EDITORIALS
Central Oxygen Delivery Systems: A Disaster Waiting to Happen?

ORIGINAL CONTRIBUTIONS
The Hospital Oxygen Supply: An "O2K" Problem

Effects of Humidifier Dead Space in Paralyzed and Spontaneously Breathing Patients

In Vitro Comparison of Circulaire and AeroTee to Traditional Nebulizer T-Piece with Corrugated Tubing

Dose and Particle Size Distributions: PEP Device plus MDI with Reservoir Using CFC vs HFA Albuterol

REVIEWS, OVERVIEWS, & UPDATES
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INDICATION AND USAGE
CUROSURF is indicated for the treatment (rescue) of Respiratory Distress Syndrome (RDS) in premature infants. CUROSURF reduces mortality and pneumothoraces associated with RDS.

CLINICAL STUDIES
The clinical efficacy of CUROSURF was demonstrated in one single-dose study (Study 1) and one multiple-dose study (Study 2) in the treatment of established neonatal RDS involving approximately 500 infants. Each study was randomized, multicenter, and controlled. REFER TO PACKAGE INSERT FOR STUDY DESCRIPTION RESULTS.

ACUTE CLINICAL EFFECTS
As with other surfactants, marked improvements in oxygenation may occur within minutes of the administration of CUROSURF.

WARNINGS
CUROSURF is intended for intratracheal use only.

THE ADMINISTRATION OF EXOGENOUS SURFACTANTS, INCLUDING CUROSURF, CAN RAPIDLY AFFECT OXYGENATION AND LUNG COMPLIANCE. Therefore, infants receiving CUROSURF should receive frequent clinical and laboratory assessments so that oxygen and ventilatory support can be modified to respond to respiratory changes. CUROSURF should only be administered by those trained and experienced in the care, resuscitation, and stabilization of pre-term infants.

TRANSIENT ADVERSE EFFECTS SEEN WITH THE ADMINISTRATION OF CUROSURF INCLUDE BRADYCARDIA, HYPOTENSION, ENDOTRACHEAL TUBE BLOCKAGE, AND OXYGEN DESATURATION. These events require stopping CUROSURF administration and taking appropriate measures to alleviate the condition. After the patient is stable, dosing may proceed with appropriate monitoring.

PRECAUTIONS
General
Correction of acidosis, hypotension, anemia, hypoglycemia, and hypothermia is recommended prior to CUROSURF administration. Surfactant administration can be expected to reduce the severity of RDS but will not eliminate the mortality and morbidity associated with other complications of prematurity.

Sufficient information is not available on the effects of administering initial doses of CUROSURF other than 2.5 mL/kg (200 mg/kg), subsequent doses other than 1.25 mL/kg (100 mg/kg), administration of more than three total doses, dosing more frequently than every 12 hours, or initiating therapy with CUROSURF more than 15 hours after diagnosing RDS. Adequate data are not available on the use of CUROSURF in conjunction with conventional therapies of RDS, e.g., high-frequency ventilation.

CARCINOGENESIS, MUTAGENESIS, IMPAIRMENT OF FERTILITY
Studies to assess potential carcinogenic and reproductive effects of CUROSURF, or other surfactants, have not been conducted.

Mutagenicity studies of CUROSURF, which included the Ames test, gene mutation assay in Chinese hamster V79 cells, chromosomal aberration assay in Chinese hamster ovary cells, unscheduled DNA synthesis in HELA S3 cells, and in vivo mouse nuclear test, were negative.

ADVERSE REACTIONS
Transient adverse effects seen with the administration of CUROSURF include bradycardia, hypotension, endotracheal tube blockage, and oxygen desaturation.

The rates of common complications of prematurity observed in Study 1 are shown below in Table 3.

<table>
<thead>
<tr>
<th>TABLE 3</th>
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<tbody>
<tr>
<td>COMPLICATIONS OF PREMATURITY</td>
</tr>
<tr>
<td>CUROSURF 2.5 mL/kg</td>
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<tr>
<td>-------------------</td>
</tr>
<tr>
<td>(200 mg/kg)</td>
</tr>
<tr>
<td>n=76</td>
</tr>
<tr>
<td>Acquired Pneumonia</td>
</tr>
<tr>
<td>Acquired Septicemia</td>
</tr>
<tr>
<td>Bronchopulmonary Dystasis</td>
</tr>
<tr>
<td>Intracranial Hemorrhage</td>
</tr>
<tr>
<td>Patent Ductus Arteriosus</td>
</tr>
<tr>
<td>Pneumothorax</td>
</tr>
<tr>
<td>Pulmonary Interstitial Emphysema</td>
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</tbody>
</table>

*Control patients were disconnected from the ventilator and manually ventilated for 2 minutes. No surfactant was instilled.

Immunological studies have not demonstrated differences in levels of surfactant-anti-surfactant immune complexes and anti-CUROSURF antibodies between patients treated with CUROSURF and patients who received control treatment.

OVERDOSAGE
There have been no reports of overdosage following the administration of CUROSURF.

In the event of accidental overdosage, and only if there are clear clinical effects on the infant’s respiration, ventilation, or oxygenation, as much of the suspension as possible should be aspirated and the infant should be managed with supportive treatment, with particular attention to fluid and electrolyte balance.

Dosing Precautions
Transient episodes of bradycardia, decreased oxygen saturation, reflux of the surfactant into the endotracheal tube, and airway obstruction have occurred during the dosing procedure of CUROSURF. These events require interrupting the administration of CUROSURF and taking the appropriate measures to alleviate the condition. After stabilization, dosing may resume with appropriate monitoring.

HOW SUPPLIED
CUROSURF® (poractant alfa) Intratracheal Suspension (NDC Numbers: 49502-180-01 [1.5 mL]; 49502-180-03 [3 mL]) is available sterile, ready-to-use rubber-stoppered clear glass vials containing 1.5 mL (120 mg phospholipids) or 3 mL (240 mg phospholipids) of suspension. One vial per carton.

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EDITORIALS

Central Oxygen Delivery Systems: A Disaster Waiting to Happen?
by Robert M Kacmarek—Boston, Massachusetts

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The Effects of Passive Humidifier Dead Space on Respiratory Variables in Paralyzed and Spontaneously Breathing Patients
by Robert S Campbell, Kenneth Davis Jr, Joy A Johannigman, and Richard D Branson—Cincinnati, Ohio

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by S David Piper—West Sacramento, California

Combining a Positive Expiratory Pressure Device with a Metered-Dose Inhaler Reservoir System Using Chlorofluorocarbon Albuterol and Hydrofluoroalkane Albuterol: Effect on Dose and Particle Size Distributions
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PFT NUGGETS

Should This Patient Use Supplemental Oxygen During Commercial Air Flight?
by James K Stoller—Cleveland, Ohio

A 72-Year-Old Smoker with Interstitial Lung Disease
by Omar A Minai and Eugene J Sullivan—Cleveland, Ohio

BOOKS, FILMS, TAPES, & SOFTWARE

Pediatric Respiratory Medicine (Taussig LM, Landau L) reviewed by Mark Heulitt and Shirley J Holt—Little Rock, Arkansas

Geriatric Respiratory Care (Sorenson HM, Thorson JA) reviewed by Pat Munzer—Topeka, Kansas

Manual of Pleural Procedures (Colt HG, Mathur PN) reviewed by Bernard J Roth—Tacoma, Washington
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Electrocardiography: The Monitoring and Diagnostic Leads, 2nd edition (Wiederhold R)
reviewed by Thomas A Barnes—Boston, Massachusetts

Cardiac Intensive Care (Brown DL, ed)
reviewed by Mark T Gladwin—Bethesda, Maryland

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Editorials, Commentaries, and Reviews to Note


Improved Outcome of ARDS Patients: Are We Really Performing Better?—Stelzer H, Kraft P. Intensive Care Med 1999 Sep;25(9):887-889.


The active cycle of breathing techniques (ACBT) in gravity-assisted drainage positions is an effective airway clearance regimen for individuals who produce excess bronchial secretions. This study compared the ACBT in positions with and without a head-down tilt. Nineteen subjects (11 men), mean age 37.1 years (range 18-76 years), with bronchiectasis who produced more than 20 g of sputum per day and had a mean forced expiratory volume in 1 s (FEV₁) of 56.9% predicted (range 23-90% pred.) were studied. There was no significant difference in the wet weight of sputum expectorated when using the ACBT in gravity-assisted drainage positions with or without a head-down tilt. Mean (SD) score for perception of breathlessness, measured on a visual analogue scale, increased significantly following treatment with a head-down tilt [2.3 (1.6) to 3.3 (2.0) cm, p = 0.02]. There was no significant difference in oxygenation or lung function (FEV₁). Eighteen subjects preferred the ACBT without a head-down tilt. The ACBT in the horizontal position is a simple airway clearance regimen suitable for individuals who produce greater than 20 g of sputum per day. Subjects were less breathless and preferred the ACBT in the horizontal position, thus providing a treatment alternative that may improve adherence in individuals who are required to carry out daily airway clearance treatments.


Cystic fibrosis (CF) is a complex illness characterized by chronic lung infection leading to deterioration in function and respiratory failure in over 85% of patients. An understanding of the risk factors for that progression and the interaction of these factors with current therapeutic strategies should materially improve the prevention of this progressive lung disease. The Epidemiologic Study of Cystic Fibrosis (ESCF) was therefore designed as a multicenter, longitudinally observational study to prospectively collect detailed clinical, therapeutic, microbiologic, and lung function data from a large number of CF treatment sites in the U.S. and Canada. The ESCF also serves an important role as a phase-IV study of dornase alfa. To be eligible for enrollment, subjects must have the diagnosis of CF and receive the majority of their care at an ESCF.
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2. Beth E, et al. Comparative respiratory deposition of 99mTc-labeled particles of albuterol using a metered dose inhaler, a metered dose inhaler with AeroChamber® spacer and OptiChamber® spacer in healthy human volunteers using gamma-spectroscopy, 1997 (Data on file, Respironics, Inc).
3. Data on file, Respironics, Inc.

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site. In this paper, the authors present the ESCF study design in detail. Further, enrollment data collected at 194 study sites in 18,411 subjects enrolled from December 1, 1993 to December 31, 1995 are presented in summary form. This comprehensive study is unique in the detail of clinical data collected regarding patient monitoring and therapeutic practices in CF care. Two companion articles present data regarding practice patterns in cystic fibrosis care, including data on resource utilization and prescribing practices.


This report describes the prescribing pattern of therapeutic interventions in the management of patients with cystic fibrosis (CF), as observed in the Epidemiologic Study of Cystic Fibrosis (ESCF). Use of 20 therapies by 12,622 patients was recorded from each health care encounter (53,024 outpatient visits and 8,561 hospitalizations) during a 1-year period (1995), and analyzed by gender, age, severity of lung disease, and presence of any Pseudomonas species in the respiratory tract. The percentage of patients using the following pulmonary therapies was observed (in descending order): airway clearance techniques (88.2%); inhaled bronchodilators (82.2%); oral antibiotics (excluding quinolones) (68.2%); dornase alfa (52.9%); intravenous antibiotics (34.4%); oral quinolones (34.4%); inhaled antibiotics (34.3%); mast cell stabilizers (29.5%); inhaled corticosteroids (25.9%); oral corticosteroids (17.1%); oral bronchodilators (16.2%); oxygen (8.1%); inhaled mucolytic agent acetylcysteine (6.5%); and diuretics (1.4%). The percentage of patients using nutritional therapies was: pancreatic enzymes (96%); oral nutritional supplements (31.1%); enteral nutrition (7.3%); and parenteral nutrition (0.7%). The percentage of patients using other therapies was: nonsteroidal anti-inflammatory drugs (7.9%); and insulin or oral hypoglycemic agents (6.1%). The general trend was for therapies to be used more by older patients, those with lower pulmonary function, and by those with Pseudomonas in their respiratory tract. Exceptions to this trend occurred for airway clearance, oral antibiotics, mast cell stabilizers, and pancreatic enzymes. Four therapies (oral nutritional supplements, parenteral nutrition, diuretics, and pancreatic enzymes) were used more by males than females. However, there was no gender difference for this group of therapies on pulmonary or nutritional status.


Chest physiotherapy (CPT) is recommended for the clearance of bronchial secretions in the management of patients with cystic fibrosis (CF). The Flutter® valve (Scandipharm, Birmingham, AL) has been introduced as an alternative method to CPT for airway mucus clearance. The objective of this study was to compare the short-term effects of CPT and the Flutter® valve on pulmonary function and exercise tolerance in patients with cystic fibrosis. Twenty-three patients, 5 to 21 years of age, were randomized to receive one of two interventions: CPT or the Flutter® valve, upon admission to the hospital for a 2-week treatment of pulmonary exacerbation. Pulmonary function testing (PFTs) and the 6-min walk test were performed on admission, day 7, and day 14 of hospitalization. Data analysis indicated no significant differences between the two groups on admission. Both groups showed improvement in pulmonary function test results, but the Flutter® group had a higher mean forced vital capacity (FVC) and forced expiratory volume in 1 sec (FEV₁) compared to the CPT group after 1 week of intervention. Both groups continued to improve during the 2-week intervention, with no significant difference in FVC or FEV₁ between groups by the end of 2 weeks. Mean forced expiratory flow rate between 25-75% of vital capacity (FEF₂₅-₇₅) was 19.3 ± 9.8 L/s vs. 18.1 ± 10.4 L/s (p = 0.05) for CPT and Flutter® respectively.


In this review, we outline the role of nitric oxide in airway inflammation in children with asthma. We also discuss the various methods reported for measuring exhaled nitric oxide and provide some insight as to the pros and cons and pitfalls of these techniques. Guidelines for measurements of exhaled nitric oxide based on our experience are provided, as well as suggestions for the use of this technique as a new "airway inflammation test."


This article reviews published evidence which addresses the relevance of cultural factors in the delivery of health services for asthma patients. In addition, it suggests a framework within which further research could be carried out to advance our knowledge on this topic.


BACKGROUND: For decades it has been assumed that postprimary tuberculosis is usually caused by reactivation of endogenous infection rather than by a new, exogenous infection. METHODS: We performed DNA fingerprinting with restriction-fragment-length polymorphism analysis on pairs of isolates of Mycobacterium tuberculosis from 16 compliant patients who had a relapse of pulmonary tuberculosis after curative treatment of postprimary tuberculosis. The patients lived in areas of South Africa where tuberculosis is endemic. Medical records were reviewed for clinical data. RESULTS: For 12 of the 16 patients, the restriction-fragment-length polymorphism banding patterns for the isolates obtained after the relapse were different from those for the isolates from the initial tuberculosis disease. This finding indicates that reinfection was the cause of the recurrence of tuberculosis after curative treatment. Two patients had reinfections with a multidrug-resistant strain. All 15 patients who were tested for the human immunodeficiency virus were seronegative. CONCLUSIONS: Exogenous reinfection appears to be a major cause of postprimary tuberculosis after a previous cure in an area with a high incidence of this disease. This finding emphasizes the importance of achieving cures and of preventing anyone with infectious tuberculosis from exposing others to the disease.


BACKGROUND: Although sleep apnea is common, it often goes undiagnosed in primary care encounters. OBJECTIVE: To test the Berlin Questionnaire as a means of identifying patients with sleep apnea. DESIGN: Survey followed by portable, unattended sleep studies in a subset of patients. SETTING: Five primary care sites in Cleveland, Ohio. PATIENTS: 744 adults (of 1008 surveyed [74%]), of whom 100 underwent sleep studies. MEASUREMENTS: Sur-
vey items addressed the presence and frequency of snoring behavior, waketime sleepiness or fatigue, and history of obesity or hypertension. Patients with persistent and frequent symptoms in any two of these three domains were considered to be at high risk for sleep apnea. Portable sleep monitoring was conducted to measure the number of respiratory events per hour in bed (respiratory disturbance index [RDI]). RESULTS: Questions about symptoms demonstrated internal consistency (Cronbach correlations, 0.86 to 0.92). Of the 744 respondents, 279 (37.5%) were in this high-risk group that was defined a priori. For the 100 patients who underwent sleep studies, risk grouping was useful in prediction of the RDI. For example, being in the high-risk group predicted an RDI greater than 5 with a sensitivity of 0.86, a specificity of 0.77, a positive predictive value of 0.89, and a likelihood ratio of 3.79. CONCLUSION: The Berlin Questionnaire provides a means of identifying patients who are likely to have sleep apnea.


The alleviation of suffering is crucial in all of medicine, especially in the care of the dying. Suffering cannot be treated unless it is recognized and diagnosed. Suffering involves some symptom or process that threatens the patient because of fear, the meaning of the symptom, and concerns about the future. The meanings and the fear are personal and individual, so that even if two patients have the same symptoms, their suffering would be different. The complex techniques and methods that physicians usually use to make a diagnosis, however, are aimed at the body rather than the person. The diagnosis of suffering is therefore often missed, even in severe illness and even when it staves physicians in the face. A high index of suspicion must be maintained in the presence of serious disease, and patients must be directly questioned. Concerns over the discomfort of listening to patients’ severe distress are usually more than offset by the gratification that follows the intervention. Often, questioning and attentive listening, which take little time, are in themselves ameliorative. The information on which the assessment of suffering is based is subjective; this may pose difficulties for physicians, who tend to value objective findings more highly and see a conflict between the two kinds of information. Recent advances in understanding how physicians increase the utility of information and make inferences allow one to reliably use the subjective information on which the diagnosis and treatment of suffering depend. Knowing patients as individual persons well enough to understand the origin of their suffering and ultimately its best treatment requires methods of empathic attentiveness and nondis-cursive thinking that can be learned and taught. The skill of suffering depends on physicians acquiring these skills.


BACKGROUND: Risk factors of delayed extubation, prolonged intensive care unit (ICU) length of stay (LOS), and mortality have not been studied for patients administered fast-track cardiac anesthesia (FTCA). The authors' goals were to determine risk factors of outcomes and cardiac risk scores (CRS) for CABG patients undergoing FTCA. METHODS: Consecutive CABG patients undergoing FTCA were prospectively studied. Outcome variables were delayed extubation > 10 h, prolonged ICU LOS > 48 h, and mortality. Univariate analyses were performed followed by multiple logistic regression to derive risk factors of the three outcomes. Simplified integer-based CRS were derived from logistic models. Bootstrap validation was performed to assess and compare the predictive abilities of CRS and logistic models for the three outcomes. RESULTS: The authors studied 885 patients. Twenty-five percent had delayed extubation, 17% had prolonged ICU LOS, and 2.6% died. Risk factors of delayed extubation were increased age, female gender, postoperative use of intraaortic balloon pump, inotropes, bleeding, and atrial arrhythmia. Risk factors of prolonged ICU LOS were those of delayed extubation plus preoperative myocardial infarction and postoperative renal insufficiency. Risk factors of mortality were female gender, emergency surgery, and poor left ventricular function. CRSs were modeled for the three outcomes. The area under the receiver operating characteristic curve for the CRS-logic models was not significantly different: 0.707/0.702 for delayed extubation, 0.851/0.855 for prolonged ICU LOS, and 0.657/0.699 for mortality. CONCLUSION: In CABG patients undergoing FTCA, the authors derived and validated risk factors of delayed extubation, prolonged ICU LOS, and mortality. Furthermore, they developed a simplified CRS system with similar predictive abilities as the logistic models.

Unintended Inhalation of Nitric Oxide by Contamination of Compressed Air: Physiologic Effects and Interference with Intended Nitric Oxide Inhalation in Acute Lung Inj-
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BACKGROUND: Compressed air from a hospital's central gas supply may contain nitric oxide as a result of air pollution. Inhaled nitric oxide may increase arterial oxygen tension and decrease pulmonary vascular resistance in patients with acute lung injury and acute respiratory distress syndrome. Therefore, the authors wanted to determine whether unintentional nitric oxide inhalation by contamination of compressed air influences arterial oxygen tension and pulmonary vascular resistance and interferes with the therapeutic use of nitric oxide.

METHODS: Nitric oxide concentrations in the compressed air of a university hospital were measured continuously by chemiluminescence during two periods (4 and 2 weeks). The effects of unintentional nitric oxide inhalation on arterial oxygen tension (n = 15) and on pulmonary vascular resistance (n = 9) were measured in patients with acute lung injury and acute respiratory distress syndrome by changing the source of compressed air of the ventilator from the hospital's central gas supply to a nitric oxide-free gas tank containing compressed air. In five of these patients, the effects of an additional inhalation of 5 ppm nitric oxide were evaluated.

RESULTS: During working days, compressed air of the hospital's central gas supply contained clinically effective nitric oxide concentrations (> 80 parts per billion) during 40% of the time. Change to gas tank-supplied nitric oxide-free compressed air decreased the arterial oxygen tension by 10% and increased pulmonary vascular resistance by 13%. The addition of 5 ppm nitric oxide had a minimal effect on arterial oxygen tension and pulmonary vascular resistance when added to hospital-supplied compressed air but improved both when added to tank-supplied compressed air. CONCLUSIONS: Unintentional inhalation of nitric oxide increases arterial oxygen tension and decreases pulmonary vascular resistance in patients with acute lung injury and acute respiratory distress syndrome. The unintentional nitric oxide inhalation interferes with the therapeutic use of nitric oxide.


Near-infrared spectroscopy has been used to monitor cerebral oxygen saturation during cerebral circulatory arrest and carotid clamping. However, its utility has not been demonstrated in more complex situations, such as in patients with head injuries. The authors tested this method during conditions that may alter the arteriovenous partition of cerebral blood in different ways. METHODS: The authors compared changes in measured cerebral oxygen saturation and other hemodynamic parameters, including jugular venous oxygen saturation, in nine patients with severe closed head injury during manipulation of arterial carbon dioxide partial pressure and after mean arterial pressure was altered by vasopressors.

RESULTS: The Bland and Altman representation of cerebral oxygen saturation versus jugular oxygen saturation showed a uniform scatter. Values for changing arterial carbon dioxide partial pressure were: bias = 1.11%, 2 SD = ±21%, absolute value; and those for alterations in mean arterial pressure: bias = 3.77%, 2 SD = ±24%, absolute value. However, a Bland and Altman plot of changes in cerebral oxygen saturation versus changes in jugular oxygen saturation had a negative slope (alteration in arterial carbon dioxide partial pressure: bias = 2.44%, 2 SD = ±17%, absolute value; alteration in mean arterial pressure: bias = -4.91%, 2 SD = ±31%, absolute value). Regression analysis showed that changes in cerebral oxygen saturation were positively correlated with changes in jugular venous oxygen saturation during the carbon dioxide challenge, whereas correlation was negative during the arterial pressure challenge. CONCLUSIONS: Cerebral oxygen saturation assessed by near-infrared spectroscopy does not adequately reflect changes in jugular venous oxygen saturation in patients with severe head injury. Changes in arteriovenous partitioning, infrared-spectroscopy contamination by extracerebral signal, algorithm errors, and dissimilar tissue sampling may explain these findings.


Health-care workers are half as likely to enter the rooms of patients in contact isolation, but are more likely to wash their hands after caring for them than after caring for patients not in isolation.


OBJECTIVE: Prognostication is central to developing treatment plans and relaying information to patients, family members, and other health care providers. The degree of confidence or certainty that a health care provider has in his or her mortality risk assessment is also important, because a provider may deliver care differently depending on their assuredness in the assessment. We assessed the performance of nurse and physician mortality risk estimates with and without weighting the estimates with their respective degrees of certainty. METH-
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ABSTRACTS


The mechanics of the chest wall was studied in seven asthmatic patients before and during histamine-induced bronchoconstriction (B). The volume of the chest wall (VC) was calculated by three-dimensional tracking of 89 chest wall markers. Plural (Plp) and gastric (Pgp) pressures were simultaneously recorded. VC was modeled as the sum of the volumes of the pulmonary-apposed rib cage (VRCp), diaphragm-apposed rib cage (VRCa), and abdomen (VAB). During B, hyperinflation was due to the increase in end-expiratory volume of the rib cage (0.63 ± 0.09 L, p < 0.01), whereas change in VAB was inconsistent (0.09 ± 0.07 L, NS) because of phasic recruitment of abdominal muscles during expiration. Changes in end-expiratory VRC and VRCa were along the rib cage relaxation configuration, indicating that both compartments shared proportionally the hyperinflation. VRCp-ppl plot during B was displaced leftward of the relaxation curve, suggesting persistent activity of rib cage inspiratory muscles throughout expiration. Changes in end-expiratory VCW during B did not relate to changes in FEV1 or time and volume components of the breathing cycle. We concluded that during B in asthmatic patients: (1) rib cage accounts largely for the volume of hyperinflation, whereas abdominal muscle recruitment during expiration limits the increase in VAB; (2) hyperinflation is influenced by sustained postinspiratory activity of the inspiratory muscles; (3) this pattern of respiratory muscle recruitment seems to minimize volume distortion of the rib cage at end-expiration and to preserve diaphragm length despite hyperinflation.


Asthma complicates up to 4% of pregnancies. Our objective was to compare emergency department (ED) visits for acute asthma among pregnant versus nonpregnant women. A prospective cohort study, as part of the Multicenter Asthma Research Collaboration. ED patients who presented with acute asthma underwent a structured interview in the ED and another by telephone 2 wk later. The study was performed at 36 EDs in 18 states. A total of 51 pregnant women and 500 nonpregnant women, aged 18 to 39, were available for analysis. Pregnant women did not differ from nonpregnant women by duration of asthma symptoms (median: 0.75 versus 0.75 d, p = 0.57) or


BACKGROUND: The functional residual capacity (FRC), the only lung volume to be routinely measured in infants, is an unreliable volume landmark. In addition to FRC, the residual volume (RV) was measured by nitrogen washout using rapid thoracoabdominal compression (RTC) in nine infants with cystic fibrosis aged 5-31 months. METHODS: A commercial system for nitrogen washout to measure lung volumes and a custom made system to perform RTC were used. Lung volume was raised to an airway opening pressure of 30 cm H2O (Vn). RTC was performed from Vn. The jet pressure (Pj) 65 to 96 cm H2O which generated the highest forced expiratory volume (range 40.2 mL/kg; 95% confidence interval CI 33.03 to 47.73) was used during the RV manoeuvre. The infants were manually hyperventilated to inhibit the respiratory drive briefly. RTC was initiated during the last passive expiration. RV was estimated by measuring the volume of nitrogen expired after end forced expiratory switching of the inspired gas from room air to 100% oxygen while jacket inflation was maintained at the time of switching into oxygen during the post-expiratory pause. RESULTS: In each infant RV and FRC measurements were reproducible and did not overlap; the difference between mean values, which is the expiratory reserve volume, was statistically significant (p < 0.05). Mean RV was 21.3% (95% CI 18.7 to 24.0), FRC was 25.5% (95% CI 22.8 to 28.1), and TLC (total lung capacity at Vn) was 61.5% (95% CI 54.4 to 68.7) mL/kg. These values were dependent on body length, weight and age. When measuring RV the period between switching to oxygen and the end of the Pj plateau was 0.301 (95% CI 0.211 to 0.391) s. The washout duration was longer for RV than for FRC measurement (80.9 s (95% CI 71.3 to 90.4) versus 72.4 s (95% CI 64.9 to 79.8)) (p < 0.001). CONCLUSIONS: A new non-invasive and reliable technique for routine measurement of RV in infants is presented.


BACKGROUND: Persisting controversy surrounds the use of transthoracic needle aspiration biopsy (TNAB) stemming from its uncertain diagnostic accuracy. A systematic review and meta-analysis was therefore conducted to evaluate the accuracy of TNAB for the diagnosis of solitary or multiple localised pulmonary lesions. METHODS: Searches for English literature papers in Index Medicus (1963-1965) and Medline (1966-1996) were performed and the bibliographies of the retrieved articles were systematically reviewed. Articles evaluating the accuracy of TNAB in series of consecutive patients presenting with solitary or multiple pulmonary lesions were considered. Only papers in which > 90% of patients were given a final diagnosis according to an appropriate reference standard were included in the meta-analysis. RESULTS: A total of 48 studies were included and five meta-analyses were conducted according to four diagnostic thresholds. From the pooled sensitivity and specificity corresponding to each diagnostic threshold, associated likelihood ratios (LRs) were derived for malignant disease as follows: (1) malignant versus all other categories, LR = 72; (2) malignant or suspicious versus all others, LR = 49; (3) suspicious versus all categories but malignant, LR = 15; (4) benign versus all others, LR = 0.07; and (5) specific benign diagnosis versus all others, LR = 0.005. Differences in methodological quality of the studies, needle types, or whether a cytopathologist participated in the procedure failed to explain the heterogeneity of the results found in almost every meta-analysis. Given a 50% probability of malignancy prior to the TNAB, post-test probabilities of malignancy upon receiving the results would be malignant, 99%; suspicious, 94%; non-specific benign, 7%; and benign with a specific diagnosis, 0.6%. CONCLUSIONS: Given the intermediate pre-test probabilities that would probably lead to performing TNAB, findings of "malignant" or of a specific diagnosis of a benign condition provide definitive results. Findings of "suspicious" markedly increase the probability of malignancy, and "benign" markedly decreases it but may not be considered definitive.
initial peak expiratory flow rate (PEFR) (51% versus 53% of predicted, p = 0.52). Despite this similarity, only 44% of pregnant women were treated with corticosteroids in the ED compared with 66% of nonpregnant women (p = 0.002). Pregnant women were equally likely to be admitted (24% versus 21%, p = 0.61) but less likely to be prescribed corticosteroids if sent home (38% versus 64%, p = 0.002). At 2-wk follow-up, pregnant women were 2.9 times more likely to report an ongoing exacerbation (95% CI, 1.2 to 6.8). Among women presenting to the ED with acute asthma, pregnant asthmatics are less likely to receive appropriate treatment with corticosteroids.


Oxygen consumption dedicated to respiratory work (VO₂RESP) during quiet breathing is small in normal patients. In the morbidly obese, at high minute ventilations, VO₂RESP is greater than in normal patients, but VO₂RESP during quiet breathing in these patients is not known. We postulated that such patients have increased VO₂RESP at rest which may predispose them to respiratory failure when additional respiratory workloads are imposed. We measured baseline VO₂ in morbidly obese patients immediately prior to gastric bypass surgery and again after intubation, mechanical ventilation, and paralysis, and compared their change in VO₂ to nonobese patients scheduled for elective abdominal surgery. Baseline VO₂ was higher in the obese patients compared with control patients (354.6 versus 221.4 mL/min; p = 0.0001) and the change in VO₂ from spontaneous breathing to mechanical ventilation was significant in the obese patients (354.6 versus 297.2 mL/min; p = 0.0002) but not the control patients (221.4 versus 219.8 mL/min; p = 0.86). We conclude that morbidly obese patients dedicate a disproportionately high percentage of total VO₂ to conduct respiratory work, even during quiet breathing. This relative inefficiency suggests a decreased ventilatory reserve and a predisposition to respiratory failure in the setting of even mild pulmonary or systemic insults.


We undertook a prospective, double-blind, placebo-controlled trial to resolve the question of the clinical effectiveness of ribavirin in previously well infants who require ventilation for respiratory distress secondary to respiratory syncytial virus (RSV) bronchiolitis. Aerosol riba-
virin or NaCl 0.9% was administered within 24 h of initiation of ventilation, 18 h/d, for a maximum of 7 d or until extubation. From March 1994 to March 1997, 42 children were randomized and 41 patients were retained for analysis. Baseline characteristics of each group-ribavirin and placebo (20:21) were not significantly different with respect to age (62.5 ± 35.9 years vs. 62.7 ± 30.9 d), sex, weight, and length of ventilation pre-aerosol. "Intent to treat" outcome analysis found no significant differences in the length of the following: ventilation (102.16 ± 65.26 vs. 126.28 ± 78.72 h; p = 0.29), aerosol therapy, stay in the intensive care unit, total oxygen therapy, and hospitalization. The aerosols were well tolerated and no deaths occurred. This trial demonstrates the lack of effectiveness of aerosolized ribavirin in reducing the length of ventilation and course of illness in infants with no underlying illness ventilated for respiratory distress secondary to RSV bronchiolitis.


Bronchoscopy is considered the most important diagnostic test for broncholithiasis. However, its role in the treatment of broncholithiasis in a large group of patients has not been studied. To evaluate the therapeutic role of bronchoscopy, we retrospectively reviewed the clinical data of patients with broncholithiasis who also underwent bronchoscopy at Mayo Clinic. Bronchoscopy revealed 127 broncholiths (free or partly eroded calcified material in the airway lumen) in 95 patients (49 men and 46 women) evaluated between 1954 and 1994. Bronchoscopic removal of 71 (56%) broncholiths was attempted in 48 patients (50.5%) during 61 bronchoscopy sessions. Forty-eight of the broncholiths selected for removal were partly eroding into the tracheobronchial lumen and 23 were free. Forty-eight percent (23 of 48) of the partly eroding broncholiths were successfully removed bronchoscopically, with a greater percentage removed with the rigid bronchoscope (67%) than with the flexible bronchoscope (30%). All free broncholiths were completely extracted regardless of the type of bronchoscope used. Complications occurred in only two patients (4% of the bronchoscopic broncholithectomy group), both with partially eroded broncholiths, and consisted of hemorrhage in one patient requiring thoracotomy and acute dyspnea in another patient, caused by a loose broncholith lodged in the trachea. We conclude that flexible and/or rigid bronchoscopic extraction of partly eroded or free broncholiths in the tracheobronchial tree can be considered safe and effective.


