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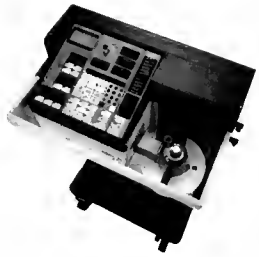

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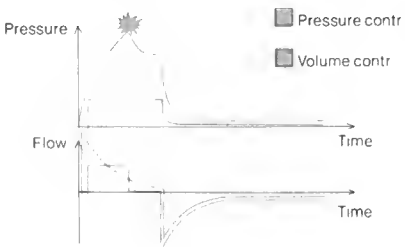

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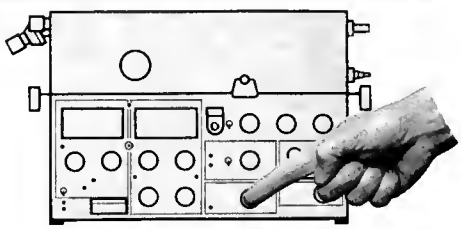

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Reviews and Statements to Note

Longitudinal Designs and their Statistical Analysis in Pediatric Pulmonary Research (editorial)—RW Helms. Pediatr Pulmonol 1990;9:69.

State of the Art: Smoking and Smoking Cessation (review)—EB Fisher Jr, D Haire-Joshu, GD Morgan, H Rehberg, K Rost. Am Rev Respir Dis 1990;142:702

Medical-Legal Definition of Occupational Asthma (special article)—DD Smith. Chest 1990;98:1007.

Infection Control for Home Health (special commentary)—B Simmons, M Trusler, J Roccaforte, P Smith, R Scott. Infect Control Hosp Epidemiol 1990;11:362.

Scientific Basis of Pulmonary Rehabilitation: Position Paper of the American Association of Cardiovascular and Pulmonary Rehabilitation—AL Ries. J Cardiopulmonary Rehabil 1990;10:418.

Diagnostic Standards and Classification of Tuberculosis (statement)—American Thoracic Society. Am Rev Respir Dis 1990;142:725.

Pulmonary Function Testing Prior to Extubation in Infants with Respiratory Distress Syndrome—KA Veness-Meehan, S Richter, JM Davis. Pediatr Pul 1990;9:2.

Pulmonary function testing was performed just prior to extubation on 50 infants mechanically ventilated for treatment of respiratory distress syndrome. All infants were ready for extubation as defined by clinical criteria. Pulmonary mechanics and energetics were measured by a computerized technique that consists of a pneumotachometer to measure flowrates and an esophageal balloon and differential transducer to estimate transpulmonary pressure. Tidal volume, minute ventilation, dynamic lung compliance, pulmonary resistance, and resistive work of breathing were then calculated by high speed microcomputer processing. Successful extubation was defined as >72 hours without respiratory decompensation requiring reinstitution of ventilatory support. Thirty-six (72%) infants were successfully extubated and 14 (28%) infants failed extubation. Infants in the success and failure groups were matched for birthweight, gestational age, age at study, weight at study, weight at study relative to birthweight, use of nasal continuous positive airway pressure (CPAP), and methylxanthines. No statistically significant differences in pulmonary mechanics were seen between the two groups. Data suggests that successful withdrawal of mechanical ventilation may be related to multiple factors such as central inspiratory drive, diaphragmatic endurance, and chest-wall stability, in addition to improved lung mechanics. Pulmonary function testing criteria alone may not be useful in determining optimal timing of extubation in premature infants.

Respiratory Mucus pH in Tracheostomized Intensive Care Unit Patients: Effects of Colonization and Pneumonia—DR Karnad, DG Mhaisekar, KV Moralwar. Crit Care Med 1990;18:699.

Daily, we studied the effects of colonization and pneumonia on the pH of respiratory mucus in 23 critically ill patients. Sixteen patients had tracheobronchial colonization, and 11 of these subsequently developed pneumonia. Two other patients

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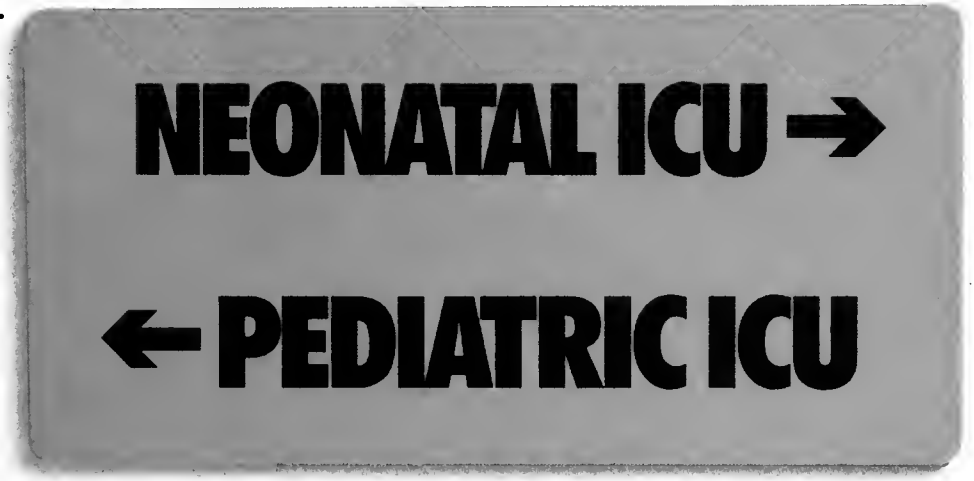

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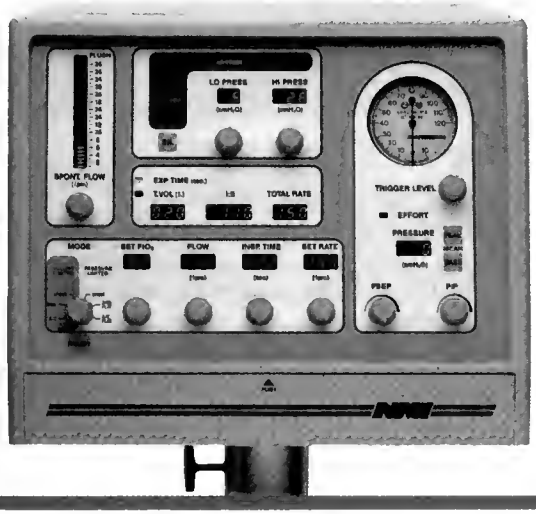

