A new prototype of an electronic jet-ventilator and its humidification system

Paul Kraincuk, Anton Kepka, Gerald Ihra, Christa Schabernig and Alexander Aloy

Background: Adequate humidification in long-term jet ventilation is a critical aspect in terms of clinical safety.

Aim: To assess a prototype of an electronic jet-ventilator and its humidification system.

Methods: Forty patients with respiratory insufficiency were randomly allocated to one of four groups. The criterion for inclusion in this study was respiratory insufficiency exhibiting a Murray score above 2. The four groups of patients were ventilated with three different respirators and four different humidification systems. Patients in groups A and B received superimposed high-frequency jet ventilation (SHFJV) by an electronic jet-ventilator either with (group A) or without (group B) an additional humidification system. Patients in group C received high-frequency percussive ventilation (HFPV) by a pneumatic high-frequency respirator, using a hot water humidifier for warming and moistening the inspiration gas. Patients in group D received conventional mechanical ventilation using a standard intensive care unit respirator with a standard humidification system. SHFJV and HFPV were used for a period of 100 h (4 days).

Results: A significantly low inspiration gas temperature was noted in patients in group B, initially (27.2 ± 2.5°C) and after 2 days (28.0 ± 1.6°C) ($P < 0.05$). The percentage of relative humidity of the inspiration gas in patients in group B was also initially significantly low (69.8 ± 4.1%; $P < 0.05$) but rose to an average of 98 ± 2.8% after 2 h. The average percentage across all four groups amounted to 98 ± 0.4% after 2 h. Inflammation of the tracheal mucosa was found in patients in group B and the mucosal injury score (MIS) was significantly higher than in all the other groups. Patients in groups A, C and D showed no severe evidence of airway damage, exhibiting adequate values of relative humidity and temperature of the inspired gas.

Conclusion: The problems of humidification associated with jet ventilation can be fully prevented by using this new jet-ventilator. These data were sustained by nondeteriorating MIS values at the end of the 4-day study period in groups A, C and D.

Address: Department of Anesthesiology and General Intensive Care, University of Vienna, Austria

Correspondence: Alexander Aloy, MD, Department of Anesthesiology and General Intensive Care Medicine, University of Vienna, 18–20 Waehringer Guertel, A-1090 Austria. Tel: +43 1 40400 4107; fax: +43 1 40400 4104; e-mail: alexander.aloy@univie.ac.at

Keywords: bronchoscopy, conventional mechanical ventilation, electronic jet ventilation, jet adapter, humidification system

Received: 18 June 1998
Revisions requested: 5 June 1999
Revisions received: 21 June 1999
Accepted: 6 July 1999
Published: 30 July 1999

Crit Care 1999, **3**:101–110

The original version of this paper is the electronic version which can be seen on the Internet (http://ccforum.com). The electronic version may contain additional information to that appearing in the paper version.

Introduction

High-frequency jet ventilation (HFJV) has proved to be an alternative to conventional mechanical ventilation (CMV) [1–4]. The major advantage of HFJV lies in the improvement of the mucociliary transport system [5], the recruitment of atelectatic areas [6], and the improvement of oxygenation [7,8], maintaining very low tidal volumes to avoid lung barotrauma [9]. However, one of the critical issues concerning HFJV is the adequate humidification and warming of the inspired gases [10]. Specific problems derive from the physical phenomena of the high velocity of the jet-stream, the Joule–Thompsen effect and the Venturi effect [11], causing low temperature and low relative humidity of the inspired gases [12,13].

There are several forms of HFJV [14]. Combined high-frequency jet ventilation (CHFJV) is a technique that requires a conventional respirator (endotracheal tube) and

HFJV = high-frequency jet ventilation; CMV = conventional mechanical ventilation; CHFJV = combined high-frequency jet ventilation; SHFJV = superimposed high-frequency jet ventilation; HFPV = high-frequency percussive ventilation; ICU = intensive care unit; ARDS = acute respiratory distress syndrome; ALI = acute lung insufficiency; PCWP = pulmonary capillary wedge pressure; CI = cardiac index; SVRI = systemic vascular resistance index; PEEP = positive end-expiratory pressure; FiO_2 = fractional inspiratory oxygen concentration; I:E = inspiration to expiration time ratio; SaO_2 = arterial oxygen saturation; MIS = mucosal injury score; Q_{H_2O} = rate of humidification; ISB = isothermic saturation boundary; LF = low frequency; HF = high frequency; CPPV = continuous positive pressure ventilation; Pmax = maximal airway pressure; PaO_2 = partial pressure of oxygen.

a high-frequency jet-ventilator (endotracheal jet tube) [15]. A special type of the CHFJV is the superimposed high-frequency jet ventilation (SHFJV), which is a time-regulated, pressure-controlled ventilation technique. It is characterized by the simultaneous application of a low-frequency jet stream and a high-frequency jet stream, resulting in the superposition of two jet streams with different frequencies [16]. This ventilation technique is managed by one respirator. Volumetric diffusive respiration is a type of ventilation (using, for example, the VDR 4, Percussionaire Corp, Idaho, USA) named high-frequency percussive ventilation (HFPV) [17]. Although these techniques have been described in the literature, they have not been rigorously compared to each other in long-term applications. There are several humidification systems for CMV [18] and HFJV [19,20], for example, hot water humidifiers, cold water humidifiers and heat and moisture exchangers. The most commonly used humidification system in our institution is the hot water humidifier (Aquapor, Type 8406640, Draeger Corp, Luebeck, Germany).

The aim of this study was to show that the problems of humidification associated with SHFJV can be prevented by using the correct humidification system. Proper methods for showing possible epithelial damage were used.

Material and methods

After approval by the institutional Ethics Committee and informed consent, 40 intensive care unit (ICU) patients (Table 1) were randomly allocated to one of four groups (A, B, C or D; 10 patients in each group). The inclusion criterion was respiratory insufficiency due to pneumonia or involvement of the lungs in multi-organ dysfunction syndrome. All patients showed a Murray score [21] (Table 2) above 2.0, fulfilling the criteria for acute respiratory distress syndrome (ARDS) or acute lung insufficiency (ALI) according to the European American Consensus Conference 1994 (Table 2). The mean number of CMV respiration days before group allocation was 2.2 days. The median age of the 16 female and 24 male patients was 67 years, ranging from 55 to 79 years.