The methods of nonbronchoscopic lung lavage used for collection of samples of epithelial lining fluid (ELF) in intubated patients are poorly standardized and incompletely validated. In infants with lung disease requiring ventilatory support, we evaluated two techniques of small volume saline lavage for the collection of a specimen suitable for pulmonary surfactant analysis. We aimed to compare apparent origin of the return fluid obtained by each method, equivalence and agreement of the estimates of measured pulmonary surfactant concentration, and the relative strength of association between surfactant indices and lung dysfunction. Fifty-three contemporaneous paired samples of lung lavage fluid suitable for surfactant analysis were collected from 31 infants using tracheal aspirate (TA, 4 ± 0.5 mL saline), and then nonbronchoscopic bronchoalveolar lavage (NB-BAL, 3 ± 1 mL/kg). Return fluid from TA had higher mean ELF concentration of total protein and IgA secretory component (SC), and a lower surfactant protein A (SP-A) concentration than NB-BAL, indicating that the TA lavage was sampling ELF more proximally in the tracheobronchial tree (protein: TA 7.7 versus NB-BAL 4.7 mg/mL; SC: 21 versus 1.8 microgram/mL; SP-A: 9 versus 19 microgram/mL; all p < 0.01). Mean concentration of surfactant indices in ELF differed only for SP-A, but for all indices, paired values showed poor agreement on Bland-Altman analysis, highlighting the potential imprecision associated with small volume lung lavage. TA return fluid yielded estimates of surfactant indices which were at least equivalent to NB-BAL in prediction of the severity of lung dysfunction. We conclude that NB-BAL return fluid has more distal origin, but analysis of TA fluid may have equal validity in the estimation of indices of pulmonary surfactant. The results of individual estimates of ELF constituents in a single sample of lavage fluid should be interpreted with caution, even when standardized sampling techniques are employed.


Limited information is available regarding the physiological response to different types of exercise training in patients with severe chronic obstructive pulmonary disease (COPD). The aim of this study was twofold: firstly, to investigate the physiological response to training at 60% of achieved peak load in patients with severe COPD; and secondly to study the effects of interval (I) versus continuous (C) training in these patients. Twenty-one patients with COPD (mean ± SD forced expiratory volume in one second: 37 ± 15% of predicted, normoxemic at rest) were evaluated at baseline and after 8 weeks' training. Patients were randomly allocated to either I or C training. The training was performed on a cycle ergometer, 5 days a week, 30 min daily. The total workload was the same for both training programmes. C training resulted in a significant increase in oxygen consumption (VO_{2}, 17%; p<0.05) and a decrease in minute ventilation (V_{E})/VO_{2} (p<0.01) and V_{E}/carbon dioxide production (VCO_{2}) (p<0.05) at peak exercise capacity, while no changes in these measures were observed after interval training. During submaximal exercise a significant decrease was observed in lactic acid production, being most pronounced in the C-trained group (-31%, p<0.01 versus -20%, p<0.05). Only in the I-trained group did a significant increase in peak work load (17%, p<0.05) and a decrease in leg pain (p<0.05) occur. Training did not result in a significant improvement in lung function, but maximal inspiratory mouth pressure increased in both groups by 10% (C: p<0.05) and 23% (I: p<0.01). The present study shows a different physiological response pattern to interval or continuous training in chronic obstruction pulmonary disease, which might be a reflection of specific training effects in either oxidative or glycolytic muscle metabolic pathways. Further work is required to determine the role of the different exercise programmes and the particular category of patients for whom this might be beneficial.


Paralysis with pancuronium bromide is used in newborn infants to facilitate ventilatory support during respiratory failure. Changes in lung mechanics have been attributed to paralysis. The aim of this study was to examine whether or not paralysis per se has an influence on the passive respiratory mechanics, resistance (Rrs) and compliance (Crs) of the respiratory system in newborn infants. In 30 infants with acute respiratory failure, Rrs was measured during paralysis with pancuronium bromide and after stopping pancuronium bromide (group A). Rrs was also measured in an additional 10 ventilated infants in a reversed fashion (group B): Rrs was measured first in nonparalysed infants and then they were paralysed, mainly for diagnostic procedures, and the Rrs measurement repeated. As Rrs is highly dependent on lung volume, several parameters, that depend directly on lung volume were recorded: inspiratory oxygen fraction (F_{I,O_2}), arterial oxygen tension/alveolar oxygen tension (a/A) ratio and volume above functional residual capacity (FRC). In group A, the Rrs was not different during (0.236±0.09 cm H_{2}O x s x mL^{-1}) and after (0.237±0.07 cm H_{2}O x s x mL^{-1}).
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Eight Hot Topics

Program #1
Pulmonary Rehabilitation: What You Need to Know
Live Videoconference - March 7, 11:30 a.m.-1:00 p.m. Central Time
Teleconference with Videotape - April 4, 11:30 a.m.-12:00 Noon Central Time
Presenters: Julien M. Roy, BA, RRT, FAACVPR, and Richard D. Bronson, BA, RRT
What constitutes a sound pulmonary rehabilitation program? How do you go about setting up a rehab program? What's the role of assessment? How can you develop an exercise prescription for the rehab of your patients? What are the issues surrounding reimbursement and what does the future hold? Learn the answers to these questions and gain an appreciation for the importance of pulmonary rehabilitation to your facility and your patients.

Program #2
Pediatric Asthma in the ER
Live Videoconference - March 28, 11:30 a.m.-1:00 p.m. Central Time
Teleconference with Videotape - April 18, 11:30 a.m.-12:00 Noon Central Time
Presenters: Timothy R. Meyers, BS, RRT and Thomas J. Kallstrom, RRT, FAARC
The prevalence of pediatric asthma has increased dramatically in the last few years. The National Asthma Education and Prevention Program has provided guidelines for management of pediatric asthma. This program will discuss these issues as well as the role of care paths in the management of the disease. Additionally, there have been some significant advances in coping with pediatric asthma in the ER.

Program #3
Drugs, Medications, and Delivery Devices of Importance in Respiratory Care
Live Videoconference - April 25, 11:30 a.m.-1:00 p.m. Central Time
Teleconference with Videotape - May 16, 11:30 a.m.-12:00 Noon Central Time
Presenters: James B. Fink, MS, RRT, FAARC and David J. Pierson, MD, FAARC
Aerosol therapy is delivered to nearly 80% of respiratory patients. There are a number of new medications in development for both local and systemic administration to those patients. Which device to use, how to negotiate care plans, and how to educate both patients and caregivers are all topics that will be discussed. Perhaps of critical importance is getting the most medication delivered to the patient's lungs, which leads to a discussion of selecting the correct delivery device.

Program #4
Cost-Effective Respiratory Care: You've Got to Change
Live Videoconference - May 23, 11:30 a.m.-1:00 p.m. Central Time
Teleconference with Videotape - June 20, 11:30 a.m.-12:00 Noon Central Time
Presenters: Kevin L. Shroake, MA, RRT, FACHS, FAAMA, FAARC and Sam P. Giordano, MBA, RRT, FAARC
Practitioners frequently confuse the implementation of protocol treatment and care management. Both programs, if successfully implemented, can lead to cost savings. The problem most practitioners face is how to identify where costs are avoided and resources are conserved. Perhaps most critical is ensuring that the correct care is delivered at the proper time. The health care practitioner is key to the ultimate success of these programs.

Program #5
Pediatric Ventilation: Kids Are Different
Live Videoconference - July 25, 11:30 a.m.-1:00 p.m. Central Time
Teleconference with Videotape - August 15, 11:30 a.m.-12:00 Noon Central Time
Presenters: Mark Heath narratives, MD, FAAP, FCCP and Richard D. Bronson, BA, RRT
There are significant differences in the anatomy and physiology of the respiratory systems between adults and children, posing problems for the practitioner attempting to mechanically ventilate a pediatric patient. Once the process is underway, the capabilities of the available mechanical ventilators and how they affect children pose additional problems. Children are so different, you need to stop and reassess actions you would normally take with an adult patient.

Program #6
What Matters in Respiratory Monitoring: What Goes and What Stays
Live Videoconference - August 22, 11:30 a.m.-1:00 p.m. Central Time
Teleconference with Videotape - September 26, 11:30 a.m.-12:00 Noon Central Time
Presenters: Dean R. Hess, PhD, RRT, FAARC and Richard D. Bronson, BA, RRT
The health care provider has an array of monitoring devices available in managing a patient. With all that technology available, which device is appropriate? What about those displays on ventilators? The availability of graphics during mechanical ventilation can provide a wealth of information. When is it essential? Under what circumstances should you pay close attention to those displays in the assessment of your patient?

Program #7
Managing Asthma: An Update
Live Videoconference - September 19, 11:30 a.m.-1:00 p.m. Central Time
Teleconference with Videotape - October 17, 11:30 a.m.-12:00 Noon Central Time
Presenters: Patti Jorgner, RRT, CCM and Mari Jones, MSN, RN, FNP, RRT
Asthma management is a hot topic for discussion. Everyone wants to implement a program at his or her facility. What will make a program work, and how do you know if it's successful? This program will provide you with the information you have been looking for in order to implement a program and determine how successful the program really is. You will be given guidance on how to analyze outcomes measures from a successful program.

Program #8
Routine Pulmonary Function Testing: Doing It Right
Live Videoconference - November 7, 11:30 a.m.-1:00 p.m. Central Time
Teleconference with Videotape - December 5, 11:30 a.m.-12:00 Noon Central Time
Presenters: Carl D. Mattam, BA, RRT, RPFT and David J. Pierson, MD, FAARC
Pulmonary function testing at the bedside is being increasingly utilized as a diagnostic tool. Is it always appropriate? How can you assure competency of the person conducting the test? How can you assure quality assurance outside the pulmonary function laboratory? This program will provide you with the information you need to assure that this diagnostic test is properly conducted outside the laboratory.
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Teleconference with Videotape Requirements
Sites must have a video monitor, a VCR, a telephone with speaker phone, and an individual to proctor the program. Participants will receive and view a 90-minute videotape and then call a toll-free number for a live 30-minute call-in question-and-answer session. Program materials for the telephone session include the toll-free telephone number, continuing education packet, attendance log, videotape and reproducible course materials, post-test with answers, evaluation, and certificate of attendance.

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H$_2$O $\times s \times mL^{-1}$) paralysis. Also, in group B, Rrs did not change (0.207±0.046 versus 0.221±0.046 cm $\times s \times mL^{-1}$ without versus with pancuronium bromide). $F_{Rrs}$ a/V ratio and volume above FRC remained constant during paralysis. These data demonstrate that paralysis does not influence the resistance of the total respiratory system in ventilated term and preterm infants when measured at comparable lung volumes.


Mucociliary clearance (MCC), the process in which airway mucus together with substances trapped within are moved out of the lungs, is an important defense mechanism of the human body. Drugs may alter this process, such that it is necessary to know the effect of the drugs on MCC. Indeed, agents stimulating MCC may be used therapeutically in respiratory medicine, especially in patients suspected of having an impairment of their mucociliary transport system. In contrast, caution should be taken with drugs depressing MCC as an undesired side-effect, independently of their therapeutic indication. Since cough clearance (CC) serves as a back-up system when MCC fails, the influence of drugs must be examined not only on MCC but also on CC. Ultimately, the clinical repercussions of alterations in mucus transport induced by drug administration must be studied. Tertiary ammonium compounds (anticholinergics), aspinir, anesthetic agents and benzodiazepines have been shown to be capable of depressing the mucociliary transport system. Cholinergics, methylxanthines, sodium cromoglycate, hypertonic saline, saline as well as water aerosol have been shown to increase MCC. Adrenergic antagonists, guaifensin, S-carboxymethylcysteine, sodium 2-mercapto-ethane sulphonate and frusemid have been reported not to alter the mucociliary transport significantly. Amiloride, aminidrin 5'-triphosphate (UTP), quaternary ammonium compounds (anticholinergics), adrenergic agonists, corticosteroids, recombinant human deoxyribonuclease (rhDNase), N-acetylcysteine, bromhexine and ambroxol have been reported either not to change or to augment MCC. Indirect data suggest that surfactant as well as antibiotics may improve the mucociliary transport system. As for the influence of drugs on CC, amiloride and rhDNase have been demonstrated to increase the effectiveness of cough. A trend towards an improved CC was noted after treatment with adrenergic agonists. The anticholinergic agent ipratropium bromide, which is a quaternary ammonium compound, has been suggested to decrease CC significantly. Bromhexine, ambroxol and neutral saline seemed not to alter CC, either positively or negatively. Finally, treatment with either amiloride, recombinant human deoxyribonuclease, bromhexine, ambroxol, N-acetylcysteine, S-carboxymethylcysteine or hypertonic saline has been suggested as a possible cause of clinical improvement in patients, such as the experience of dyspnea, the case of expectoration or the frequency of infective exacerbations. Other agents did not show a clinical benefit.


CONTEXT: As the world's largest producer and consumer of tobacco products, China bears a large proportion of the global burden of smoking-related disease and may be experiencing a tobacco epidemic. OBJECTIVE: To develop an evidence-based approach supporting tobacco control initiatives in China. DESIGN AND SETTING: A population-based survey consisting of a 52-item questionnaire that included information on demographics, smoking history, smoking-related knowledge and attitudes, cessation, passive smoke exposure, and health status was administered in 145 disease surveillance points in the 30 provinces of China from March through July 1996. PARTICIPANTS: A nationally representative random sample of 128766 persons, aged 15 to 69 years were asked to participate; 120298 (93.8%) provided data and were included in the final analysis. About two thirds of those sampled were from rural areas and one third were from urban areas. MAIN OUTCOME MEASURES: Current smoking patterns and attitudes; changes in smoking patterns and attitudes compared with results of a previous national survey conducted in 1984. RESULTS: A total of 41187 respondents smoked at least 1 cigarette per day, accounting for 34.1% of the total number of respondents, an increase of 3.4 percentage points since 1984. Current smoking continues to be prevalent among more men (63%) than women (38%). Age at smoking initiation declined by about 3 years for both men and women (from 28 to 25 years). Only a minority of smokers recognized that lung cancer (36%) and heart disease (4%) can be caused by smoking. Of the nonsmokers, 53.5% were exposed to environmental tobacco smoke at least 15 minutes per day on more than 1 day per week. Respondents were generally supportive of tobacco control measures. CONCLUSION: The high rates of smoking in men found in this study signal an urgent need for smoking prevention and cessation efforts; tobacco control initiatives are needed to maintain or decrease the currently low smoking prevalence in women.


BACKGROUND: Inhaled nitric oxide improves oxygenation and lessens the need for extracorporeal-membrane oxygenation in full-term neonates with hypoxemic respiratory failure and persistent pulmonary hypertension, but potential adverse effects are intracranial haemorrhage and chronic lung disease. We investigated whether low-dose inhaled nitric oxide would improve survival in premature neonates with unresponsive severe hypoxemic respiratory failure, and would not increase the frequency or severity of intracranial haemorrhage or chronic lung disease. METHODS: We did a double-blind, randomised controlled trial in 12 perinatal centres that provide tertiary care. 80 premature neonates (gestational age ≤34 weeks) with severe hypoxemic respiratory failure were randomly assigned inhaled nitric oxide (n=48) or no nitric oxide (n=32, controls). Our primary outcome was survival to discharge. Analysis was by intention to treat. We studied also the rate and severity of intracranial haemorrhage, pulmonary haemorrhage, duration of ventilation, and chronic lung disease at 36 weeks' postconceptional age. FINDINGS: The two groups did not differ for baseline characteristics or severity of disease. Inhaled nitric oxide improved oxygenation after 60 min (p=0.03). Survival at discharge was 52% in the inhaled-nitric-oxide group and 47% in controls (p=0.65). Causes of death were mainly related to extreme prematurity and were similar in the two groups. The two groups did not differ for adverse events or outcomes (intracranial haemorrhage grade 2-4, 28% inhaled nitric oxide and 33% control; pulmonary haemorrhage 13% and 9%; chronic lung disease 60% and 80%). INTERPRETATION: Low-dose inhaled nitric oxide improved oxygenation but did not improve survival in severely hypoxemic premature neonates. Low-dose nitric oxide in the most critically ill premature neonates does not increase the risk of intracranial haemorrhage, and may decrease risk of chronic lung injury.


BACKGROUND: Inhaled nitric oxide improves oxygenation in severely hypoxemic term neonates, which lessens the need for extracorporeal-membrane oxygenation. Improvement in other relevant outcomes remains unknown, and safety of inhaled nitric oxide is uncertain in preterm neonates. We did a randomised controlled trial to assess use of inhaled nitric oxide in preterm and near-term neonates. METHODS: We randomly assigned 204 preterm (<33 weeks)
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weeks) and near-term (≥ 33 weeks) neonates with oxygenation indices from 12.5 to 30.0 and 15 to 40, respectively, 10 parts per million (ppm) inhaled nitric oxide (n=105) or control ventilation therapy without nitric oxide (n=99). The primary endpoint was the oxygenation index at 2 h. Analysis was done by intention to treat. FINDINGS: 12 neonates were excluded, leaving 97 (45 preterm) in the nitric-oxide group and 95 (40 preterm) in the control group. The difference in oxygenation index at 2 h was greater in the nitric-oxide group than in the control group (IQR 6.2 [median 8.4] vs -2.9 [12.4], p=0.005), but was significant only in near-term neonates (p=0.03). Survivors assigned nitric oxide spent fewer days on mechanical ventilation and in the neonatal intensive-care unit, but this was also significant only in near-term neonates (6 [3] vs 7 [3] days, p=0.05, and 9 [6] vs 12 [9] days, p=0.02, respectively). INTERPRETATION: Low-dose inhaled nitric oxide early in the course of neonatal respiratory failure improves oxygenation and shortens duration of mechanical ventilation and the length of stay in intensive care. Inhaled nitric oxide was not, however, significantly beneficial in preterm neonates.


BACKGROUND: Little is known about the risk factors, outcome, and impact of pneumonia and other lower respiratory tract infections (LRTIs) in residents of long-term care facilities. OBJECTIVE: To determine the risk factors and the effect of these infections on functional status and clinical course. METHODS: Active surveillance for these infections was conducted for 475 residents in 5 nursing homes from July 1, 1993, through June 30, 1996. Information regarding potential risk factors for these infections, functional status, transfers to hospital, and death was also obtained. RESULTS: Two hundred seventy-two episodes of pneumonia and other LRTIs occurred in 170 residents during 228,757 days of surveillance for an incidence of 1.2 episodes per 1000 resident-days. Multivariable analysis revealed that older age (odds ratio [OR], 1.7; 95% confidence interval [CI], 1.1-2.6 per 10-year interval; p = 0.01), male sex (OR, 1.9; 95% CI, 1.1-3.5; p = 0.03), swallowing difficulty (OR, 2.0; 95% CI, 1.2-3.3; p = 0.01), and the inability to take oral medications (OR, 8.3; 95% CI, 1.4-50.3; p = 0.02) were significant risk factors for pneumonia; receipt of influenza vaccine (OR, 0.4; 95% CI, 0.3-0.5; p = 0.01) was protective. Age (OR, 1.6 [95% CI, 1.0-2.5]) per 10-year interval; p = 0.05) and immobility (OR, 2.6; 95% CI, 1.8-3.8; p = 0.01) were significant risk factors for other LRTIs, and influenza vaccination was protective (OR, 0.3; 95% CI, 0.2-0.4; p = 0.01). Residents with pneumonia (OR, 0.7; 95% CI, 0.3-1.4; p = 0.31) or with other LRTIs (OR, 0.5; 95% CI, 0.2-1.1; p = 0.43) were no more likely to have a deterioration in functional status than individuals in whom infection did not develop. CONCLUSIONS: Swallowing difficulty and lack of influenza vaccination are important, modifiable risks for pneumonia and other LRTIs in elderly residents of long-term care facilities. Our findings challenge the commonly held belief that pneumonia leads to long-term decline in functional status in this population.


BACKGROUND: There are several nicotine replacement products on the market, and physicians are likely to be asked with increasing frequency about which of these products their patients should use. OBJECTIVE: To provide a basis for rational advice by comparing nicotine polacrilex (gum), a transdermal patch, nasal spray, and an inhaler. DESIGN: Randomized trial with assessments at the quit date and 1, 4, and 12 weeks later. SETTING: Hospital smokers' clinic. PATIENTS: Male and female community volunteers (N = 504) smoking 10 or more cigarettes per day and seeking help to stop smoking. INTERVENTIONS: Patients were given brief advice, and purchased their nicotine replacement treatment at approximately half the regular retail price. MAIN OUTCOME MEASURES: Nicotine replacement treatment use, ratings of withdrawal symptoms, ratings of product characteristics and helpfulness, and biochemically validated continuous lapse-free abstinence. RESULTS: The products did not differ in their effects on withdrawal discomforts to smoke, or rates of abstinence. The continuous validated 12-week abstinence rates were 20%, 21%, 24%, and 24% in the gum, patch, spray, and inhaler groups, respectively. Compliance with recommended nicotine replacement treatment use was high for the patch, low for gum, and very low for the spray and the inhaler. The spray was underused because of adverse effects more often than the other products. In the subjects using the spray, the level of use among abstainers at week 1 predicted outcome at week 12. The inhaler was rated as more embarrassing to use than the other products, but provided at least as much nicotine as the gum. CONCLUSION: When asked about nicotine replacement treatment products available, physicians should note that, despite low compliance with the recommended dose of the spray and inhaler and differences in product ratings, overall, there are no notable differences between the products in their effects on withdrawal discomfort, perceived helpfulness, or general efficacy.


Stable chronic hypercapnic patients are often prescribed long-term mask noninvasive pressure support ventilation (NPSV). There is a lack of information on the effects of posture on NPSV. Therefore posture induced changes in physiological effects of NPSV in awake stable chronic hypercapnic patients were evaluated. In 12 awake chronic obstructive pulmonary disease (COPD) patients breathing pattern, respiratory muscles, mechanics and dyspnoea (by visual analogue scale: VAS) were evaluated during spontaneous breathing (SB) in sitting posture and during NPSV in sitting, supine and lateral positions randomly assigned. Arterial blood gases were evaluated during SB and at the end of the last NPSV session (whatever the posture). As expected NPSV resulted in a significant improvement in carbon dioxide tension in arterial blood (P_{CO2}) (from 7.4±0.85 to 6.9±0.7 KPa). When compared with SB, sitting NPSV resulted in a significant increase in tidal volume and minute ventilation and in a significant decrease in breathing frequency. Inspiratory muscle effort as assessed by oesophageal pressure swings and pressure-time product per minute (from 14±4.8 to 6.2±3.5 cm H_{2}O, and from 240±81 to 96±60 cm H_{2}O x s x min^{-1} respectively), intrinsic dynamic positive end expiratory pressure (from 2.7±2.3 to 1.4±1.3 cm H_{2}O) and expiratory airway resistance (from 18±7 to 5±3 cm H_{2}O X L X s^{-1}) decreased during sitting NPSV, whereas VAS did not change. Changing posture did not significantly affect any parameter independently of the patients weight, whether obese or not. In awake stable hypercapnic chronic obstructive pulmonary disease patients changing posture does not significantly influence breathing pattern and respiratory muscles during noninvasive pressure support ventilation suggesting that mask ventilation may be performed in different positions without any relevant difference in its effectiveness.


Excessive inflammation seems important in chronic obstructive pulmonary disease (COPD), particularly during exacerbations of the disease. Exhaled nitric oxide concentration (NO_{ex}) is a sensitive marker of bronchial inflammation in asthma; it is unclear if this is also the case in...
COPD. This study: 1) quantifies NO<sub>exh</sub> in patients with COPD (during an exacerbation and while clinically stable); 2) investigates the response of NO<sub>exh</sub> to i.v. steroid therapy, and its potential relationship with other relevant physiological variables; and 3) assesses the relative contributions of the central and peripheral airways to NO<sub>exh</sub> by collecting exhaled air in two different bags connected in series. Seventeen COPD patients (forced expiratory volume in one second (FEV<sub>1</sub>) 37.6±3.4% of the predicted value ±SEM) hospitalized because of an exacerbation of the disease (arterial oxygen tension (P<sub>O2</sub>) 7.41±0.02 kPa 42.3±2.8 mm Hg), arterial carbon dioxide tension (P<sub>CO2</sub>) 5.63±0.37 kPa 42.3±2.8 mm Hg), pH 7.41±0.02 and 10 healthy subjects that served as controls were studied. On admission, NO<sub>exh</sub> in COPD was higher than normal (41.0±5.1 Versus 13.3±0.8 parts per billion (ppb), respectively, p<0.001). Despite i.v. steroid therapy, NO<sub>exh</sub> remained elevated throughout recovery (37.9±4.8 ppb, p<0.001) until discharge (40.9±4.3 ppb, p<0.001). In contrast, when the patients were clinically stable (several months later), NO<sub>exh</sub> was significantly reduced (15.8±3.8 ppb, p<0.001), and no longer different from control values. NO<sub>exh</sub> was not related to any of the physiological variables measured during recovery (pulmonary gas exchange) or at discharge (forced spirometry, lung volumes, diffusing capacity). Finally, the contribution of the central and peripheral airways to NO<sub>exh</sub> was not different at any point in time. These results indicate that during exacerbations of chronic obstructive pulmonary disease, the exhaled nitric oxide concentration: 1) is higher than normal; 2) is not reduced acutely by i.v. steroids but is normalized several months after discharge; 3) is unrelated to several physiological indices of disease severity; and 4) appears to be produced homogeneously in central and peripheral airways. Overall, these results are different from those reported in asthma, suggesting that different inflammatory mechanisms are operating in both diseases.


This study compared estimates of the severity and impact of asthma recorded using global questions of the type used in diary cards with health status measurements obtained using comprehensive questionnaires. Seventy-four outpatients with asthma, aged 17-76 yrs (mean 48 yrs) participated. Mean±SD forced expiratory volume in one second (FEV<sub>1</sub>) was 72±26% predicted. Patients recorded morning and evening peak expiratory flow rate (PEFR) and scaled their responses to the questions: "How bad is your asthma this morning/this evening?" (asthma severity) and "How much effect has your asthma had on your life today?" (asthma impact) for 2 weeks. They then completed Juniper's Asthma Quality of Life Questionnaire (AQLQ) and the St George's Respiratory Questionnaire (SGRQ). Diary card scores for asthma impact were less severe than for asthma severity (p<0.0001). Both correlated with AQLQ and SGRQ total scores (r=0.7; p<0.0001). Some patients responded 'none' for asthma severity (n=10) or asthma impact (n=13) on all 14 days of recording. For these patients, FEV<sub>1</sub> was <80% predicted, morning PEFR was <90% predicted and their AQLQ and SGRQ scores indicated significant health impairment. Diary card scores for asthma severity and impact were correlated with health status, but these global questions were insensitive in mild disease. Responses to these questions were influenced by their wording, so the number of symptom-free days calculated from diary cards will depend on the questions used. Standardization is required before symptom-free days can be used as a reliable measure of treatment efficacy.


It has been postulated that hypertonic saline (HS) might impair the antimicrobial effects of defensins within the airways. Alternative non-ionic osmotic agents such as mannitol may thus be preferable to HS in promoting bronchial mucus clearance (BMC) in patients with cystic fibrosis (CF). This study reports the effect of inhalation of another osmotic agent, dry powder Mannitol (300 mg), compared with its control (empty capsules plus matched voluntary cough) and a 6% solution of HS on BMC in 12 patients with cystic fibrosis (CF). Mucus clearance was measured using a radioaerosol/gamma camera technique. Post-intervention clearance was measured for 60 min, followed by cough clearance for 30 min. However during the post-intervention measurement there was a significant improvement in BMC for both the mannitol (8.7±3.3% versus 2.8±0.7%) and HS (10.0±2.3% versus 3.5±0.8%). There was also a significant improvement in cough clearance with the Mannitol (9.7±2.4%) compared with its control (2.5±0.8%). Despite premedication with a bronchodilator, a small fall in forced expiratory volume in one second (FEV<sub>1</sub>) was seen immediately after administration of both the mannitol (7.3±2.5%) and HS (5.8±1.2%). Values of FEV<sub>1</sub> returned to baseline by the end of the

PURPOSE: The clinical literature on the incidence and subsequent mortality of adult respiratory distress syndrome (ARDS) has come primarily from the experiences of large tertiary referral centers, particularly in Western Europe and North America. Consequently, very little has been published on the incidence, management, and outcome of ARDS in smaller community-based intensive care units. We aimed to delineate early clinical respiratory predictors of death in children with ARDS on the modest scale of a community hospital. MATERIALS AND METHODS: A retrospective chart review of children with ARDS needing conventional mechanical ventilation admitted to our pediatric intensive care unit from 1984 to 1997. The diagnosis of ARDS was based on acute onset of diffuse, bilateral pulmonary infiltrates of non-cardiac origin and severe hypoxemia defined by partial pressure of oxygen <200 mm Hg during positive end-expiratory pressure (PEEP) of 6 cm H2O or greater for a minimum of 24 hours. Demographic, clinical, and physiological data including PaO2/FiO2, A-aDO2, and ventilation index were retrieved. RESULTS: Fifty-six children with ARDS aged 8 ± 5.5 years (range, 50 days to 21 years) were identified. The mortality rate was 50%. Early predictors of death included the peak inspiratory pressure (PIP), ventilation index, and PEEP on the third day after diagnosis: Nonsurvivors had significantly higher PIP (35.3 ± 10.5 cm H2O vs 44.4 ± 10.7 cm H2O, p < 0.001), PEEP (8 ± 2.8 cm H2O vs 10.7.0 ± 3.5 cm H2O, p < 0.01), and ventilation index (49.14 ± 20.4 mm Hg x cm H2O/min vs 61 ± 51.1 mm Hg cm H2O/min) than survivors. In contrast, PaO2/FiO2 and A-aDO2 were capable of predicting outcome by day 5 and thereafter. CONCLUSIONS: A small-scale mortality outcome for ARDS is comparable to large tertiary referral institutions. The PIP, PEEP, and ventilation index are valuable for predicting outcome in ARDS by the third day of conventional therapy. The development of a local risk profile may assist in decision-making of early application of supportive therapies in this population.


The aim of this essay was to demonstrate the thoracic venous anatomy as delineated by malpositioned central venous catheters on plain chest radiographs. We therefore used the didactic advantage of clinically inadvertent catheter positions. This approach was chosen to illustrate venous anatomy with plain chest radiographs, and, thereby, to recognize malpositions promptly on the modality with which positions of central venous catheters is routinely performed.


Venricular assist devices (VAD) allow for long-term circulatory support of patients with end-stage heart failure. With the increasing duration of circulatory support, diagnostic imaging plays an important role in the management of patients on a VAD. The aim of our review was to analyze the radiologic features of different VADs. From 1987 to 1996, 319 patients (mean age 42 years, range 3 to 74 years) were treated with a VAD. A Berlin Heart VAD was implanted in 263 of the patients, the univentricular Baxter Novacor was implanted in three patients, and the univentricular CI Heartmate was implanted in 19 patients. All patients were studied by serial chest radiographs. In addition, 70 patients underwent computed tomography (CT), and five patients underwent electron beam CT. The Berlin Heart VAD was used as a biventricular support system in 218 patients. In all cases, the position of the wire-directed cannulae was identified on the chest radiographs, while the exact position of the cannula tip could be visualized by CT only. The plastic cannulae of both the Novacor and the Heartmate were not discernible on radiographs, but required CT for evaluation. Computed tomography also resolved the metal components of the pumps. The titanium-made pump housing of the Heartmate caused beam-hardening artefacts that might conceal fluid accumulations in the pump pocket. Computed tomography is the standard of reference for examinations of cannula position, pump position, and pump components of ventricular assist devices.
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closely predicted actual absorption times for experimental intravascular gas embolisms and was more accurate than a model based on spherical shape. We computed absorption times for cerebrovascular gas embolism assuming a range of bubble geometries, initial volumes, and parameters relevant to brain blood flow. Results of the simulations demonstrated absorption time maxima and minima based on initial geometry, with several configurations taking as much as 50% longer to be absorbed than would a comparable spherical bubble.


The response to inspiratory resistance loading (IRL) of the upper airway during sleep in children is not known. We, therefore, evaluated the arousal responses to IRL during sleep in children with the obstructive sleep apnea syndrome (OSAS) compared with controls. Children with OSAS aroused at a higher load than did controls (23 ± 8 vs. 15 ± 7 cm H2O, 1 s; p < 0.05). Patients with OSAS had higher arousal thresholds during rapid eye movement (REM) vs. non-REM sleep (p < 0.001), whereas normal subjects had lower arousal thresholds during REM (p < 0.005). Ventilatory responses to IRL were evaluated in the controls. There was a marked decrease in tidal volume both immediately (56 ± 17% of baseline at an IRL of 15 cm H2O, 1 s; p < 0.001) and after 3 min of IRL (67 ± 23%, p < 0.005). The duty cycle increased. We conclude that children with OSAS have impaired arousal responses to IRL. Despite compensatory changes in respiratory timing, normal children have a decrease in minute ventilation in response to IRL during sleep. However, arousal occurs before gas-exchange abnormalities.