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developed pneumonia without prior colonization. For the 13 patients with pneumonia, there was a significantly greater decrease in pH below the value recorded on the day of intubation vs that for the 10 patients who did not have pneumonia. In the 11 patients who developed initial colonization and then pneumonia, the pH did not change after colonization, but became acidic with the development of pneumonia. The pH returned to basal levels after recovery from pneumonia. We tested the pH value to differentiate between colonization and infection in these intubated critically ill patients. A decrease in pH $\geq$ 0.2 below the pH value on the day of intubation could predict the presence of pneumonia with a positive predictive value of 90%. This decrease in pH occurred before or on the same day as the development of radiologically detectable pneumonia in most cases. Daily monitoring of the pH of tracheal mucus may be of value in critically ill intubated patients.

Role of Molecular Diffusion in Conventional and High Frequency Ventilation—RA Klocke, AR Saltzman, BJB Grant, AT Aquilina, S Zhang. Am Rev Respir Dis 1990;142:802.

The influence of molecular diffusion on gas-mixing during conventional mechanical ventilation (CMV) and high frequency ventilation (HFV) was studied by observing the wash-in of six poorly soluble, inert gases in arterial blood. Anesthetized dogs were ventilated either with CMV or HFV. Following a step change in inspired gas composition, the increase in arterial concentrations of hydrogen, helium, methane, ethane, isobutane, and sulfur hexafluoride was determined by gas chromatography. The relative gas diffusivities encompassed a range of almost one order of magnitude. Propane, present in inspired gas during both the control and wash-in phases, served as an internal reference for calculation of blood tracer concentrations. The wash-in of all six inert gases followed a single exponential time course during both CMV and HFV. The rate of wash-in of each gas decreased with increasing molecular weight (MW). The relationship of rate constants to a measure of relative diffusivity ($MW^{-0.5}$) was significantly different than zero for both types of ventilation. The slope of this relationship was three times larger for CMV than HFV, indicating that molecular diffusion has a greater role in gas mixing during ventilation with large tidal volumes. Diffusion has a minor role in gas mixing during high frequency ventilation with small tidal volumes. Demonstration of the presence of gas separation secondary to molecular diffusion during HFV is enhanced by measuring wash-in, rather than wash-out, of inert gases because gas separation is likely to be obscured as exhaled gases pass through the well-mixed central airways during gas wash-out.

Oxygen Supplementation during Exercise in Cystic Fibrosis—PA Nixon, DM Orenstein, SE Curtis, EA Ross. Am Rev Respir Dis 1990;142:807.

Fourteen female and 22 male patients with cystic fibrosis (CF), 8 to 29 years of age, performed two progressive exercise tests to exhaustion on a cycle ergometer, breathing normoxic air (21% O_2) for one test, and hyperoxic air (30% O_2) for the other test. The order of gas administration was randomized. Minute ventilation ($\dot{V}_E$), oxygen uptake ($\dot{V}O_2$), end-tidal CO_2 tension (P_{etCO_2}), work rate, oxyhemoglobin saturation (S_{aO_2}), and heart rate (HR) were measured throughout the tests. The S_{aO_2} of 11 patients at peak exercise was 90% or less (''Low Sat'' group). The S_{aO_2} of 23 patients remained above 90% throughout the exercise (''High Sat'' group). Hyperoxic air minimized desaturation during exercise in the Low Sat group to 2 $\pm$ 2% compared to a decrease of 10 $\pm$ 5% with normoxic air. The decrease in saturation was not significant for the High Sat group (1 $\pm$ 1% for both 21% and 30% O_2). Peak work rate and $\dot{V}O_2$ did not differ significantly between normoxic and hyperoxic conditions. However, $\dot{V}_E$ and HR at peak exercise tended to be lower, and P_{etCO_2} was higher during peak exercise with 30% O_2 than 21% O_2 for both groups. During submaximal exercise, O_2 desaturation was diminished and HR was significantly lower with supplemental O_2, specifically in the Low Sat group. $\dot{V}_E$ was significantly lower for both groups during submaximal exercise with hyperoxic air. The results suggest that O_2 supplementation minimizes O_2 desaturation and enables patients with CF to exercise with reduced ventilatory and cardiovascular work.

Efficacy of Positive vs Negative Pressure Ventilation in Unloading the Respiratory Muscles—MJ Belman, GW Soo Hoo, JH Kuei, R Shadmehr. Chest 1990;98:850.

We compared the efficacy of positive pressure ventilation (PPV) vs negative pressure ventilation (NPV) in providing ventilatory muscle rest for 5 normal subjects and 6 patients with chronic obstructive pulmonary disease (COPD). All participants underwent measurement of transdiaphragmatic pressure (P_{di}), pressure time integral of the diaphragm (PTI), integrated diaphragmatic electromyogram (iEMG), minute ventilation $\dot{V}_E$, tidal volume (V_T), and end-tidal CO_2 ($etCO_2$) during 15 minutes of PPV and NPV. For each subject, ventilator