All patients were sedated with midazolam 0.1–0.15 mg/kg per h and sufentanil 0.01–0.015 mg/kg per h, and optionally with ketamine 1–2 mg/kg per h. Fluid and catecholamines were administered to achieve a pulmonary capillary wedge pressure (PCWP) between 12 and 18 mmHg, a cardiac index (CI) above 2.5 l/min per m^2, and a systemic vascular resistance index (SVRI) between 600 and 1500 dyn × s/cm^5 per m^2. Antibiotic therapy was adapted according to the results of bacterial cultures of the tracheobronchial secretion. Red packed cells where given to restore blood volume when required and to maintain the hematocrit >32%. Hemodynamic variables were assessed through central venous and peripheral arterial lines. In eight patients, a Swan-Ganz pulmonary artery catheter was inserted.

Table 1

Patient characteristics and demographic data at study entry

Characteristic	
Age (years)	67 ± 12
Sex (male/female)	24/16
Etiology of acute respiratory failure (*n*)	
Postoperative	20
Pneumonia	12
Sepsis	8
Drug treatment	
Midazolam (*n*, mg/kg per h)	40, 0.12 ± 0.03
Sufentanil (*n*, mg/kg per h)	40, 0.012 ± 0.003
Ketamine (*n*, mg/kg per h)	15, 1.5 ± 0.5
Respirator therapy (CPPV, pressure controlled mode, by Evita*)	
Pmax (*n*, mbar)	40, 26 ± 4
PEEP (*n*, mbar)	40, 10 ± 2
FiO_2 (*n*, %)	40, 70 ± 16
I:E (*n*, time ratio)	40, 1:1

*Draeger Corp, Luebeck, Germany. CPPV, continuous positive pressure ventilation; Pmax, maximal airway pressure; PEEP, positive end-expiratory pressure; FiO_2, fractional inspiratory oxygen concentration; I:E, inspiration to expiration time ratio.

Table 2

Respiratory insufficiency as the inclusion criterion (Murray score [21] and European American Consensus Conference 1994 values)

Murray Score (mean values of 40 patients)	Measured value	Points
PaO_2/FiO_2	160 ± 15	3
PEEP (mbar)	10 ± 2	2
Compliance (ml/cmH_2O)	36 ± 3	3
X-ray quadrants	2 ± 1	2
Sum total		10
Score (Sum total/4)		2.5

European American Consensus Conference 1994

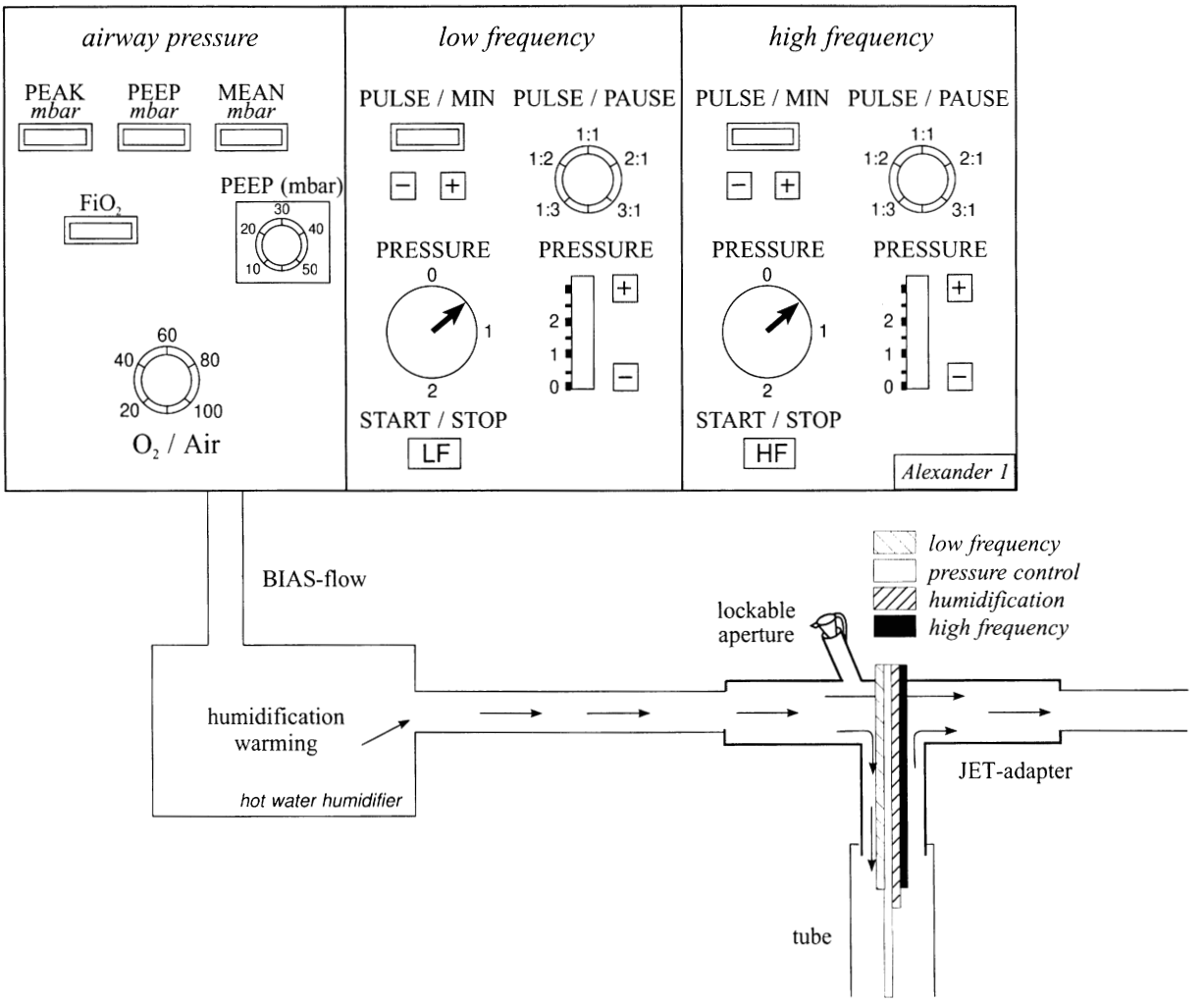
ARDS:
- acute onset
- PaO_2/FiO < 200 mmHg (PEEP independent)
- Bilateral infiltration on ap chest X-ray
- PCWP < 18 mmHg or absence of left atrial hypertension

ALI:
- acute onset
- PaO_2/FiO_2 < 300 mmHg (PEEP independent)
- Bilateral infiltration on ap chest x-ray
- PCWP < 18 mmHg or absence of left atrial hypertension

PaO_2, partial pressure of oxygen; FiO_2, fractional inspiratory oxygen concentration; PEEP, positive end-expiratory pressure; ARDS, acute respiratory distress syndrome; ALI, acute lung insufficiancy; ap, anterior–posterior; PCWP, pulmonary capillary wedge pressure.