During dynamic hyperinflation with induced bronchoconstriction, there is a reduction in lung elastic recoil at constant lung volume (R. Pellegrino, O. Wilson, G. Jenourri, and J. R. Rodarte, J. Appl. Physiol. 81: 964-975, 1996). In the present study, lung elastic recoil at control end inspiration was measured in normal subjects in a volume displacement plethysmograph before and after voluntary increases in mean lung volume, which were achieved by one tidal volume increase in functional residual capacity (FRC) with constant tidal volume and by doubling tidal volume with constant FRC. Lung elastic recoil at control end inspiration was significantly decreased by approximately 10% within four breaths of increasing FRC. When tidal volume was doubled, the decrease in computed lung recoil at control end inspiration was not significant. Because voluntary increases of lung volume should not produce airway closure, we conclude that stress relaxation was responsible for the decrease in lung recoil.


The original presentation of what we know as Boyle’s law has several interesting features. First, the technical difficulties of the experiment were considerable, because Boyle used a glass tube full of mercury that was nearly 2.5 m long, and the large pressures sometimes shattered the glass. Next, Boyle’s table of results contains extremely awkward fractions, such 10/13, 2/17, 13/19, and 18/23, which look very strange to us today. This was because he calculated the pressure for a certain volume of gas by using simple multiplication and division, keeping the vulgar fractions. Boyle was not able to express the numbers as decimals because this notation was not in common use at the time. Finally, his contention that pressure and volume were inversely related depended on the reader’s comparing two sets of numbers in adjacent columns to see how well they agreed. Today we would plot the data, but again orthogonal graphs were not in general use in 1662. When Boyle’s data are plotted by using modern conventional methods, they strongly support his hypothesis that the volume and pressure of a gas are inversely related.


Permissive hypercapnia (acceptance of raised concentrations of carbon dioxide in mechanically ventilated patients) may be associated with increased survival as a result of less ventilator-associated lung injury. Conversely, hypocapnia is associated with many acute illnesses (e.g., asthma, systemic inflammatory response syndrome, pulmonary edema), and is thought to reflect underlying hyperventilation. Accumulating clinical and basic scientific evidence points to an active role for carbon dioxide in organ injury, in which raised concentrations of carbon dioxide are protective, and low concentrations are injurious. We hypothesise that therapeutic hypercapnia might be tested in severely ill patients to see whether supplemental carbon dioxide could reduce the adverse effects of hypocapnia and promote the beneficial effects of hypercapnia. Such an approach could also expand our understanding of the pathogenesis of disorders in which hypocapnia is a constitutive element.


PURPOSE: Home care is increasingly being used as a substitute for hospital care. This study examined older patients’ perceptions of the home and of the hospital as treatment sites for acute illness and the patient characteristics that are associated with these perceptions. SUBJECTS AND METHODS: A series of questions derived from open-ended interviews supplemented by literature review were administered by telephone in a cross-sectional, descriptive study to community-dwelling persons age 65 years or older who had been hospitalized 2 months earlier with congestive heart failure, chronic obstructive pulmonary disease, or pneumonia. RESULTS: Among 246 participants, nearly equal proportions agreed with statements that the home and the hospital would be comfortable sites of care (54% versus 55%), that the home and the hospital would provide rapid recovery (41% versus 37%), and that home treatment and hospital treatment would be burdensome on family and friends (40% versus 33%). Although 93% would feel safe in the hospital, only 42% would feel safe at home. Perceptions were not associated with sociodemographic characteristics, primary diagnosis, self-rated health, depression, or social support. Functionally dependent patients had more positive perceptions of treatment at home. CONCLUSIONS: Evaluation of perceptions of home and hospital can facilitate assessing the acceptability of shifting acute care from hospital to home. Our findings suggest that successful expansion of acute home care will require flexibility in the use of home and hospital as well as education to change perceptions about the safety and efficacy of treatment at home.


PURPOSE: We sought to assess the yield of chest roentgenography for the detection of pneumothorax among hospitalized patients with pleural effusion who have undergone diagnostic or therapeutic thoracentesis. SUBJECTS AND METHODS: We performed a prospective study of 506 thoracentesis procedures in 370 patients. After the procedure, each operator filled out a note recording patient data and the characteristics of the thoracentesis. A chest radiograph was performed within 12 hours after the procedure in all patients. RESULTS: Eighteen (4%) pneumothoraces occurred in 17 patients, 9 (2%) of which required chest tube drainage. Of the 488 patients without symptoms, only 5 (1%)...
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developed a pneumothorax, only 1 of which required chest tube drainage. By contrast, of the 18 patients with symptoms, 13 developed a pneumothorax, 8 of which required chest tubes. There were two independent predictors of pneumothorax: presence of symptoms (odds ratio [OR] = 250; 95% confidence interval [CI]: 65 to 980) and male gender (OR = 5.4; 95% CI: 1.9 to 69). CONCLUSIONS: Among the symptom-free patients in our sample, the risk of developing pneumothorax with clinical consequences was so low that the practice of routine chest roentgenography may not be justified.


PURPOSE: Food asphyxiation is a common problem whenever and wherever people eat. A knowledge of predisposing factors might help to prevent this problem. SUBJECTS AND METHODS: We reviewed 34,476 consecutive autopsies done during a 14-year period (1984 to 1997) at the Institute of Forensic Medicine, Vienna. Demographic features and predisposing factors were determined for the 191 cases of fatal foreign body asphyxiation. RESULTS: Old age, poor dentition, and alcohol consumption were frequent findings. Other risk factors included chronic disease, sedation, and eating risky foods. On 120 (63%) of the 191 occasions, observers were present at the time of the incident and subsequently called the Emergency Service. In 110 (92%) cases, neither the observers nor the majority of the emergency medical technicians and physicians who would have been able to intervene recognized the definite diagnosis. Only 10 cases were correctly identified during cardiopulmonary resuscitation. CONCLUSIONS: These fatal accidents could be prevented easily. Effective prevention depends on understanding the nature and frequency of accidental deaths due to asphyxiation and the factors that lead to their occurrence and having a high degree of suspicion.


PURPOSE: To review the literature to determine whether inhaled ipratropium bromide provides additive benefits to adults with acute asthma who are being treated with beta-agonists in an emergency department. SUBJECTS AND METHODS: English-language studies, both published (1978 to 1999) and unpublished, were retrieved using MEDLINE, Science Citation Index, Current Contents, bibliographic reviews of primary research, review articles, consultation with experts, and the register of Medical Editors’ Trial Amnesty. Only randomized, double-blind, controlled trials that enrolled patients having an exacerbation of asthma were included. The main outcome measure was pulmonary function; hospital admission rate was also evaluated. RESULTS: Ten studies including 1,483 adults with acute asthma were selected (mean age 32 ± 13 years, 36% men). The overall effect size in SD units of pulmonary function showed a significant benefit from ipratropium (effect size 0.14, 95% confidence interval [CI]: 0.04 to 0.24, p = 0.008). Study-specific effect sizes ranged from 0.03 to 0.63. This pooled effect size was equivalent to a 10% (95% CI: 2% to 18%) increase in forced expiratory volume in 1 second (FEV1) or peak expiratory flow in the ipratropium group compared with the control group. Analysis of the four studies that included patients with extreme obstruction (FEV1 or peak flow <35% of predicted at presentation) showed substantial improvement with ipratropium therapy (effect size 0.38, 95% CI: 0.09 to 0.67). In the five trials (1,186 patients) that studied the effect of ipratropium administration on hospital admissions, pooled results revealed that ipratropium reduced admission rates significantly (odds ratio 0.62, 95% CI: 0.44 to 0.88, p = 0.007). CONCLUSIONS: The addition of ipratropium to beta-agonist therapy offers a statistically significant, albeit modest, improvement in pulmonary function, as well as a reduction in the rate of hospital admissions.


Study objectives: To investigate the prevalence of gastroesophageal reflux (GER) among patients with asthma and to determine the effect of omeprazole on the outcome of asthma in patients with GER. DESIGN: A double-blind, placebo-controlled crossover study. SETTING: Asthmatic patients who attended the pulmonary outpatient clinic of Turku University Central Hospital, Finland. PATIENTS: One hundred seven asthmatic patients. INTERVENTIONS: The patients who were found to have GER in ambulatory esophageal pH monitoring were randomized to receive either omeprazole, 40 mgqd, or placebo for 8 weeks. After a 2-week washout period, the patients were crossed over to the other treatment. Spirometry was performed at baseline and immediately after both treatment periods. Peak expiratory values, use of sympathomimetics, and pulmonary and gastric symptoms were recorded daily in a diary. RESULTS: Pathologic GER was found in 53% of the asthmatic patients. One third of these patients had no typical reflux symptoms. Daytime pulmonary symptoms did not improve sig-
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OBJECTIVE: Assess the frequency and outcome of inhospital resuscitation and determine the relationship between patient age and survival and whether it is affected by initial rhythm. DESIGN: Retrospective, single-institution, registry study of inhospital resuscitation. SETTING: A 550-bed, tertiary-care, teaching hospital in Macon, GA. PATIENTS: All admissions for which a resuscitation was attempted was the Medical Center of Central Georgia during the period of January 1, 1987 through December 31, 1993. The registry sample included 2,394 admissions, for which 2,813 resuscitation attempts were made; only the first resuscitation attempt during an admission was analyzed. INTERVENTIONS: None. MEASUREMENTS AND MAIN RESULTS: Rates of survival to discharge steadily increased from 24.4% in 1987 to 38.6% in 1993; the overall survival rate was 26.8%. Age, used as a continuous variable, was strongly related to survival (odds ratio = 0.984; p < 0.0001). Categorically, overall survival rates for pediatric, adult, and geriatric patients were 56.4%, 29.0%, and 24.0%, respectively. Survival rates also varied significantly (odds ratio = 0.469; p < 0.0001) among initial rhythms, i.e., supraventricular tachycardia (60.7%), ventricular tachycardia (57.6%), perfusing rhythms (49.8%), ventricular fibrillation (32.0%), pulseless electrical activity (14.6%), and asystole (9.1%). The relationship between age and survival did not change across the years included in the study, but did vary as a function of initial rhythm (p < 0.0001). Age was positively related to survival when the initial rhythm was supraventricular tachycardia (p = 0.04), negatively related to survival when the initial rhythm was perfusing (p < 0.0001) or pulseless electrical activity (p = 0.0002), and not related to survival when the initial rhythm was ventricular tachycardia (p = 0.98), ventricular fibrillation (p = 0.14), or asystole (p = 0.21). CONCLUSIONS: The relationship between patient age and a successful resuscitation attempt is not as simple as reported earlier. Whether age is related to increased or decreased survival, or is unrelated to survival, depends on the rhythm extent when resuscitation attempts begin. Survival rates were higher than most reported elsewhere and improved significantly over time. Multicentered studies are needed to determine whether these results are unique to the institution studied.


Study objectives: Health-related quality of life associated with intestinal lung disease has received little attention in clinical studies because there have been no validated methods for directly measuring it. We have assessed the validity of several generic and respiratory-specific quality-of-life instruments in patients with intestinal lung disease. DESIGN: Cross-sectional study. SETTING: Outpatient pulmonary clinic at a university referral center. PATIENTS: Fifty patients with intestinal disease such as idiopathic pulmonary fibrosis, sarcoidosis, hypersensitivity pneumonitis, and asbestosis. INTERVENTIONS: Patients were administered four quality-of-life questionnaires, the Medical Outcomes Study Short Form 36 (SF-36), the Quality of Well-being scale (QWB), the Chronic Respiratory Questionnaire (CRQ), and the St. George’s Respiratory Questionnaire (SGRQ). Patients concomitantly underwent pulmonary function testing and performed a 6-min walk. Measurements and results: Validation of these instruments was based on testing an a priori hypothesis that worse quality-of-life scores should correlate with more severe physiologic impairment demonstrated by pulmonary function tests, exercise tolerance on the 6-min walk, and dyspnea scores. Our patients, on average, had a moderate degree of physiologic impairment and demonstrated moderately decreased quality-of-life scores. Scores from all four quality-of-life instruments correlated significantly with 6-min walk distance and dyspnea score. Scores from the SF-36, QWB, and SGRQ showed significant correlation with FVC, FEV1, and diffusing capacity as well. The SF-36 and SGRQ consistently showed the strongest correlation with physical impairment. CONCLUSIONS: Our findings indicate that preexisting quality-of-life instruments can be applied to patients with intestinal lung disease and suggest that the SF-36 and the SGRQ, in particular, are sensitive tools for assessing quality of life in these patients. Future intervention studies of patients with intestinal lung disease should consider using these measures.


Study objectives: To evaluate lung function in patients cured from childhood acute lymphoblastic leukemia (ALL) with chemotherapy alone or plus bone marrow transplantation (BMT). Pulmonary toxicity is a well-recognized side effect of many ALL treatments. DESIGN: Cross-sectional study conducted at least 3 years after cessation of therapy. SETTING: Outpatient pulmonology department of the University Hospital. PATIENTS: Forty-four subjects (age range at observation, 6 to 23 years): 21 treated only with intensive Berlin-Frankfurt-Munster (BFM)-type chemotherapy for newly diagnosed ALL (group A), and 23 treated with chemotherapy plus BMT (group B). MEASUREMENTS: A detailed history of smoking habit, respiratory symptoms, and diseases was recorded directly from the patients with the aid of their parents. A complete physical examination and lung function testing (lung volumes and diffusion capacity for carbon monoxide [DLCO]) were performed in all subjects. RESULTS: No patient reported acute or chronic respiratory symptoms or diseases. In group A patients, lung function was in the normal range, except for three subjects in whom there was an isolated impairment of DLCO. In group B patients, lung function was markedly impaired, with more than half the patients having an abnormal DLCO. A statistically significant difference was found between the two groups for FVC (p = 0.022) and DLCO (p = 0.004). CONCLUSIONS: Intensive, BFM-type frontline chemotherapy is not associated with late pulmonary dysfunction; however, retreatment including BMT can frequently injure the lung. Thus, in patients who undergo BMT and whose life expectancy is long, careful monitoring of lung function and counseling about avoiding additional lung risk factors is recommended.


BACKGROUND: Older age is associated with less aggressive treatment and higher short-term mortality due to serious illness. It is not known whether less aggressive care contributes to this survival disadvantage in elderly persons. OBJECTIVE: To determine the effect of age on short-term survival, independent of baseline patient characteristics and aggressiveness of care. DESIGN: Secondary analysis of data from a prospective cohort study. SETTING: Five aca-
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OBJECTIVE: To determine whether a ventilatory strategy of permissive hypercapnia (PHC) reduces the duration of assisted ventilation in surfactant-treated neonates weighing 601 to 1250 g at birth. DESIGN: Forty-nine surfactant-treated preterm infants (birth weight: 854 ± 163 g; gestational age: 26 ± 1.4 weeks) receiving assisted ventilation were randomized during the first 24 hours of age to a PHC group (PETCO2: 45-55 mm Hg) or to a normocapnia group (NC; PETCO2: 35-45 mm Hg). The primary outcome measure was the total number of days on assisted ventilation. Uniform extubation and reintubation criteria were used for both groups. All patients received aminophylline before extubation. RESULTS: The total number of days on assisted ventilation expressed as median (25th-75th percentiles) was 2.5 (1.5-11.5) in the PHC group and 9.5 (2.0-22.5) in the NC group (Mann-Whitney U test). The number of patients on assisted ventilation throughout the first 96 hours after randomization was lower in the PHC group (log rank test). During that period, the ventilated patients in the PHC group had a higher PETCO2 and lower peak inspiratory pressure, mean airway pressure, and ventilator rate than did those in the NC group. The percentage of patients requiring reintubation within 24 hours postextubation (PHC 17% vs NC 28%) and supplemental oxygen at 28 days of life (PHC 43% vs NC 64%) and the total days of oxygen supplementation (PHC 15 [4-53] vs NC 32 [17-50]) did not differ between the groups. There were no differences in mortality, air leaks, intraventricular hemorrhage, periventricular leukomalacia, retinopathy of prematurity, or patent ductus arteriosus. CONCLUSION: A ventilatory strategy of PHC in preterm infants who receive assisted ventilation is feasible, seems safe, and may reduce the duration of assisted ventilation.


OBJECTIVE: This study was an assessment of potential exposures of medical personnel to nitrogen oxides during simulated and actual inhaled nitric oxide treatment of newborn and pediatric patients. DESIGN: Breathing zone exposures to nitric oxide (NO) and nitrogen dioxide (NO2) were monitored using data-logging personal dosimeters during simulated and actual administration of NO gas to patients in an intensive care setting. Sample. A total of 28 bedside nurses and 18 respiratory therapists were monitored during 6 different patient treatments. ANALYSIS: The highest measured concentrations of NO and NO2 in the personal breathing zones of the nurses and respiratory therapists were peak readings (<1 minute in duration) of 6.7 parts per million (ppm) NO and 3.1 ppm NO2. Exposures averaged throughout 15 minutes and throughout the work shift were below the limit of detection (0.8 ppm NO and 0.5 ppm NO2). CONCLUSION: Detectable exposures to NO and NO2 were brief, infrequent, and well below Occupational Safety and Health Administration permissible exposure limits or any other exposure guideline, eg, American Conference of Governmental Hygienists Threshold Limit Values.


OBJECTIVE: To investigate the association of collaboration between intensive care unit (ICU) physicians and nurses and patient outcome. DESIGN: Prospective, descriptive, correlational study using self-report instruments. SETTINGS: A community teaching hospital medical ICU, a university teaching hospital surgical ICU, and a community non-teaching hospital mixed ICU, all in upstate New York. SUBJECTS: Ninety-seven attending physicians, 63 resident physicians, and 162 staff nurses. PROCEDURE: When patients were ready for transfer from the ICU to an area of less intensive care, questionnaires were used to assess care providers' reports of collaboration in making the transfer decision. After controlling for severity of illness, the association between interprofessional collaboration and patient outcome was assessed. Unit-level organizational collaboration and patient outcomes were ranked. MEASURES: Healthcare providers' reported levels of collaboration, patient severity of illness and individual risk, patient outcomes of death or readmission to the ICU, unit-level collaboration, and unit patient risk of negative outcome. MAIN RESULTS: Medical ICU nurses' reports of collaboration were associated positively with patient outcomes. No other associations between individual reports of collaboration and patient outcome were found. There was a perfect rank order correlation between unit-level organizational collaboration and patient outcomes across the three units. CONCLUSIONS: The study offered some support for the importance of physician-nurse collaboration in ICU care delivery, a variable susceptible to intervention and further study.


OBJECTIVE: To determine with meta-analysis the effects of nursing-delivered smoking cessation interventions. RESULTS: Fifteen studies comparing nursing intervention with a control or usual care found intervention to significantly increase the odds of smoking cessation. There was heterogeneity among the study results, but pooling by using a random effects model did not alter the estimate of effect. There was no evidence from indirect comparison that interventions classified as intensive had a larger effect than less intensive ones. There was evidence that interventions were more effective for hospital inpatients with cardiovascular disease than for inpatients with other conditions. Interventions in nonhospitalized patients also showed evidence of efficacy. Nurse counseling on smoking cessation during a screening health check was likely to have less effect. The results indicate the potential benefits of smoking cessation advice and counseling given by nurses to their patients, with reasonable evidence that intervention can be effective.
Central Oxygen Delivery Systems: A Disaster Waiting to Happen?

I began writing this editorial as I sat in my office on Friday night, December 31st, 1999, awaiting the new millennium and all of its "potential" Y2K problems. As we now know, the fears of massive computer failures leading to disruption of hospital operations were grossly exaggerated and essentially did not occur. Many will look back on the massive effort to prepare for Y2K as a waste of time and money because no problems occurred. However, the preparations for the new millennium did help all of us to identify outdated systems and equipment, and most of us were able to update these systems and purchase new equipment as we prepared for the turn of the century, thus eliminating the potential for problems.

SEE THE ORIGINAL STUDY ON PAGE 300

As illustrated in the paper by Stoller et al1 in this issue of Respiratory Care, we may all need to place as much energy in evaluating and ensuring our readiness for potential oxygen delivery system disruptions as we did for Y2K issues. In this age of ongoing concerns regarding terrorism and the normal construction/accident related disruptions of primary support systems, it was interesting to note that of the 32 hospitals surveyed by Stoller et al1 only 9 had their primary and reserve oxygen delivery systems in separate locations, and in only 6 of these institutions did the primary and reserve system enter the hospital via different delivery pipes. Clearly, those institutions with both the primary and reserve systems located at the same site are potentially disasters waiting to happen. In these institutions, if a construction or motor vehicle accident, or a deliberate disruption of the single gas delivery pipeline were to occur, it is hard to imagine that the results would be anything but disastrous. In addition, Stoller et al1 report that only 63% of the institutions surveyed had an external connector where an oxygen delivery tanker truck could hook up to provide emergency oxygen to the institution's complete system if a disruption in oxygen delivery were to occur.

Although infrequent disruption of oxygen delivery has been a problem in many institutions, 16% of the hospitals surveyed by Stoller et al1 and 31% of respondents to an earlier survey2 reported disruption of bulk oxygen delivery systems, resulting in three reported deaths.2 In addition, many of the inadvertent oxygen delivery system disruptions that have occurred over the years have never been reported in the medical literature.

Guidelines for the setup of oxygen delivery systems specify that a primary and reserve system must be available, but they do not specify that they be at separate locations or that they be connected to the hospital by independent piping systems. As we move into the new millennium, it seems reasonable that we revisit these guidelines and regulations and update them based on the modern day concerns in our society.

Similar to the recommendations made by Anderson and Brock-Urne3 and Stoller et al,1 I recommend the following guidelines for central oxygen delivery systems be implemented:

1. That the primary and reserve systems be physically separated from each other and enter the institution through independent pipelines.
2. That all hospitals incorporate an external connection to the central oxygen piping system, into which an oxygen tanker truck could attach to provide emergency oxygen for the entire institution.
3. That the reserve system, whether it is gaseous tanks or liquid, be of sufficient size to be capable of supplying oxygen long enough to allow an oxygen tanker truck to arrive at the hospital.
4. That those institutions where the primary and reserve systems are located at the same site install a gaseous reserve at a separate location with sufficient volume to provide oxygen to all locations until an oxygen tanker truck can arrive.

With all of the fuss over the New Year and its potential problems behind us, it is now time to ensure that our oxygen delivery systems are appropriately upgraded to minimize both "potential" and real problems in the new millennium.

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Massachusetts General Hospital
Department of Anaesthesia
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Boston, Massachusetts

REFERENCES
The Hospital Oxygen Supply: An "O2K" Problem
James K Stoller MD, Mark Stefanak PE, Douglas Orens MBA RRT, and John Burkhart RRT

BACKGROUND: As an essential hospital facility, the central oxygen supply system should be designed with features allowing backup and/or redundancy in the event of system failure. As part of an organized institutional review of The Cleveland Clinic Foundation hospital inpatient central oxygen supply system, we undertook a survey of all hospitals in two Ohio cities to determine the characteristics of hospital central supply systems. METHODS: The questionnaire was developed and completed by structured telephone interview during calls placed to managers of facilities engineering departments in 35 hospitals in the greater Cleveland and Columbus, Ohio, metropolitan areas. To encourage candid responses to the telephone interview, respondents were assured that institutional names would not be presented in published reports. The questionnaire addressed the type of primary and reserve oxygen sources in the hospital, whether a backup system exists, and if so, in what configuration. The questionnaire also addressed whether any unplanned interruption or other problem (such as contamination of the piped-in oxygen supply) had ever occurred in the facility. RESULTS: Of the 35 eligible hospitals, responses were available from 32 (91.4%). The mean number of beds in the hospitals responding was 397 ± 251 (standard deviation), and the original construction dates of the responding hospitals ranged from 1887 to 1982. All 32 responding institutions reported a reserve system, described as a liquid reservoir in 72% (23/32), manifolds in 16% (5/32), and both in 13% (4/32). Twenty-six (81%) of those responding reported having the reserve system liquid or manifolded gas cylinders at the same location as the primary liquid vessel. The supply lines of these contiguous primary and reserve containers were reported to join proximal to entering the hospital structure, so at each of these 26 hospitals the primary and reserve systems depend on a single length of pipe. Only 4 (13%) of the hospitals have manifolded cylinders in addition to the primary and reserve liquid supplies. These manifolds are in different locations from the primary and reserve, have physically separate feed lines, and represent the only true examples of redundant piped-in oxygen supplies recorded during the survey. Of the 32 hospitals surveyed, 5 (16%) reported having experienced mishaps with the bulk liquid supply. CONCLUSIONS: (1) Not surprisingly, most of the hospitals in these two urban areas use bulk liquid oxygen systems (with primary and reserve liquid reservoirs) as the main central supply source, with some providing manifolded cylinders as backup. (2) Mishaps regarding the main supply line from the bulk oxygen reservoir were reported by 16% (5/32) of responding institutions. (3) In this context, the fact that most main and reserve tanks were contiguous and fed through a single line to the hospital facility suggests ongoing risk for interruption of an oxygen supply by line mishaps (e.g., street repair). (4) Contingency planning to lessen the risk of an interrupted supply should involve back-up systems with physically separated feed lines, as well as tanks of manifolded cylinders along the course of the main hospital oxygen circuit line. [Respir Care 2000;45(3):300–305] Key words: oxygen, central oxygen supply, oxygen reserve system.
THE HOSPITAL OXYGEN SUPPLY: AN “O2K” PROBLEM

Background

Providing supplemental oxygen to hospital inpatients requires a carefully engineered system, whether by liquid oxygen fed from a common source tank or by multiple pressurized cylinders localized throughout the hospital. Although supplemental oxygen provides a critical life-support need for many hospitalized inpatients (eg, those in the operating room or intensive care unit), relatively little attention has been given to issues of backup systems or disaster strategies in the event of failure of the main supply system. Indeed, the possibility of such a system failure, given the critical need for a reliable and redundant hospital inpatient oxygen supply, likens supplying inpatient oxygen to the “Y2K” problem of preparing computers for the new millennium. In particular, the similarities are:

1. Like “Y2K” issues, problems with the hospital oxygen supply may be invisible until they occur.
2. As with computer problems, by the time the oxygen supply is interrupted, the consequences may be severe.
3. Because any reaction at the time the problem occurs could be inadequate to avert serious consequences, an expectant approach is recommended.

See the Related Editorial on Page 299

As part of a recent organized institutional review of The Cleveland Clinic Foundation inpatient hospital oxygen supply, we conducted a survey of all hospitals in the greater metropolitan Cleveland and Columbus, Ohio, areas to ascertain the status of hospital central oxygen supply systems. Specifically, for all responding hospitals, we surveyed hospitals’ facilities engineering personnel by a structured telephone survey to learn whether (1) any adverse conditions had ever arisen regarding interruption or malfunction of the hospital oxygen central supply system, (2) whether a backup system or plan was available, and, if so, (3) what backup measures exist. The results of this survey suggest that mishaps regarding the central supply line have been surprisingly common. In the context that the consequences of failure of the inpatient hospital supply system could be grave, our survey suggests that significant attention and planning is warranted to assure the security and back-up of central hospital oxygen supplies. Likening this problem to that of preparing computers for the new millennium, we might dub this “the O2K problem.”

Methods

A questionnaire was administered by structured telephone interviews during calls placed to the chiefs of facilities engineering at all 34 other hospitals in the greater Cleveland and Columbus, Ohio, metropolitan areas. Responses for The Cleveland Clinic Foundation were provided by the study investigators, who were members of the Department of Facilities Engineering and Section of Respiratory Therapy. The questionnaire addressed what type of central hospital oxygen supply system exists, whether mishaps involving the central oxygen supply system had occurred, to the respondent’s knowledge, whether a backup system exists, and, if so, details of its configuration. To maximize compliance with questionnaire response, the anonymity of responding institutions and responding individuals in published reports was assured.

The questionnaires were administered by two of the study investigators (JB and MS), both of whom were knowledgeable regarding hospital oxygen supplies in their respective capacities as supervisory registered respiratory therapist and senior engineer in The Cleveland Clinic Foundation facilities engineering group.

If the director of facilities engineering could not be reached, a follow-up call for questionnaire completion was placed to the director of respiratory care.

Results

As listed in Table 1, 35 hospitals (24 in Cleveland, 11 in Columbus) were surveyed, and responses were available from up to 32 (91.4%) of the institutions. Respondents were directors of facilities engineering in 81% (26/32), directors of respiratory therapy in 13% (4/32), and respiratory therapy instrumentation coordinators in 6% (2/32).

The size of responding hospitals ranged from 26 to 1,000 beds (mean 397 ± 251 [standard deviation], median 356). All respondents reported that central oxygen supply systems consisted of bulk medical gas with a primary liquid cylinder. The hospital construction dates ranged from 1887 to 1982 (median 1955), though central oxygen supplies were considerably more recent (1959 to 1989, median 1973).

Although the amount of oxygen used per day was infrequently known by respondents (3/32), hospitals for which the daily oxygen consumption was known reported using 9,000 cubic feet, 4,300 gallons, and 133,333 cubic feet per day. All 32 responding institutions reported a reserve system, described as a liquid reservoir in 79% (23/32), manifolded cylinders in 16% (5/32), and both in 13% (4/32).

Twenty-six (81%) of those responding reported having the reserve supply liquid or manifolded gas cylinders at the same location as the primary liquid vessel. The supply lines of these contiguous primary and reserve containers join proximal to entering the hospital structure, so that the primary and reserve of these 26 hospitals are dependent on
The Hospital Oxygen Supply: An "O2K" Problem

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<th>Year Oxygen System Constructed</th>
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<td>11</td>
<td>Chardon</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
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<td>NA</td>
</tr>
<tr>
<td>12</td>
<td>Lakewood</td>
<td>Director, Facilities Eng</td>
<td>400</td>
<td>1920</td>
<td>1975</td>
<td>Liquid + cylinder manifold</td>
</tr>
<tr>
<td>13</td>
<td>Cleveland</td>
<td>RT Instrumentation Coord.</td>
<td>220</td>
<td>1940</td>
<td>1977</td>
<td>Bulk liquid</td>
</tr>
<tr>
<td>14</td>
<td>Garfield Hts.</td>
<td>Director, Facilities Eng</td>
<td>280</td>
<td>1948</td>
<td>1972</td>
<td>Bulk liquid</td>
</tr>
<tr>
<td>15</td>
<td>Euclid</td>
<td>Director, Resp. Therapy</td>
<td>371</td>
<td>NA</td>
<td>NA</td>
<td>Bulk liquid</td>
</tr>
<tr>
<td>16</td>
<td>Mayfield Hts.</td>
<td>Director, Resp. Therapy</td>
<td>347</td>
<td>1968</td>
<td>NA</td>
<td>Bulk liquid</td>
</tr>
<tr>
<td>17</td>
<td>Cleveland</td>
<td>Director, Resp. Therapy</td>
<td>387</td>
<td>NA</td>
<td>NA</td>
<td>Bulk liquid</td>
</tr>
<tr>
<td>18</td>
<td>Warrensville Hts.</td>
<td>Director, Facilities Eng</td>
<td>166</td>
<td>1958</td>
<td>1970</td>
<td>Bulk liquid</td>
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<tr>
<td>19</td>
<td>Cleveland</td>
<td>Director, Facilities Eng</td>
<td>680</td>
<td>1974</td>
<td>1974</td>
<td>Bulk liquid</td>
</tr>
<tr>
<td>20</td>
<td>Columbus</td>
<td>Director, Facilities Eng</td>
<td>300</td>
<td>1972</td>
<td>1972</td>
<td>Bulk liquid</td>
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<tr>
<td>21</td>
<td>Columbus</td>
<td>Director, Facilities Eng</td>
<td>525</td>
<td>1905</td>
<td>1972</td>
<td>Cylinder manifold</td>
</tr>
<tr>
<td>22</td>
<td>Columbus</td>
<td>Director, Facilities Eng</td>
<td>1,000</td>
<td>NA</td>
<td>NA</td>
<td>Bulk liquid</td>
</tr>
<tr>
<td>23</td>
<td>Columbus</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>24</td>
<td>Parma</td>
<td>Director, Facilities Eng</td>
<td>364</td>
<td>1961</td>
<td>1961</td>
<td>Bulk liquid</td>
</tr>
<tr>
<td>25</td>
<td>Cleveland</td>
<td>Director, Facilities Eng</td>
<td>316</td>
<td>1929</td>
<td>1976</td>
<td>Cylinder manifold</td>
</tr>
<tr>
<td>26</td>
<td>Richmond Hts.</td>
<td>Director, Facilities Eng</td>
<td>210</td>
<td>1969</td>
<td>1983</td>
<td>Bulk liquid</td>
</tr>
<tr>
<td>27</td>
<td>Cleveland</td>
<td>Director, Facilities Eng</td>
<td>405</td>
<td>NA</td>
<td>NA</td>
<td>Bulk liquid</td>
</tr>
<tr>
<td>28</td>
<td>Cleveland</td>
<td>Director, Facilities Eng</td>
<td>100</td>
<td>1887</td>
<td>1979</td>
<td>Bulk liquid</td>
</tr>
<tr>
<td>29</td>
<td>Columbus</td>
<td>Director, Facilities Eng</td>
<td>800</td>
<td>1969</td>
<td>1969</td>
<td>Cylinder manifold</td>
</tr>
<tr>
<td>30</td>
<td>Middleburg Hts.</td>
<td>Director, Facilities Eng</td>
<td>325</td>
<td>1974</td>
<td>1974</td>
<td>Bulk liquid</td>
</tr>
<tr>
<td>31</td>
<td>Westlake</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>32</td>
<td>Westerville</td>
<td>Director, Facilities Eng</td>
<td>189</td>
<td>1982</td>
<td>1982</td>
<td>Bulk liquid</td>
</tr>
<tr>
<td>33</td>
<td>Cleveland</td>
<td>Director, Facilities Eng</td>
<td>474</td>
<td>1921</td>
<td>1959</td>
<td>Liquid + cylinder manifold</td>
</tr>
<tr>
<td>34</td>
<td>Cleveland</td>
<td>Director, Facilities Eng</td>
<td>440</td>
<td>1962</td>
<td>NA</td>
<td>Liquid + cylinder manifold</td>
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<tr>
<td>35</td>
<td>Cleveland</td>
<td>Director, Facilities Eng</td>
<td>799</td>
<td>1928</td>
<td>NA</td>
<td>Bulk liquid</td>
</tr>
</tbody>
</table>

**RT = respiratory therapy.**  
**NA = not available.**

A single length of pipe. Only 4 (13%) of the hospitals reported having manifolds in addition to the primary and reserve liquid supplies. These manifolds are in different locations than the primary and reserve, have physically separate feed lines, and represent the only true examples of redundant piped-in oxygen supplies recorded during the survey.