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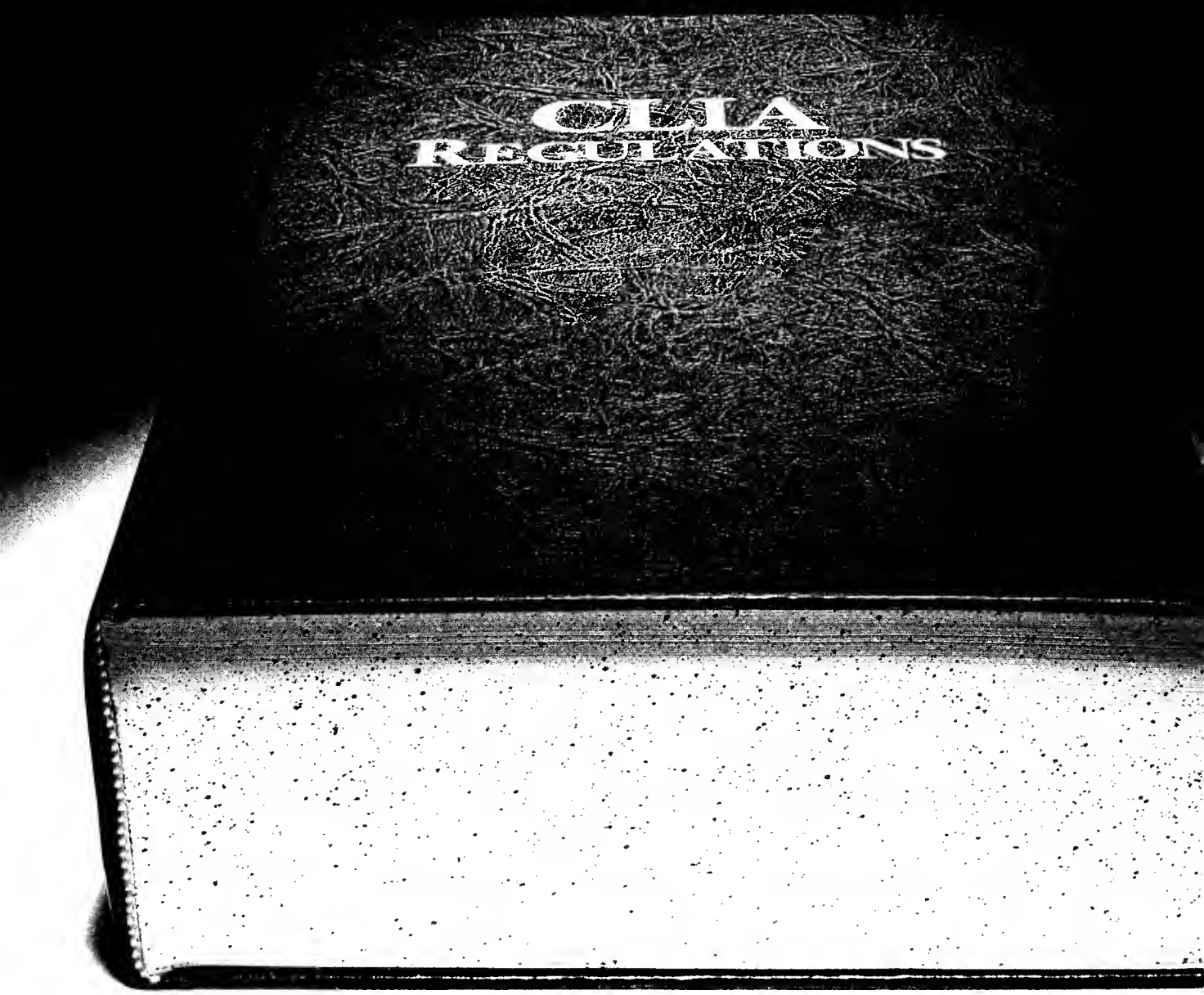

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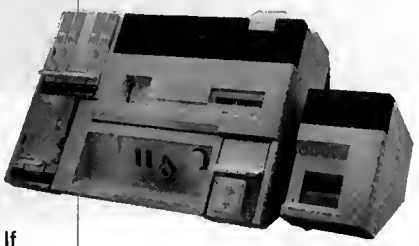

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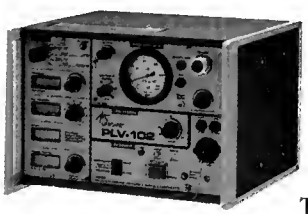

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adjustments were made to obtain $\dot{V}_E$ similar to levels measured during quiet breathing (QB). We found that the iEMG, P_{di}, PTI, and average coefficient of variation of the tidal volume (CV-V_T) were consistently lower during PPV as compared with NPV ($p = 0.01$). The iEMG normalized for $\dot{V}_E$ and V_T was also significantly lower during PPV ($p = 0.01$). During PPV, subjects were mildly hyperventilated (lower $etCO_2$ and higher $\dot{V}_E$) compared with QB and NPV, but no significant correlation was noted between the change in $etCO_2$ and the change in iEMG. The change in PTI was significantly correlated with the change in iEMG ($p = 0.01$). We conclude that in the short term, PPV is more effective than NPV in reducing diaphragmatic activity. Positive pressure ventilation may be the preferred method of assisted ventilation in future studies of ventilatory muscle rest therapy.

Surfactant Replacement Therapy in Respiratory Distress Syndrome: Meta-Analysis of Clinical Trials of Single-Dose Surfactant Extracts—HM Hennes, MB Lee, AA Rimm, DL Shapiro. Am J Dis Child 1991;145:102.

Replacement therapy with surfactant extracts in premature infants with respiratory distress syndrome has been evaluated in several clinical trials. The results of individual trials do not provide conclusive evidence that administration of a single dose of surfactant improves morbidity or mortality. Meta-analysis is a statistical method to combine the results of such clinical trials, and combined analysis provides a means to overcome the problem of not being able to detect significant small differences in individual trials due to these small sample sizes. Seven clinical trials (277 patients treated with nonhuman surfactant extract and 263 controls) met the criteria for analysis; five outcome measurements (mortality, patent ductus arteriosus, pneumothorax, intraventricular hemorrhage, and bronchopulmonary dysplasia) were selected to estimate the treatment effect. The meta-analysis showed that a single dose of surfactant administered before the first breath or within 15 hours of birth significantly decreased the mortality rate (95% confidence interval = -0.19 to -0.03) and the risk of developing pneumothorax (95% confidence interval = -0.28 to -0.14) in infants with respiratory distress syndrome. Further clinical trials are needed to evaluate other aspects of surfactant replacement therapy in premature infants because inconsistent results were observed among the seven analyzed studies.

Circadian Basis of the Late Asthmatic Response—AA Mohiuddin, RJ Martin. Am Rev Respir Dis 1990;142:1153.