Patients in group A received SHFJV by a prototype of an electronic jet-ventilator (Alexander 1, Festo Corp, Vienna, Austria) (Fig. 1). The Alexander 1 works in an open system, providing a low-frequency jet stream ranging from 4 to 40 breaths/min that influences the peak pressure, and a high-frequency jet stream ranging from 60 to 990 breaths/min that influences the positive end-expiratory

Figure 1



A prototype of an electronic jet ventilator (Alexander 1; Festo Corp, Vienna, Austria). This ventilator delivers a high-frequency and a low-frequency jet stream simultaneously to the patient: superimposed high-frequency jet ventilation (SHFJV). PEEP, positive end-expiratory pressure; FiO_2, fractional inspiratory oxygen concentration; LF, low frequency; HF, high frequency.

pressure (PEEP). Both jet streams are simultaneously applied to the patient using a newly developed jet adapter [22]. This plastic jet adapter (T-connector with a luer lock, Willy RãSCH AG, Kermen, Germany) consists of a T-piece and four central small-bore cannulas that can be connected to any commercially available endotracheal tube, avoiding reintubation with an endotracheal jet tube when beginning SHFJV. The high-frequency and low-frequency ventilation is performed with two jet nozzles which have been designed according to optimal dynamic flow measurements. Two further jet nozzles are used for continuous airway pressure monitoring and for passing saline solution into the jet stream. Next to the T-piece there is a lockable aperture that can be used for suctioning or bronchoscopy, without loss of PEEP.

For humidification, we used a combined humidification and warming system for patients in group A. The entrained gases (bias flow) were humidified by a hot water humidifier (Aquapor) and the inspiration gas bubbles through a waterbath heated by an immersed heating element. Gas leaving the device is saturated with water vapor and is heated to a pre-set temperature. The absolute humidity can be altered by changing the temperature of the waterbath.

Humidification of the jet gas was achieved by a continuous infusion of 0.9% saline via a separate cannulla in the jet adapter. This was propelled and nebulized by the high-pressure jet stream, starting at 20 ml/h. The saline solution was warmed to 39°C by a fluid warmer (HL-90 INT, Level

1 Technologies Corp, Rockland, Massachusetts, USA) before it reached the jet adapter (ie, an additional humidification system). This allows for a possible warm-up of 42°C to compensate for the temperature drop of the gas after decompression (the Joule–Thompsen effect).

Patients in group B also used the Alexander 1, with a hot water humidifier for the bias flow (Aquapor) and a continuous saline infusion for the jet flow (the additional fluid warmer mentioned above was not used in patients in group B).

Patients in group C received HFPV using the VDR 4, which is a pneumatic time-regulated, pressure-controlled respirator that works in a closed system to provide two oscillating pressure plateaus. The HFPV is generated by a phasitron, causing gases in the airways to oscillate back and forth between the respirator and the patient, with the fresh gas entrained from the bias flow. Because of the continuous flow applied during HFPV the patient is able to breathe spontaneously at any time during the respiratory cycle, and respiratory weaning can be performed. This method allows conventional heater humidifiers to be used. In our study we used a hot water humidifier (Aquapor).

Patients in group D received CMV using a conventional intensive care respirator (Evita, Draeger Corp) in a pressure-controlled mode using a hot water humidifier (Aquapor).

In all four groups, PEEP, the fractional inspiratory oxygen concentration (FiO_2) and inspiration to expiration time ratio (I:E) were adjusted to keep arterial oxygen saturation (SaO_2) above 90% with the lowest possible peak airway pressure and FiO_2.

On the first day of the study, relative humidity measurements were performed at jet ventilation commencement, and after 20 min and 2 h. Consequently, data were taken only twice a day until the end of the 4-day study. Values were accurate to ±2% of relative humidity. Relative humidity was measured inside the tube, at its distal end, with an electronic device (E20-FXD, E u. E Elektronik Corp, Unterwiesen, Austria). This very small sensor consists of a condensator with a capacity of 600 picoFarad at a relative humidity of 70%. The polymer sensor changes its capacity according to the amount of moisture inside the area that is measured. This chemical reaction is translated into an electronic signal, which is shown on a liquid crystal display (ie, monitor) outside the tube as a percentage of the relative humidity inside the inspired gas flow.

The temperature was measured at the same location following the same timetable as above with a temperature sensor line (21076 A, Hewlett Packard, Palo Alto, California, USA). Values were accurate to ±0.1°C.

The tracheobronchial mucosa was inspected twice a day by bronchoscopy (Olympus BF Type P20D, Olympus Corp Ltd, Shirakwa, Japan) over the entire 100-h study period according to a mucosal injury score (MIS). The MIS takes into account the area and the extent of macroscopic epithelial damage in the tracheobronchial system: area A represents the mucosa around the cuff (for inspection, the tube has to be withdrawn a little); area B represents the mucosa from below the cuff down to the carina; area C represents the mucosa of the right mainstem bronchus; and area D represents the mucosa of the left mainstem bronchus. Each area is given a number, representing the amount of mucosal damage (0, no exsiccosis, no inflammation and no necrosis; 1, exsiccosis as shown by a pale mucosa with no signs of epithelial damage; 2, inflammation of the mucosa shown by mucosal redness and tumor; 3, necrosis shown by a pale mucosa with signs of epithelial damage). Thus, the possible value of the MIS ranges from 0 to 12 with damage classified as: 1–3, simple damage; 4–8, significant damage; 9–12, severe damage.

The humidification rate (initially 20 ml/h) was set depending on the bronchoscopic aspect of the tracheobronchial mucosa; classification above 'simple damage' (ie, MIS above 3) led to an increase in the humidification rate up to and above 45 ml/h.

Statistical analysis

For demographic data (Table 1) and measured values of relative humidity and gas temperature a Duncan test was calculted to determine statistical significance. All values are expressed as mean ± standard deviation. $P < 0.05$ was regarded as statistically significant.