In the context that few respondents knew the average daily institutional oxygen consumption, it was not surprising that the mean duration of the backup system supply was infrequently known to respondents (6/32, 19%). In 6 instances, the mean backup duration was estimated to be 16 hours. Availability of an emergency outside low-pres-
Table 2. Summary of Selected Reported Mishaps with Hospital Central Oxygen Supply Systems

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Hospital</th>
<th>Mishap</th>
</tr>
</thead>
<tbody>
<tr>
<td>The Toronto Star4</td>
<td>1974</td>
<td>NA</td>
<td>Crossed oxygen and nitrous oxide lines in an emergency room caused at least 9 deaths.</td>
</tr>
<tr>
<td>The Times of London5</td>
<td>1975</td>
<td>NA</td>
<td>Crossed oxygen and nitrous oxide pipes caused inhalation of hypoxic gas and brain damage in a 26-year-old.</td>
</tr>
<tr>
<td>Eichhorn et al6</td>
<td>1977</td>
<td>Beth Israel Hospital wing, Boston, Massachusetts</td>
<td>Contamination of new oxygen piping system by a hydrocarbon gas and dust containing silicon, aluminum, and titanium; filling the main hospital oxygen supply vessel to replace gas used in purging caused a high-pressure relief valve to freeze, allowing high pressure to develop (90 psi).</td>
</tr>
<tr>
<td>Smith FP6</td>
<td>1987</td>
<td>NA</td>
<td>Contamination of central oxygen supply by argon, causing the death of two patients who inspired the contaminated oxygen.</td>
</tr>
<tr>
<td>The Orange County Register7</td>
<td>1988</td>
<td>University of California, Irvine Medical Center</td>
<td>Liquid oxygen leaked from the storage vessel when the valve froze in the open position during delivery.</td>
</tr>
<tr>
<td>Gilmour et al8</td>
<td>1990</td>
<td>University of Minnesota Hospital</td>
<td>Carbon tetrachloride (used to clean the truck delivery tank) contaminated the hospital oxygen supply, causing disruption of the hospital oxygen systems for 15 hours.</td>
</tr>
<tr>
<td>Shaw et al9</td>
<td>1991</td>
<td>Walton Hospital, England</td>
<td>Air entered oxygen pipeline into operating room, lowering fraction of inspired oxygen.</td>
</tr>
<tr>
<td>Lawler and Newman10</td>
<td>1992</td>
<td>South Cleveland Hospital, England</td>
<td>Air entered oxygen pipeline, lowering fraction of inspired oxygen.</td>
</tr>
<tr>
<td>The Sacramento Bee11</td>
<td>1993</td>
<td>Oroville Hospital, Sacramento, California</td>
<td>Oxygen from liquid oxygen vessel leaked when a pipe broke, caused by delivery truck’s driving away without releasing the delivery hose.</td>
</tr>
<tr>
<td>Los Angeles Times12</td>
<td>1994</td>
<td>Three hospitals (Olive View, Holy Cross Medical Center, Sepulveda VA Hospital)</td>
<td>Earthquake disrupted oxygen pipes.</td>
</tr>
</tbody>
</table>

NA = not available.

adjacent structure during a windstorm damaged the supply line from the primary liquid reservoir, but the reserve bulk reservoir was activated as designed, averting an interruption of oxygen supply. In the 4 instances in which the main supply line was interrupted, the reported duration of interruption was 3–4 hours in three hospitals, but was unknown in the fourth. In no instance of central oxygen supply interruption was an adverse patient outcome reported.

Responses to the 4 main supply-line interruptions included delivery of a tanker truck (n = 1), activation of a manifolded cylinder bank (n = 2), and back-feeding oxygen from cylinders (n = 1).

Problems other than interruption of the oxygen supply were described in one instance in this series. The event regarded inadvertent and unsuspected crossing of oxygen and nitrous oxide lines to anesthesia machines in the operating room at a time before oxygen analyzers were routinely in use. The resulting inadvertent delivery of nitrous oxide instead of oxygen reportedly contributed to the deaths of two patients. Among the remaining 31 respondents, problems other than interruption of the main supply were not reported (88%, 28/32) or were unknown to the respondents (9%, 4/32).

Discussion

The main finding from this survey regarding hospital central oxygen supply systems is that mishaps regarding the central oxygen supply were surprisingly common (16% [5/32] of responding institutions). Fortunately, despite interruptions of the main oxygen supply for up to 4 hours in the current series, contingency plans were exercised, with no adverse clinical effects from main supply line interruption. One reported gas mishap that was unrelated to the central oxygen supply consisted of crossing oxygen and nitrous oxide in the operating room and was associated with serious consequences.

Although similar mishaps have been described in past case reports, the current study extends prior understanding by surveying hospitals in two large cities to estimate the rate of such mishaps. In one earlier (1976) survey regarding hospital central gas mishaps, Feeley and Hedley-Whyte administered a mail survey to 200 hospital directors of anesthesiology to ascertain the frequency of malfunction of hospital oxygen and nitrous oxide systems. Based on the 88% of responses, 31% (n = 59) of respondents reported 76 incidents, which caused 3 deaths. One of the fatal mishaps regarded gas lines that were crossed during
new hospital construction, and 2 fatalities related to contamination of an existing oxygen supply (with nitrogen gas). Overall, at least 6 (8%) of the 76 incidents involved malfunction of existing central gas supply systems. Insufficient oxygen pressure accounted for 51% (n = 37) of the 76 incidents, while excessive pressures were reported in 9%. Failure of low-pressure alarms, predisposing to depletion of oxygen supplies, was reported in 4 instances (5%), and low oxygen flow occurred twice (3%). Excluding the instances of crossed pipes, contamination of the oxygen supply in an established central supply system was reported in 2 instances (3%), once when the supply vessel was filled with nitrogen (causing 2 deaths) and once when water contaminated the line. Finally, one instance of a leak in the oxygen pipeline was reported.

Of the 37 instances when oxygen pressure was reportedly low, causes were specified in 30, and included: pipeline damage during nearby construction activity (n = 8), pipeline blockage (n = 6), oxygen depletion because of insufficient capacity (n = 6), freezing of a regulator during delivery (n = 4), unannounced system shutdowns by engineering (n = 3), lightning damage to the main reservoir vessel, (n = 1), regulator malfunction (n = 1), and installation of wrong wall connectors (n = 1). Although the status of backup oxygen supply systems was not the subject of the 1976 survey, it appears that at least 26 of these 37 instances of low or absent oxygen pressure might have been averted by availability of adequate backup oxygen supplies.

Since publication of the results of the 1976 survey, MEDLINE and Lexis-Nexis searches of the indexed medical and popular literature found 8 additional reports of mishaps with central oxygen supply systems (Table 2). Five of these instances involved contamination of the central oxygen supply, either by introduction of toxic substances (eg, carbon tetrachloride, argon) during delivery of liquid oxygen to the main storage vessels (n = 3), or by contamination with air through existing pipes (n = 2). Three instances regarded oxygen leaks, either due to human error during delivery of liquid oxygen (n = 2) or due to earthquake damage to pipes (n = 1). Two instances regarded inadvertent crossing of supply lines containing oxygen and nitrous oxide.

Conclusions

Overall, available results from the current survey and prior reports suggest that inadvertent interruption of the main oxygen supply line from the bulk reservoir to the hospital occurs relatively frequently (ie, in 12.5% [4/32] of the responding institutions in this series) and is the most common cause of oxygen main supply interruption. The need for greater attention to labeling and protecting the main supply line is clearly suggested. The results of our survey also suggest several specific measures to lessen the risk of mishaps involving the hospital central oxygen supply:

1. Hospitals should conduct a systematic audit of their central gas supply systems regarding the rate of daily oxygen consumption, the presence and adequacy of a backup system, and the existence of a specific contingency plan in the event of an interruption.

2. Central oxygen supply systems should incorporate several important design features:
   a. Prominent labeling and shielding of the oxygen feed lines that connect the main supply vessel to the hospital, so as to avert accidental interruption (eg, during street repair),
   b. Availability of a backup supply vessel, ideally located remote from the main vessel and with separate feed lines to the hospital, and
   c. Ample valves along the oxygen supply line within the hospital, so that leaks can be isolated without interrupting the central supply to the entire institution.

Although the present research is only the second survey that, to our knowledge, has systematically addressed the rate and spectrum of mishaps, several important shortcomings of this study are noteworthy. First, the response rate was incomplete, with some responses available in as few as 22% (7/32) of hospitals surveyed. In instances in which responses were available from the minority of institutions addressed, sampling bias remains a concern. Another potential bias affecting our results is that, despite the promise of nondisclosure of institutional identity by the investigators, reluctance by respondents to candidly volunteer adverse hospital experiences might be expected, causing the rates and even severity of adverse events to be underreported in this series. Finally, our survey did not systematically address gas mishaps unrelated to the central hospital gas supply (eg, interruptions of oxygen lines in anesthesia machines serving individual patients). However, as has been discussed by Anderson and Brock-Utne,13 such interruptions also may occur, and it is critically important for the managing clinician to have a systematic approach for identifying the specific cause of oxygen interruption.

Notwithstanding these potential study biases, we believe that the reported frequency of oxygen supply mishaps should prompt clinicians, administrators, and hospital facilities personnel to review the status and contingency plans regarding their hospitals’ central oxygen supplies, so as to avert the substantial risks of unanticipated failure. In this way, the “O2K problem” can be avoided.
REFERENCES

3. The Toronto Star; Apr 10, 1974.
The Effects of Passive Humidifier Dead Space on Respiratory Variables in Paralyzed and Spontaneously Breathing Patients

Robert S Campbell RRT FAARC, Kenneth Davis Jr MD, Jay A Johannigman MD, and Richard D Branson RRT

BACKGROUND: Passive humidifiers have gained acceptance in the intensive care unit because of their low cost, simple operation, and elimination of condensate from the breathing circuit. However, the additional dead space of these devices may adversely affect respiratory function in certain patients. This study evaluates the effects of passive humidifier dead space on respiratory function. METHODS: Two groups of patients were studied. The first group consisted of patients recovering from acute lung injury and breathing spontaneously on pressure support ventilation. The second group consisted of patients who were receiving controlled mechanical ventilation and were chemically paralyzed following operative procedures. All patients used 3 humidification devices in random order for one hour each. The devices were a heated humidifier (HH), a hygroscopic heat and moisture exchanger (HHME) with a dead space of 28 mL, and a heat and moisture exchanger (HME) with a dead space of 90 mL. During each measurement period the following were recorded: tidal volume, minute volume, respiratory frequency, oxygen consumption, carbon dioxide production, ratio of dead space volume to tidal volume ($V_T/V_t$), and blood gases. In the second group, intrinsic positive end-expiratory pressure was also measured. RESULTS: Addition of either of the passive humidifiers was associated with increased $V_T/V_t$. In spontaneously breathing patients, $V_T/V_t$ increased from 59 ± 13 (HH) to 62 ± 13 (HHME) to 68 ± 11% (HME) (p < 0.05). In these patients, constant alveolar ventilation was maintained as a result of increased respiratory frequency, from 22.1 ± 6.6 breaths/min (HH) to 24.5 ± 6.9 breaths/min (HHME) to 27.7 ± 7.4 breaths/min (HME) (p < 0.05), and increased minute volume, from 9.1 ± 3.5 L/min (HH) to 9.9 ± 3.6 L/min (HHME) to 11.7 ± 4.2 L/min (HME) (p < 0.05). There were no changes in blood gases or carbon dioxide production. In the paralyzed patient group, $V_T/V_t$ increased from 54 ± 12% (HH) to 56 ± 10% (HHME) to 59 ± 11% (HME) (p < 0.05) and arterial partial pressure of carbon dioxide ($P_{acO_2}$) increased from 43.2 ± 8.5 mm Hg (HH) to 43.9 ± 8.7 mm Hg (HHME) to 46.8 ± 11 mm Hg (HME) (p < 0.05). There were no changes in respiratory frequency, tidal volume, minute volume, carbon dioxide production, or intrinsic positive end-expiratory pressure. DISCUSSION: These findings suggest that use of passive humidifiers with increased dead space is associated with increased $V_T/V_t$. In spontaneously breathing patients this is associated with an increase in respiratory rate and minute volume to maintain constant alveolar ventilation. In paralyzed patients this is associated with a small but statistically significant increase in $P_{acO_2}$. CONCLUSION: Clinicians should be aware that each type of passive humidifier has inherent dead space characteristics. Passive humidifiers with high dead space may negatively impact the respiratory function of spontaneously breathing patients or carbon dioxide retention in paralyzed patients. When choosing a passive humidifier, the device with the smallest dead space, but which meets the desired moisture output requirements, should be selected. [Respir Care 2000;45(3):306–312] Key words: passive humidifier, dead space, respiratory function, mechanical ventilation, alveolar ventilation, humidification, respiratory equipment.

Background

Humidification of inspired gases following tracheal intubation for mechanical ventilation is required to prevent the untoward effects of cool, dry gases on the tracheobronchial epithelium.1-3 For nearly 4 decades, heated humidification has been the preferred method of conditioning inspired gases. In the past decade, passive humidifiers (PHs,
also known as “artificial noses”) have gained some popularity because of their low cost and simple operation. During introduction of PHs to our practice, we became concerned about the potential for increase in the work of breathing (WOB) due to the inherent flow resistance of the device. This increased resistance was studied by others and found to have a small, clinically unimportant effect on respiratory function under normal conditions. However, clinically important differences have been reported in the case of accumulated secretions or water in the PH. More recently, several reports have shown that PH dead space may adversely affect respiratory mechanics, blood gases, and WOB. We evaluated the effects of two PHs, one with a small dead space (28 mL) and one with a larger dead space (90 mL), on respiratory mechanics, lung volumes, and blood gases during spontaneous and controlled mechanical ventilation.

Materials and Methods

All patients were in the surgical intensive care unit at the University of Cincinnati. Informed consent was obtained from the patient or next of kin prior to study entry. Two groups of patients were studied. Group I consisted of 15 patients considered to be weaning from mechanical ventilation and who were receiving 5 cm H₂O positive end-expiratory pressure, fraction of inspired oxygen ≤ 0.50, and pressure support of 10 cm H₂O. All Group I patients were recovering from acute lung injury and breathing spontaneously at near minimal ventilatory support during the study. Group II consisted of 11 patients receiving controlled mechanical ventilation who were pharmacologically paralyzed and sedated following operative procedures.

Each patient used 3 humidification devices in random order for a period of one hour each. The devices included a heated humidifier (HH, an MR 730, Fisher & Paykel, Panmure, New Zealand), a hygroscopic heat and moisture exchanger (HHME, a Humid-Vent 2, Gibeck, Indianapolis, Indiana), and a heat and moisture exchanger (HME, an Extended Use HME, Mallinckrodt, Pleasanton, California). The HH was a passover device set to maintain proximal airway temperature at 34°C. A conventional (not a heated-wire) 60-inch breathing circuit (Hudson-RCI, Temecula, California) was used during all 3 study periods. The HHME and HME were placed between the endotracheal tube and circuit Y-piece. The HH was removed from the circuit when each PH was in use. The resistance of each device was measured at a constant flow of 1 L/s using a calibration analyzer (RT-200 Timeter Calibration Analyzer, Allied Health Care Products, St Louis, Missouri) prior to use. The dead space of each PH was measured according to International Organization for Standardization standard 9360. Table 1 shows characteristics of the 3 devices.

Patients were maintained in the semi-Fowler’s position throughout the study. All patients were ventilated using a Puritan-Bennett 7200ae ventilator (Mallinckrodt, Pleasanton, California), and ventilator settings remained constant during the study periods. Each ventilator used was up-to-date with regard to preventive maintenance schedules, and the flow and volume monitoring accuracy was assured within 10% prior to data collection. Pressure-triggering was used during the entire study period to eliminate bias flow in the breathing circuit, and the sensitivity was set to the lowest level that would not result in auto-triggering. All patients in Group II were ventilated using volume-controlled mandatory breaths. When a new humidification device was placed, a complete extended self test was performed to assure circuit integrity (eliminate leaks) and calculate circuit compressible volume. Airway pressures, volumes, and flows were measured using sensors integral to the ventilator. Data for these measurements were an average of the values obtained over the last 5 minutes of each one-hour period. Continuous measurements of oxygen consumption (VO₂) and carbon dioxide production (VCO₂) were accomplished using a commercially available, indirect calorimeter (Delta Trac, Sensormedics, Yorba Linda, California). The fraction of inspired oxygen was stabilized by using an inspiratory mixing chamber (MRM 250, Fisher & Paykel, Panmure, New Zealand), and expiratory gases were collected from the expiratory port of the ventilator. The accuracy of the calorimeter is ± 3% for VCO₂ and ± 7% for VO₂. Values for VO₂ and VCO₂ are based on an average of the last 10 minutes of each one-hour period. Mixed expired carbon dioxide (Pako₂) was measured during the final 3 minutes of observation. During the final minute, an arterial blood gas sample was drawn and immediately analyzed for pH, Pao₂, and Paco₂. Dead space to tidal volume ratio (VD/VT) was calculated using the Bohr equation:

\[
V_D/VT = \frac{P_{aCO_2} - P_{ECO_2}}{P_{aCO_2}}
\]
Table 2. Patient Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Group I (n = 15)</th>
<th>Group II (n = 11)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>37 ± 12</td>
<td>54 ± 15</td>
</tr>
<tr>
<td>Gender (male/female)</td>
<td>9/6</td>
<td>8/3</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>86 ± 9</td>
<td>83 ± 12</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>164 ± 18</td>
<td>152 ± 17</td>
</tr>
<tr>
<td>Duration of ventilation (days)</td>
<td>11 ± 3</td>
<td>1 ± 2</td>
</tr>
</tbody>
</table>

Dead space ventilation was calculated as the product of minute ventilation and $V_L/V_T$. Alveolar ventilation was calculated as the difference between minute ventilation and dead space ventilation. The ventilatory equivalents for oxygen and carbon dioxide were measured by dividing the minute ventilation by the $V_{O_2}$ and $V_{CO_2}$, respectively. In patients receiving controlled mechanical ventilation, intrinsic positive end-expiratory pressure (PEEP) was measured using the expiratory port occlusion technique. PEEP was measured twice, with 2–3 minutes between measurements. PEEP was the last measurement to be accomplished at the end of each study period.

All data are reported as mean ± standard deviation. Data were compared using analysis of variance, and a p < 0.05 was considered significant.

Results

Table 1 shows the resistance and dead space characteristics of each humidification device. Table 2 shows patient characteristics.

Spontaneously Breathing Patients (Group I)

The addition of either an HHME or an HME increased respiratory frequency, compared to breathing via an HH. This increase in frequency was accompanied by increased $V_T$ in the HME group. Minute ventilation, $V_L/V_T$, and the ventilatory equivalent of both oxygen and carbon dioxide were significantly greater with the HME than with the HH (p < 0.05). Alveolar ventilation remained constant during all 3 study periods. Although respiratory frequency, minute ventilation, and $V_L/V_T$ were higher with HHME than with HH, the differences did not reach statistical significance. There were no changes in blood gases between study periods resulting from the humidification techniques employed. Figure 1 shows the relationship between minute ventilation, alveolar ventilation, and dead space volume in spontaneously breathing patients resulting from each humidification technique. All data for Group I patients are shown in Table 3 as mean ± standard deviation.

Controlled Mechanical Ventilation Patients (Group II)

The addition of either an HHME or an HME increased both $V_L/V_T$ and $P_{aCO_2}$ and decreased alveolar ventilation, compared to HH, and the difference was statistically significant when comparing HH to HME (p < 0.05). There were no changes in $P_{aO_2}$. Figure 2 shows the relationship between minute ventilation, alveolar ventilation, and dead space volume in paralyzed patients receiving controlled mechanical ventilation, with each of the humidification techniques employed. By virtue of the study design, minute ventilation, frequency, and $V_T$ remained constant between study periods. There were no differences in PEEP. All data for Group II patients are shown in Table 4 as mean ± standard deviation.

Discussion

Passive humidifiers have had growing acceptance in recent years because of their low cost, simple operation, and elimination of circuit condensation. Concern over adverse ventilatory effects of PHs has predominately been aimed at the increased resistance imposed by the foam or paper insert. Those studies, however, failed to show significant changes in the WOB associated with PH resistance, in the absence of partial occlusion by secretions.
blood, or saline. More recently, PH dead space has been implicated as a source of ventilatory impairment.12-14

Our findings indicate that increasing PH dead space increases $V_D/V_T$. In spontaneously breathing patients, this requires increased minute ventilation to maintain constant alveolar ventilation and $P_{aCO_2}$. In our patients, this increase was mainly accomplished by increasing respiratory frequency. During controlled mechanical ventilation, the increase in $V_D/V_T$ resulted in a small but statistically significant increase in $P_{aCO_2}$, indicating decreased alveolar ventilation. These findings are consistent with the work of Le Bourdelles et al.,12 Pelosi et al.,13 and Iotti et al.14

Pelosi et al compared the effects of two PHs on ventilatory mechanics and volumes in 14 patients ventilated using pressure support ventilation.13 Characteristics of the HHMEs used in that study were: dead space 95 mL and resistance 1.9 cm H$_2$O/L/s, and dead space 65 mL and resistance 2.5 cm H$_2$O/L/s. All patients in that study were recovering from acute lung injury. They found that both HHMEs increased minute ventilation approximately 2.5 L/min over that seen with HH use.13 These authors also found that patient WOB increased 66% with the larger HHME and 37% with the smaller HHME, compared to HH. Pelosi et al also noted that the pressure generated in the first 100 milliseconds ($P_{0.1}$) of inspiration significantly increased during use of the HHMEs. During use of the large HHME, $P_{0.1}$ doubled, and during use of the small HHME, $P_{0.1}$ increased by 60%. Their findings indicate that the additional minute ventilation required to overcome the increased dead space significantly impacts patient WOB. In an effort to overcome the increased patient WOB, the authors increased the pressure support level. The use of pressure support returned WOB per breath to baseline values, but the WOB per minute remained elevated. The authors suggested that an additional 10 cm H$_2$O pressure support should eliminate the increased WOB caused by increased HHME dead space.13

Le Bourdelles et al compared blood gases and ventilatory volumes during ventilation using an HH and an HHME (dead space 75 mL). All patients in this study were spontaneously breathing on 10–15 cm H$_2$O pressure support. They found that during HHME use, minute ventilation increased from $8.1 \pm 0.8$ L/min to $9.3 \pm 0.8$ L/min, with $V_T$ remaining constant and respiratory frequency increasing from $19 \pm 2$ breaths/min to $21 \pm 2$ breaths/min. They also found that, despite the increase in minute ventilation, $P_{aCO_2}$ increased from $42 \pm 2$ mm Hg to $44 \pm 2$ mm Hg.12 Le Bourdelles et al did not measure WOB, $V_D/V_T$, or
V\textsubscript{CO\textsubscript{2}}. The authors suggested that, though in most patients the addition of HHME dead space is probably insignificant, patients with respiratory muscle fatigue could be negatively affected during weaning.

In a study very similar to ours, lotti et al compared respiratory mechanics, blood gases, and respiratory volumes during use of an HH, an HHME with a flex tube (dead space 60 mL), and an HHME filter (HHMEF) with a dead space of 100 mL\textsuperscript{14}. Unique to their study was use of a closed loop controller that automatically adjusted pressure support to maintain a constant P\textsubscript{p,1}. Like our study, they measured V\textsubscript{T}/V\textsubscript{T} and calculated alveolar ventilation and dead space ventilation. They also measured WOB, airway resistance, PEEP\textsubscript{T}, and lung compliance. During this study, minute ventilation increased from 10.6 \pm 2.3 L during use of an HH, to 10.9 \pm 1.6 L during use of an HHME, to 11.9 \pm 1.6 L during use of an HHMEF\textsuperscript{14}. They also noted a significantly increased total WOB per minute between devices. The total WOB (patient work and ventilator work) was 13.6 \pm 8.6 j/min using the HH, 19.2 \pm 9.1 j/min using the HHME, and 22.3 \pm 9.9 j/min using the HHMEF. Because the closed loop controller increased and decreased pressure support to maintain constant P\textsubscript{p,1}, patient WOB remained constant. Respiratory rate was unchanged, but the required pressure support level increased from 13 cm H\textsubscript{2}O (HH) to 15 cm H\textsubscript{2}O (HHME) to 18 cm H\textsubscript{2}O (HHMEF) in order to keep P\textsubscript{p,1} constant. The changes in minute ventilation and dead space ventilation observed by lotti et al are similar to our results. In their study, alveolar ventilation was kept constant at the cost of a 2 L/min increase in minute volume when using the 100 mL dead space HHMEF. Our results, using an HME with 90 mL of dead space, averaged a 2.6 L/min increase in minute ventilation. In our patients, increased frequency led to higher minute ventilation, whereas in lotti’s patients the ventilator automatically adjusted the pressure support level in order to maintain constant P\textsubscript{p,1}, thus utilizing V\textsubscript{T} changes to increase minute ventilation. During constant pressure support, increasing frequency is easier for the patient than increasing V\textsubscript{T}\textsuperscript{19}

Another interesting finding in lotti’s study relates to changes in expiratory resistance and total WOB. During use of the large HHMEF, expiratory resistance increased by 30%, yet the total inspiratory WOB increased by 60%. Although we did not measure in vivo airways resistance, no indication of significantly increased resistance could be associated with the HHME or HME, compared to HH, by measurement of peak inspiratory pressure, peak expiratory flow, or PEEP\textsubscript{T}. We did not directly measure patient mechanical WOB, but indirect measures of patient work (ven-
Table 4. Comparison of Ventilatory and Blood Gas Parameters in Paralyzed Patients, Using Three Humidification Devices

<table>
<thead>
<tr>
<th>Variable</th>
<th>HH</th>
<th>HHME</th>
<th>HME</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency (breaths/min)</td>
<td>11.7 ± 3.1</td>
<td>11.7 ± 3.1</td>
<td>11.7 ± 3.1</td>
</tr>
<tr>
<td>Tidal volume (mL)</td>
<td>794 ± 321</td>
<td>801 ± 322</td>
<td>791 ± 312</td>
</tr>
<tr>
<td>Minute volume (L/min)</td>
<td>9.3 ± 3.1</td>
<td>9.4 ± 3.2</td>
<td>9.3 ± 3.1</td>
</tr>
<tr>
<td>PIP (cm H₂O)</td>
<td>32.6 ± 7.4</td>
<td>32.6 ± 7.0</td>
<td>32.9 ± 7.1</td>
</tr>
<tr>
<td>P₀₂ (mm Hg)</td>
<td>104 ± 36</td>
<td>104 ± 31</td>
<td>96 ± 34</td>
</tr>
<tr>
<td>Pₐcarbon- dioxide (mm Hg)</td>
<td>43.2 ± 8.5</td>
<td>43.9 ± 8.7</td>
<td>46.8 ± 11.1*</td>
</tr>
<tr>
<td>V₀₂ (mL/min)</td>
<td>308 ± 87</td>
<td>310 ± 87</td>
<td>312 ± 83</td>
</tr>
<tr>
<td>Vₐcarbon-dioxide (mL/min)</td>
<td>257 ± 60</td>
<td>254 ± 58</td>
<td>237 ± 57</td>
</tr>
<tr>
<td>Vₑ/V₀₂ (L/L V₀₂)</td>
<td>30.2 ± 7.6</td>
<td>30.3 ± 7.9</td>
<td>29.8 ± 8.3</td>
</tr>
<tr>
<td>Vₑ/Vₐcarbon-dioxide (L/L Vₐcarbon-dioxide)</td>
<td>36.2 ± 7.1</td>
<td>37.0 ± 6.9</td>
<td>39.2 ± 7.6</td>
</tr>
<tr>
<td>Vₑ/Vₑ (Vₑ) (%)</td>
<td>54 ± 12</td>
<td>56 ± 10</td>
<td>59 ± 11†</td>
</tr>
<tr>
<td>PEEP</td>
<td>0.8 ± 0.4</td>
<td>1.0 ± 0.7</td>
<td>1.1 ± 0.5</td>
</tr>
</tbody>
</table>

HH = heated humidifier.
HHME = hypoxicographic heat and moisture exchanger.
HME = heat and moisture exchanger.
PIP = peak inspiratory pressure.
P₀₂ = arterial partial pressure of oxygen.
Pₐcarbon-dioxide = arterial partial pressure of carbon dioxide.
V₀₂ = oxygen consumption.
Vₐcarbon-dioxide = carbon dioxide production.
Vₑ/V₀₂ = ratio of minute ventilation to oxygen consumption.
Vₑ/Vₐcarbon-dioxide = ratio of minute ventilation to carbon dioxide production.
Vₑ/Vₑ (Vₑ) = ratio of dead space volume to tidal volume.
PEEP = positive end-expiratory pressure.
* p < 0.05 HHME and HH vs HME.
† p < 0.05 HH vs HME.

The devices used in the other studies all had filters, with the exception of the small HHME in the study by Iotti et al.12-14 Therefore, the devices in our study posed less impediment to expiratory flow. Second, in the other studies, pressure support was increased to supplement Vₑ and maintain alveolar ventilation. In this instance, higher Vₑ coupled with increased expiratory resistance would have increased the risk of PEEP₁. Increased PEEP₁ may also lead to increased patient WOB, because ventilator triggering would become more difficult. Additionally, the patients in our study who had PEEP₁ measured were neuromuscularly blocked. In these patients, changes in frequency and inspiration-expiration ratio were not possible and exhalation was always passive.

The effect of PEEP₁ caused by PH resistance might be expected to impact respiratory function differently, depending on the presence of obstructive pulmonary disease. In a patient with chronic obstructive pulmonary disease, the additional resistance of a PH might counterbalance the intrinsic flow resistance (similar to pursed-lip breathing) and allow more complete emptying of the lung and thus no net change in end-expiratory alveolar pressure. In a patient with normal airways, any additional expiratory resistance may lead to a net increase in PEEP₁. In an earlier study of chronic obstructive pulmonary disease patients, Conti et al failed to find any increase in PEEP₁ in a group of patients with chronic obstructive pulmonary disease using an HHME.21 This may explain the apparent contradiction.

Conclusions

In conclusion, our findings suggest that the increased dead space of a PH can negatively impact ventilatory function by increasing Vₑ/Vₑ. In spontaneously breathing patients, alveolar ventilation is maintained by increasing minute ventilation. Minute ventilation is increased by increasing respiratory frequency, which may result in increased WOB. The addition of 5-10 cm H₂O of pressure support may be helpful in normalizing patient WOB and breathing pattern during ventilation with a high dead space PH. In paralyzed patients, the additional dead space may reduce alveolar ventilation, increasing Pₐcarbon-dioxide. The effect of PH dead space may be exacerbated during ventilation at low Vₑ and with larger dead space PH devices. When choosing between available humidification devices, clinicians should consider dead space, in addition to the resistance and moisture output characteristics of each available PH.

REFERENCES

In Vitro Comparison of the Circulaire and AeroTee to a Traditional Nebulizer T-Piece with Corrugated Tubing

S David Piper PE

BACKGROUND: Nebulizers are a popular means of delivering aerosolized medication, primarily albuterol, to the bronchial Airways of patients, and there has been extensive research done on numerous nebulizers used with nebulizer T-pieces and corrugated tubing. Very little research has been performed on other types of nebulizer delivery systems and there is no substantial information on how effective various nebulizer delivery systems are in terms of the quantity and particle size of aerosolized medication delivered to the patient. In this study the Circulaire and the AeroTee, two devices that rely on bags to store aerosol during patient exhalation, are evaluated and compared to the conventional nebulizer T-piece with corrugated tubing. METHODS: Three each of the nebulizer T-piece with corrugated tubing, the Circulaire, and the AeroTee were sampled using 3 Vixone nebulizers. Each one of the 3 nebulizer delivery systems used the same 3 Vixone nebulizers. Each nebulizer delivery system was evaluated by connecting a constant-flow vacuum and compressed gas source cycled to simulate patient breathing at a respiratory rate of 14 breaths/min and an inspiration-expiration ratio of 1:2. Medication delivered was determined by sampling a portion of the simulated patient’s flow onto a membrane filter and calculating the total medication received by the patient. Particle size was determined by sampling with a cascade impactor under ambient conditions. RESULTS: The Circulaire delivered significantly less medication than the nebulizer T-piece with corrugated tubing (p < 0.001), whereas the AeroTee delivered substantially more medication than the nebulizer T-piece with corrugated tubing (p < 0.001). The particle size delivered by the Circulaire was significantly smaller than that of the nebulizer T-piece with corrugated tubing (p < 0.001), whereas the AeroTee delivered particle size equivalent to a nebulizer T-piece with corrugated tubing (p = 0.82). CONCLUSION: There are clinically important differences between nebulizer delivery systems. When evaluating the optimum means for delivering aerosolized medication, equal consideration should be given to both the brand of nebulizer and the nebulizer delivery system. [Respir Care 2000;45(3):313–319] Key words: nebulizer, aerosol, Circulaire, AeroTee, bronchodilator, aerodynamic particle size.