The late asthmatic response (LAR) to an allergen challenge has a marked impact on lung function in the patient with asthma. Virtually all studies on the LAR have been done during the daytime. This study evaluated the LAR as a function of the time of day an inhaled allergen challenge was performed. An allergen challenge given in the morning produced a LAR in 4 of 10 subjects, while the same challenge in the evening caused a LAR in 9 of 10 ($p < 0.05$). The time to onset of the LAR following the morning and evening challenges was 9.4 ± 2.0 h versus 3.1 ± 0.3 h, respectively ($p < 0.05$). The maximal decrease in FEV_1 for the LAR was $32.8 \pm 5.6\%$ for the morning challenge versus $43.0 \pm 3.1\%$ in the evening ($p < 0.05$). Additionally, the bronchial responsiveness to methacholine was significantly greater at 24 h following evening allergen challenge than after the morning ($p < 0.05$) challenge. Thus, it is important to take into account the time of day a patient is exposed to an allergen in regard to the development of the LAR.

Design and Validation of an Indicator Gas Injector for Multiple Gas Washout Tests in Mechanically Ventilated Patients—PEM Huygen, BWA Feenstra, WPJ Holland, C Ince, H Stam, HA Bruining. Crit Care Med 1990;18:754.

A device to produce a stepwise indicator-gas-fraction variation to initiate a washout test in mechanically ventilated patients is described. The device, which can be used in conjunction with the commonly used Siemens-Elema series 900 ventilators, is based on simple, off-the-shelf technology. It features the simultaneous use of two indicator gases (so that the influence of diffusion processes in the gas exchange to the patient can be measured) and maintains a nearly constant F_{IO_2} during a washout procedure. With this indicator gas injector, the transition time of the indicator gas fraction at the beginning of a washout proved to be short enough to detect ventilation inhomogeneity by visual inspection of the washout curves. Functional residual capacity measurements using this device are presented on a test lung with known volume, on healthy volunteers, and on critically ill patients.

Bronchodilator Response to Ipratropium Bromide in Infants with Bronchopulmonary Dysplasia—KL Brundage, KG Mohsini, AB Froese, JT Fisher. Am Rev Respir Dis 1990;142:1137.

Although the muscarinic antagonist ipratropium bromide is used clinically as a bronchodilator in infants ventilated because of bronchopulmonary dysplasia (BPD), no studies have compared the response or efficacy of

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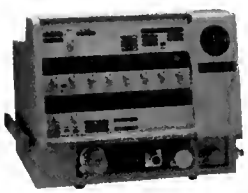

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different dosages or its effectiveness in combination with β-adrenergic agonists. We measured the response of respiratory system mechanics in 10 ventilated infants (25 ± 2 days of age) to 75, 125, and 175 μg ipratropium bromide (IB), 125 μg IB plus 0.04 mg salbutamol (SAL), 175 μg IB plus 0.04 mg SAL, and saline vehicle, delivered via nebulizer into the ventilator circuit. Respiratory system resistance (R_{rs}) and compliance (C_{rs}) were measured by the passive flow-volume technique. R_{rs} and C_{rs} were measured before and at 1 to 2 h and at 4 h after delivery of the five drug dosages or saline. All six studies were completed within a 72-h period. Saline had no significant effect on mechanics. Significant responses to ipratropium alone were seen only after 175 μg where Rrs decreased 20 ± 3% (SEM) ($p < 0.05$) at 1 to 2 h and 16 ± 5% ($p < 0.05$) at 4 h. After 125 μg IB + SAL and 175 μg IB + SAL, R_{rs} was significantly decreased both at 1 to 2 h and at 4 h, and C_{rs} was significantly increased 20 ± 6% and 20 ± 6% and 20 ± 4%, respectively, at 1 to 2 h. The greatest decrease in R_{rs} (26 ± 6%) was seen 1 to 2 h after 175 μg IB + SAL. We conclude that muscarinic receptors contribute to the increased bronchomotor tone of infants with BPD and that a combined dose of 175 μg IB and SAL should be used with this delivery system to ensure that the most effective and long-lasting bronchodilation is obtained in the majority of premature infants.

Metered Dose Inhaler Aerosol Characteristics Are Affected by the Endotracheal Tube Actuator/ Adapter Used—MJ Bishop, RP Larson, DL Buschman. Anesthesiology 1990;73:1263.

The authors studied the particle size of aerosols of metaproterenol produced by three different actuators designed for use in patients with endotracheal tubes in place. These were compared with the metaproterenol aerosol produced by the actuator (provided by Boehringer-Ingelheim [BI]) that was supplied by the manufacturer for use in patients whose tracheas are not intubated. The volume of particles in the respiratory size range (1.0-5.1 μm) delivered to the end of the endotracheal tube were measured using adapters designed by Intec (IT), Instrumentation Industries (II), and Monaghan (MAIS). Particle numbers were measured using a CSAS 100 scattering-aerosol laser spectrometer, and volumes were calculated by assuming the particles were spheres. The authors found that the volume of particles in the respiratory range with the IT, II, and MAIS adapters plus endotracheal tube were 11, 31, and 66%, respectively, of the volume produced in the respiratory range by the BI. When particles likely to impact before reaching the lower airways (> 5 μm) were measured, almost none was produced by the adapters plus endotracheal tube, whereas the majority of drug volume in the BI aerosol was in the > 5 μm range. It was concluded that the aerosol produced by different actuators differ from each other, that all three produced less drug in the respiratory range than was produced by the manufacturer-supplied actuator, and that large particles are effectively removed by the adapter plus endotracheal tube.

Things To Remember:

Take Advantage of the Early Deadline for Open Forum—March 20. Abstracts submitted by March 20 will be reviewed, critiqued, and their authors notified by April 26—to allow revision if necessary before the June 5th Final Deadline.