Since homogeneity of variances were seen, statistical evaluation was performed using single factorial analysis. However, as the small number of spot samples impaired the standard distribution, a nonparametric method (variance analysis by Kruskal–Wallis) was also used. Both methods showed the same significant differences between the groups. Furthermore, a pathanalysis was calculated to check the statistical relevance of the hypothetical influence of the forms of ventilation on temperature and humidity.

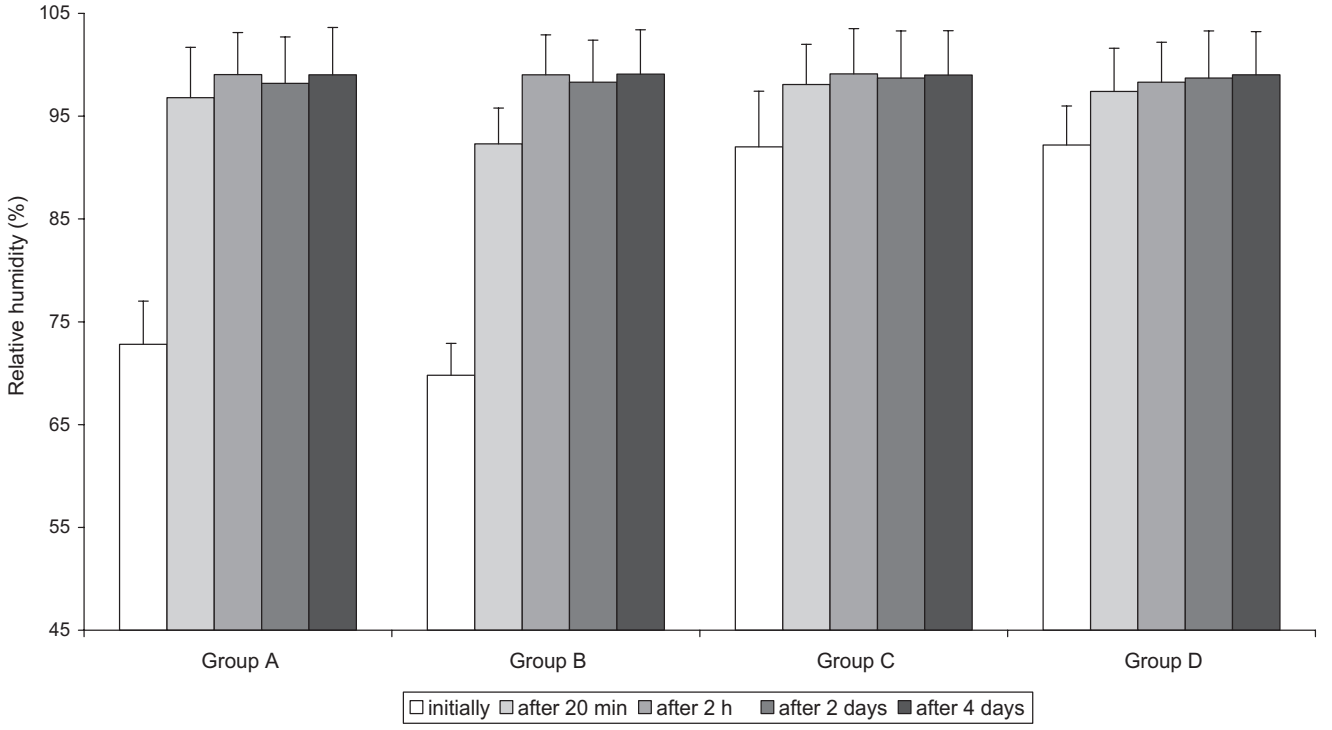
Results

Prior to random allocation to the four groups, all patients were conventionally ventilated (CMV using Evita) for 48–72 h. The average number of ventilation days in each group was 10 days (the duration of mechanical ventilation before weaning started). The overall period of ventilation was 18 ± 4 days.

Relative humidity

Initially, the lowest percentage of mean relative humidity (Fig. 2) was seen in patients in group B (69.8 ± 4.1%;

Figure 2



Percentage of relative humidity (RH) of the inspiration gas for each group initially, after 20 min, 2 h, after 2 days and after 4 days of ventilation (mean ± standard deviation).

$P < 0.05$). Patients in group A showed a mean relative humidity of 71.2%, patients in group C showed a mean relative humidity of 92.2% and patients in group D showed a mean relative humidity of 92.0%.

After 20 min of ventilation the mean relative humidity of the inspiration gas was still lower in patients in group B (92.8%) compared with patients in group A (96.8%), group C (97.4%), and group D (98.6%).

After 2 h of ventilation patients in all four groups showed almost equivalent mean values (98 ± 0.4%). All measurements taken from this point until the end of the 100-h study protocol showed no more significant changes compared to the values measured after 2 h.

Temperature

Initially, the lowest temperature measured inside the tube (Fig. 3) was seen in patients in group B (27.2 ± 2.5°C; $P < 0.05$). Patients in group A had a mean gas temperature of 31.4 ± 2.8°C, those in group C had a mean gas temperature of 32.1 ± 2.6°C, and those in group D had a mean gas temperature of 34.2 ± 2.7°C.

After 20 min of ventilation the inspiration gas temperature was still lower in patients in group B (27.1 ± 1.8°C) compared with patients in group A (32 ± 1.8°C), patients in group C (32.5 ± 2.3°C), and patients in group D (34.1 ± 2.5°C).

After 2 h of ventilation the trend was similar (patients in group B, 27.1 ± 1.8°C; patients in group A, 32.4 ± 1.1°C; patients in group C, 32.6 ± 1.6°C; and patients in group D, 34.3 ± 2.3°C).