Background

Aerosolized bronchodilators have been shown to be an effective means of treating severe reversible airway disease. Quantity of medication delivered, aerosol particle size (mass median aerodynamic diameter [MMAD]), and treatment time are all important factors in evaluating the performance of an aerosol delivery device. The majority of research on aerosol delivery devices has focused on the nebulizer itself, traditionally equipped with a nebulizer T-piece and a 6-inch length of corrugated tubing. One of the most comprehensive studies was performed by Hess et al., in which the performance of 17 different nebulizers configured in the traditional way was evaluated under simulated patient conditions. Recently, a number of different nebulizer delivery systems have been developed that are intended to improve and replace the traditional nebulizer T-piece and 6-inch length of corrugated tubing. Two new nebulizer delivery systems, the Circulaire (Westmed, Tucson, Arizona) and AeroTee (Hudson Respiratory Care, Temecula, California), both use a bag to store aerosol during patient exhalation, which is delivered on the subsequent inhalation. The devices differ in their methods of mini-
mizing the retention of carbon dioxide. The Circulaire uses a one-way flapper valve to prevent the backflow of any exhaled gas into the aerosol storage bag. The AeroTee uses a purging technique to effectively flush all retained carbon dioxide from the aerosol storage bag prior to inhalation. The present study compares the performance of these two new nebulizer delivery systems to the traditional nebulizer T-piece with corrugated tubing.

Methods

Nebulizer Delivery Systems Evaluated

Figure 1 shows the three nebulizer systems evaluated. Three of each nebulizer delivery system were selected, along with three Vixone nebulizers (Westmed, Tucson, Arizona). The Vixone nebulizer was selected for this study because, unlike the AeroTee, which may be used with any small volume nebulizer, the Circulaire comes equipped only with the Vixone and is not available without it. Both the Circulaire and the AeroTee use a 750 mL bag to store aerosol during exhalation so that the stored aerosol is delivered to the patient on the subsequent inhalation. The two devices differ in the manner in which they prevent the patient from rebreathing exhaled gas. The Circulaire (Fig. 2) uses a one-way flapper valve so that gas can travel only from the direction of the aerosol storage bag toward the patient, thus ensuring that exhaled gas goes directly from the patient to the ambient environment. The AeroTee (Fig. 3) allows some exhaled gas to enter the aerosol storage bag, which is then effectively purged by aerosolized medication using a coherent laminar jet prior to the beginning of inhalation. The nebulizer T-piece with corrugated tubing (Fig. 4) does not use collapsible aerosol storage means of any sort, but relies on the corrugated tubing (~60 mL) to capture some of the aerosol produced by the nebulizer during exhalation.
Evaluation of Quantity of Medication Delivered to the Patient and Total Treatment Time

Each of the three sets of nebulizer delivery systems were tested with the same three Vixone nebulizers. Each of the Vixone nebulizers was tested initially with a different nebulizer delivery system to prevent any bias in data due to deterioration in performance of the nebulizer after multiple uses. Prior to each test, each nebulizer was rinsed with filtered water, dried with compressed air, and filled with 3 mL of normal saline containing 2.5 mg of albuterol sulfate (Sigma, St Louis, Missouri) and 3.0 mg of fluorescein (Sigma, St Louis, Missouri). Despite the fact that Hess et al.1 showed increasing medication delivery with increases in fluid volume, 3 mL of fluid was used for each test because that is what is still most commonly used under clinical conditions. As described by the manufacturer of the Vixone nebulizer, each nebulizer was set up and run at 6 L/min until all visual evidence of aerosol ceased and sputtering had ceased. Total treatment time was recorded. Simulated patient conditions were achieved with two solenoid valves, one attached to a compressor and one to a vacuum pump (Fig. 5). The solenoid valves were controlled by the function generator, which was set to cycle on a breathing rate of 14 breaths/min with a constant inspiration-expiration ratio (I:E) of 1:2. The flows of the compressed gas (exhalation) and the vacuum source (inhalation) were set to simulate a minute ventilation of 10 L/min (inhalation flow 30 L/min, exhalation flow 15 L/min). Prior to each test, a Wright Mark 14 respirometer (Ferraris, England) was used to verify that the volume of gas inhaled was equivalent to the volume of gas exhaled (tidal volume = 714 mL ± 5%). Each nebulizer and accompanying delivery device was attached via a mouthpiece to 22 mm of tubing, the middle of which was equipped with a T-piece for sampling aerosol at 1.0 L/min through a 47 mm membrane filter (Micro Filtration Systems, Dublin, California). The opposite end of the 22 mm tube was equipped with a Y-piece, the branches of which led to the inhalation and exhalation solenoid valves. The dead space volume between the Y-piece and the mouthpiece of the nebulizer delivery system was measured to be approximately 50 mL.
The flow of simulated exhalation gas was shown to purge the dead space volume in approximately 0.2 second, so as to ensure that minimal aerosol was sampled during the exhalation phase. Sampling occurred continuously throughout the entire treatment time. Fluorescein collected was measured using a spectrophotometer (Barnstead, Dubuque, Iowa). Medication delivery rates were calculated from the product of the total amount of mass collected and the ratio of the inspiratory flow to the sampling flow.

**Evaluation of Aerodynamic Particle Size**

As with the evaluation of quantity of medication delivered, each of the three sets of nebulizer delivery systems was tested with the same three Vixone nebulizers. Each of the Vixone nebulizers was tested initially with a different nebulizer delivery system to prevent any bias in data due to deterioration in nebulizer performance after multiple uses. Prior to each test, each nebulizer was rinsed with filtered water, dried with compressed air, and then filled with 3 mL of normal saline containing 2.5 mg of albuterol sulfate and 3.0 mg of fluorescein. The nebulizer was run at 6 L/min. Aerosol was sampled at a flow of 0.7 L/min using a 7-stage cascade impactor (In-Tox Products, Albuquerque, New Mexico) immediately proximal to the mouthpiece as the aerosol exited into ambient room conditions. Aerodynamic cut-off sizes for the stages of the cascade impactor were 6.19 μm, 3.89 μm, 2.5 μm, 2.0 μm, 1.31 μm, 0.85 μm, and 0.40 μm, in addition to a membrane filter that captured all aerosol < 0.40 μm. The relative amount of medication collected on each stage of the cascade impactor was determined fluorometrically using the spectrophotometer. MMAD was then calculated from a best-fit plotting of cumulative mass percentage versus aerodynamic diameter on log-probability graph paper, using the spectrophotometer data for each cascade impactor stage.

**Percent of Aerosol Delivered in the 1–5 μm Range**

The percent of aerosol delivered in the 1–5 μm range was determined directly from the cascade impactor particle sizing data. Because the cascade impactor did not have stages with aerodynamic cutoffs at exactly 1 μm and 5 μm, results were logarithmically interpolated using the relative mass collected for the stages immediately around 1 μm and 5 μm, as is consistent with cascade impactor data.

**Mass of Aerosol Delivered in the 1–5 μm Range**

The mass of aerosol delivered in the 1–5 μm range was calculated as the percent of aerosol between 1 μm and 5 μm, multiplied by the medication rate determined previously, and represents the amount of medication most available for deposition to the bronchial airways and alveoli. 1–5 Although this is a simplified model of deposition rate, it provided the means for achieving one of the objectives of this study, which was to compare the variation of these nebulizer delivery systems with the variation identified by Hess et al1 for various nebulizers.

**Volume of Gas Rebreathed by the Patient**

Testing was performed using the previously described setup (see Fig. 5). Exhalation flow was simulated using 100% oxygen. Nebulizers were run without fluids at 6 L/min, using compressed air. Measurements were taken with an oxygen sensor inside a two-liter bag. The two-liter bag was used to capture two complete inhalations from the outlet of the vacuum source, and the increase in oxygen concentration above ambient was used to calculate the volume of gas rebreathed. The volume of gas rebreathed with the simulated patient breathing directly into ambient conditions was subtracted from the volume of gas rebreathed with each nebulizer delivery system to obtain a final result. The technique was checked using a known dead space of 60 mL, and the volume of gas rebreathed was determined to be 56.5 mL, which was considered to be in good agreement.

**Statistical Analysis**

Summary statistics are reported as mean ± standard error. Differences between groups were determined by one-tailed or two-tailed tests, as appropriate. Statistical significance was set at p < 0.05.

**Results**

**Quantity of Medication Delivered and Treatment Time**

Figure 6 shows the quantity of medication delivered by each nebulizer delivery system. The Vixone nebulizer equipped in the traditional manner delivered 0.53 ± 0.03 mg. The Circulaire delivered 0.32 ± 0.01 mg, which was significantly less than the nebulizer T-piece with corrugated tubing (p < 0.001). During testing it was observed that the Circulaire retained a pool of medication proximal to an internal one-way valve. Further testing was performed on the medication retained proximal to the one-way valve, and it was found to contain a mean 0.62 ± 0.05 mg of albuterol. The AeroTee delivered 0.80 ± 0.02 mg, which was significantly more than the Circulaire or the nebulizer T-piece with corrugated tubing (p < 0.001). The nebulizer T-piece with corrugated tubing treatment time was not significantly different than the Circulaire (p = 0.81) or the AeroTee (p = 0.84) (Fig. 7).
Aerodynamic Particle Size Evaluation

Figure 8 shows the measured MMAD for the three systems. The nebulizer T-piece with corrugated tubing and the AeroTee had MMADs of 2.9 ± 0.2 μm and 3.0 ± 0.2 μm, respectively, and the difference was not significant (p > 0.82). The MMAD of the Circulaire was measured to be 0.7 ± 0.1 μm and found to be significantly less than either the nebulizer T-piece with corrugated tubing (p < 0.001) or the AeroTee (p < 0.001).

Percent of Aerosol Delivered in the 1–5 μm Range

Figure 9 shows the percent of aerosol in the 1–5 μm range. The percent of aerosol in the 1–5 μm range for the nebulizer T-piece with corrugated tubing and the AeroTee were 44 ± 2.5% and 48 ± 2.0%, respectively, and the difference was significant (p = 0.024). The percent of aerosol in the 1–5 μm range for the Circulaire was 24 ± 1.7%, which was significantly less than that of the nebulizer T-piece with corrugated tubing or the AeroTee (p < 0.001).

Mass of Aerosol in the 1–5 μm Range

Figure 10 shows the mass of aerosol in the 1–5 μm range for each nebulizer delivery system. The AeroTee delivered 0.38 ± 0.01 mg of aerosol in the 1–5 μm range, which was more than the nebulizer T-piece with corrugated tubing or the Circulaire (p < 0.001). The nebulizer T-piece with corrugated tubing delivered 0.24 ± 0.02 mg of aerosol in the 1–5 μm range, whereas the Circulaire delivered only 0.08 ± 0.01 mg. The difference between all devices was significant (p < 0.001).

Volume of Rebreathed Gas Evaluation

Figure 11 shows the volumes of gas rebreathed by the simulated patient. The AeroTee and the nebulizer T-piece
**Circulaire vs AeroTee vs Traditional Nebulizer**

![Graph](image)

**Fig. 10.** Effect of nebulizer delivery system on aerosol mass delivered to the patient in the aerodynamic particle size range of 1–5 μm (respirable mass) (mean and standard deviation). Neb T = nebulizer with T-piece.

![Graph](image)

**Fig. 11.** Effect of nebulizer delivery system on rebreathed volumes delivered to the patient (mean and standard deviation). Neb T = nebulizer with T-piece.

with corrugated tubing were measured to have rebreathed volumes of 47.9 ± 3.5 mL/breath and 47.7 ± 3.1 mL/breath, respectively. The difference was not significant (p = 0.93). The volume of rebreathed gas for the Circulaire was 15.3 ± 3.4 mL/breath, which was significantly less than the AeroTee (p < 0.001) or the nebulizer T-piece with corrugated tubing (p < 0.001).

**Discussion**

This study found that the nebulizer delivery system used to deliver aerosolized medication can have a significant effect on the amount and quality of aerosol delivered to the patient. Most noteworthy is how much the AeroTee and Circulaire appear similar from casual observation, and yet the performance of each was strikingly different. The AeroTee, when used with the Vixone nebulizer, was shown to deliver almost 5 times the mass of 1–5 μm-range aerosol as the Circulaire, despite the fact that both are equipped with identical aerosol storage means. Furthermore, the MMAD of the AeroTee, when used with the Vixone nebulizer, was strikingly different than that of the Circulaire. Particularly noteworthy was the finding that the percent difference in the mass of aerosol in the 1–5 μm range and MMAD between the AeroTee and the Circulaire when used with identical nebulizers was larger than the difference for any two of the 17 nebulizers studied by Hess et al.1

The Vixone nebulizer equipped in the traditional manner delivered 0.53 ± 0.03 mg of albuterol, compared to 0.75 ± 0.02 mg as measured by Hess et al.1 In their study, Hess et al.4 used a sinusoidal waveform to simulate patient breathing that had an I:E of 1:1.5, compared to the I:E of 1:2 simulated in the present study. It is reasonable to expect an increase in inspiratory time to cause an increase in quantity of medication delivered, because less aerosol would be wasted during exhalation. When corrected for the difference in I:E, the results of Hess et al.4 and the results of this study vary only by 14.7%.

The smaller particle size of the Circulaire was a key factor in the dramatically lower mass of aerosol in the 1–5 μm range (see Fig. 5). The MMAD of 0.7 ± 0.1 μm measured in this study is not much different than the MMAD of 0.51 μm reported by the manufacturer of the Circulaire. The mass of aerosol in the 1–5 μm range is an important parameter because it represents the range of particle sizes most likely to deposit in the bronchial airways. Raabe et al.2,3 in addition to defining a respirable range of aerosol particle sizes as 1–5 μm, also published data showing that the pulmonary (alveolar) deposition fraction for 0.7 μm and 3.0 μm particles is 0.2 and 0.7, respectively. Although there is some deposition for submicron particles, the rate of deposition is three times less than for particles in the peak respirable range.

Several clinical studies have compared the Circulaire to a conventional nebulizer configuration, and all reported equal or slightly greater clinical effectiveness with the Circulaire.2,6–8 Those clinical evaluations would seem to contradict the results of this study. However, in none of those studies was the Circulaire delivery system evaluated separately from the nebulizer used, because in each case the Circulaire with a Vixone nebulizer was compared to a traditional configuration using an Airlife Misty-Neb (Baxter Healthcare, Valencia, California). Hess et al.4 found that the Airlife Misty-Neb produces approximately half as much 1–5 μm range aerosol as the Vixone. A more objective clinical evaluation of the Circulaire would have been performed had both nebulizer delivery systems been equipped with the same brand of nebulizer.

**Conclusions**

Nebulizer delivery systems have at least as great an effect on the quantity and quality of aerosol delivered to the patient as do the individual nebulizers themselves. In
this bench study, the AeroTee delivered superior performance, compared to a nebulizer T-piece with corrugated tubing, whereas the Circulaire, which is similar in form to the AeroTee, delivered less medication than a nebulizer T-piece with corrugated tubing. Further clinical research should be performed on these and other nebulizer delivery systems. When comparing different nebulizer delivery systems, it is important to conduct the work under reasonably similar circumstances, using the same or comparable nebulizers and dosing strategies.

REFERENCES


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Deadline for Submission: April 28, 2000
Combining a Positive Expiratory Pressure Device with a Metered-Dose Inhaler Reservoir System Using Chlorofluorocarbon Albuterol and Hydrofluoroalkane Albuterol: Effect on Dose and Particle Size Distributions

Joseph L Rau PhD RRT FAARC and Mervi Torniainen MS CRT

BACKGROUND: Combining a positive expiratory pressure (PEP) device with inhalation of albuterol via metered dose inhaler (MDI) may improve drug delivery to the lung, but may also affect dose availability. PURPOSE: Determine the effect of interposing a PEP device on dose availability of albuterol via MDI and reservoir with either a chlorofluorocarbon (CFC) or hydrofluoroalkane (HFA) propellant. METHODS: MDI dose availability of CFC albuterol (Proventil) and HFA albuterol (Proventil HFA) using an Aerosol Cloud Enhancer (ACE) reservoir with and without a PEP device (TheraPEP) attached was determined. Drug availability was assessed using an Andersen 8-stage cascade impactor operated at 28.3 ± 0.5 L/min. The PEP device was inserted between the reverse-firing ACE and the United States Pharmacopeia induction throat. Drug collected on impactor plates was analyzed spectrophotometrically at 276 nm, and the fine particle fraction was determined as the mass of drug < 4.7 μm. RESULTS: With CFC albuterol, total dose and drug mass < 4.7 μm (means and standard deviations) for the MDI-ACE alone were 44.4 ± 7.7 μg and 33.4 ± 2.2 μg, respectively, and for the MDI-ACE with TheraPEP were 50.1 ± 6.4 μg and 39.8 ± 14.3 μg, respectively. With HFA-albuterol sulfate, total drug and drug mass < 4.7 μm for the MDI-ACE alone, expressed as base drug, were 41.7 ± 4.2 μg and 35.2 ± 6.3 μg, respectively, and for the MDI-ACE with TheraPEP were 48.9 ± 8.0 μg and 44.2 ± 6.2 μg, respectively. There was no significant difference in dose availability between the MDI-ACE alone and with the PEP device attached (Wilcoxon signed-rank test, p > 0.05), for either CFC or HFA albuterol. CONCLUSION: Interposing the TheraPEP device at the MDI-ACE outlet does not change total dose, drug mass < 4.7 μm, or mass median aerodynamic diameter of MDI albuterol, with either CFC or HFA propellants. [Respir Care 2000;45(3):320–326] Key words: positive expiratory pressure, albuterol, metered dose inhaler, Aerosol Cloud Enhancer, TheraPEP, particle size distribution, mass median aerodynamic diameter.

Background

Positive expiratory pressure (PEP) therapy is a bronchial hygiene technique used to mobilize secretions and treat atelectasis.1–2 With PEP therapy, using either a mouthpiece or mask, the subject inspires normally, or with a greater than normal tidal volume, and then exhales actively but not forcefully against a fixed-orifice resister, generating pressures of 10–20 cm H2O, and with a 1:3 or 1:4 inspiration-expiration ratio. Application of expiratory resistance may improve airway patency, reduce airway collapse, and promote collateral ventilation.3 With the prevention of premature airway collapse, expiratory airflow may mobilize secretions toward the larger airways for cough and expectoration.4 The

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breathing maneuver has been used in asthma and chronic obstructive pulmonary disease to reduce air trapping, as well as in cystic fibrosis and chronic bronchitis to mobilize secretions. The technique has also been shown effective in preventing or reversing atelectasis.

The same physiologic effects of PEP therapy may be beneficial in optimizing aerosolized drug delivery, especially bronchodilators. Improved volumes and longer expiratory times could enhance distribution and deposition of drug in the airway. Several clinical trials have examined the effect of combining an inhaled bronchodilator and PEP therapy in both asthma and chronic obstructive pulmonary disease, with conflicting results. Both metered-dose inhalers (MDIs) with reservoir devices and small volume nebulizers were used. However, no previous study of the effect of PEP devices on aerosol particle size distribution and dose availability could be found. The present study examined the effect of interposing a PEP device on dose availability from an MDI-reservoir system, using a β agonist with either chlorofluorocarbon (CFC) or hydrofluoroalkane (HFA) propellant.

Materials and Methods

Study Design

CFC-formulated albuterol (Proventil, Key Pharmaceuticals, Kenilworth, New Jersey) and HFA-formulated albuterol sulfate (Proventil HFA, Key Pharmaceuticals, Kenilworth, New Jersey), both administered via MDI, were used as β agonists. Both types of MDI canisters were used with the Aerosol Cloud Enhancer (ACE, DHD Healthcare, Canastota, New York), a cone-shaped reverse-firing reservoir that contains an integral MDI adapter. The PEP device studied was the TheraPEP (DHD Healthcare, Canastota, New York) positive expiratory pressure system. MDI dose availability with 4 samples of the ACE reservoir were tested both with and without the TheraPEP device attached at the mouthpiece, using CFC-formulated albuterol. Four samples of TheraPEP devices were used for the testing. Three additional samples of the ACE and TheraPEP were tested similarly, using HFA-formulated albuterol sulfate. The order of testing, with or without TheraPEP, was alternated for each pair of trials to control for an order effect due to canister fullness. All canisters were new and unused at the beginning of testing. The same MDI was used in a paired fashion for the two modes of delivery (TheraPEP and no TheraPEP). No MDI was exhausted beyond 50% of capacity, as verified by a log of all actuations. Separate measures of drug delivery from the MDI alone were made for reference dose and particle size distribution.

Measurement

Measurement of particle size distributions was performed using an Andersen 8-stage cascade impactor (Andersen Instruments Inc, Smyrna, Georgia) with a United States Pharmacopeia induction throat. The cascade impactor has cut points providing cumulative drug mass < 4.7 μm as well as < 2.1 μm, which are considered to be in the range of particle sizes with a higher probability of deposition in the lower respiratory tract than larger particle sizes. The mouthpiece containing the one-way valve in the ACE was retained when testing without TheraPEP. This mouthpiece was then replaced with the TheraPEP device. The one-way inspiratory valve in the TheraPEP was retained, and the device connected directly to the outlet of the ACE. The mouthpiece of the TheraPEP was removed for connection to the impactor and no other valves were interposed between the MDI-reservoir and the impactor. The TheraPEP T-piece connecting to the pressure indicator was left in line, although the tubing connection was capped. Figure 1 shows the testing configuration of the combined MDI-ACE and TheraPEP with impactor.

Ten MDI actuations were discharged to waste before using a new MDI canister. For all dose delivery trials, a total of 5 actuations, each 30 seconds apart, were conducted, with shaking of the canister between each actuation, per package insert instructions. Two waste actuations were first discharged if an MDI had not been used for more than 4 hours prior to testing. To minimize flow variation, the impactor pump was activated just prior to the first actuation, and the impactor sampling pump was allowed to run for 20 seconds following the final actuation. A flow of 28.3 ± 0.5 L/min was maintained through the impactor, and verified using a Fischer & Porter calibrated flowmeter (ABB Automation, New Berlin, Wisconsin). Drug from each impactor stage was washed with 20 mL of 0.1 N HCl solvent, and the resulting solution analyzed using a Beckman DU 640 spectrophotometer (Beckman Instruments, Fullerton, California) at a wavelength of 276 nm. Serial dilutions of known concentrations of drug solution were measured for albuterol and albuterol sulfate, with a least squares regression line fitted to the data points. The regression equation was used to predict drug mass from the measured absorption of the samples. All measures of the HFA formulation with sulfate drug were expressed as the base equivalent, to allow direct comparison between the CFC and the HFA formulations.

Data Analysis

Differences between aerosol dose from the MDI-reservoir with and without the PEP device attached were tested using the nonparametric Wilcoxon signed-rank test, at a significance level of 0.05, because of the small number of
samples and unknown sampling distribution of the drug dose. Calculations were made with Systat 7.0 software (SPSS Inc, Chicago, Illinois). Means and standard deviations for the total drug mass delivered, drug mass < 4.7 μm and < 2.1 μm, and the mass median aerodynamic diameter (MMAD) were determined. Average particle size distributions for the MDI-reservoir with and without the PEP device attached are given for CFC-formulated albuterol and for HFA-formulated albuterol sulfate.

Results

Table 1 summarizes the results of cascade impaction testing for MDI dose availability of albuterol for the ACE reservoir alone and for the ACE with TheraPEP attached. Means and standard deviations for total mass recovered, drug mass < 4.7 μm, drug mass < 2.1 μm, and MMAD are listed. Using the Wilcoxon signed-rank test, for CFC-formulated albuterol, there was no significant difference (p > 0.05) between MDI-ACE alone and MDI-ACE with PEP in total drug mass recovered, drug mass < 4.7 μm, or drug mass < 2.1 μm. Although the difference was not significant, the MDI-ACE with PEP yielded slightly more drug on average.

Similar results were obtained for HFA-formulated albuterol sulfate, which is reported as the base equivalent in Table 1, with 100 μg of albuterol base equivalent to 120 μg of the sulfate (1 μg of sulfate = 0.833 μg base). There was no statistically significant difference in drug availability for total drug, drug mass < 4.7 μm, or drug mass < 2.1 μm between MDI-ACE alone and MDI-ACE with PEP (p > 0.05). On average, slightly more drug was recovered with the TheraPEP device attached.

Figures 2 and 3 show the average amount of drug recovered from each impactor stage, for the MDI-ACE alone, and in combination with the TheraPEP, for both CFC-formulated albuterol and HFA-formulated albuterol sulfate. Drug mass at each stage is reported as a percentage of total mass recovered.

Discussion

No difference in drug availability was detected from the MDI-ACE system when the TheraPEP device was interposed at the outlet of the ACE reservoir, in the tested configuration. Since the aerosol path is changed minimally when the PEP device is attached to the outlet of the ACE,
the distribution $< 4.7 \, \mu m$ for the CFC formulation, after averaging the MDI-ACE alone with the TheraPEP (see Fig. 2). However, with the HFA formulation, drug mass in the size range $< 4.7 \, \mu m$ was 87.6% of the total for the combined MDI-ACE and TheraPEP curves (see Fig. 3). Results were similar if the particle size distribution curves were considered separately for either the CFC or the HFA formulation, with and without the PEP device attached (see Figs. 2 and 3). When tested using either a Mann-Whitney procedure or a Student's $t$ test, the difference in percentage of mass $< 4.7 \, \mu m$ was not significant ($p = 0.37$), possibly because of the small number of samples tested and the attendant standard errors. The increase noted was in the fraction (percent) of total mass $< 4.7 \, \mu m$ rather than in the mass itself, which we interpret as improved efficiency in the available dose. When considering drug mass $< 2.1 \, \mu m$, no increase was seen. For the CFC formulation, an average of 49.3% of drug was in the size range $< 2.1 \, \mu m$, and for the HFA formulation an average of 43.6% of drug was in the size range $< 2.1 \, \mu m$. In a comparison of CFC and HFA albuterol availability with both a large and small volume holding chamber, Mitchell et al found an increase in the fine particle fraction (considered as the drug mass $< 4.7 \, \mu m$) from the pressurized MDI alone with the HFA formulation. Their study found that with the CFC formulation, 42.0 $\mu g$ (40.5%) of a total of 103.6 $\mu g$ from the MDI was in the size range $< 4.7 \, \mu m$. 

Fig. 2. Drug mass recovered at each impactor stage as a percent of total mass recovered for chlorofluorocarbon-formulated albuterol (means and standard errors). Open circles: metered-dose inhaler and Aerosol Cloud Enhancer (MDI-ACE) with TheraPEP attached. Closed circles: MDI-ACE without TheraPEP.
Fig. 3. Drug mass recovered at each impactor stage as a percent of total mass recovered for hydrofluoroalkane (HFA) formulated albuterol sulfate (means and standard errors). Open circles: metered-dose inhaler and Aerosol Cloud Enhancer (MDI-ACE) with TheraPEP attached. Closed circles: MDI-ACE without TheraPEP.

Table 1. Summary of Performance of MDI-ACE With and Without TheraPEP Device Attached

<table>
<thead>
<tr>
<th>Configuration</th>
<th>Chlorofluorocarbon-formulated albuterol (Proventil), n = 4</th>
<th>Hydrofluoroalkane-formulated albuterol (Proventil HFA), n = 3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total Mass (µg)</td>
<td>Drug Mass &lt; 4.7 µm</td>
</tr>
<tr>
<td>MDI-ACE alone</td>
<td>44.4 ± 7.7</td>
<td>33.4 ± 2.2</td>
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<tr>
<td></td>
<td></td>
<td>(76.4%)</td>
</tr>
<tr>
<td>MDI-ACE with TheraPEP</td>
<td>50.1 ± 6.4</td>
<td>39.8 ± 14.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(78.0%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(p = 0.14)</td>
</tr>
<tr>
<td></td>
<td>Hydrofluoroalkane-formulated albuterol (Proventil HFA), n = 3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total Mass (µg)</td>
<td>Drug Mass &lt; 4.7 µm</td>
</tr>
<tr>
<td>MDI-ACE alone</td>
<td>41.7 ± 4.2</td>
<td>35.2 ± 6.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(84.6%)</td>
</tr>
<tr>
<td>MDI-ACE with TheraPEP</td>
<td>48.9 ± 8.0</td>
<td>44.2 ± 6.2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(90.5%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(p = 0.29)</td>
</tr>
</tbody>
</table>

MDI-ACE = metered dose inhaler with Aerosol Cloud Enhancer.
MMAD = mass median aerodynamic diameter.
Figures are means and standard deviations.
Numbers in parentheses represent drug mass as a percent of the total mass recovered for each configuration for particle sizes <4.7 µg and <2.1 µg.
Observed probability levels (p) are for differences between the two configurations with and without the TheraPEP device attached.
*Expressed as albuterol base, 100 µg = 120 µg albuterol sulfate.
With the HFA formulation, 38.7 µg (47.6%) of a total of 81.3 µg from the MDI was in the size range < 4.7 µm. This increase in dose efficiency (ie, more of the available drug dose in the fine particle size range < 4.7 µm) was not seen when a holding chamber was added to the MDI, which is in contrast to our study. Mitchell et al used an AeroChamber and a Volumatic in their study, whereas we tested an ACE reservoir. With the AeroChamber, which is more comparable to the ACE than the Volumatic, there was an increase in both total and fine particle mass (62.0 ± 2.9 µg and 64.2 ± 3.0 µg, respectively) with the HFA compared to the CFC formulation (45.4 ± 0.3 µg and 47.2 ± 0.1 µg, respectively). However, there was no difference in the fraction of the total dose < 4.7 µm between the HFA and CFC formulations with the AeroChamber. It is possible that, in our study, design differences between the AeroChamber and the ACE (volume, spray direction, MDI actuator) accounted for the differing results in dose fraction from the reservoir with the two albuterol formulations.

Drug mass < 4.7 µm and < 2.1 µm are both given in Table 1. The particle size range < 5 µm is considered to be of interest for drugs able to reach the lower respiratory tract and has often been termed the “respirable fraction”; this is now referred to as the “fine particle fraction.” As particle size decreases to < 5 µm, there is a corresponding shift in lung deposition from more central portions of the airway to the peripheral and alveolar regions. Cutpoints provided by the Andersen cascade impactor give convenient measures of drug mass < 4.7 µm as well as < 2.1 µm, and the particles in the smaller size range (< 2.1 µm) have a high probability of peripheral lung deposition. Other than characterizing particle size ranges, data from the present study cannot translate directly into dose deposition in the human lung. The cascade impactor technique, with constant flow, does not simulate spontaneous human ventilation with varying inspiratory/expiratory flow, or tidal volume, or age or disease changes. However, data from the present study show no difference in dose availability from the MDI-ACE system when the TheraPEP device is interposed at the reservoir outlet. Establishing the dose equivalence in vitro, as we have done, offers the basis for better interpretation of clinical studies examining response to the combined PEP/aerosol therapy. Results obtained in the present study cannot be generalized to other reservoir or PEP systems or to nebulizer-PEP combination systems without additional testing.

At least three clinical trials have used MDIs and cone spacers (Nebulhaler) with PEP valves, in configurations similar to the one tested here. There is a difference in reservoir volume; the Nebulhaler has a 750 mL chamber, whereas the ACE chamber is approximately 175 mL. Two of the studies by Frischknecht-Christensen et al, of asthmatic subjects, reported conflicting results, with one showing improved bronchodilation with combined PEP and inhaled β2 agonist therapy, and the second study finding no additional improvement with PEP if sufficient doses of inhaled terbutaline were used. A third study, also by Frischknecht-Christensen et al, of chronic bronchitics, found a small but significant improvement in bronchodilation when PEP was combined with inhaled terbutaline, using the cone spacer. However another study by Frischknecht-Christensen et al, using PEP with a small-volume nebulizer, found no difference in peak expiratory flows compared to nebulized terbutaline alone in 10 patients with chronic obstructive pulmonary disease. In contrast, Andersen and Klausen found that the addition of positive end-expiratory pressure (a maneuver somewhat different than PEP) to nebulized terbutaline improved pulmonary function in severe bronchospasm. It is unfortunate that laboratory testing of drug output and size distributions was not performed prior to the clinical trials, to provide better understanding of the variable results obtained.

**Conclusions**

Combining an MDI, a reservoir chamber, and a PEP therapy device into a single aerosol/PEP treatment device is attractive in terms of time-efficiency. The present study shows that there is no change in dose availability in vitro with a bronchodilator, using either a CFC or an HFA albuterol formulation, with the combination system of MDI-ACE and TheraPEP. Clinical study of specific patient populations is needed to determine actual lung deposition and if improved bronchodilator response can be obtained with the combined system.