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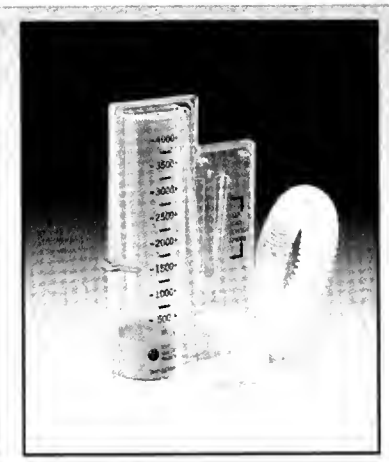

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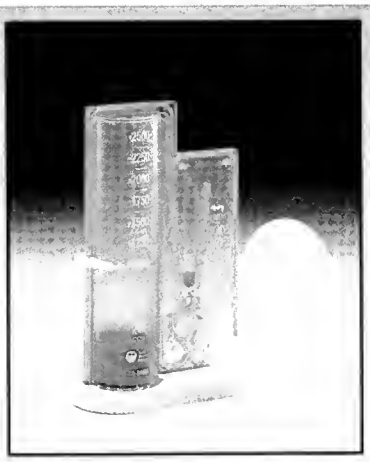

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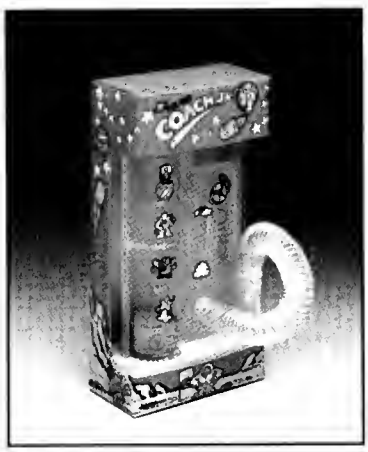

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Evaluation of Ten Manual Resuscitators across an Operational Temperature Range of –18°C to 50°C

Thomas A Barnes EdD RRT and Deborah L Stockwell

Because of the temperature extremes encountered during emergency resuscitation and transport in the field, we sought to evaluate the performance and safety of 10 adult resuscitators (5 permanent units: Hope 4, Laerdal, Lifesaver, Mark 3, and PMR; and 5 disposable units: BagEasy, Code Blue, CPR Bag, DMR, and SPUR) across an operational temperature range of –18°C to 50°C. METHOD: We tested the devices against the American Society for Testing and Materials (ASTM) Standard F-920 and the International Organization for Standardization (ISO) Standard 8382. We tested each resuscitator by using a lung model, the Bio-Tek VT-1 Ventilator Tester. RESULTS: All of the resuscitators met the ventilation requirements for V_T and f (600 mL × 20) and I:E < 1:1 except the SPUR at –18°C. Standards ASTM F-920 and ISO 8382 specify a fractional delivered oxygen concentration (F_{DO_2}) of ≥ 0.85 with attachments and ≥ 0.40 without attachments at oxygen flow of 15 L/min and $\dot{V}_E$ of 7.2 L/min (600 mL × 12). Nine resuscitators met Standards ASTM F-920 and ISO 8382 for F_{DO_2} with attachments at 21°C and 50°C, but only 3 units (Code Blue, DMR, and PMR) passed at –18°C. At 21°C, the Hope 4 had an F_{DO_2} of 0.77 ± 0.03, which was significantly lower ($p < 0.001$) than that of the other 9 resuscitators, all of which were ≥ 0.93. Nine resuscitators met the F_{DO_2} standard without attachments. All 10 resuscitators passed the tests for valve function after contamination with simulated vomitus (at an oxygen flow of 30 L/min) and for backward leakage. At the ventilation pattern recommended by the American Heart Association (AHA) (800 mL × 12) the PMR's mean F_{DO_2} dropped to 0.86 ± 0.03 because of air leaking into the bag where it attaches to the patient-valve assembly. All 10 resuscitators passed the test for mechanical shock at 21°C and 50°C, but 3 units failed at –18°C. CONCLUSION: We conclude that only the Code Blue and DMR meet the ASTM and ISO standards for operator-powered adult resuscitators across the operational temperature range of –18°C to 50°C. (Respir Care 1991;36:161-172.)

Dr Barnes is Director of Clinical Education and Associate Professor of Respiratory Therapy, College of Pharmacy and Allied Health Professions, Northeastern University; and Ms Stockwell is a Respiratory Therapist, Beth Israel Hospital—Boston, Massachusetts.

This study was completed at Northeastern University, Boston, Massachusetts; and the U.S. Army Research and Development Center, Natick, Massachusetts. Neither of the authors has a financial interest in any of the products tested.

Dr Barnes and Ms Stockwell presented some of the material in this paper at the RESPIRATORY CARE Open Forum during the 1990 AARC Annual Meeting in New Orleans, Louisiana.

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Introduction

Manual resuscitators are used widely by hospital and emergency medical personnel.[1,2] These bag-valve devices, whether disposable or permanent, should meet a series of minimum performance and safety specifications developed by the American Society for Testing and Materials (ASTM) and the International Organization for Standardization (ISO).[3,4] During emergency, transport, or maintenance ventilation, it is imperative that bag-valve units perform adequately to optimize patient oxygenation and ventilation. With the introduction of disposable manual resuscitators in recent years, health care providers with various degrees of training and experience have more options available to them when selecting bag-valve devices.

Abbreviations Used in this Paper

F_{DO_2}	— Fractional oxygen concentration
f	— Ventilatory rate
I:E	— Inspiratory-expiratory time ratio
P_{aCO_2}	— Arterial carbon dioxide tension
PEEP	— Positive end-expiratory pressure
$\dot{V}_E$	— Minute volume
V_T	— Tidal volume

A Guide to the Use of SI in this Paper*

The SI unit for compliance is liters/ kilopascal (L/kPa). (L/cm H_2O) (10.20) = L/kPa.

The SI unit for resistance is the kilopascal (kPa · s · L^{-1}.). (cm H_2O · s · L^{-1}) (0.098 06) = kPa · s · L^{-1}.

*For further information on SI (le Systeme International d'Unites), see Respir Care 1988;33:861-873 (October 1988) and Respir Care 1989;34:145 (February 1989—Correction).

According to the manufacturers, disposable bag-valve devices do offer the advantages of reduced cross-contamination and lower cost. However, recent reports by the Emergency Care Research Institute (ECRI)[5] and other investigators (personal communication, D Theron Van Hooser, 1990) suggest that disposable bag-valve devices perform poorly at extremely cold temperatures.

When ventilation with manual resuscitators is initiated, many variables may alter performance both in the field and in the clinical setting. Varying hand size and technique (one-hand vs two-hand ventilation) has been shown to significantly affect the tidal volume that can be delivered by adult bag-valve units.[6,7] It has also been reported by several investigators that ventilation pattern, resuscitator design, oxygen flow, and operator technique affect delivered oxygen concentration (F_{DO_2}).[8-12] We found only one study,[5] however, that reported on resuscitator performance at the extremely cold (–18°C) or hot (50°C) operating temperatures specified by the ASTM and ISO standards. In this report of 8 disposable manual resuscitators, the ECRI recommended not using the Ambu SPUR at –5°C because the cycling rate was reduced to 8 compressions/min.