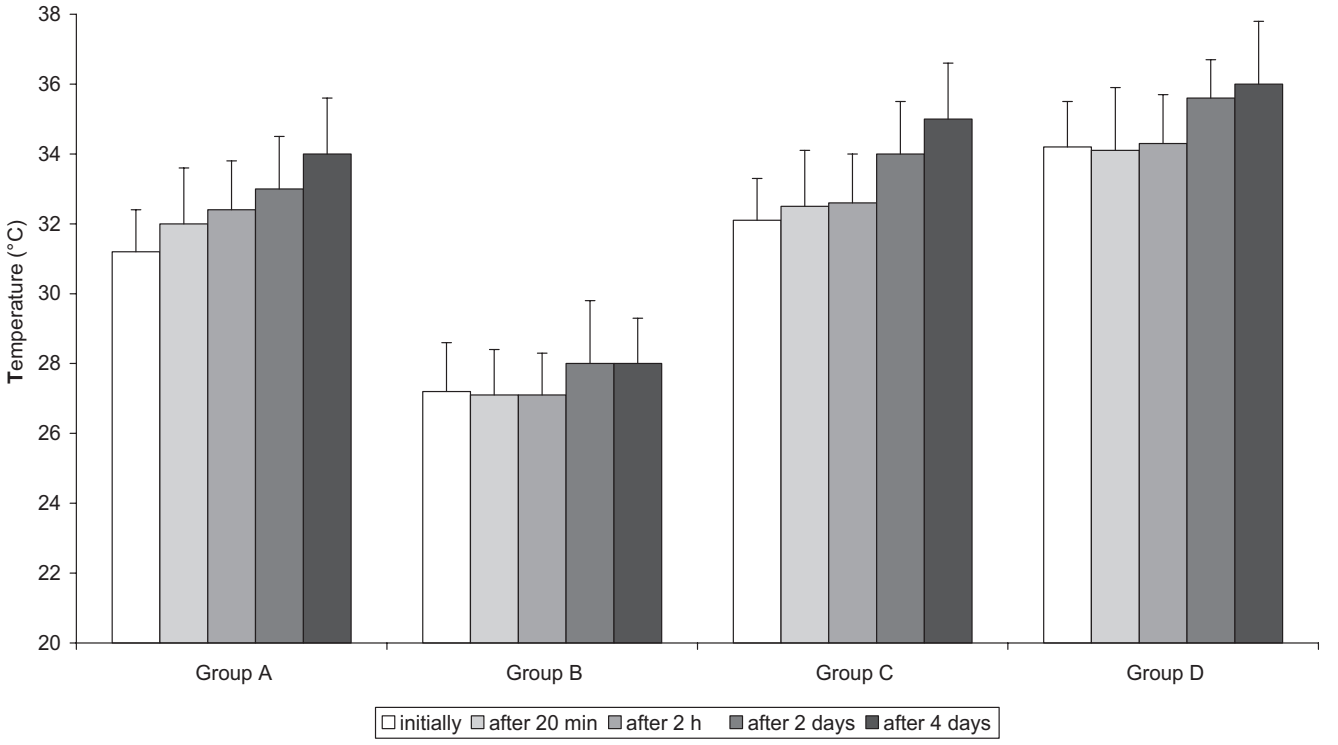
After 2 days the values were: patients in group B, 28.0 ± 1.6°C; patients in group A, 33.2 ± 1.7°C; patients in group C, 33.2 ± 2.5°C; patients in group D, 34.5 ± 1.8°C.

After 4 days the values were: patients in group B, 28.0 ± 1.9°C; patients in group A, 33.0 ± 1.7°C; patients in group C, 33.6 ± 2.5°C; patients in group D, 34.3 ± 1.8°C.

Mucosal injury score

During the course of SHFJV, patients in group B showed signs of epithelial lesions and inflammation of the mucosa in area B within hours (MIS = 2), deteriorating on the third and fourth day (MIS = 3) (Fig. 4). During bronchoscopy, a

Figure 3



Temperature of the inspiration gas of each group initially, after 20 min, 2 h, after 2 days and after 4 days of ventilation (mean ± standard deviation).

tracheobronchial secretion was removed. The bronchial epithelium (area C) showed no pathologic evidence. Typical changes to the mucosa were not detected in patients in any group other than those in group B.

In group A, C and D no epithelial damage could be found in any area. Eight patients (group independent) who were in an intermittent prone position (usually turned every 12 h) showed little tracheal damage at the end of the tube.

Although one patient in group C died as a result of severe sepsis, their tracheobronchial mucosa showed no adverse tissue change when compared to the control group. As the period of ventilation (72 h) was probably sufficient to induce any injury as a result of the humidification system, data from this patient were not excluded in our comparison. The statistical relevance of the hypothetical influence of the forms of ventilation on temperature and humidity were checked by pathanalysis. Only group B appeared to show any influence.

Table 3

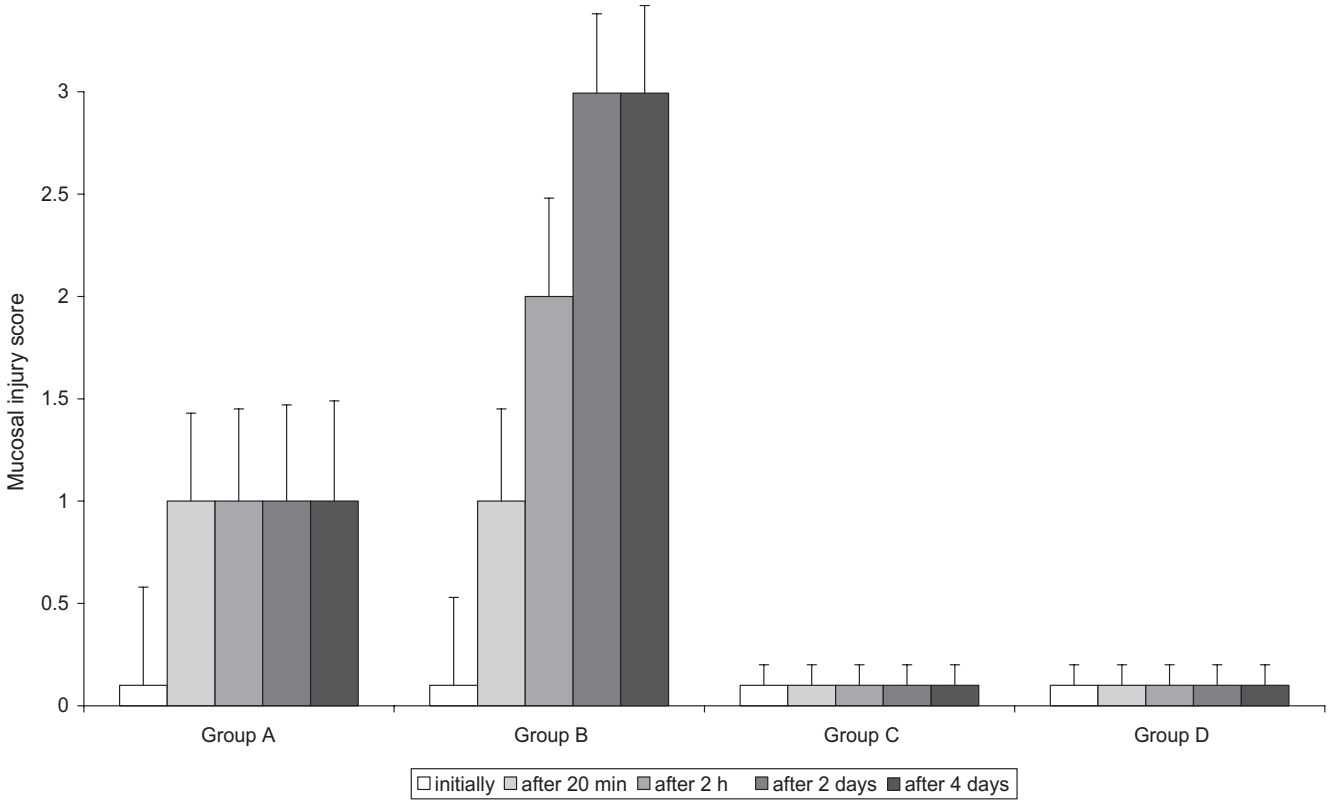
Mucosal injury score and humidification rate settings during the course of superimposed high-frequency jet ventilation