**ACKNOWLEDGMENTS**

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Case Reports

Spontaneous Pneumothorax and Alpha$_1$-Antitrypsin Deficiency

Reena Daniel MD and Luis Teba MD

Spontaneous pneumothorax has been observed in patients with abnormal levels of alpha$_1$-antitrypsin. We report the case of a young woman with a low level of alpha$_1$-antitrypsin who presented with recurrent episodes of spontaneous pneumothorax and who required pleuroscopy, apical lung resection, and pleurodesis. [Respir Care 2000;45(3):327-329] Key Words: alpha$_1$-antitrypsin deficiency, spontaneous pneumothorax, emphysema.

Introduction

Primary spontaneous pneumothorax (SpPTx) is believed to result from rupture of subpleural emphysematous blebs in patients without underlying pulmonary disease. The incidence of SpPTx in the general population can be as high as 9/100,000 cases per year. It occurs more commonly in men than in women, and an inherited tendency for the development of primary SpPTx has been reported.$^{2,3}$

Alpha$_1$-antitrypsin (alpha$_1$AT) is a protease inhibitor that protects the lungs from destruction by the lung neutrophilic elastases. alpha$_1$AT deficiency can cause a wide spectrum of diseases, with emphysema being the major clinical sequela.$^4$ Although an association of SpPTx with abnormal levels of alpha$_1$AT has been observed,$^{5,6}$ other studies have failed to confirm this association.$^7$

We report a case of recurrent SpPTx in a patient with a low level of alpha$_1$AT, and ZZ phenotype.

Case Summary

An 18-year-old white woman presented to the emergency room with the sudden onset of left-sided pleuritic chest pain, which developed at rest. The pain was exacerbated by inspiration and was unrelated to body movements. She denied dyspnea, cough, hemoptysis, nausea, vomiting, or fever. Medical and surgical history was unremarkable. She was not taking any medication, and denied alcohol or tobacco use. Family history was negative for lung diseases. Physical examination showed that she was not in any respiratory distress. Temperature was 36.5°C, blood pressure 120/70 mm Hg, pulse 88 b/min, and respiratory rate 16 breaths/min. Her oxygen saturation, measured via pulse oximetry, was 97% on room air. She weighed 115 pounds and was 5'4" tall. Decreased breath sounds with hyperresonance were appreciated in the left upper hemithorax, but no crackles or wheezes were heard. The rest of the physical examination was unremarkable. Serum electrolytes, cell blood count with differential, blood urea nitrogen, creatinine, and arterial blood gases were normal. Chest radiograph revealed a 10% left-sided pneumothorax, and she was treated with analgesics and 100% oxygen via face mask. By the next day there was complete resolution of the pneumothorax, and the patient was discharged home.

One week later she was readmitted with a 15% left-sided pneumothorax. A chest tube was inserted and the pneumothorax resolved. A computerized axial tomography of the chest with multiple high-resolution images through the lungs did not report any evidence of emphysema. However, two small (<1 cm) blebs were identified in the apex of the left lung. Spirometry results were within normal limits and without any indication of airway obstruction (forced vital capacity was 3.87 L, and forced expiratory volume in the first second was 3.69 L, 92% and 108% of predicted values, respectively). Serum level of alpha$_1$AT was markedly decreased (24 mg/dL, normal = 100-275 mg/dL).

Three weeks after the initial presentation, she had a third episode of left-sided pneumothorax (Fig. 1), and underwent video-assisted thoracoscopic. Apical blebs were observed and a wedge resection of the apex of the left lung

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and mechanical pleurodesis were performed. Microscopic examination of the specimen found some enlarged, thin-walled air spaces, with fragmented alveolar septa protruding from the walls of the lesions into the air space. The final pathology diagnosis was emphysema.

The \( \alpha_1 \)-AT level was rechecked, and was again markedly low (32 mg/dL). Phenotyping of \( \alpha_1 \)-AT revealed ZZ variant. Familial screening found the patient's brother to have the MZ phenotype.

**Discussion**

The relationship between \( \alpha_1 \)-AT deficiency and emphysema was first described in 1963 by Laurell and Eriksson.\(^8\) \( \alpha_1 \)-AT deficiency is the result of a gene defect in the long arm of chromosome 14. \( \alpha_1 \)-AT deficiency is more common in people of European descent, and it has been estimated that there are 20,000 Americans with \( \alpha_1 \)-AT deficiency.\(^9\) Although most of the estimated cases are asymptomatic, enrollees in the national registry of \( \alpha_1 \)-AT deficiency are symptomatic, and 97% of the enrollees have the ZZ variant.\(^10\) Furthermore, patients with \( \alpha_1 \)-AT deficiency may remain unidentified as such because they may have been misclassified as having asthma or cigarette-induced lung disease. The average time from onset of symptoms to diagnosis of \( \alpha_1 \)-AT deficiency has been reported to be 7.2 years.\(^1\)

The initial complaints of patients with \( \alpha_1 \)-AT deficiency are usually dyspnea, wheezing, with or without upper respiratory infections, and cough. Patients with \( \alpha_1 \)-AT deficiency appear to have increased risk of pneumonia.\(^2\) Presentation can also mimic or be associated with bronchial asthma, bronchectasis, or chronic bronchitis. The chest radiograph may initially be normal but it may also present bibasilar hyperlucency and bullous changes.\(^3\) Lung function can be preserved in \( \alpha_1 \)-AT deficiency patients, but smokers have a greater decline in forced expiratory volume in the first second, compared to nonsmokers.\(^4\) Emphysema is usually observed in \( \alpha_1 \)-AT deficiency patients during their fourth or fifth decade of life. Diagnosis of \( \alpha_1 \)-AT deficiency is confirmed by measuring \( \alpha_1 \)-AT plasma level. Normal levels in persons with MM, MS, and SS phenotypes are 150–350 mg/dL. A severe deficiency, which most frequently includes the ZZ and null/null variants, is defined by a plasma level of < 40 mg/dL.\(^5\)

SpPTx is not described as one of the clinical manifestations of \( \alpha_1 \)-AT deficiency. We reviewed all cases of primary SpPTx seen at our institution in the years 1995–1998, and an \( \alpha_1 \)-AT level (normal) was obtained in only one of the 27 patients in addition to the one reported here. A previous report identified an SpPTx in a 43-year-old black patient who had an intermediate form of deficiency of the MZ genotype,\(^5\) and Pawłowiez et al found two patients with partial \( \alpha_1 \)-AT deficiency in a group of 56 cases of SpPTx.\(^6\) In contrast, Lenler-Petersen et al obtained normal \( \alpha_1 \)-AT level in 8 patients with primary SpPTx,\(^5\) and Bense et al did not find \( \alpha_1 \)-AT deficiency in 27 non-smoking patients with history of SpPTx.\(^7\)

In conclusion, we presented a case of SpPTx with \( \alpha_1 \)-AT deficiency. Did the deficiency in \( \alpha_1 \)-AT result in the development of SpPTx or, on the contrary, was the association of these two entities anything but mere coincidence? Although this potential association has been observed and suggested in previous reports, it has not been confirmed. The true frequency of \( \alpha_1 \)-AT deficiency among patients with primary SpPTx needs to be studied so that the occurrence of anything beyond the chance association can be better evaluated.

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Spontaneous Pneumothorax and Alpha₁-Antitrypsin Deficiency

Smoking Cessation in Hospitalized Patients

Michael T Halpern MD PhD, Jordana K Schmier MA, Kenneth D Ward PhD, and Robert C Klesges PhD

Background

Background

Cigarette smoking is a significant contributor to morbidity and premature mortality in the United States, responsible for over 400,000 deaths annually, 20% of all deaths in the United States.1 Smokers face a higher relative risk of cancer as well as cardiovascular and respiratory disease.1 In addition, smoking-related illnesses impose substantial economic burdens; smoking-attributable costs for medical care in the United States are estimated to be $50.0 billion (1993 data), with over half of that cost being hospital expenditures.2 Though two thirds of the 45 million adult smokers have expressed interest in quitting,1 the addictive nature of cigarette smoking makes it challenging to quit.

A number of studies have considered the advantages of using inpatient hospital stays for low-impact cessation interventions.4 Hospitalization of a smoker represents a “teachable moment,” an optimal chance to present a smoking cessation intervention. Patients are generally not allowed or able to smoke while in the hospital, enforcing a period of cessation. Further, they are likely to be ill or recovering from illness; thus, they may be more amenable to interventions that will improve their health. Hospitalized patients also come in contact with a broad range of health care professionals who can deliver or reinforce smoking cessation interventions.

Many studies have described or evaluated smoking cessation programs. Most successful programs address multiple components of smoking behavior, and in evaluating smoking cessation programs it is useful to have a framework for categorizing program components. Ward et al6 published such a framework, identifying 4 components that smoking cessation programs should address: the demographics and smoking history components include characteristics such as age, gender, ethnicity, psychological dependence on nicotine, previous quit attempts, and diagnosis of smoking-related disease; biological components include withdrawal symptoms, fear of weight gain, and cigarette cravings; intrapersonal components encompass

References

self-efficacy, beliefs about the risk of smoking, and depression; and situation/interpersonal components include environmental exposure, access to tobacco, and social support. These components, both individually and in conjunction, categorize the interventions that form most smoking cessation programs and ultimately contribute to successful smoking cessation outcomes. Using the 4-component framework, we reviewed the literature regarding smoking cessation in hospitalized patients.

Literature Review and Study Selection

Published studies included in this review were identified using the MEDLINE computerized database and references from identified studies. Inclusion criteria were similar to those used by the Agency for Healthcare Research and Quality (formerly called the Agency for Health Care Policy and Research) in developing its smoking cessation guidelines: only randomized, controlled trials published in English in peer-reviewed journals between 1975 and 1998 were included. Additionally, each study reported on cessation outcomes at least 5 months after the initial intervention. Eleven studies met these criteria (Table 1).

Factors Associated with Smoking Cessation among Hospitalized Patients

Not surprisingly, the 4 factors associated with smoking cessation in other populations also characterize influences on cessation in hospitalized patients. Demographics and smoking history, generally recognized as strong predictors of cessation success, are difficult to examine in hospitalized populations. Although numerous studies have delineated the sociodemographic characteristics and smoking history of their participants, the narrow age range and presence of multiple comorbidities constrain variance and make analysis difficult. One study identified age as a predictor of cessation among hospitalized patients; smokers over the age of 65 were more likely to quit than younger smokers.16 However, many of the reviewed studies excluded patients over the age of 70,6,7,14,15 whereas others showed a tendency for older patients to have higher quit rates, but were not powered to detect differences.10 Some studies have found no demographic or clinical variables predictive of quitting behavior in this population; in several studies, neither age nor gender were predictors of quitting.8,11,12 and the three studies that reported race found race not to be predictive of cessation outcome.11,15,16 Current understanding suggests that these factors are generally less important in the hospitalized population than in a general population of smokers. However, one patient-based aspect is clearly important: smoking-related diagnosis. This strong predictor of cessation is discussed below.

Biological components, such as experiencing nicotine withdrawal symptoms, affect cessation rates in the general population. However, among hospitalized patients this component does not appear to play a substantial role in successful cessation programs. For example, the introduction of nicotine replacement therapy (NRT) has not significantly affected quit rates. Miller et al8 permitted the use of nicotine gum or patch for participants who met criteria for nicotine dependence or who had severe withdrawal symptoms. There was no significant difference between the quit rate of the NRT group and the no NRT group. Lewis et al8 described a study of three groups (nicotine patch, placebo patch, and no NRT), and reported similar outcomes among the groups. One program found that craving for a cigarette at baseline was a significant (p = 0.02) predictor of validated smoking cessation at 6 months, independent of sociodemographic factors and length of stay.11 More research is needed regarding the importance of this component in smoking cessation among hospitalized patients.

Intrapersonal components are clearly important predictors of success in hospitalized patients. Increased self-efficacy, demonstrated by high adherence to lifestyle changes and rehabilitation programs, is associated with cessation.14 Confidence in one’s ability to quit is a predictor of success (p < 0.05).11,16 In addition, knowledge of increased health risks involved with smoking is a precursor to success.17-19 Discussion of smoking risk among patients with ischemic heart disease appears to be an important factor in cessation outcomes.17 Smokers understand their higher risk of heart attack, cancer, and stroke compared to nonsmokers, but they still tend to underestimate their risk.18 Perkins19 hypothesizes that a smoker’s myocardial infarction (MI) experience facilitates abstinence through the certain recognition of the health risks of continued smoking. This relationship between knowledge of health risks and quitting success has also been shown in pregnant women attending prenatal hospital care.20 These factors are particularly important to the hospitalized smoker, exceptionally so if the hospitalization is because of a smoking-related diagnosis.

Finally, situational/interpersonal components also appear to be predictive of cessation among hospitalized patients. Both increased social support and reduced exposure to smoking cues have been associated with higher quit rates,19 although neither were significant predictors of cessation outcomes in the articles reviewed. The restrictions on smoking in hospitals and difficulty in reaching smoking areas are also important for cessation in this population. There was a nonsignificant trend for patients subject to a smoking ban to quit in higher rates than those permitted to smoke while in the hospital.7 It is difficult to evaluate the specific situational aspects of smoking cessation programs...
## Table 1. Literature Summary

<table>
<thead>
<tr>
<th>Author, Patient Population</th>
<th>Components of Intervention(s)</th>
<th>Length of Follow-up/ Method of Confirmation</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>DeBusk et al. 1994^6 Post-myocardial infarction patients 79% male</td>
<td>Special intervention (nurse-initiated telephone contacts, computer-generated mailed progress reports, counseling, relapse prevention manual) (n = 293) Usual care (n = 292)</td>
<td>12 months Cotinine levels</td>
<td>At baseline, 43% of the patients were smokers. At 6 months, no significant difference in quit rates between intervention group and usual care group. At 12 months, significantly more quitters in intervention group (70% vs 53%, p = 0.03). Special intervention group also received nutritional counseling and lipid-lowering drug therapy.</td>
</tr>
<tr>
<td>Joseph et al. 1993^7 Patients undergoing substance abuse treatment 100% male</td>
<td>Intervention (hospital smoking ban, drug and nicotine dependency treatment) (n = 92) Control (smoking permitted in designated areas) (n = 105)</td>
<td>8-21 months after completing treatment Self-report</td>
<td>Over 80% of patients were smokers at time of hospitalization. No significant difference between groups in proportion of patients who reported smoking “more,” “less,” or “the same” since hospital admission. Quit rates: intervention group 60%, control group 66%.</td>
</tr>
<tr>
<td>Lewis et al. 1998^8 Patients who expressed interest in quitting smoking upon admission 54% male</td>
<td>MC—Minimal care (physician counseling) CAP—Counseling plus active nicotine patch (physician counseling, extended bedside and phone counseling) CPP—Counseling plus placebo patch</td>
<td>6 months Self-report, confirmed by expired breath carbon monoxide</td>
<td>No statistically significant difference in quit rates between treatment groups. Biochemically confirmed abstinence at 6 months was MC 4.9%, CAP 9.7%, CPP 6.5%. Patients hospitalized with a respiratory diagnosis were more likely to quit than were patients with other conditions (p &lt; 0.00001).</td>
</tr>
<tr>
<td>Miller et al. 1997^9 Patients hospitalized for a variety of conditions: 32% cardiovascular disease, 12% pulmonary, 28% other internal medicine 51% male</td>
<td>Minimal intervention (nurse-mediated counseling and one post-discharge telephone contact) (n = 460) Intensive intervention (nurse-mediated counseling and 4 post-discharge telephone contacts) (n = 560) Usual care (n = 942) Nicotine replacement therapy (NRT) offered to patients who met the criteria for nicotine dependence</td>
<td>12 months Self-report, confirmed by plasma cotinine levels or family member report</td>
<td>Higher quit rates in intensive (27%) and minimal (22%) intervention than in usual care (20%) (p = 0.009, intensive vs usual care). No significant difference between intensive and minimal intervention, nor between minimal intervention and usual care. Odds of cessation higher for patients with cardiovascular disease, internal medical conditions, or pulmonary disease than for patients hospitalized with other conditions.</td>
</tr>
<tr>
<td>Pederson et al. 1991^10 Respiratory disease patients: 43% chronic bronchitis, 57% emphysema 70% male</td>
<td>Treatment group (initial and follow-up in-hospital counseling, self-help manual) (n = 37) Usual care (n = 37)</td>
<td>6 months Self-report, validated by carboxyhemoglobin analysis</td>
<td>Quit rates were similar in both groups at 6 months (treatment 33.3%, control 21.4%).</td>
</tr>
<tr>
<td>Rigotti et al. 1997^11 Newly-defined medical and surgical patients 55% male</td>
<td>Intervention (counseling, self-help materials, chart prompt for cessation reminders, up to 3 post-discharge phone calls) (n = 325) Usual care (n = 325)</td>
<td>6 months Self-report, validated by saliva cotinine levels</td>
<td>At 6 months, no statistically significant differences between quit rates (intervention 8.1%, usual care 8.7%). At 1 month, intervention group had higher quit rates (22.3% vs 16.1%). At 6 months, among patients who had never tried to quit before, intervention appeared to lead to significantly higher quit rate (intervention 15.3%, usual care 37%: p = 0.01).</td>
</tr>
<tr>
<td>Simon et al. 1997^12 Post-surgery patients 98% male</td>
<td>Multicomponent intervention (in-hospital counseling, videotape, self-help literature. NRT, 3-month phone follow-up) (n = 168) Self-help literature and brief counseling (n = 156)</td>
<td>12 months Self-report, validated by serum or saliva cotinine levels</td>
<td>Quit rate higher in intervention group, both as measured by biochemical assay (intervention 15%, comparison 8%; p = 0.04) and by self-report (intervention 27%, comparison 13%; p &lt; 0.01).</td>
</tr>
</tbody>
</table>
SMOKING CESSATION IN HOSPITALIZED PATIENTS

Table 1. Continued

<table>
<thead>
<tr>
<th>Author, Patient Population</th>
<th>Components of Intervention(s)</th>
<th>Length of Follow-up/Method of Confirmation</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stevens et al, 1993</td>
<td>Intervention (in-hospital counseling, videotape, self-help literature, follow-up telephone call)</td>
<td>12 months</td>
<td>Abstinence at both 3-month and 12-month assessments showed significantly higher quit rate in the intervention group (13.5%) than in the usual care group (9.2%, p = 0.023).</td>
</tr>
<tr>
<td>Newly-admitted patients 38% male</td>
<td>(n = 666)</td>
<td></td>
<td>At 26 weeks, patients in the training group had quit at rate similar to patients without exercise training (training 31%, no training 39%), but training patients reported significantly fewer cigarettes per day (11 vs 22 cigarettes per day; p &lt; 0.03).</td>
</tr>
<tr>
<td>Taylor et al, 1988</td>
<td>Training (treadmill testing and home exercise, treadmill testing and group exercise) (n = 107)</td>
<td>6 months</td>
<td>Significantly higher quit rate in the intervention group, both at the 12-month assessment (71% vs 45%; p = 0.003) and considering sustained abstinence (at both 3-month and 12-month assessment; 65% vs 35%; p = 0.024).</td>
</tr>
<tr>
<td>Post-myocardial infarction patients</td>
<td>No training (treadmill testing only, control) (n = 53)</td>
<td></td>
<td>Significantly higher confirmed quit rate in the intervention group (31% vs 21%, p = 0.006). Age and confidence to quit were significant (p &lt; 0.05) predictors of success.</td>
</tr>
<tr>
<td>Percent male not reported</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Taylor et al, 1990</td>
<td>Nurse-managed intervention (in-hospital counseling, manual, audio tapes, follow-up phone calls)</td>
<td>12 months</td>
<td></td>
</tr>
<tr>
<td>Post-myocardial infarction patients 86% male</td>
<td>(n = 86)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Usual care (n = 87)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Taylor et al, 1996</td>
<td>Nurse-managed intervention (counseling, videotape, relaxation audiotope, workbook, NRT offered to patients who met criteria for nicotine dependence, post-discharge phone contacts) (n = 315)</td>
<td>12 months</td>
<td></td>
</tr>
<tr>
<td>Patients admitted for a variety of conditions 55% male</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Usual care (n = 313)</td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

among hospitalized patients, because all such patients are in the same situation (ie, a hospital).

Impact of Smoking-Related Diagnosis on Smoking Cessation

The impact of smoking-related diagnosis on smoking cessation has been extensively evaluated among hospitalized patients. Patients with smoking-related diagnoses routinely have higher quit rates than patients with smoking-unrelated diagnoses. For example, one study comparing three interventions (including one with nicotine replacement therapy) found no significant difference in quit rates. However, patients with respiratory diagnoses quit in higher rates than all other patients, regardless of treatment group assignment. Another study, one that did find differences in quit rates among three interventions, also found that patients with pulmonary disease quit in high rates, regardless of treatment group assignment. Another nurse-managed year-long study found that patients hospitalized for all smoking-related conditions quit in greater numbers than patients with smoking-unrelated conditions, regardless of the type of cessation intervention.

Cardiac disease patients represent an especially recept-
interested in quitting than smokers in a general medical unit.\textsuperscript{9,24}

Because of the greater impact of smoking cessation programs among cardiac patients, it has been suggested that smoking cessation should be a standard component of hospitalization for MI patients.\textsuperscript{25} Essential elements of the cessation program include prohibiting smoking throughout the hospital, beginning the intervention before discharge, including physician advice as well as counseling from other health care providers, and integrating motivational and skill-based programs.\textsuperscript{25} It is clear that the health benefits of cessation after MI are substantial.\textsuperscript{26–29}

**Limitations on Smoking Cessation Interventions for Hospitalized Patients**

There are certain populations in which in-hospital smoking cessation programs may not be appropriate. Requiring a smoke-free inpatient unit may present a problem for patients undergoing psychiatric care, as nicotine withdrawal can exacerbate symptoms of psychiatric illness, such as anxiety or depression.\textsuperscript{30} There are also concerns that smoking cessation programs may interfere with substance abuse treatment. However, limited empirical evidence suggests that hospital-based cessation programs may improve quit rates for patients in substance abuse programs\textsuperscript{31} and do not appear to interfere with treatment for other substance abuse disorders.\textsuperscript{7}

**Relapse Prevention**

Unfortunately, many patients who stop smoking while hospitalized relapse following discharge. Burling et al\textsuperscript{21} reported that although one third to one half of MI patients quit smoking or decrease their level of smoking, over one third resume smoking within 5 days. This high level of relapse may reflect the extrinsic motivation provided by the situational components (smoke-free policies and other restrictions) within hospitals; such motivation is not likely to be as successful for cessation as is intrinsic motivation more related to intrapersonal components.\textsuperscript{32}

Given the large percentage of smokers who quit for only a short period of time following hospital discharge, the inclusion of relapse prevention techniques is an important factor in smoking cessation programs for this population. Two year-long studies with similar follow-up components (printed literature and frequent nurse-initiated telephone calls), found significantly higher quit rates at 12 months in the follow-up intervention groups than in the usual care groups.\textsuperscript{13,16} Miller et al\textsuperscript{9} also examined intensive follow-up compared with minimal intervention or usual care, and found significantly higher quit rates at one year among patients receiving intensive intervention and follow-up. Interventions that included little follow-up or relapse prevention focus have not shown improvements in long-term quit rates.\textsuperscript{9,10}

**Costs of Cessation Programs for Hospitalized Patients**

Krumholz et al\textsuperscript{33} assessed the cost-effectiveness of a smoking cessation program for post-MI patients using a decision tree, with resource and efficacy parameters based on the program described by Taylor et al.\textsuperscript{15} The program consisted of a nurse-managed program including counseling, self-help materials, and post-discharge telephone calls for 5 months. Their results indicated that this smoking cessation program is highly cost-effective, with an estimate of $220 per year of life saved. Sensitivity analysis confirmed that the program remains cost-effective (less than $20,000 per year of life saved) if the success rate is as low as 0.3% or if the program costs as much as $8,840 per patient. Another cost-effectiveness analysis of a hospital-based cessation program consisting of counseling, self-help materials, and up to two post-discharge telephone calls, found that the incremental cost per life-year saved by the intervention was $3,697, based on a 5% discount rate and an incremental quit rate of 4.3%, much lower than the incremental costs of commonly accepted interventions.\textsuperscript{34}

**Recent Advances**

Although many of the studies addressed in this review included NRT, NRT was provided exclusively in the form of a patch or gum. Other forms of NRT are now available, including nasal spray and inhaler. Hughes et al\textsuperscript{35} provide a thorough summary of advantages of each method. In addition, bupropion hydrochloride is a nicotine-free pharmacotherapy available for cessation. Sustained-release bupropion, in dosages of ≥ 150 mg, significantly improves smoking cessation rates, compared with placebo, in the short term (7 weeks) and long term (one year).\textsuperscript{36} It is likely, suggest Hughes et al,\textsuperscript{35} that patients will try one of the methods available over the counter (gum, patch), and consult their physicians only after failure with those methods.

**Recommendations for Clinical Practice**

No hospitalized smoker should be discharged without receiving smoking cessation information. Although several of the studies reviewed found no significant differences between interventions and usual care (often operationalized only as provision of cessation literature), admission to the hospital in itself may promote readiness to quit by emphasizing that smoking has health consequences, as well as by surrounding the smoker with health care providers and enforcing abstinence during the hospitalization. This is confirmed by the higher rate of smoking...
cessation among hospitalized patients than among the general population. The opportunity presented by hospitalization must not be squandered.

Results from smoking cessation interventions in this population show high short-term quit rates but common relapse following hospital discharge. An important determinant of intervention success described in these articles was the amount and length of follow-up. With long-term follow-up and relapse prevention, interventions have been much more successful. Cessation interventions may begin in the hospital, but successful programs emphasize relapse prevention.

These studies have identified that the 4 components of smoking cessation in hospitalized patients are similar to those of the general smoking population. Demographics, smoking history, and intrapersonal components are important predictors of success in cessation. As more is learned about these components, information on predictors of smoking cessation, especially predictors in specific subgroups of smokers, should be considered in designing future cessation programs.

Combined therapies should also be considered; studies of these therapies have not been reported among hospitalized patients, but the results among other groups of smokers are promising. Jorenby et al. found higher 12-month quit rates among patients receiving both sustained-release bupropion and a nicotine patch than among patients receiving either one of the pharmacotherapies singly (combined treatment vs bupropion only, not significant; combined treatment vs patch, p < 0.001). Similarly, the use of nicotine nasal spray with nicotine patch was shown to increase quit rates compared with a patch alone over a 6-year follow-up period. Though earlier studies cautioned against the use of multiple pharmacotherapies, combined therapies are now often prescribed. We suggest, and we concur, that combined pharmacotherapies be considered routinely for hospitalized smokers.

**Recommendations for Further Research**

All of these studies except one presented biochemically-confirmed abstinence rates. Of studies also presenting self-reported quit rates, these rates were, as expected, substantially higher than confirmed quit rates. These findings emphasize the importance of including biochemical verification of smoking status in research. Hospitalized smokers, who are likely to return for follow-up medical care, present an especially convenient opportunity for biochemical validation of self-reported cessation.

Several studies have indicated that smoking-related diagnoses, such as respiratory or cardiovascular disease, significantly impact cessation. Studies enrolling patients with a variety of conditions must be careful to consider this in randomization strategies. Specifically, cessation interventions enrolling patients with both smoking-related and smoking-unrelated diagnoses should evaluate cessation rates in the groups separately as well as pooled.

Some of the studies considered drop-outs in power calculations, but it is not clear that all did so. Particularly in hospitalized patients and in those with life-threatening illness, it is important not to underestimate the proportion deceased or otherwise lost to follow-up before the final evaluation. The planned number of enrolled patients must be large enough to account for this drop-out rate, which is likely to be greater than that of the general population.

**Summary**

The components of readiness to change for smoking cessation that are found in the general population are also applicable to hospitalized smokers. Smoking cessation interventions must be specifically tailored to subgroups among hospitalized patients, with emphasis on smoking-related diagnosis when applicable. Interventions should include key components related to smoking cessation, such as knowledge, self-efficacy, exposure to smoking, and social support. Interventions that include relapse prevention and are conducted in the context of other risk reduction strategies should be developed.

**REFERENCES**


Should This Patient Use Supplemental Oxygen During Commercial Air Flight?

James K Stoller MD

Case Summary

A 65-year-old former smoker with stable chronic obstructive pulmonary disease (COPD) lives in a Midwestern city (20 feet above sea level) and reports to you that he plans to fly to his daughter’s wedding in California. He has no history of anemia, coronary artery disease, or stroke, and he is eucapnic. Table 1 shows the results of his spirometry (without prior administration of a bronchodilator).

Although he does not require supplemental oxygen currently, he asks whether you recommend his using oxygen while he is traveling aboard the airplane to the wedding.

Discussion

Because commercial air travel by COPD patients is common,1-5 pulmonary clinicians are often called upon to evaluate patients regarding their need for supplemental in-flight oxygen. Though available data suggest that the incidence of adverse medical occurrences during commercial travel is low (ie, approximately 1/30,000 travel-days),1,5,6-8 current recommendations suggest prescribing supplemental in-flight oxygen when the patient’s predicted arterial partial pressure of oxygen (P\textsubscript{aO\textsubscript{2}}) during flight is ≤ 50 mm Hg. In turn, predicting whether the patient’s in-flight P\textsubscript{aO\textsubscript{2}} will be ≤ 50 mm Hg requires knowledge of the physiology of commercial airflow and of available prediction methods. Specifically, by Federal Aviation Administration requirements, the cabin pressurization must maintain the cabin altitude at ≤ 8,000 feet equivalent during commercial air flight. Indeed, actual measurement of cabin altitude during commercial air flight on different airplane types suggests variation in the true cabin altitude to levels generally between 5,000 and 7,000 feet equivalent, but uniformly ≤ 8,000 feet.

Various techniques exist for estimating in-flight P\textsubscript{aO\textsubscript{2}} so as to allow a clinical decision about recommending in-flight supplemental oxygen. The three available techniques include: (1) actual measurement of the patient’s P\textsubscript{aO\textsubscript{2}} during so-called hypobaric hypoxia (eg, during actual ascent to altitude or while inside a hypobaric chamber),9,10 (2) measurement of the patient’s P\textsubscript{aO\textsubscript{2}} while breathing a hypoxic gas mixture3,11 (so-called hypoxic hypoxia), and (3) use of regression equations that predict the P\textsubscript{aO\textsubscript{2}} at 8,000 feet based on the patient’s sea level resting room air P\textsubscript{aO\textsubscript{2}} and spirometry values.1,3,5,8,9,11

Of these three available techniques, actual measurement of P\textsubscript{aO\textsubscript{2}} during hypobaric hypoxia is usually confined to research settings or to special facilities where hypobaric chambers are available for clinical purposes. Far more commonly, the in-flight P\textsubscript{aO\textsubscript{2}} is predicted by using regression equations or by measuring the patient’s P\textsubscript{aO\textsubscript{2}} while breathing a hypoxic gas mixture made to simulate the partial pressure of oxygen in the airplane cabin.

Specifically, two techniques have been proposed for breathing hypoxic gas mixtures. The first involves inhal-

Table 1. Results of Spirometry and Room Air Blood Gas (Close to Sea Level) in a 65-Year-Old with Stable Chronic Obstructive Pulmonary Disease

<table>
<thead>
<tr>
<th>Test</th>
<th>Predicted</th>
<th>Measured</th>
<th>% Predicted</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEV\textsubscript{1} (L)</td>
<td>3.41</td>
<td>1.43</td>
<td>42</td>
</tr>
<tr>
<td>FVC (L)</td>
<td>4.41</td>
<td>3.70</td>
<td>84</td>
</tr>
<tr>
<td>FEV\textsubscript{1}/FVC</td>
<td>0.81</td>
<td>0.39</td>
<td>48</td>
</tr>
<tr>
<td>P\textsubscript{aO\textsubscript{2}} (mm Hg)</td>
<td>—</td>
<td>62</td>
<td>—</td>
</tr>
<tr>
<td>P\textsubscript{aCO\textsubscript{2}} (mm Hg)</td>
<td>—</td>
<td>40</td>
<td>—</td>
</tr>
<tr>
<td>pH</td>
<td></td>
<td>7.41</td>
<td>—</td>
</tr>
</tbody>
</table>

Predicted = mean predicted values as per Crapo et al.1 (standard error of estimate is 0.486 for FEV\textsubscript{1} for males). P\textsubscript{aO\textsubscript{2}} = arterial partial pressure of oxygen. P\textsubscript{aCO\textsubscript{2}} = arterial partial pressure of carbon dioxide. FVC = forced vital capacity. FEV\textsubscript{1}/FVC = ratio of FEV\textsubscript{1} to FVC.
ing a 15.1% oxygen mixture from a cylinder, which simulates the ambient partial pressure of oxygen at 8,000 feet.\textsuperscript{3,9–12} Using this technique, the patient inspires a hypoxic gas mixture through a tight-fitting face mask, so as to avoid entraining room air (with fraction of inspired oxygen 20.9%). As an alternative technique, Vohra and Klocke\textsuperscript{13} determined that approximately 16% oxygen can be produced by the patient’s breathing from a 35% air-entrainment mask through which nitrogen (instead of oxygen) is bled. Entrainment of room air into this nitrogen stream creates a hypoxic gas mixture that is 16.1–16.5% oxygen, thereby allowing estimation of the $P_{aO_2}$ under conditions of cabin altitude exposure to approximately 6,700 feet.\textsuperscript{13}

Using either of these techniques, the patient is asked to inhale the hypoxic mixture to a steady state, after which an arterial blood gas sample is obtained. If $P_{aO_2}$ is < 50 mm Hg, supplemental oxygen can be prescribed.\textsuperscript{5,5,11,12} For patients not requiring supplemental oxygen on room air at sea level, available data suggest that a flow of 2 L/min of supplemental oxygen usually suffices during commercial air flight.\textsuperscript{13}

As an alternative to actually measuring the $P_{aO_2}$ while the patient breathes a hypoxic gas mixture, regression equations can be used to predict the $P_{aO_2}$ at cabin altitude. As demonstrated by Dillard et al, measurements necessary to predict the $P_{aO_2}$ at 8,000 feet include spirometry measurements (ie, forced expiratory volume in the first second [FEV\textsubscript{1}] or the percent of predicted FEV\textsubscript{1}, and the ratio of FEV\textsubscript{1} to forced vital capacity) and a room air resting $P_{aO_2}$ at sea level.\textsuperscript{10–12} The available regression equations (Table 2) were determined in patients with eucapnic COPD, and the spirometry measurements were performed without prior bronchodilator administration. As such, these equations should be applied only to the type of patients included in the studies from which the equations were derived, and under the same testing conditions. Specifically, patients to whom the equations apply have stable eucapnic COPD and lack other comorbidities (eg, coronary artery disease or cerebral vascular disease). Do not apply the equations to patients with hypercapnia or pulmonary diseases other than COPD, for which validated equations are not currently available.