Temperatures of ≤ 0°F (≤ –18°C) were recorded by more than ten weather stations in each of 42 states of the USA during 1988, and temperatures of ≥ 113°F (≥ 45°C) were recorded in two states (Arizona and California).[13] We were particularly interested in resuscitator performance at the temperature extremes encountered by firefighters, emergency medical technicians, police, ski patrol, and medics of the armed forces stationed world-wide. First-responder personnel must often store rescue equipment in unheated garages or in outside compartments or trunks of their vehicles. Thus, it would be reasonable to assume that resuscitators are not always stored and utilized under ideal room temperature conditions of 21°C. To evaluate resuscitator performance across the operating temperature range of –18°C to 50°C specified by the ASTM and ISO standards, we investigated 5 permanent units: Hope 4, Laerdal, Lifesaver, Mark 3, and PMR; and 5 disposable units: BagEasy, Code Blue, CPR Bag, DMR, and SPUR (Figs. 1-4).*

Materials and Methods

We investigated all the resuscitators at 70°F (21°C) for (1) F_{DO_2} with oxygen reservoir attached,

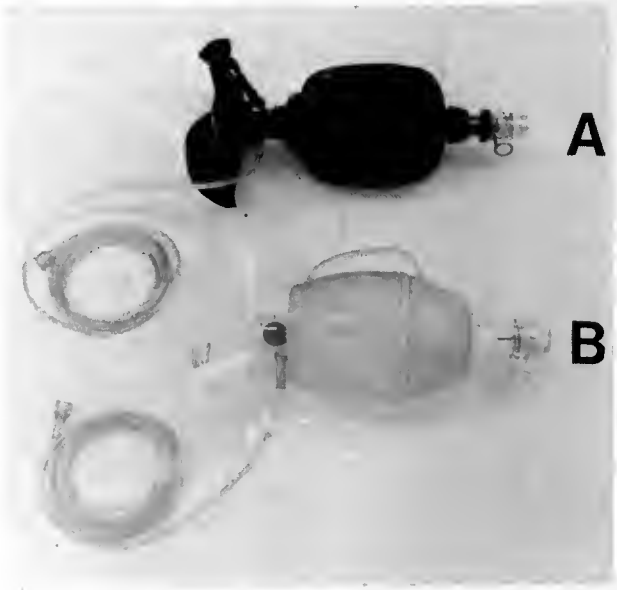

Fig. 1. Two adult manual resuscitators tested. A-Mark 3 (permanent), B-SPUR (disposable). (See Figs. 2, 3, & 4 for the other resuscitators tested.)

*Suppliers are identified in the Product Sources section at the end of the text.

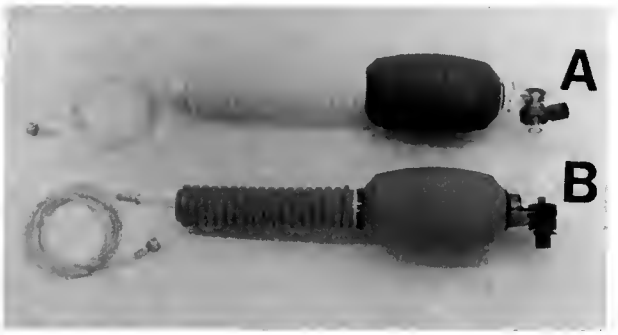

Fig. 2. Two adult manual resuscitators tested. A-DMR (Disposable), B-PMR (Permanent). (See Figs. 1, 3, & 4 for the other resuscitators tested.)

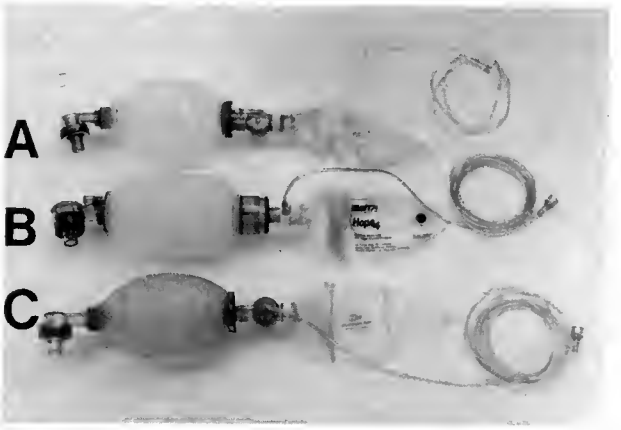

Fig. 3. Three permanent adult manual resuscitators tested. A-Laerdal, B-Hope 4, C-Lifesaver. (See Figs. 1, 2, & 4 for the other resuscitators tested.)

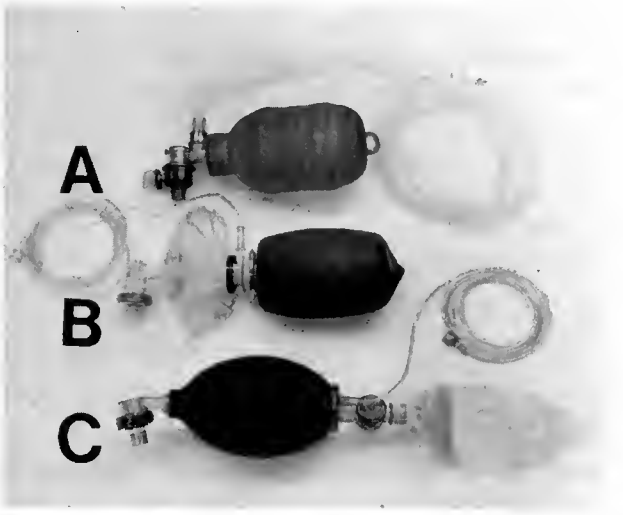

Fig. 4. Three disposable adult manual resuscitators tested. A-Code Blue, B-BagEasy, C-CPR Bag. (See Figs. 1, 2, & 3 for the other resuscitators tested.)