	Group A	Group B
Initially		
Mucosal injury score	0	0
Humidification rate setting (ml/h)	20	20
After 20 min		
Mucosal injury score	0	1
Humidification rate setting (ml/h)	20	30
After 2 h		
Mucosal injury score	1	2
Humidification rate setting (ml/h)	30	40
After 2 days		
Mucosal injury score	2	3
Humidification rate setting (ml/h)	40	45
After 4 days		
Mucosal injury score	1	3
Humidification rate setting (ml/h)	35	50

Humidification rate of the additional humidification system

Initially, the humidification rate of the additional humidification system was set at 20 ml/h saline solution via the jet adapter (Table 3). This was changed depending on the bronchoscopic aspect of the tracheobronchial mucosa and the presence of dry secretion. Patients in group A showed an average setting of 30 ± 10 ml/h, starting at

Figure 4



Mucosal injury score of each group initially, after 20 min, 2 h, after 2 days and after 4 days of ventilation (mean ± standard deviation).

20 ml/h and rising to 40 ml/h on the second day. Patients in group B showed the highest demand for saline solution, with an average setting of 45 ± 5 ml/h and a peak of 50 ml/h on the fourth day. Patients in groups C and D were served by a hot water humidifier without an additional humidification system.

Discussion

This study yielded two major findings. Firstly, adequate humidification without sufficient warming of the inspiration gas does not prevent epithelial damage or inflammation. Secondly, the amount of humidification needed has to be changed almost every day, sometimes even within hours. Thus, regular bronchoscopy is necessary. Leaving the inspiration gas temperature significantly below 30°C ('cold ventilation'), even under high humidity (relative humidity above 90%), leads to epithelial damage, inflammation or even necrotizing tracheobronchitis [23].

The continuous 0.9% saline infusion into the humidification line of the jet adapter started at 20 ml/h. Regular checks on the mucosa showed that over 75% of all patients needed higher humidification over the whole study period, in some cases an increase of 200%. This increase was dependent on the bronchoscopic aspect of the tracheobronchial mucosa and the presence of dry secretions. Detecting these changes for alteration of the humidification settings requires a lot of experience and cannot be explained merely by facts, figures and equations.

To humidify a dry gas and reach 100% relative humidity, however, requires 44 mg water to be added per litre of inspired gas. As a consequence, the rate of humidification (Q_{H_2O}; ml/h) equals 2.64 × V, where V is the minute ventilation. Because minute ventilation during high-frequency ventilation ranges between 20 and 40 l/min, Q_{H_2O} may vary between 50 and 100 ml/h. Of course, any change in the ventilatory parameters (tidal volume, ventilation frequency per min, driving pressure, I:E) modifies minute ventilation and, therefore, Q_{H_2O}. These basic physical principles of humidification are well known, and are accepted in ventilation therapy. The saline solution was warmed by a fluid warmer to 39°C before it reached the jet adapter, allowing for a possible warm up of 42°C if necessary, to compensate for the temperature drop of the gas after decompression (Joule–Thompsen effect) and possible epithelial lesions.

In fact, increasing the temperature of the inspired gas delivered during high-frequency jet ventilation from 39°C–42°C might appear insufficient. After decompression of the gas into the trachea, there is a sudden drop in the temperature of 5–10°C. As a consequence, to reach 37°C in the tracheobronchial tree, inspired gas should be warmed to at least 45°C.

In the face of this argument, our humidification system seems to face a serious drawback by providing insufficient temperature of the inspired gas, especially in terms of inducing lesions in the tracheobronchial tree after a prolonged period of SHFJV. However, our period of observation covered approximately 4 days and no signs of persisting inflammation or even necrotizing tracheobronchitis were observed in any group; only tracheobronchial damage (epithelial lesions and transitory inflammation) were detected. These alterations could be compensated for by increasing the humidification rate (Table 3) of the additional humidification system, which differed more than 50% from patient to patient.

The etiology of these different demands can be seen in the multifactorial reasons that influence it, the most important of which is the drift of the so-called isothermic saturation boundary (ISB). This boundary usually lies above the carina during spontaneous breathing and represents the point where the inspired air is fully saturated with vapor and has a temperature of 36°C. Turbulent flow also changes to laminar flow below this point [24], and intubation and ventilation bring this ISB further back along the tracheobronchial tract. Thus, the ability of the lungs to facilitate an adequate temperature and amount of moisture in the inspiration gas is dramatically impaired, leading to the problems that are associated with inadequate humidification and warming.

At present, equipment to monitor humidity is not sufficiently sophisticated to allow accurate breath to breath measurements of humidity within the airway. The estimation of humidification requirements must therefore be based on scientific evidence and clinical impression. Humidification of inspired gases should not be considered in isolation but as part of total airway management. It should be associated with careful fluid balance, physiotherapy, bronchial aspiration and appropriate drug therapy.