In the patient described above, use of Equation 4 from Table 2\textsuperscript{11} allows the following calculation (based on a room air $P_{aO_2}$ at sea level of 62 mm Hg and an FEV\textsubscript{1} of 42% of predicted):

$$P_{aO_2,8000} = 0.294 \times (62) + 0.086 \times (42) + 23.211$$

$$P_{aO_2,8000} = 45.05 \text{ mm Hg}$$

Notably, although the weight of evidence suggests that room air sea level $P_{aO_2}$ and spirometry measurements are both important independent variables,\textsuperscript{10–12} use of Equation 3 (in which the only independent variable is the $P_{aO_2}$ on room air at sea level) produces a very similar estimate, 43.04 mm Hg.

Because the predicted in-flight $P_{aO_2}$ is below 50 mm Hg, supplemental in-flight oxygen is recommended. In general, 2 L/min of oxygen in-flight will suffice to maintain adequate arterial oxygen saturation.\textsuperscript{14} Actual measurement of the required liter flow can be performed by measuring an arterial blood sample taken while the patient breathes supplemental oxygen (at the specified liter flow) in a hypobaric chamber or while the patient breathes a hypoxic gas mixture.

REFERENCES

A 72-Year-Old Smoker with Interstitial Lung Disease

Omar A Minai MD and Eugene J Sullivan MD

Case Summary

The patient is a 72-year-old white female ex-smoker (40–50 pack-years) with a medical history notable for coronary artery disease, status post coronary artery bypass grafting in 1988. She was referred for evaluation of progressive dyspnea on exertion over a two year period. She would have to stop after walking approximately half a block, more from leg claudication than dyspnea. She was able to climb one flight of stairs. There were no significant complaints of cough, fevers, night sweats, or weight loss. She did not keep pets. On examination she was in no apparent distress. Her oxygen saturation on room air was 98%. Her lung examination revealed crackles over the lower one third of the lung fields bilaterally. Early clubbing was noted. A chest roentgenogram showed bilateral increased interstitial markings with lower lobe predominance. There were bullous changes in the right upper lobe. A recent dobutamine echocardiogram showed no ischemia and good left ventricular systolic function. Table 1 shows the pulmonary function test results.

What is your diagnosis?

Discussion

The most striking feature of the pulmonary function test results is the severely reduced diffusing capacity in the presence of normal spirometry and only mildly reduced total lung capacity.

Decreases in the diffusing capacity for carbon monoxide ($D_{LCO}$) can be produced by a wide variety of clinical disorders and may reflect any of the following physiologic processes, either alone or in combination: (1) compromise of the pulmonary vasculature (eg, thromboembolic disease), (2) alteration or destruction of the normal alveolar architecture (eg, pulmonary fibrosis or emphysema), (3) loss of lung parenchyma (eg, surgical resection), (4) mismatching of ventilation and perfusion, or (5) abnormally low oxygen carrying capacity (anemia). As a matter of routine practice, certain “corrections” are commonly made in order to help separate these processes. Correction for the patient’s measured hemoglobin will help to eliminate the effect of anemia on the measure. Further, if the $D_{LCO}$ is reduced because of loss of lung parenchyma, correction for simultaneously measured lung volume will not normalize the measure.

When the $D_{LCO}$ does not normalize on correction for hemoglobin and lung volume (as in this case), one must attempt to determine which physiologic process is operative. Measures of lung volume can help. When lung volumes are elevated, particularly in association with evidence of spirometric air flow limitation, emphysema is the most common cause of reduced diffusing capacity per unit of alveolar volume. When lung volumes are reduced, the most common cause is diffuse parenchymal lung disease (eg, fibrosis). When lung volumes are normal, the most common cause is pulmonary vascular disease.

In this case, were the clinical information not available, one might be inclined to attribute the physiologic abnormality to pulmonary vascular disease. However, the smoking history, crackles on physical examination, and chest radiograph abnormalities suggest other processes. Given these clues, the pulmonary function test results are most

Table 1. Pulmonary Function Test Results

<table>
<thead>
<tr>
<th>Test</th>
<th>Predicted</th>
<th>Measured</th>
<th>% Predicted</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC (L)</td>
<td>2.31</td>
<td>2.17</td>
<td>94</td>
</tr>
<tr>
<td>FEV₁ (L)</td>
<td>1.89</td>
<td>1.61</td>
<td>85</td>
</tr>
<tr>
<td>FEV₁/FVC</td>
<td>0.78</td>
<td>0.70</td>
<td>90</td>
</tr>
<tr>
<td>FRC (L)</td>
<td>2.47</td>
<td>1.85</td>
<td>75</td>
</tr>
<tr>
<td>RV (L)</td>
<td>1.91</td>
<td>1.24</td>
<td>65</td>
</tr>
<tr>
<td>TLC (L)</td>
<td>4.41</td>
<td>3.26</td>
<td>74</td>
</tr>
<tr>
<td>$D_{LCO}$ (mL/mm Hg/s)</td>
<td>16.74</td>
<td>3.85</td>
<td>23</td>
</tr>
<tr>
<td>$D_{LCO}/N_{A}$</td>
<td>4.33</td>
<td>1.17</td>
<td>27</td>
</tr>
</tbody>
</table>

Predicted = mean predicted values as per Crapo et al
FVC = forced vital capacity
FEV₁ = forced expiratory volume in the first second.
FEV₁/FVC = ratio of FEV₁ to FVC
FRC = functional residual capacity.
RV = residual volume.
TLC = total lung capacity by helium dilution.
$D_{LCO}$ = Diffusing capacity for carbon monoxide.
$D_{LCO}/N_{A}$ = ratio of diffusing capacity to alveolar volume.

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consistent with a combined disorder. The combination of obstructive physiology due to emphysema and restrictive physiology due to pulmonary fibrosis contributes to the disproportionate decrease in $D_{LCO}$ relative to lung volumes. A computed tomography scan of the chest showed evidence of both apical bullous disease and basal fibrosis.

It is well known that emphysema patients have decreased pulmonary parenchymal elastic recoil, increased resistance to air flow, increased lung volumes, and decreased $D_{LCO}$. In contrast, patients with idiopathic pulmonary fibrosis (IPF) have increased elastic recoil, decreased lung volumes, normal or supra-normal air flow parameters, and decreased $D_{LCO}$. Because of the opposing effects on lung compliance, the concurrent existence of these two disorders may result in relatively normal overall lung compliance. This may result in normal measures of air flow and lung volumes. However, because both disorders result in diffusion impairment, the $D_{LCO}$ will be significantly reduced.

Approximately 60–70% of IPF patients are current or former smokers.2,3 Schwartz et al4 found evidence of air trapping (increased residual volume and functional residual capacity) in their IPF patients who were smokers. Further, the ratio of the forced expiratory volume in the first second to the forced vital capacity was not related to past smoking in those IPF patients. They postulated that IPF and emphysema may exert opposite physiologic effects on both lung compliance and airway function.3 Data from Hanley et al5 support the idea that smoking alters the lung compliance characteristics of IPF patients. These authors found that in smokers with IPF, pressure-volume curves are shifted upwards and to the left, compared to nonsmokers with IPF, indicating larger lung volumes at a given transpulmonary pressure. They also found that the maximal transpulmonary pressure was lower in IPF patients who had a history of smoking.

In conclusion, we can say that patients with mixed obstructive and restrictive parenchymal disease may have severe functional limitation despite relatively preserved lung volumes and normal spirometry. The finding of an "isolated" reduced $D_{LCO}$ may indicate pulmonary vascular disease, but in the appropriate clinical setting may indicate a mixed disorder. High-resolution computed tomography of the chest may also be helpful in such cases.6

REFERENCES


This comprehensive textbook of pediatric respiratory care medicine provides an in-depth discussion of the expansive field of respiratory medicine. The book promises a pediatric emphasis with an international flavor. It is organized into 14 main parts and 81 chapters. The 14 main parts attempt to group related topics, but the logic of the grouping and order is sometimes confusing.

The first 4 parts include general, applied physiology, assessment, and therapeutic principles. These parts lay the groundwork for later sections, which are disease-specific. When we received this book, we eagerly attacked the first parts for pediatric-specific discussions of respiratory physiology. This topic is given only passing coverage in other textbooks. However, since this is a pediatric textbook, we felt certain that the long awaited answers to our questions would be found here. To our disappointment, we found varying levels of coverage. The initial chapters on molecular genetics, developmental anatomy and physiology, and lung cell biology were excellent. However, the chapters on host defense systems mechanisms, acute lung injury and repair, and applied clinical respiratory physiology were lacking. They were good reviews but offered only a glancing (if any) discussion of pediatric and neonatal issues. We understand that page space is limited in a textbook this size, and that there are limited pediatric data available, but this is a pediatrics textbook, so the differences between children and adults should have been addressed.

The next part of the book, “Assessment,” addresses a wide range of techniques used in physical diagnosis. The first chapter in this part, “Clinical Assessment and Diagnostic Approach to Common Problems,” was especially thorough in addressing the many techniques used in basic physical assessment of pediatric patients. It very nicely emphasized the importance of the art of physical assessment, which sometimes is too readily replaced with new technologic means of diagnosis. Technologic advances in diagnosis are not to be undervalued, but physical assessment must never be overlooked. This point is well made by the coverage of more advanced assessment techniques in the next chapter, “Imaging of the Respiratory System.” This chapter gives a comprehensive overview of the use of several types of imaging over the entire respiratory tract. We appreciated the next two chapters discussing pulmonary function testing in infants and pediatrics. It is a good reference tool for those not involved on a daily basis with pulmonary function tests but who need a good understanding of the limitations and information available in that patient population. “Gas Exchange and Acid-Base Physiology” is another chapter in this part, and provides an excellent background and review for anyone wishing to learn more about this subject or just needing to refresh his or her knowledge base. It included not only the obvious blood gas interpretation section, but also covered capnography and pulse oximetry saturation interpretation.

The following parts address issues of therapeutic principles and disease-specific discussions. The “Therapeutic Principles” section has some excellent discussions of pharmacology, aerosol therapy, chest physiotherapy (CPT), and lung transplantation. The chapter on CPT is timely and well written. It has an important section entitled “Scientific Basis of CPT,” which begins “Over two decades ago, a conference on the scientific basis of CPT was opened by the statement that CPT lacks an established scientific basis and evidence of lasting clinical benefit.” It then goes on to offer a complete discussion of the physiologic basis and recent studies of CPT. This is a chapter that should be copied and handed out to every new house officer, as well as to many attendings. The chapter on assisted ventilatory support and oxygen therapy, however, is dated and does a poor job of discussing this important topic. This chapter demonstrates a general weakness of the entire textbook, and that is its discussion of mechanical ventilatory support. The discussions of ventilators are lacking and very dated. These may be passable for someone wanting only the most rudimentary understanding of ventilators, but beyond that, they are inadequate. Again, we were sure the authors were limited by space, but offered dated materials in an area that is evolving quickly. The remaining chapter, on home care, is good but should be expanded.

The next part discusses respiratory insults and intensive care. To a pediatric intensivist (MJH), this section was of great interest. We recognized that this was not a critical care textbook, but we looked forward to well written summaries. We were only slightly disappointed. The first chapter, “Lung Trauma: Toxic Inhalation and ARDS,” offered a nice review of lung trauma and toxic inhalation. Even though it lacked pediatric focus, it gave a nice general overview of the topic. However, the section on acute respiratory distress syndrome was poor. Not only did it offer very little pediatric data, it was wrong. It stated that “mortality rates in pediatric patients were 50%” with morbidity in the survivors substantial.” Neither of these assertions is correct, though they are frequently repeated in textbooks.

The authors owe it to the readers to offer the latest information, even if some of the information is preliminary. The remaining chapters in this part include respiratory failure, foreign body aspiration, aspiration syndromes, respiratory effect of anesthesia and sedation, and drug-induced pulmonary disease. All of these chapters are well written and are good reviews.

The remainder of the textbook is dedicated to respiratory disease-specific issues in pediatrics. These are the strengths of the book and are the reason we would recommend it to a wide range of readers. The first part includes a section on respiratory disorders of the neonate and infants. The remaining sections are disease-specific, including infectious, immunologic, cardiopulmonary and pulmonary vascular disorders, asthma, cystic fibrosis, sudden infant death syndrome, structural and mechanical abnormalities, and miscellaneous disorders. These topics make up the mainstay of a pediatric pulmonologist’s practice, and are timely and well presented.

Each of the remaining chapters is detailed and offers a wealth of pediatric information. Some reorganization might improve its readability as a textbook. For instance, there is an excellent chapter on fluid balance in the developing lung, but it should be
moved to the part on respiratory disorders of the neonate and infants. After reading this chapter, the reader would be better prepared to understand the pathophysiology of neonatal lung disease.

The remaining chapters each read as authoritative discussion of their topics. However, there are still some shortcomings that will need to be addressed in future editions. For example, the part on asthma offers an excellent presentation of all the outpatient aspects of asthma. What is lacking is a detailed discussion of inpatient asthma care, including intensive care. Even though in some institutions this may be delegated to the intensivist rather than to the pulmonologist, it still requires a detailed discussion.

This book is very good despite its shortcomings. The quality of illustrations is excellent. The radiographs are well reproduced and findings are clearly illustrated. The price is in line with other textbooks of this quality. We would recommend this book for anyone who wants to expand his or her knowledge of respiratory medicine in pediatrics. It would be an excellent resource for medical students, residents, fellows, and attendings. Respiratory therapists and nurses could make this an excellent addition to their libraries, but will have to go to other resources for some respiratory care issues such as ventilator principles and management. The authors and editors should be complimented on an excellent textbook.

REFERENCES


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Current data indicate that the population over age 60 is growing fast and that this growth will accelerate in the next several decades, with the aging of the baby boom generation. The percentage of people over age 85 (the old old) is currently the fastest growing segment of the aged population. Because they are also heavy users of health services, the need for health care practitioners who are knowledgeable about and caring toward elderly people is critical.

This book on gerontology and geriatrics focuses on respiratory care. It provides a good introduction to the aging process and the clinical management and treatment of older persons by respiratory therapists. The intended readership includes college educators, those who provide continuing education, and respiratory therapists, nurses, and other health care practitioners who deal with many elderly patients, such as those working in home care or in nursing homes.

Each of the 8 chapters has these sections: key terms, learning objectives, introduction, case studies, summary, review questions, and references. This format furthers the coauthors’ stated purpose for the book, that it be used as a text for a freestanding course, as part of another course, or for a continuing education program.

Chapters 1 and 2 examine the social and psychological aspects of the aging process and the best practices in dealing with cognitively impaired older adults.

Chapter 3 addresses the biological causes of aging and includes the pulmonary changes associated with the aging process and their implications. Chapter 4 examines cardiac, cerebrovascular, and restrictive lung diseases as well as tuberculosis and cancer. Chapter 5 covers pharmacology, including common drug reactions that affect the elderly and why polypharmacy is prevalent in this population. Chapter 6 examines the acute cardiovascular and acute respiratory disease diagnoses, hypothermia, and treatment of postoperative complications.

Chapter 7 discusses issues of long-term care (both at home and within institutions) and the signs of failure to thrive among older institutionalized patients, and describes predictors for and signs of elder abuse. Chapter 8 provides a discussion on the end of life. As respiratory therapists, our job is to maintain life. This chapter describes death and the termination of life support, but unfortunately does not discuss or even set the parameters of the ethical problems associated with termination of life support.

I find this book a good introductory text for both respiratory therapy educators and practitioners, and really any health care professionals who work with elderly patients. It is easily read and provides pertinent information about the aged that one does not normally get in a respiratory therapy educational program. This type of information is a must in today’s society and will be increasingly important as our elderly population becomes larger.

The main concern I have is with the references, many of which date back to the 1960s. One wonders if more current, more refined data could not be found. For example, on Page 178, Sudnow (1976) is used to support the comment that “very poor and very unattractive patients were the least likely to get much interpersonal care.” Is there not more current data about this and other predictors of interpersonal care?

In summary, the authors provide a good basic text dealing with elderly patients. Principally for respiratory therapy practitioners, the book is also appropriate for any health care practitioner who deals with elderly people. The authors did a fine job making the reader sensitive to elderly people and the stressful health situations they may face.

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The Manual of Pleural Procedures, by Colt and Mathur, is an excellent addition to the pocket paperbacks that are so popular among medical trainees. Although I think it will be most useful to internal medicine residents or pulmonary fellows who are learning how to do pleural procedures, the information presented would also be useful to respiratory therapists, nurses, and other technical staff who may be involved in support of someone performing these procedures. It is an excellent reference for how to prepare for, how to perform, how to document, and the supplies needed for performing thoracentesis, closed-needle pleural biopsy, fine-needle aspiration biopsy, closed-tube thoracostomy, pleurodesis, and thoracotomy.
The book is broken into two parts: “Background” and “Pleural Procedures.” The background section starts out with a concise review of the anatomy and physiology of the thoracic cage and pleura, followed by a chapter on physical examination of the chest. These chapters are very readable, with useful illustrations and pictures that complement the text. The next two chapters cover radiology and ultrasonography of the pleura. These are short but pithy. The next chapter, on thoracic drainage devices, should be required reading for anyone participating in the care of a patient with a chest tube. The commercially available drainage systems are explained and some very practical advice is given, such as what to do if the drainage device tips over. I found the next two chapters (on skin degerming, disinfection, sterilization, and pharmacologic agents) the least useful, but our lead respiratory technician and our nurse who is in charge of managing the procedure suite and conscious sedation, respectively, found these chapters very helpful. The last chapter in the background section is a “Primer of Differential Diagnosis of Pleural Diseases.” I was impressed by how much information the authors managed to cram into 10 pages of text. This would be good quick reading material to brush up on before rounds and dazzle the attending.

The remainder of the book is devoted to step-by-step instructions on how to perform the procedures listed above. Each chapter is broken down into clear sections, including definition of the procedure, indications, contraindications, equipment, techniques, and complications. The techniques section is further broken down regarding what to do before, during, and after the procedure. A unique feature of the post-procedure section is a description of what should be included in the ideal procedure note. Each chapter has a collection of clinical pearls and a group of questions with answers called “Heard on the Wards.” These are very practical points such as “How do I avoid a dry tap?” or “Is there a rule of thumb for selecting chest tube size?” Each chapter ends with an annotated bibliography of recommended reading. A very helpful bonus in each chapter is a table (called a procedural guideline) that distills the important points of each procedure into a user-friendly diagram. I could envision house officers photocopying these pages to put in their “pocket brains” to help remind them of the important tasks involved in performing these procedures on the ward.

The technique sections are very easy to follow, and most of the procedures (except for thoracoscopy) could be performed by skilled persons with little other instruction than reading the book. I was impressed by the inclusion of many newer products, including the Argyle-Turkel safety needle for thoracentesis and the Tru-Close Thoracic Vent for pneumothorax. A conspicuous product that was missing was the Raja needle for closed-needle pleural biopsy. In my own practice I have found this needle to be easier to use and more reliable than either the Cope or Abrams needle. Another product that was absent and deserves mention is the PleuRx pleural catheter, which is used for long-term drainage of malignant effusions on an outpatient basis. This frequently leads to spontaneous sclerosis of the pleural space, without the hospital stay or pain that usually marks palliative chemical sclerotherapy.

The entire text is loaded with real-life practical instruction that should make performing pleural procedures fun. I only disagreed with the authors in two instances. On page 108, in the description of Abrams needle biopsy technique, the text reads “The needle is inserted through a small stab incision into the pleural space using a gentle rotating forward movement.” This does not adequately describe the amount of muscular effort it takes to get an Abrams needle to pop into the chest. Most of the fellows I have taught to do this procedure were surprised by how hard they had to push. Then, on page 158, under “Complications of Bedside Pleurodesis,” the statement, “Pain is uncommon and can usually be alleviated by prophylactic administration of analgesics and sedatives,” is misleading. Despite use of intrapleural lidocaine and large doses of intravenous narcotics and sedatives, patients may have severe pain, which was once described to me as “a fire inside!” I have routinely used midazolam for its amnestic properties so that at least they don’t remember experiencing it.

The authors state in the preface, “The purpose of this manual is to give physicians a practical, step-by-step guide of various techniques and procedures to diagnose and treat patients with pleural disease.” I believe they have achieved that purpose, and I highly recommend the book to internal medicine residents and pulmonary fellows. In addition, physicians who are putting together a service to perform pleural procedures should get this book and share it with the respiratory therapists, technicians, and nurses who are part of the team who make the care we give our patients so great.

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The author of this book is the Battalion Chief for the Brevard County Public Safety Department, Rockledge, Florida. The book is designed to be an introductory electrocardiography text for the student health care provider. The book will be useful to all health care professionals who need to assume responsibilities as advanced cardiac life support (ACLS) providers. The text may be appropriate for prehospital first responders to 911 calls, such as emergency medical technicians (EMTs) and public safety workers, because the author covers cardiovascular anatomy, the electrocardiogram (ECG), and normal sinus rhythm, assuming very little prior knowledge. However, the remainder of the book is appropriate for all health care professionals who need to complete ACLS training (eg, paramedics, registered respiratory therapists, and registered nurses specializing in critical care). The text will not replace the American Heart Association ACLS textbook because it does not cover the treatment of the dysrhythmias discussed. ACLS instructors encourage students to treat the patient and not the ECG, but beginning students and clinicians often have a great deal of difficulty sorting out the ECGs of their patients. In the short space of 116 pages the author systematically covers the major dysrhythmias seen with monitoring and 12-lead ECGs. The text proceeds in a logical manner, with separate chapters on cardiovascular anatomy, the ECG, normal sinus rhythm, ectopy, sinus and atrial dysrhythmias, junctional dysrhythmias, heart blocks, ventricular dysrhythmias, pacemaker rhythms, limitations of the monitoring lead, recognition and interpretation, and the 12-

*The views expressed herein reflect only the views of the author and are not the official views of the Department of the Army or the Department of Defense.
lead ECG. The text has an extensive glossary, index, 471 practice monitoring ECG rhythms with answer key, 100 practice 12-lead ECG rhythms with answer key, and 21 flash cards with primary differential criteria and the interpretation on the back of each flash card. The anatomical drawings of the heart in Chapter 1 are among the best I have encountered, and the figures throughout the book enhance the material discussed. The text makes good use of boxes to present tables that summarize information. The text accompanying the photographs of several popular monitor-defibrillators makes for interesting reading. The ECGs shown in the figures are large and printed without distortion or fading, which makes it easier for the reader to understand the dysrhythmia under discussion. Each chapter begins with a list of objectives, key terminology, and an interesting quotation. Margin comments liberally spread throughout each chapter successfully reiterate the most important principles discussed. A summary and self-study questions appear at the end of each chapter. The reader will also be able to make good use of the glossary, index, practice ECGs, and flash cards. The practice ECGs are a valuable resource for students who need to practice ECG interpretation in preparation for ACLS Mega-Code evaluations.

The writing is clear, with nothing taken for granted. The author has used his experience as a teacher of electrocardiography to anticipate questions students usually ask when electrocardiography concepts are presented for the first time. It is also quite clear from the organization of the material that the text was written by a“master” teacher. For example, tables in Chapter 11 organize dysrhythmias by bradyarrhythmia and pacemaker location, normocardia (rate 60–100) and pacemaker location, tachycardia and pacemaker location, rate alone, regularity (regular/irregular), PR ratios (P:R = 0:1; P:R = 1:1; P:R > 1:1), PR interval (normal, elongated, irregular), QRS interval (wide, normal, absent). Chapter 11 also includes the Wiedenhold algorithm, which organizes dysrhythmias by primary differential criteria and serves as a creative alternative to the widely used American Heart Association ACLS algorithms.

The second edition expands the text to include a chapter on diagnostic 12-lead ECG in the prehospital and emergency department setting. This chapter does a good job of explaining the difference between a monitoring ECG and a 12-lead diagnostic ECG. The author devotes space to explaining the electrical action of the leads, using the Einthoven’s triangle with figures showing vector analysis of limb leads with anatomical reference. The remainder of Chapter 12 covers axis orientation, Q waves, ST segment changes, T wave changes, electrocardiographic signs of infarction, localizing infarction, infarction analysis by lead, evolution of the myocardial infarction, and ECG evidence of other conditions. This chapter, along with the 100 practice 12-lead ECGs, will take a student a long way toward understanding this complex topic. The author should replace the limited number of references in this text with a bibliography for each chapter.

In summary, if you teach ACLS or electrocardiography, this text deserves a place on your bookshelf. Although you may not decide to make this a required text for your course, it definitely should be listed in your syllabus as a recommended reference. Beginning health care students learning ECG recognition and interpretation will consider the 571 practice ECGs and flash cards worth the price of the book, and the 12 well-written chapters will be a bonus.

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Cardiac Intensive Care is a reference textbook devoted to coronary intensive care medicine. A full one half of the book is devoted to coronary artery disease diagnosis, treatment, and complications. The second half covers noncoronary cardiac diseases such as congestive heart failure, dysrhythmias, hypertension, pericardial disease, and pulmonary embolism. The book concludes with chapters on pharmacologic agents and diagnostic and therapeutic techniques. This text is truly a cardiac intensive care unit (CCU) reference and therefore is best suited for medical professionals working in that setting. However, the concise, clear, well-referenced chapters, replete with data-derived tables, make it a highly valuable resource for the medical/surgical ICU as well.

The strength of the book lies in its numerous tables and figures that highlight the textual content and provide for rapid data acquisition for the busy practicing intensivist. This is typified in Chapter 4, which covers the criteria for admission to the cardiac ICU. Tables highlight clinical and electrocardiograph (ECG) prognostic criteria, and a data-derived flow diagram outlines risk prediction. This is followed by a handful of chapters covering cardiac, coronary, and coagulation system pathophysiology, and then 19 chapters on coronary artery disease. Chapter 12 covers the diagnosis of myocardial infarction and includes tables covering sensitivity and specificity of clinical symptoms and ECG abnormalities. Full 12-lead ECGs cover specific infarct localization, including right-sided ECGs for the diagnosis of right ventricular infarction. I was impressed to find a table with the criteria for diagnosis of myocardial infarction in the setting of left bundle branch block. Though comprehensive data are presented on biochemical markers of myocardial infarction, there is little information on the effects of renal failure on troponin levels or the clinical approach to patients with low-level troponin release.

All the chapters were comprehensive yet concise. For example, Chapter 13, on thrombolysis, included an algorithm for the approach to the bleeding patient. A chapter on right ventricular infarction provided ECGs, pulmonary artery catheter data and tracings, and excellent treatment algorithms. The second half of the textbook covers noncoronary disease such as congestive heart failure, sudden death, pacemaker placement (complications and troubleshooting), valvular disease (again providing pulmonary artery catheter waveforms, radiographs, and tables), and pericardial disease. The chapter on pericardial disease shows classic pulmonary artery catheter tracings for tamponade versus constriction. The final chapters cover a number of diagnostic and therapeutic procedures, such as pulmonary artery catheters (falls short here on providing representative tracings and interpretive pitfalls), intra-aortic balloon counterpulsation, ventricular assist devices, and a very comprehensive airways section that includes difficult airway management (laryngeal mask airway, transtracheal jet ventilation, and algorithms).

In summary, this textbook presents a wealth of data packaged into concise, well-written chapters, fully supported by summary charts and illustrative figures. It will be a very useful reference and educational textbook for cardiac disease in the ICU.

Mark T Gladwin MD
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Nebulizer Mask. PARI Respiratory Equipment Inc introduces a new nebulizer mask called BUBBLES THE FISH™. The company says that the new device is now available to help deliver aerosol medicine to children with asthma or other respiratory diseases. PARI describes the new mask as a good alternative to the "press-and-breath" MDI method because its front-loading design minimizes medication waste, and treatments take only 7-8.5 minutes, compared to 10-15 minutes with traditional nebulizers. For more information from PARI Respiratory Equipment, circle number 182 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Pulse Oximeter and CO₂ Detector. Nonin Medical Inc announces the release of the 9840 Series Pulse Oximeter and CO₂ Detector. According to Nonin, the 9840 Series combines proven pulse oximetry technology with innovative CO₂ detection, eliminating the need for two devices. The 9840 Series, Nonin says, is designed specifically for short-term monitoring and is ideal for patient transport and emergency applications. For more information from Nonin Medical, circle number 183 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Blood Glucose Data Management System. AVL Medical Instruments announces its DataCare™ Critical Care Information Management System now provides point-of-care (POC) connectivity and data management functionality for blood glucose instrumentation. According to AVL, DataCare is compatible with all POC blood glucose testing instruments, regardless of manufacturer, as long as the instrumentation has a method for connectivity. Company literature says this system will enable hospital laboratory managers to oversee one data management system for near patient blood gas, electrolytes, and glucose testing. For more information from AVL Medical Instruments, circle number 184 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Obstructive Sleep Apnea Therapy System. Respironics Inc has introduced the BiPAP Duet LX, a full-featured bi-level therapy system. The company says this new therapy system can provide more personalized treatment for obstructive sleep apnea (OSA) patients ideally suited for adult patients who have difficulty complying with CPAP therapy. According to Respironics literature, this system improves therapy compliance with the Auto-Trak Sensitivity™. For more information from Respironics, circle number 185 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/
AARC & AFFILIATES

April 7-9 — Franklin, Tennessee
The TSRC convention will be held at the Marriott Cool Springs just outside of Nashville. Topics include asthma management, ECMO, nitric oxide ventilation, a mock trial, fast-track weaning protocols, and home care topics. Contact: Patti Joyner, RRT, CCM, at (901) 725-7100, ext. 3101, Pjoyner@mmcc-tlc.com.

April 13-14 — Orange Beach, Alabama
The ASRC is hosting a clinical conference at the Hilton Garden Inn. Topics include therapist-driven protocols, patient management, noninvasive positive pressure ventilation in the acute care setting, pharmacology, and mechanical ventilation. Eight CRCE hours have been requested. Contact: Bill Pruitt at (334) 434-3405 or wprritt@jaguarl.usouthal.edu.

April 13-14 — King of Prussia, Pennsylvania
The Pennsylvania Society for Respiratory Care presents their Third Annual Eastern Regional Conference and Exhibition at the Holiday Inn. “Diversity, Dimension, Design 2000” will feature speakers William J. Malley, MS, RRT, and AARC President Gary W. Kauffman, MPA, CHE, RRT. Ten CEUs are pending. Contact: Angie Herstine at (609) 784-0340, or Ann Cusano at (215) 646-7300, ext. 428.

April 17-19 — Bismarck, North Dakota
The North Dakota Society for Respiratory Care presents their annual educational symposium at the Radisson Inn. “On the Horizon of Respiratory Care” will offer 12 CRCE hours and will feature speakers Heidi Heitkamp; North Dakota Attorney General James Fink, MS, RRT; George Gaebler, MSEd, RRT; and Trish Blakely, RRT. Contact: For more information, contact Mike Runge at (701) 530-8556.

April 26-28 — Indianapolis, Indiana
Region II for Respiratory Care will hold its 27th annual program at the Hyatt Convention Center. The conference will feature psychiatrist Dr. Clifford Kuhn (alias “The Laugh Doctor”), Dr. James Stoller, Dr. Neil MacIntyre, and Vijay Deshpande. Topics on pediatrics, home care, and management will be included; and an exhibit hall will feature the latest respiratory care technology and resources. Contact: For information, call (800) 691-3041, Mailbox #1, or www.bright.net/~dsibb/reg2rc.htm.

April 28 — Erie, Pennsylvania
The Pennsylvania Society for Respiratory Care presents their 19th annual Northwest District Educational Seminar and Equipment Exhibition at the Quality Inn. Contact: Sue Sheakley at (814) 452-5406 or sshmekley@svhs.org for more information.

May 5 — San Antonio, Texas
The University of Texas Health Science Center at San Antonio, in conjunction with the TSRC (Alamo District) and Wilford Hall Medical Center, announce the 5th Annual Respiratory Care Symposium to be held at The University of Texas Health Science Center at San Antonio. Topics include the state of the profession, newer modes of mechanical ventilation, care of the adult asthmatic, pediatric assessment, COPD, mechanical ventilation of the neonate, shock/truma, and pediatric asthma. Six CRCE credits provided. Contact: UTHSCSA, Department of Respiratory Care-MSC 6248, 7703 Floyd Curl Dr., San Antonio, TX 78229-3900, (210) 567-8850.