(2) F_{DO_2} without oxygen reservoir attached, (3) valve function in the presence of simulated vomitus, (4) valve function during high flow of 30 L/min, (5) backward leakage of exhaled gas, (6) tolerance of mechanical shock (wet and dry), (7) tidal volume capability, (8) cycle-rate capability, and (9) operation after water immersion.

The resuscitators were also investigated at 0°F (–18°C) and 123°F (50°C) for (1) F_{DO_2} with oxygen reservoir attached, (2) tidal volume capability, (3) cycle-rate capability, (4) valve function during high flow of 30 L/min, (5) valve function when wet with exhaled gas condensate (–18°C only), and (6) tolerance of mechanical shock. Four specifications tested at 21°C (F_{DO_2} without oxygen reservoir, patient-valve function with vomitus, patient-valve backward leakage, and water immersion) were not evaluated at –18°C or 50°C due to limited time available in the environmental chamber. Patient-valve function when wet with condensate is not an ISO or ASTM specification and was not tested at 21°C or 50°C because we were only interested in the effect of ice formation. The relative humidity was maintained at 53% for tests at –18°C and at 20% for tests at 50°C. Wind speed was < 3 mph during testing. The resuscitators were allowed to stabilize for a minimum of 4 hours at the ambient conditions used during testing.

F_{DO_2}

We evaluated the performance of each resuscitator by means of the test apparatus shown in Figure 5. A pressure-compensated Thorpe-tube flowmeter supplied oxygen to the resuscitator. The oxygen

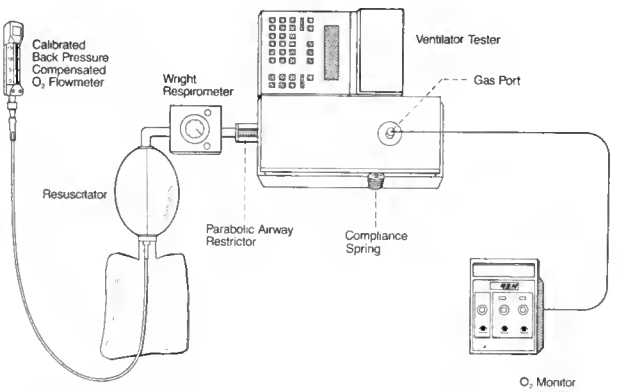

Fig. 5. Test apparatus used to evaluate manual resuscitators.

flowrate was verified before and after each test run with a Timeter Model RT-200 Calibration Analyzer. The resuscitator was manipulated to ventilate a Bio-Tek VT-1 Ventilator Tester. For tests at 21°C, a sampling probe from a Beckman OM-15 polarographic oxygen analyzer was connected to the gas port of the VT-1 to measure F_{DO_2}. A 3-point calibration of the oxygen monitor was performed immediately prior to testing each resuscitator with test gases having oxygen concentrations of 0.21, 0.80, and 1.00 to assure linearity across the entire scale. Each resuscitator was tested at 21°C for F_{DO_2} at Ventilation Patterns 1, 2, and 3 with oxygen flow of 15 L/min to the unit. For each run, the VT-1 was ventilated until the F_{DO_2} was constant (3 to 4 min). Five runs at 21°C were made for each resuscitator at each ventilation pattern (Table 1).

Because of the slow response time and inaccuracy of the OM-15 polarographic sensor at –18°C and 50°C, we used an Ametek S-3A/I oxygen analyzer system with a ceramic zirconium oxide cell heated to 650°C for F_{DO_2} measurements made in the environmental chambers at these extreme temperatures. The S-3A/I system had a response time of 100 ms to 90% of final reading and 0.01% resolution and accuracy. A 2-point calibration of the S-3A/I oxygen analyzer was performed with test gases having an oxygen concentration of 0.21 and 1.00. Gas was pumped from the gas port of the VT-1 at 100 mL/min to the oxygen analyzer system, which was located outside the environmental chamber. Only Ventilation Patterns 1 and 3 were used for tests of F_{DO_2} at –18°C and 50°C because of the limited time available in the environmental chambers.

Ventilation

Cycle Rate and Tidal Volume. At 21°C, the cycle-rate and tidal volume requirements were tested using Ventilation Patterns 1, 2, and 3 and an oxygen flow of 15 L/min (Table 1). At –18°C and 50°C, these requirements were tested using Ventilation Patterns 1 and 3 and an oxygen flow of 15 L/min. Each resuscitator was evaluated against ASTM and ISO specifications for tidal volume and cycle rate by ventilating the VT-1 for 4 minutes—configured for adult units.

Table 1. Ventilation Patterns Used To Test Resuscitators

Ventilation Pattern	Tidal Volume (V_T) (mL)	Ventilatory Rate (f) (cycles/min)	Minute Volume ($\dot{V}_E$) (L)
1	600	12	7.2
2	800	12	9.6
3	600	20	12.0

The VT-1 was set at a compliance of 0.02 L/cm H_2O [0.20 L/kPa] and a resistance of 20 cm $H_2O \cdot s \cdot L^{-1}$ [2 $kPa \cdot s \cdot L^{-1}$] as specified in the ASTM and ISO standards. The precision of the VT-1 display of tidal volume (V_T) was verified with a calibrated super syringe, and the display of ventilatory rate (f) was confirmed with a chronometer. A Wright Model L-D panel-mounted respirometer was placed between the VT-1 and the resuscitator to provide an approximate indication of the delivered V_T, which was monitored breath-by-breath by the VT-1 status display. The primary control of the ventilation pattern was the VT-1 display, which indicated the V_T, f, minute volume ($\dot{V}_E$), and inspiratory-expiratory time ratio (I:E). We ventilated the VT-1 by squeezing the resuscitator bag while observing the V_T displayed by the VT-1, and using the chronometer to determine f. When the desired V_T was reached, the bag was released and allowed to fill without restriction. Immediately after the bag had been released, the VT-1 status display was checked to confirm that the V_T, f, and $\dot{V}_E$ were correct. We were able to control the ventilatory pattern by making small adjustments in the V_T and f based on immediate feedback from the VT-1 display.