Functional studies [24,25] have shown adverse effects of dry inspiratory gas on the tracheal mucosa during ventilation. Chalon *et al* [12] studied the cytology of epithelial cells from tracheal washings taken during anesthesia via an endotracheal tube with gases at different humidities. Abnormal cytology was found within 2 h of ventilation with dry gas but not at 60% humidity or higher. Doyle *et al* [18] demonstrated that extensive epithelial damage to the trachea with destruction of the cilia, tissue inflammation and necrosis occurred after 72 h of CMV with dry gas, but no damage was observed at high humidity. Other studies concerning jet ventilation [26,27] demonstrated epithelial damage at the level of the carina and left and right mainstem bronchus. There are multiple effects of passing warm, dry gas over the tracheal mucosa. Initially, there is an increase in blood supply to this region [28], probably as a result of release of local vasodilators [29]. Subsequently, there is an increase in the osmolality of the mucus secretion as the airway mucosa humidifies the gas before it enters the lungs [30]. Mucus secretions are under parasympathetic control and increase in volume after vagal nerve stimulation [31] or acetylcholine treatment [32]. As the mucus secretion becomes increasingly hyperosmolar and dry, it is trapped and encapsulated beneath the surface. Cellular injury occurs and neutrophil sequestration is exaggerated due to the increase in blood supply. In this situation, with aggravating tissue damage, an exsudate is formed that appears as blisters below the mucosal surface. Because of the external force from mechanical ventilation or jet ventilation, the encapsulated mucus penetrates and causes sloughing of the tracheal epithelial cells.

The clinical implications of inadequate humidification during mechanical ventilation or jet ventilation are very important. The use of dry gas during anesthesia is still common during surgical procedures. Furthermore, dry oxygen is used during resuscitation of asphyxiated patients and preterm infants. The use of dry gas during routine respiratory changes may impair already compromised lungs, especially in infants with chronic obtructive pulmonary disease and adults with ARDS. Tarnow-Mordi *et al* [32,33] showed an increase in the incidence of pneumothorax and chronic lung disease in patients who were ventilated with inspired gas at low temperatures, implying a low inspired absolute humidity. Necrotizing tracheobronchitis [34], which has been described in several ICUs, has also been attributed to low inspired humidity leading to sloughing of tracheal and bronchial cells [35]. Buchdahl *et al* [36] showed an increase in chronic upper respiratory tract problems in children with reduced ciliary beat frequency. Damage to the tracheal mucosa occurs during endotracheal intubation and ventilation whatever the inspired humidity. Using dry inspired gas the damage is dramatically worse; therefore, clinical procedures should aim to reduce the use of dry gases in ventilator circuits.

Warming of the inspiration gas is just as important as its humidification to avoid bronchotracheal damage. A lack of warming of the inspired gas is said to be responsible for necrotizing tracheobronchitis among many other cofactors, especially under treatment with HFJV [37,38]. However, Keszler *et al* [39] showed in a multicenter clinical trial that the incidence of necrotizing tracheobronchitis is similar comparing HFJV with CMV in neonates.

Although multiple factors are associated with necrotizing tracheobronchits, Hanson *et al* [40] proposed that regional or generalized airway ischemia was the mechanism for airway damage, based on the appearance of mucosal damage with a lack of warming.

Cavanagh *et al* [41] found an increase in regional tracheal blood flow (10.3-fold), measured by radioactive microspheres, under HFJV compared with spontaneous breathing, increasing further when using dry gas at 22°C. The authors claim the enhanced local hyperemia effect is because of greater water and heat extraction. A moderate hyperventilation that occurred during high-frequency jet ventilation in this study [41] might also have affected tracheal blood flow, although the authors concede that the hyperemia effects appear to relate to shear stress and water removal rather than alveolar partial pressure of carbon dioxide. They found blood flow improvements in the tracheal mucosa, with the highest increase using dry gas.

Although the study by Cavanagh *et al* covers only a period of 2.5 h, another study [42] shows that longer periods of ventilation (33 h) produce no significant differences in airway damage when comparing CMV with HFJV.

Another explanation for the hyperemia effects under HFJV is given by Baile *et al* [43]. They claim that greater shear stresses with HFJV may also alter mucosal epithelia permeability and secondarily affect the blood flow.

Our study showed that, by providing proper humidification and warming of the inspiration gases, epithelial damage to the tracheobronchial mucosa can be prevented, as can possible inflammation and necrotizing tracheobronchitis, even in long-term applications.

The damage to the mucosa observed in patients in group B relates well with the outcome of the studies by Rouby *et al* [7,8] and Chatburn and McClellan [19], who have made a very interesting contribution to the problems associated with humidification and warming of high-frequency jet ventilation. Doyle *et al* [18] and Chatburn and McClellan [19] showed that, by providing adequate moisture and proper temperature of the inspiration gases, deleterious effects on the tracheobronchial mucosa can be prevented. Although their mechanical expenditures and material costs were much higher than ours, the outcome of their studies were just as satisfactory as the results of this study. SHFJV has been shown to be a serious alternative to CMV. Using the Alexander 1, the problems of humidification and warming of the ventilation gas can be handled very well.

