(1): 125.

39. Prasad KK, Chen L. Complications related to the use of a heat and moisture exchanger. *Anesthesiology* 1990; **72:** 958.

40. Campbell RS, Davis K jun, Johannigman JA, Branson RD. The effects of passive humidifier dead space on respiratory variables in paralyzed and spontaneously breathing patients. *Respir Care* 2000; **45:** 306-312.

41. Prat G, Renault A, Tonnelier JM, et al. Influence of the humidification device during acute respiratory distress syndrome. *Intensive Care Med* 2003; **29:** 2211-2215.

42. Jaber S, Chanques G, Matecki S, et al. Comparison of the effects of heat and moisture exchangers and heated humidifiers on ventilation and gas exchange during non-invasive ventilation. *Intensive Care Med* 2002; **28:** 1590-1594.

43. Lellouche F, Maggiore SM, Deye N, et al. Effect of the humidification device on the work of breathing during noninvasive ventilation. *Intensive Care Med* 2002; **28:** 1582-1589.

44. Thomachot L, Vialet R, Viguier JM, Sidier B, Roulier P, Martin C. Efficacy of heat and moisture exchangers after changing every 48 hours rather than 24 hours. *Crit Care Med* 1998; **26:** 477-481.

45. Boisson C, Viviand X, Arnaud S, Thomachot L, Miliani Y, Martin C. Changing a hydrophobic heat and moisture exchanger after 48 hours rather than 24 hours: a clinical and microbiological evaluation. *Intensive Care Med* 1999; **25:** 1237-1243.

46. Davis K jun, Evans SL, Campbell RS, et al. Prolonged use of heat and moisture exchangers does not affect device efficiency or frequency rate of nosocomial pneumonia. *Crit Care Med* 2000; **28:** 1412-1418.

47. Thomachot L, Boisson C, Arnaud S, Michelet P, Cambon S, Martin C. Changing heat and moisture exchangers after 96 hours rather than after 24 hours: a clinical and microbiological evaluation. *Crit Care Med* 2000; **28:** 714-720.

48. Thomachot L, Leone M, Razzouk K, Antonini F, Vialet R, Martin C. Randomized clinical trial of extended use of a hydrophobic condenser humidifier: 1 vs. 7 days. *Crit Care Med* 2002; **30:** 232-237.

49. Broach SD, Durbin CG jun. A randomized, controlled, clinical trial of a chemically-reactive heated humidifier. *Respir Care* 2001; **46**(1): 37-42.

50. Kirton OC, DeHaven B, Morgan J, Morejon O, Civetta J. A prospective, randomized comparison of an in-line heat moisture exchange filter and heated wire humidifiers: rates of ventilator-associated early-onset (community-acquired) or late-onset (hospital-acquired) pneumonia and incidence of endotracheal tube occlusion. *Chest* 1997; **112:** 1055-1059.

51. Dreyfuss D, Djedaini K, Gros I, et al. Mechanical ventilation with heated humidifiers or heat and moisture exchangers: effects on patient colonization and incidence of nosocomial pneumonia. *Am J Respir Crit Care Med* 1995; **151:** 986-992.

52. Branson RD, Davis K jun, Brown R, Rashkin M. Comparison of three humidification techniques during mechanical ventilation: Patient selection, cost and infection considerations. *Respiratory Care* 1996; **41:** 809-816.

53. Kola A, Eckmanns T, Gastmeier P. Efficacy of heat and moisture exchangers in preventing ventilator-associated pneumonia: meta-analysis of randomized controlled trials. *Intensive Care Med* 2005; **31:** 5-11.

54. Martin C, Papazian L, Perrin G, Bantz P, Gouin F. Performance evaluation of three vaporizing humidifiers and two heat and moisture exchangers in patients with minute ventilation > 10 l/min. *Chest* 1992; **102:** 1347-1350.

55. Luchetti M, Stuani A, Castelli G, Marraro G. Comparison of three different humidification systems during prolonged mechanical ventilation. *Minerva Anestesiol* 1998; **64:** 75-81.

56. Food and Drug Administration. *Hazards of Volume Ventilators and Heated Humidifiers*. Department of Health and Human Services, 15 September 1993.

57. Gowardman JR, Moriarty B. Explosion and fire in the expiratory limb of a Fisher and Paykel 'three in one' respiratory care system. *Anaesth Intensive Care* 1998; **26:** 427-430.

58. Wong DH. Melted delivery hose – a complication of a heated humidifier. *Can J Anaesth* 1988; **35:** 183-186.

59. Lellouche F, Taille S, Maggiore SM, et al. Influence of ambient and ventilator output temperatures on performance of heated-wire humidifiers. *Am J Respir Crit Care Med* 2004; **170:** 1073-1079.

60. Carter BG, Kemp T, Mynard J, Hochmann M, Osborne A. Compensating for the effect of inlet gas temperature on heated humidifier performance. *Anaesth Intensive Care* 2003; **31**(1): 54-57.

61. Oh TE, Lin ES, Bhatt S. Resistance of humidifiers, and inspiratory work imposed by a ventilator-humidifier circuit. *Br J Anaesth* 1991; **66:** 258-263.

62. Prin S, Chergui K, Augarde R, Page B, Jardin F, Vieillard-Baron A. Ability and safety of a heated humidifier to control hypercapnic acidosis in severe ARDS. *Intensive Care Med* 2002; **28:** 1756-1760.

63. Vesely D, Anderson J, Halbert M, Wyman L. Bacterial output from three respiratory therapy humidifying devices. *Respir Care* 1979; **24:** 228-234.

64. Rhame FS, Streifel A, McComb C, Boyle M. Bubbling humidifiers produce microaerosols which can carry bacteria. *Infect Control* 1986; **7:** 403-407.

65. Waugh JB, Granger WM. An evaluation of 2 new devices for nasal high-flow gas therapy. *Respir Care* 2004; **49:** 902-906.

66. Larsson A, Gustafsson A, Svanborg L. A new device for 100 per cent humidification of inspired air. *Crit Care* 2000; **4**(1): 54-60.

67. Kapadia F, Shelly MP, Anthony JM, Park GR. An active heat and moisture exchanger. *Br J Anaesth* 1992; **69:** 640-642.

68. Boots RJ, Howe S, George N, Harris FM, Faoagali J. Clinical utility of hygroscopic heat and moisture exchangers in intensive care patients. *Crit Care Med* 1997; **25:** 1707-1712.

69. Thomachot L, Vialet R, Arnaud S, Barberon B, Michel-Nguyen A, Martin C. Do the components of heat and moisture exchanger filters affect their humidifying efficacy and the incidence of nosocomial pneumonia? *Crit Care Med* 1999; **27:** 923-928.