May 9-12 — Grand Rapids, Michigan
The Michigan Society for Respiratory Care will host their Annual Spring Conference at the Grand Center/Amway Grand Plaza Hotel. Contact: For more information, call (517) 336-7570 or visit their web site at www.michiganrc.com.

May 15-17 — Southington, Connecticut
The CTSRC and the Connecticut Thoracic Society will co-host their spring conference, “Branching Forth, Reaching Up, Speaking Out,” at the Aqua Turf Club. Speakers include Richard Branson, Dr. James Stoller, and AARC President-Elect Carl Wiezalis. Sessions will cover topics on pulmonary rehab, sleep disorders, and critical care. CRCE credits for RTs, CE credits for physicians, and CEU credits for nurses are being applied for. Contact: Frank Salvatore for more information at (860) 827-1958, ext. 5706, or access www.ctsrc.org.

May 19 — Brainerd, Minnesota
The Minnesota Society for Respiratory Care host their Spring Fling — “Rev It Up” at the Breezy Point Resort. Five CRCEs will be requested. Contact: For more information, contact Laurie Tomaszewski at (651) 232-1922, Carolyn Dunow at dunowc@fhpcare.com, or Carl Mottram at motttram.carl@mayo.edu.

May 21-22 — Spokane, Washington
The Respiratory Care Society of Washington will hold their 27th Annual Pacific Northwest Regional Respiratory Care Conference at Cavanaugh’s Inn at the Park. Topics will include current government issues and respiratory care, high frequency, and open lung strategies. Contact: For more information, contact Larry Knisley at (509) 921-6560, Garth Arkell at (509) 924-1197 or e-mail arkel4@gateway.net.

September 20-22 — Rochester, Minnesota
The Minnesota Society for Respiratory Care host their 31st Annual Fall State Conference — “Too Hot to Handle.” Contact: For more information, contact Laurie Tomaszewski at (651) 232-1922, Carolyn Dunow at dunowc@fhpcare.com, or Carl Mottram at motttram.carl@mayo.edu.

Other Meetings

May 19—21 — Atlanta, Georgia
Children’s Healthcare of Atlanta- Egleston will sponsor “Moving ECMO into the New Millennium” — SEECMO 2000, the 10th Annual Southeastern ECMO Conference, at the Grand Hyatt Buckhead. Presentations will cover adult ECMO, ECMO flow direction, CVVH techniques, and hands-on water drills. Contact: Micheal Heard, RN, at (404) 315-2593 or micheal.heard@choa.org.
CALL FOR ABSTRACTS

RESPIRATORY CARE • OPEN FORUM 2000

The American Association for Respiratory Care and its science journal, RESPIRATORY CARE, invite submission of brief abstracts related to any aspect of cardiorespiratory care. The abstracts will be reviewed, and selected authors will be invited to present posters at the OPEN FORUM during the AARC International Respiratory Congress in Cincinnati, Ohio, October 7-10, 2000. Accepted abstracts will be published in the August 2000 issue of RESPIRATORY CARE. Membership in the AARC is not required for participation. All accepted abstracts are automatically considered for ARCF research grants.

SPECIFICATIONS—READ CAREFULLY!

An abstract may report (1) an original study, (2) the evaluation of a method, device or protocol, or (3) a case or case series. Topics may be aspects of adult acute care, continuing care/rehabilitation, perinatology/pediatrics, cardiopulmonary technology, or health care delivery. The abstract may have been presented previously at a local or regional— but not national—meeting and should not have been published previously in a national journal. The abstract will be the only evidence by which the reviewers can decide whether the author should be invited to present a poster at the OPEN FORUM. Therefore, the abstract must provide all important data, findings, and conclusions. Give specific information. Do not write such general statements as “Results will be presented” or “Significance will be discussed.”

ESSENTIAL CONTENT ELEMENTS

Original study. Abstract must include (1) Background: statement of research problem, question, or hypothesis; (2) Method: description of research design and conduct in sufficient detail to permit judgment of validity; (3) Results: statement of research findings with quantitative data and statistical analysis; (4) Conclusions: interpretation of the meaning of the results.

Method, device, or protocol valuation. Abstract must include (1) Background: identification of the method, device, or protocol and its intended function; (2) Method: description of the evaluation in sufficient detail to permit judgment of its objectivity and validity; (3) Results: findings of the evaluation; (4) Experience: summary of the author’s practical experience or a lack of experience; (5) Conclusions: interpretation of the evaluation and experience. Cost comparisons should be included where possible and appropriate.

Case report. Abstract must report a case that is uncommon or of exceptional educational value and must include (1) Introduction: relevant basic information important to understanding the case. (2) Case Summary: patient data and response, details of interventions. (3) Discussion: content should reflect results of literature review. The author(s) should have been actively involved in the case and a case-managing physician must be a co-author or must approve the report.

FORMAT AND TYPING INSTRUCTIONS

Accepted abstracts will be photographed and reduced by 40%; therefore, the size of the original text should be at least 10 points. A font like Helvetica or Times makes the clearest reproduction. The first line of the abstract should be the title in all capital letters. Title should explain content. Follow title with names of all authors (including credentials), institution(s), and location; underline presenter’s name. Type or electronically print the abstract single spaced in one paragraph on a clean sheet of paper, using margins set so that the abstract will fit into a box no bigger than 18.8 cm (7.4") by 13.9 cm (5.5"), as shown on the reverse of this page. Insert only one letter space between sentences. Text submission on diskette is allowed but must be accompanied by a hard copy. Data may be submitted in table form, and simple figures may be included provided they fit within the space allotted. No figure, illustration, or table is to be attached to the abstract form. Provide all author information requested. Standard abbreviations may be employed without explanation; new or infrequently used abbreviations should be spelled out on first use. Any recurring phrase or expression may be abbreviated, if it is first explained. Check the abstract for (1) errors in spelling, grammar, facts, and figures; (2) clarity of language; and (3) conformance to these specifications. An abstract not prepared as requested may not be reviewed. Questions about abstract preparation may be telephoned to Linda Barcus at (972) 406-4667.

Early Deadline Allowing Revision. Authors may choose to submit abstracts early. Abstracts postmarked by February 29, 2000 will be reviewed and the authors notified by letter only to be mailed by March 31, 2000. Rejected abstracts will be accompanied by a written critique that should, in many cases, enable authors to revise their abstracts and resubmit them by the Final Deadline (April 28, 2000).

Final Deadline. The mandatory Final Deadline is April 28, 2000 (postmark). Authors will be notified of acceptance or rejection by letter only. These letters will be mailed by July 12, 2000.

Mailing Instructions. Mail (Do not fax!) 2 clear copies of the completed abstract form, diskette (if possible), and a stamped, self-addressed postcard (for notice of receipt) to:

2000 RESPIRATORY CARE OPEN FORUM
11030 Ables Lane
Dallas TX 75229-4593

Submit your OPEN FORUM abstract electronically
visit www.rcjournal.com
### RESPIRATORY CARE OPEN FORUM 2000 Abstract Form

1. Title must be in all upper case (capital) letters, authors’ full names and text in upper and lower case.
2. Follow title with all authors’ names including credentials (underline presenter’s name), institution, and location.
3. Do not justify (ie, leave a ‘ragged’ right margin).
4. Do not use type size less than 10 points.
5. All text and the table, or figure, must fit into the rectangle shown. (Use only 1 clear, concise table or figure.)
6. Submit 2 clean copies.

Mail original & 1 photocopy (along with postage-paid postcard) to:

**2000 RESPIRATORY CARE OPEN FORUM**
11030 Abies Lane
Dallas TX 75229-4593

*Early deadline is February 28, 2000 (postmark)*

*Final deadline is April 28, 2000 (postmark)*

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### Presenter

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### Corresponding Author if Different from Presenter

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American Association for Respiratory Care

MEMBERSHIP APPLICATION

Please read the eligibility requirements for each of the classifications in the right-hand column, then complete the applicable section. All information requested below must be provided, except where indicated as optional. See other side for more information and fee schedule. Please sign and date application on reverse side and type or print clearly. Processing of application takes approximately 15 days.

☐ Active
  Associate
  ☐ Foreign
  ☐ Physician
  ☐ Industrial
  ☐ Special
  ☐ Student

Last Name ____________________

First Name ____________________

Social Security No. ____________________

Home Address ____________________

City ____________________

State __________ Zip ____________________

Phone No. ( ________ )

Primary Job Responsibility (check one only)

☐ Technical Director
☐ Assistant Technical Director
☐ Pulmonary Function Specialist
☐ Instructor/Educator
☐ Supervisor
☐ Staff Therapist
☐ Staff Technician
☐ Rehabilitation/Home Care
☐ Medical Director
☐ Sales
☐ Student
☐ Other, specify ____________________

Type of Business

☐ Hospital
☐ Skilled Nursing Facility
☐ DME/HME
☐ Home Health Agency
☐ Educational Institution
☐ Manufacturer or supplier
☐ Other, specify ____________________

Date of Birth (optional) ____________ Sex (optional) ____________

U.S. Citizen? Yes ______ Na ______

Have you ever been a member of the AARC? ____________________

If so, when? From ____________________ to ____________________

Preferred mailing address: ☐ Home ☐ Business

For office use only

FOR ACTIVE MEMBER

An individual is eligible if he/she lives in the U.S. or its territories or was an Active Member prior to moving outside its borders or territories, and meets ONE of the following criteria: (1) is legally credentialed as a respiratory care professional if employed in a state that mandates such, OR (2) is a graduate of an accredited educational program in respiratory care, OR (3) holds a credential issued by the NBRC. An individual who is an AARC Active Member in good standing on December 8, 1994, will continue as such provided his/her membership remains in good standing.

PLEASE USE THE ADDRESS OF THE LOCATION WHERE YOU PERFORM YOUR JOB, NOT THE CORPORATE HEADQUARTERS IF IT IS LOCATED ELSEWHERE.

Place of Employment ____________________

Address ____________________

City ____________________

State __________ Zip ____________________

Phone No. ( ________ )

Medical Director/Medical Sponsor ____________________

FOR ASSOCIATE OR SPECIAL MEMBER

Individuals who hold a position related to respiratory care but do not meet the requirements of Active Member shall be Associate Members. They have all the rights and benefits of the Association except to hold office, vote, or serve as chair of a standing committee. The following subclasses of Associate Membership are available: Foreign, Physician, and Industrial (individuals whose primary occupation is directly or indirectly devoted to the manufacture, sale, or distribution of respiratory care equipment or supplies). Special Members are those not working in a respiratory care-related field.

PLEASE USE THE ADDRESS OF THE LOCATION WHERE YOU PERFORM YOUR JOB, NOT THE CORPORATE HEADQUARTERS IF IT IS LOCATED ELSEWHERE.

Place of Employment ____________________

Address ____________________

City ____________________

State __________ Zip ____________________

Phone No. ( ________ )

FOR STUDENT MEMBER

Individuals will be classified as Student Members if they meet all the requirements for Associate Membership and are enrolled in an educational program in respiratory care accredited by, or in the process of seeking accreditation from, an AARC-recognized agency.

SPECIAL NOTICE — Student Members do not receive Continuing Respiratory Care Education (CRCE) transcripts. Upon completion of your respiratory care education, continuing education credits may be pursued upon your reclassification to Active or Associate Member.

School/RC Program ____________________

Address ____________________

City ____________________

State __________ Zip ____________________

Phone No. ( ________ )

Length of program

☐ 1 year ☐ 4 years
☐ 2 years ☐ Other, specify ____________________

Expected Date of Graduation (REQUIRED INFORMATION)

Month __________ Year __________

American Association for Respiratory Care • 11030 Ables Lane • Dallas, TX 75229-4593 • (972) 243-2272 • Fax (972) 484-2720
Demographic Questions
We request that you answer these questions in order to help us design services and programs to meet your needs.

Check the Highest Degree Earned
- High School
- RC Graduate Technician
- Associate Degree
- Bachelor's Degree
- Master's Degree
- Doctorate Degree

Number of Years in Respiratory Care
- 0-2 years
- 3-5 years
- 6-10 years
- 11-15 Years
- 16 years or more

Job Status
- Full Time
- Part Time

Credentials
- RRT
- CRT
- Physician
- CRNA
- RN
- LVN/LPN
- CPFT
- RPFT
- Perinatal/Pediatric

Salary
- Less than $10,000
- $10,001-$20,000
- $20,001-$30,000
- $30,001-$40,000
- $40,000 or more

Please Sign
I hereby apply for membership in the American Association for Respiratory Care and have enclosed my dues. If approved for membership in the AARC, I will abide by its bylaws and professional code of ethics. I authorize investigation of all statements contained herein and understand that misrepresentations or omissions of facts called for is cause for rejection or expulsion.

A yearly subscription to RESPIRATORY CARE Journal and AARC Times magazine includes an allocation of $11.50 from my dues for each of these publications.

NOTE: Contributions or gifts to the AARC are not tax deductible as charitable contributions for income tax purposes. However, they may be tax deductible as ordinary and necessary business expenses subject to restrictions imposed as a result of association lobbying activities. The AARC estimates that the non-deductible portion of your dues — the portion which is allocable to lobbying — is 26%.

Signature
Date

Membership Fees
Payment must accompany your application to the AARC. Fees are for 12 months. (NOTE: Renewal fees are $75.00 Active, Associate-Industrial or Associate-Physician, or Special status; $90.00 for Associate-Foreign status; and $45.00 for Student status).

- Active $87.50
- Associate (Industrial or Physician) $87.50
- Associate (Foreign) $102.50
- Special $87.50
- Student $45.00

TOTAL

Specialty Sections
Established to recognize the specialty areas of respiratory care, these sections publish a bi-monthly newsletter that focuses on issues of specific concern to that specialty. The sections also design the specialty programming at the national AARC meetings.

- Adult Acute Care Section $15.00
- Education Section $20.00
- Perinatal-Pediatric Section $15.00
- Diagnostics Section $15.00
- Continuing Care—Rehabilitation Section $15.00
- Management Section $20.00
- Transport Section $15.00
- Home Care Section $15.00
- Subacute Core Section $15.00

TOTAL

GRAND TOTAL = Membership Fee plus optional sections

Payment Method
- Total Amount Enclosed/Charged
- Please charge my dues (see below)

To charge your dues, complete the following:
- MasterCard
- Visa

Card Number

Card Expires ______________________

Signature ______________________

Mail application and appropriate fees to:
American Association for Respiratory Care • 11030 Ables Lane • Dallas, TX 75229-4593 • (972) 243-2272 • Fax (972) 484-2720
Manuscript Preparation Guide

RESPIRATORY CARE welcomes original manuscripts related to the science and technology of respiratory care and prepared according to the following instructions and the Uniform Requirements for Manuscripts Submitted to Biomedical Journals (available at http://www.acponline.org/journals/resource/unifreq.htm). Manuscripts are blinded and reviewed by professionals who are experts in their fields. Authors are responsible for obtaining written permission to publish previously—published figures and tables from the original copyright holder. Accepted manuscripts are copyedited for clarity, concision, and consistency with RESPIRATORY CARE format. Before publication, authors receive page proofs for minor correction. Published papers are copyrighted by Daedalus Inc and may not be published elsewhere without permission. Editorial consultation is available at any stage of planning or writing for any submission; contact the Editorial Office.

Categories of Articles

Research Article: A report of an original investigation (a study). Must include Title Page, Abstract, Key Words, Background, Methods, Results, Discussion, Conclusions, and References. May also include Tables, Figures (if so, must include Figure Legends), Acknowledgments, and Appendices.

Review Article: A comprehensive, critical review of the literature and state-of-the-art summary of a topic that has been the subject of at least 40 published research articles. Must include: Title Page, Outline, Key Words, Introduction, Review of the Literature, Summary, and References. May also include: Tables, Figures (if so, must include Figure Legends), and Acknowledgments.

Overview: A critical review of a pertinent topic that has fewer than 40 published research articles. Same structure as Review Article.

Update: A report of subsequent developments in a topic that has been critically reviewed in RESPIRATORY CARE or elsewhere. Same structure as a Review Article.

Special Article: A pertinent paper not fitting one of the other categories. Consult with the Editor before writing or submitting such a paper.

Editorial: A paper addressing an issue in the practice or administration of respiratory care. It may present an opposing opinion, clarify a position, or bring a problem into focus.

Letter: A brief, signed communication responding to an item published in RESPIRATORY CARE or about other pertinent topics. Tables, Figures, and References may be included. The letter should be marked "For Publication."

Case Report: Report of an uncommon clinical case or a new or improved method of management or treatment. A case-managing physician must either be an author or furnish a letter approving the manuscript. Must include: Title Page, Abstract, Introduction, Case Summary, Discussion, and References. May also include: Tables, Figures (if so, must include Figure Legends), and Acknowledgments.

Point-of-View Paper: A paper expressing personal but substantiated opinions on a pertinent topic. Must include: Title Page, Text, and References. May also include Tables and Figures (if so, must include Figure Legends).

Drug Capsule: A miniature review paper about a drug or class of drugs that includes discussions of pharmacology, pharmacokinetics, or pharmacotherapy.

Graphics Corner: A brief case report discussing and illustrating waveforms for monitoring or diagnosis. Should include Questions, Answers, and Discussion sections.

Kittredge's Corner: A brief description of the operation of respiratory care equipment. Should include information from manufacturers and editorial comments and suggestions.

PFT Corner: A brief, instructive case report including pulmonary function testing, accompanied by a review of the relevant physiology and appropriate references to the literature.

Test Your Radiologic Skill: A brief, instructive case report involving pulmonary medicine radiography and including one or more radiographs. May involve imaging techniques other than conventional chest radiography.

Review of a Book, Film, Tape, or Software: A balanced, critical review of a recent release. RESPIRATORY CARE does not accept unsolicited book reviews; please contact the Editor if you have a suggestion for a book review.

Preparing the Manuscript

Print on one side of white 8.5 ×11 inch paper, with margins of at least 1 inch on all sides. Double-space the text and number the pages. Do not include author names, author institutional affiliations, or allusions to institutional affiliations anywhere except on the title page. On the Abstract page include the title but do not include author names. Begin each of the following on a new page: Title Page, Abstract, Text, Acknowledgments, References, each Table, each Figure, and each Appendix. Use standard English in the first person and active voice. Type all headings in initial-capital letters (eg. Background, Methods, Patients, Equipment, Statistical Analysis, Results, Discussion). Center the main section headings and place second-level headings on the left margin.
Abstract. Please ensure that the abstract does not contain any facts or conclusions that do not also appear in the body text. Limit the abstract to no more than 400 words.

Key Words. Research, Review, Overview, and Special Articles require Key Words. On the Abstract or Outline page, include a list of 6 to 10 key words or two-word phrases.

References. Assign reference numbers in the order that articles are cited in your manuscript. At the end of your manuscript, list the cited works in numerical order. Abbreviate journal names as in Index Medicus. List all authors. The following examples show RESPIRATORY CARE’s style for references.

Article in a journal carrying pagination throughout the volume:


Article in a publication that numbers each issue beginning with Page 1:


Corporate author journal article:


Article in journal supplement: (Journals differ in numbering and identifying supplements. Supply information sufficient to allow retrieval.)

Reynolds HY. Idiopathic interstitial pulmonary fibrosis. Chest 1986;89(3 Suppl):139S-143S.

Abstract in journal: (Abstracts citations are to be avoided, and those more than 3 years old should not be cited.)

Stevens DP. Scavenging ribavirin from an oxygen hood to reduce environmental exposure (abstract). Respir Care 1990;35(11):1087-1088.

Editorial in a journal:


Editorial with no author given:


Letter in journal:


Corporate author book:


Book: (For any book, specific pages should be cited whenever reference is made to specific statements or other content.)


Chapter in book with editor(s):


Paper accepted but not yet published:

Hess D. New therapies for asthma. Respir Care (year, in press).

Personal communication of unpublished data not yet accepted for publication: You must obtain written permission to cite unpublished data received via personal communication. Do not number such references, but instead make parenthetical reference in the body text of your manuscript. Example: “Recently, Jones found this treatment effective in 45 of 83 patients (Jones HI, University of the Cascades, 1999, personal communication).”

Tables. Tables should be consecutively numbered. Start each table on a separate page. Number and title the table and give each column a brief heading. Place explanations in footnotes, including all non-standard abbreviations and symbols. Key the footnotes with the following symbols, superscripted, in the table body, and in the following order: *, †, ‡, §, ¶, ‡‡, ‡‡‡, ‡‡‡‡. Do not use horizontal or vertical rules or borders. Do not submit tables as photographs, reduced in size, or on oversize paper.

Figures (illustrations). Figures include graphs, line drawings, photographs, and radiographs. Use only illustrations that clarify and augment the text. Number figures consecutively as Figure 1, Figure 2, etc. All the figures must be mentioned in the text. Every figure should have a legend (a title and/or description explaining the figure). Figure legends should appear as separate paragraphs at the end of the manuscript (after the References section), in the same computer file as the manuscript (not in a separate file, as with the tables and figures). Do not create scanned versions of figures borrowed from other publications; clear photocopies are preferable. To include figures previously published in other publications, you must obtain permission from the original copyright holder (see below). Figures must be of professional quality and a copy of the article from which the figure came should be available. If color is essential to the figure, consult the Editor for more information. In reports of animal experiments, use schematic drawings, not photographs. A letter of consent must accompany any photograph of an identifiable person. If possible, submit radiographs as prints and full-size copies of film.

Drugs. Precisely identify all drugs and chemicals used, giving generic names, doses, and methods of administration. Brand names may be given in parentheses after generic names.

Commercial Products. In the text, parenthetically identify commercial products only on first mention, giving the manufacturer’s name, city, and state or country. Example: “We performed spirom-
Permissions: You must obtain written permission to use pictures of identifiable individuals or to name individuals in the Acknowledgments section. You must obtain written permission from the original copyright holder to use figures and tables from other publications. Copies of all applicable permissions must be on file at RESPIRATORY CARE before a manuscript goes to press. Copyright is most often held by the journal or book in which the figure or table originally appeared and applies to the creativity, style, and form in which the facts/data are presented to the reader; the facts themselves are not copyright-protectable. Therefore, if you were asking permission to reproduce a table or figure directly from a journal or book, or with minor adaptations, permission would be necessary. However, if you intend to extract some data from text or illustrations and present them in an entirely new form, permission would not be needed. Simply cite the source of the data using the following statement: "Figure adapted from data published in ..."

Ethics. When reporting experiments on human subjects, indicate that procedures were conducted in accordance with the ethical standards of the World Medical Association Declaration of Helsinki (see Respir Care 1997;42(6):635-636) or of the institution’s committee on human experimentation. State that informed consent was obtained. Do not use patient’s names, initials, or hospital numbers in text or illustrations. When reporting experiments on animals, indicate that the institution’s policy, a national guideline, or a law on the care and use of laboratory animals was followed.

Statistics. Identify the statistical tests used in analyzing the data and give the prospectively determined level of significance in the Methods section. Report actual p values in the Results section. Cite only textbook and published article references to support choices of tests. As with commercial products (see above), parenthetically identify any general-use or commercial computer programs used.

Units of Measurement. Express measurements of length, height, weight, and volume in metric units appropriately abbreviated; temperatures in degrees Celsius; and blood pressures in millimeters of mercury (mm Hg). Report hematologic and clinical-chemistry measurements in conventional metric and in SI (Système Internationale) units (units and conversion factors listed at Respir Care 1997;42(6):640). Show gas pressures (including blood gas tensions) in millimeters of mercury (mm Hg).

Conflict of Interest. On the cover page, authors must disclose any liaison or financial arrangement they have with a manufacturer or distributor whose product is addressed in the manuscript or with the manufacturer or distributor of a competing product. Such arrangements do not disqualify a paper from consideration and are not disclosed to reviewers. Reviewers are screened for possible conflict of interest.

Abbreviations and Symbols. Use standard abbreviations and symbols, listed at Respir Care 1997;42(6):637-642. Do not create new abbreviations. Do not use abbreviations in the title, in section headings, and do not use unusual abbreviations in the abstract. Use an abbreviation only if the term occurs 4 or more times in the paper. Define all abbreviations (ie, write out the full term on first mention, followed by the abbreviation in parentheses) and thereafter use only the abbreviation. Standard units of measurement and scientific terms can be abbreviated without explanation (eg, L/min, mm Hg, pH, O2). Please use the following forms: cm H2O (not cmH2O), f (not bpm), L (not l), L/min (not LPM, l/min, or lpm), ml (not ml), mm Hg (not mmHg), pH (not Ph or PH), p > 0.001 (not p<0.001), s (not sec), SPO2 (arterial oxygen saturation measured via pulse-oximetry).

Prior and Duplicate Publication. In general, do not submit work that has been published or accepted elsewhere, though in special instances the Editor may consider such material if the original publisher grants permission. Please consult the Editor before submitting such work.

Authorship. All persons listed as authors should have participated in the reported work and in the shaping of the manuscript, all must have proofread the submitted manuscript, and all should be able to publicly discuss and defend the paper’s content. A paper of corporate authorship must specify the key persons responsible for the article. Attribution of authorship is not based solely on solicitation of funding, collection or analysis of data, provision of advice, or similar services. Persons who provide such ancillary services may be recognized in an Acknowledgments section.

Reviewers: Please supply the names, credentials, affiliations, addresses, and phone/fax numbers of 3 professionals whom you consider expert on the topic of your paper. Your manuscript may be sent to one or more of them for blind peer review.

Submitting the Manuscript

Submit three printed copies and one (3.5-inch) computer diskette. The printed copies should each include photocopies of all of the Figures, Tables, and Appendices. On the diskette, the manuscript should be in one file and the tables in a separate file. If soft copies of the figures are available, they should also be in a separate file. However, do not create scanned versions of figures borrowed from other publications; clear photocopies are preferable. Include the completed Cover Letter and Checklist (see next page) and permission letters. Mail to RESPIRATORY CARE, 600 Ninth Avenue, Suite 702, Seattle WA 98104. Do not fax manuscripts. Receipt will be acknowledged.
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Publication Category: 

Corresponding Author: 

Mailing Address: 

Reprints:  Yes  No 

E-mail Address: 

"We, the undersigned, have all participated in the work reported, proofread the accompanying manuscript, and approve its submission for publication." Please print and include credentials, title, institution, academic appointments, city and state. If more than 4 authors, please use another copy of this form.*

*First Author: 

Author Signature/Date 

*Second Author: 

Author Signature/Date 

*Third Author: 

Author Signature/Date 

*Fourth Author: 

Author Signature/Date 

Has this research been presented in any public forum?  Yes  No 

If yes, where, when and by whom? 

Has this research received any awards?  Yes  No 

If yes, please describe. 

Has this research received any grants or other support, financial or material?  Yes  No 

If yes, please describe. 

Do any of the authors of this manuscript have a financial interest in (or a commercial or consulting relationship to) any of the products or manufacturers mentioned in this paper or any competing products or manufacturers?  Yes  No 

If yes, please describe. 

☐ Have you enclosed a copy of the manuscript on diskette? 

☐ Is double-spacing used throughout entire manuscript? 

☐ Are all pages numbered in upper-right corners? 

☐ Are all references, figures, and tables cited in the text? 

☐ Has the accuracy of the references been checked, and are they correctly formatted? 

☐ Have SI values been provided? 

☐ Has all arithmetic been checked? 

☐ Have generic names of drugs been provided? 

☐ Have necessary written permissions been provided? 

☐ Have authors' names been omitted from text and figure labels? 

☐ Have copies of 'in press' references been provided? 

☐ Has the manuscript been proofread by all the authors? 

☐ Have the manufacturers and their locations been provided for all devices and equipment used?
For VOLUNTARY reporting by health professionals of adverse events and product problems

### A. Patient Information

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  Date of birth: | 3. Sex  
  female: lbs  
  male: kgs | 4. Weight  
  lbs |   |

**In confidence**

### B. Adverse event or product problem

1. **Adverse event** and/or **Product problem** (e.g., defects/malfunctions)
2. **Outcomes attributed to adverse event** (check all that apply)
   - death (mo/day)
   - congenital anomaly
   - life-threatening
   - hospitalization - initial or prolonged
   - other:  
3. **Date of event** (mo/day)
4. **Date of this report** (mo/day)
5. **Describe event or problem**
6. **Relevant tests/laboratory data**, including dates
7. **Other relevant history**, including preexisting medical conditions (e.g., allergies, race, pregnancy, smoking and alcohol use, hepatic/renal dysfunction, etc.)

### C. Suspect medication(s)

1. **Name** (give labeled strength & mfr/lbl, if known)
2. **Dose, frequency & route used**
3. **Therapy dates** (if unknown, give duration)  
   (mo/day) or (best estimate)
4. **Diagnosis for use** (indication)
5. **Event abated after use** stopped or dose reduced
6. **Lot #** (if known)
7. **Exp. date** (if known)
8. **Event reappeared after reintroduction**
9. **NDC #** (for product problems only)
10. **Concomitant medical products and therapy dates** (exclude treatment of event)

### D. Suspect medical device

1. **Brand name**
2. **Type of device**
3. **Manufacturer name & address**
4. **Operator of device**  
   - health professional
   - lay user/patient
   - other:
5. **Expiration date** (mo/day)
6. **Model #**
7. **If Implanted, give date** (mo/day)
8. **If explanted, give date** (mo/day)
9. **Device available for evaluation?** (Do not send to FDA)
   - yes  
   - no  
   - returned to manufacturer on  
10. **Concomitant medical products and therapy dates** (exclude treatment of event)

### E. Reporter (see confidentiality section on back)

1. **Name & address**
2. **Phone #**
3. **Health professional?**
4. **Also reported to**  
   - manufacturer
   - user facility
   - distributor
5. **If you do NOT want your identity disclosed to the manufacturer, place an “X” in this box.**
ADVICE ABOUT VOLUNTARY REPORTING

Report experiences with:
- medications (drugs or biologics)
- medical devices (including in-vitro diagnostics)
- special nutritional products (dietary supplements, medical foods, infant formulas)
- other products regulated by FDA

Report SERIOUS adverse events. An event is serious when the patient outcome is:
- death
- life-threatening (real risk of dying)
- hospitalization (initial or prolonged)
- disability (significant, persistent or permanent)
- congenital anomaly
- required intervention to prevent permanent impairment or damage

Report even if:
- you're not certain the product caused the event
- you don't have all the details

Report product problems — quality, performance or safety concerns such as:
- suspected contamination
- questionable stability
- defective components
- poor packaging or labeling
- therapeutic failures

How to report:
- just fill in the sections that apply to your report
- use section C for all products except medical devices
- attach additional blank pages if needed
- use a separate form for each patient
- report either to FDA or the manufacturer (or both)

Important numbers:
- 1-800-FDA-0178 to FAX report
- 1-800-FDA-7737 to report by modem
- 1-800-FDA-1088 to report by phone or for more information
- 1-800-822-7967 for a VAERS form for vaccines

If your report involves a serious adverse event with a device and it occurred in a facility outside a doctor's office, that facility may be legally required to report to FDA and/or the manufacturer. Please notify the person in that facility who would handle such reporting.

Confidentiality: The patient's identity is held in strict confidence by FDA and protected to the fullest extent of the law. The reporter's identity, including the identity of a self-reporter, may be shared with the manufacturer unless requested otherwise. However, FDA will not disclose the reporter's identity in response to a request from the public, pursuant to the Freedom of Information Act.

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Washington, D.C. 20204

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Scheduled Professor's Rounds 2000

Pulmonary Rehabilitation: What You Need to Know—Julien M Roy RRT; Host, Richard Branson RRT—Video March 7; Audio April 4

Pediatric Asthma in the ER—Tim Myers RRT; Host, Richard Branson RRT—Video March 28; Audio April 18

Drugs, Medications and Delivery Devices of Importance in Respiratory Care—Jim Fink MS RRT; Host, David Pierson MD—Video April 25; Audio May 16

Cost Effective Respiratory Care: You've Got to Change—Kevin Shlake MA RRT FACHE; Host, Sam P Giordatio MBA RRT—Video May 23; Audio June 20

Pediatric Ventilation: Kids Are Different—Mark Hultin MD; Host, Richard Branson RRT—Video July 25; Audio August 15

What Matters in Respiratory Monitoring: What Goes and What Stays—Dean Hess PhD RRT FAARC; Host, Richard Branson RRT—Video August 22; Audio September 26

Managing Asthma: An Update—Patti Joyner RRT CCM; Host, Mari Jones MSN RN RRT—Video September 19; Audio October 17

Routine Pulmonary Function Testing: Doing It Right—Carl D Mottram RRT RPFT; Host, David Pierson MD—Video November 7; Audio December 5

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American Association for Respiratory Care
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— American Respiratory Care Foundation fellowships, grants, and awards
— Clinical Practice Guidelines

National Board for Respiratory Care
http://www.nbrc.org

RESPIRATORY CARE online
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Asthma Management Model System
http://www.nhlbi.nih.gov

Keys to Professional Excellence
http://www.aarc.org/keys/

The National Board for Respiratory Care—Examination Dates and Fees for 2000

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² Protocol of the Month, University of Pittsburgh Medical Center, Department of Respiratory Care. AARC Times, May 1997.