References

1. Klain M, Smith RB: **High frequency percutaneous transtracheal jet ventilation.** *Crit Care Med* 1977, **5**:230–236.
2. Schuster DP, Klain M, Snyder JV: **Comparison of high frequency jet ventilation to conventional ventilation during severe acute respiratory failure.** *Crit Care Med* 1982, **10**:625–631.
3. Nordin U, Klain M, Keszler H: **Electron-microscopic studies of tracheal mucosa after high frequency jet ventilation.** *Crit Care Med* 1981, **10**:211–215.
4. Neu J, Hamilton L, Linehan J: **Long-term high frequency jet ventilation in neonates.** *Crit Care Med* 1984, **12**:833–839.
5. Chang HK, Harf A: **High-frequency jet ventilation: a review.** *Respir Physiol* 1984, **57**:135–152.
6. Nordin U, Keszler H, Klain M: **How does high frequency jet ventilation effect the muciliary transport ?** *Crit Care Med* 1981, **19**:160–165.
7. Rouby JJ, Fusciardi J, Bourgain JL, Viars P: **High-frequency jet-ventilation in postoperative respiratory failure: determinants of oxygenation.** *Anaesthesiology* 1983, **59**:281–287.
8. Rouby JJ, Simonneau G, Benhamou D, *et al:* **Factors influencing pulmonary volumes and CO_2 elimination during high-frequency jet-ventilation.** *Anaesthesiology* 1985, **63**:473–482.
9. Herridge MS, Slutsky AS, Colditz GA: **Has high-frequency ventilation been inappropriately discarded in adult acute respiratory distress syndrome?** *Crit Care Med* 1998, **26**:2073–2077.
10. Kahn RC: **Humidification of the airways: adequate for function and integrity?** *Chest* 1983, **5**:510–514.
11. Chalon J: **Low humidity and damage to tracheal mucosa.** *Bull NY Acad Med* 1980, **56**:314–319.
12 Chalon J, Loew D, Malebranche J: **Effects of dry anesthetic gases on tracheobronchial ciliated epithelium.** *Anesthesiology* 1972, **37**: 338–343.
13. Shelly MP, Lloyd GM, Park GR: **A review of mechanisms and methods of humidification of inspired gases.** *Int Care Med* 1988, **14**:118–123.
14. Klain M, Smith RB: **High frequency percutaneous transtracheal jet ventilation.** *Crit Care Med* 1977, **5**:280–287.
15. Roustan JP: **High frequency jet ventilation combined with conventional mechanical ventilation in the treatment of adult respiratory distress syndrome.** *Ann Fr Anesth Reanim* 1995, **14**:276–288.
16. Aloy A, Schachner M, Cancura W: **Tubeless translaryngeal superimposed jet ventilation.** *Eur Arch Otorhinolaryngol* 1991, **248**:475–478.
17. Gallagher TJ, Boysen PG, Davidson DD, Miller JR, Leven SB: **High frequency percussive ventilation compared with conventional mechanical ventilation.** *Crit Care Med* 1989, **17**:364–366.
18. Doyle H, Napolitano A, Lippmann R: **Different humidification systems for high frequency jet ventilation.** *Crit Care Med* 1984, **12**:815–819.
19. Chatburn RL, McClellan LD: **A heat and humidification system for high frequency jet ventilation.** *Repir Care* 1982, **27**:1386–1391.
20. Burton JDK. **Effects of dry anaesthetic gases on the respiratory mucus membrane.** *Lancet* 1962, **11**:235–240.
21. Murray JF, Matthay MA, Luce JM, Flick MR: **An expanded definition of the adult respiratory distress syndrome.** *Am Rev Respir Dis* 1988, **138**:720–723.
22. Ihra G, Kepka A, Lanzenberger E, Schabernig C, Zimpfer M, Aloy A: **SHFJV via a newly developed jet adaptor in the ICU [in German].** *Anaesthesist* 1998, **47**:209–219.
23. Shelly MP, Lloyd GM, Park GR, *et al:* **A review of mechanisms and methods of humidification of inspired gases.** *Int Care Med* 1988, **14**:1–9.
24. Hirsch JA, Tokayer JL, Robison MJ, *et al:* **Effects of dry air and subsequent humidification on tracheal mucous velocity in dogs.** *J Appl Physiol* 1975, **39**:242–249.
25. Mammel MC, Ophoven JP, Lewallen PK, *et al:* **High frequency ventilation and tracheal injuries.** *Pediatrics* 1986, **77**:608–612.
26. Wiswell TE, Clark RM, Null DM, *et al:* **Tracheal and bronchial injury in high-frequency oscillatory ventilation and high-frequency flow-interruption compared with positive pressure ventilation.** *J Pediatr* 1988, **112**:249–253.
27. Bail EM, Dahlby RW, Wiggs BR, *et al:* **Effect of cold and warm dry air hyperventilation on canine airway blood flow.** *J Appl Physiol* 1987, **62**:526–531.
28. Bail EM, Godden DJ, Pare PD: **Mechanism for increase in tracheobronchial blood flow induced by hyperventilation of dry air in dogs.** *J Appl Physiol* 1990, **68**:105–111.
29. Man SFP, Adams GK, Proctor DF, *et al:* **Effects of temperature, relative humidity and mode of breathing in canine airway sections.** *J Appl Physiol* 1979, **46**:205–211.
30. Phillips RJ, Richardson PS: **The effect of autonomic nerve system and related drugs on tracheal mucous secretion.** *J Physiol (Lond)* 1975, **247**:270–274.

31. Strugess J, Reid L: **An organ culture study of the effect of drugs on the secretory activity of the human bronchial submucosal gland.** *Clin Sci* 1972, **43**:533–537.
32. Tarnow-Mordi WO, Reid E, Griffiths PR, *et al:* **Low inspired gas temperature and respiratory complications in very low birthweight infants.** *J Pediatr* 1989, **114**:438–442.
33. Tarnow-Mordi WO, Sutton P, Wilkinson AR: **Inadequate humidification of respiratory gases during mechanical ventilation of the newborn.** *Arch Dis Child* 1986, **61**:698–704.
34. Kirpalani H, Higa T, Perlman M, *et al:* **Diagnosis and therapy of necrotizing tracheobronchitis in ventilated neonates.** *Crit Care Med* 1985, **13**:792–798.
35. Metlay LA, MacPherson TA, Doshi N, *et al:* **A new iatrogenous lesion in newborns requiring assisted ventilation.** *N Engl J Med* 1983, **309**:111–116.
36. Buchdahl RM, Reiser J, Ingram D, *et al:* **Ciliary abnormalities in respiratory disease.** *Arch Dis Child* 1988, **63**:238–244.
37. Ophoven JP, Mammel MC, Gordon MJ, *et al:* **Tracheobronchial histopathology associated with high-frequency jet-ventilation.** *Crit Care Med* 1984, **12**:829–832.
38. Boros SJ, Mammel MC, Coleman JM, *et al:* **Neonatal high-frequency jet-ventilation: four year's experience.** *Pediatrics* 1985, **75**:657–663.
39. Keszler M, Donn SM, Bucciarelli RL, *et al*: **Multicenter controlled trial comparing high-frequency jet-ventilation and conventional mechanical ventilation in newborn infants with pulmonary interstitial emphysema.** *J Pediatr* 1991, **119**:85–93.
40. Hanson JB, Waldstein G, Hernandez JA, *et al*: **Necrotizing tracheobronchitis: an ischemic lesion.** *Am J Dis Child* 1998, **142**:1094–1098.
41. Cavanagh KA, Hill HF, Wojciechowsky WV, Parker JC: **Regional tracheal blood flow during conventional and high-frequency jet-ventilation in suckling pigs.** *Crit Care Med* 1996, **24**:280–286.
42. Naglie RA, Donn SM, Nicks JJ, *et al:* **Tracheobronchial and pulmonary histopathology following conventional ventilation and high-frequency jet-ventilation.** *J Perinatol* 1986, **10**:46–51.
43. Baile EM, Guillemi S, Pare PD: **Tracheobronchial and upper airway blood flow in dogs during thermally induced panting.** *J Appl Physiol* 1987, **63**:2240–2246.