For extreme temperature testing in the environmental chamber, the RS232 data-output port of the VT-1 was connected by cable to a Macintosh IIX computer located at a window outside the chamber. The data were displayed on a two-page Apple monitor that was easily read from inside the chamber. A Bio-Tek thermal printer was located outside the chamber and connected by cable to the VT-1. A Vent-Aid Training Test Lung (TTL) was used to evaluate tidal volume and cycle-rate specifications following the drop and ice-formation tests in the environmental chambers because the VT-1 was in continuous use for the F_{DO_2} test. The TTL was also used to evaluate ventilation specifications whenever the patient valve became wet during testing (ie, from vomitus, water immersion, ice formation).

Valve Performance

High Supplemental Flows. To verify that the patient valve would not lock at high flow, oxygen flow to each resuscitator was set at 30 L/min, and the VT-1 was ventilated with Patterns 1 and 3. The flow of 30 L/min was verified with a Timeter Model RT-200 Calibration Analyzer.

Patient-Valve Backward Leakage. The potential for rebreathing was tested by connecting the resuscitator (without attachments or oxygen flow) to a 2-L anesthesia bag supplied with an oxygen flow of 15 L/min. The resuscitator was cycled at f 30/min for 3 minutes, and then the oxygen concentration in the resuscitator bag was measured with a Beckman OM-15 Oxygen Monitor. The ASTM backward leak requirement limits the increase of F_{O_2} in the resuscitator bag to less than 10 percentage points (ie, to $F_{O_2} < 0.31$).

Valve Function after Contamination by Vomitus. To evaluate this requirement, we poured 175 mL of simulated vomitus into the patient-connection port while cycling the resuscitator at f 12/min for 30 seconds. Vomitus was simulated by a mixture of two parts of baby food (Gerber Toddler Meal, Beef with Vegetables) and one part water. The patient valve was cleared of vomitus by squeezing the bag briskly and shaking any remaining obstructing material out of the exhalation port and patient-connection port. Immediately following removal of the vomitus from the patient-valve assembly, performance was assessed by using the resuscitator to ventilate the TTL with Ventilation Patterns 1 and 3. The TTL was configured with the same compliance and resistance setting as the VT-1.

Valve Function at Extreme Cold Temperature (–18°C). A spontaneously breathing healthy volunteer was ventilated with each bag-valve-mask device for 10 min at –18°C. Ventilation was accomplished with communication and cooperation between the experimenter who squeezed the bag and the subject who held the mask tightly to his face. The patient valve was checked for ice formation and normal function during the 10-min trial when exhaled gas with 100% relative humidity (at body temperature) passed through the exhalation port. Following manual ventilation of the subject for 10 min, each resuscitator was set aside with the patient-valve assembly wet from exhaled gas condensate. The wet valves were checked for valve function after 5 min and 30 min by ventilating the TTL test lung with Ventilation Patterns 1 and 3. The TTL was configured with the same compliance and resistance setting as the VT-1.

Mechanical Shock (Drop Test)

Each resuscitator was dropped 5 times from a height of 1 meter onto a concrete floor at 21°C and onto a heavy steel plate inside the environmental chamber at –18°C and 50°C. The unit was dropped in a worst-case mode so that it landed on the patient-valve and gas-intake-valve assemblies. The units were dropped five times to assure that mechanical shock was delivered to both patient-valve and gas-intake-valve assemblies at a minimum of two impact angles. Following the shock test, we inspected the resuscitator for damage and checked its performance by ventilating the TTL with Ventilation Patterns 1 and 3.

Immersion in Water

Each resuscitator was arranged in its ready-for-use configuration and dropped from a height of 1 meter into a water reservoir. After 10 seconds, the resuscitator was removed from the reservoir and shaken for not more than 20 seconds to remove water. Once free of water, the resuscitator was tested for normal function by ventilating the TTL with Ventilation Patterns 1 and 3.

The effect of resuscitator design, ventilation pattern, and temperature on F_{DO_2} was evaluated by three-way analysis of variance. A paired *t* test was used to determine the independent effect of temperature or ventilation pattern on F_{DO_2}. The effect of resuscitator design on F_{DO_2} was evaluated by one-way analysis of variance; $p < 0.05$ was considered significant. All statistical tests were performed with Exstatix version 1.0.1 software.

Results

Tables 2 and 3 list the mean (SD) F_{DO_2} values for the 10 resuscitators we studied. Tables 4 and 5 list the resuscitators' pass/fail results for the ISO

Table 2. Fractional Oxygen Concentration Delivered by Ten Resuscitators at 21°C

	With Reservoir			Without Reservoir
	600 × 12*	800 × 12	600 × 20	600 × 12
Permanent Resuscitators				
Mark 3	1.00 (0.00)†	0.99 (0.01)	1.00 (0.00)	0.44 (0.01)
Lifesaver	1.00 (0.01)	0.99 (0.02)	1.00 (0.00)	0.47 (0.01)
Laerdal	0.99 (0.00)	0.99 (0.00)	1.00 (0.00)	0.40 (0.01)
PMR	0.98 (0.01)	0.86 (0.01)	0.88 (0.04)	0.35 (0.01)
Hope 4	0.77 (0.03)	0.75 (0.04)	0.72 (0.02)	0.42 (0.02)
Disposable Resuscitators				
BagEasy‡	1.00 (0.00)	1.00 (0.00)	1.00 (0.00)	-
Code Blue‡	1.00 (0.00)	0.98 (0.00)	0.97 (0.01)	-
DMR‡	0.97 (0.00)	0.89 (0.01)	0.91 (0.01)	-
CPR Bag	0.96 (0.00)	0.96 (0.01)	0.94 (0.02)	0.43 (0.02)
SPUR‡	0.93 (0.01)	0.95 (0.01)	0.96 (0.00)	-

*Tidal volume (mL) × ventilatory rate (cycles/min).
Compliance 0.020 L/cm H_2O [0.20 L/kPa].
Resistance 20 cm $H_2O \cdot s \cdot L^{-1}$ [2 kPa · s · L^{-1}].
Oxygen flow 15 L/min.
†Mean (SD).
‡The reservoir is permanently attached.

Table 3. Fractional Oxygen Concentration Delivered by Ten Resuscitators at −18°C and 50°C

	−18°C (0°F)		50°C (123°F)	
	600 × 12*	600 × 20	600 × 12	600 × 20
Permanent Resuscitators				
PMR	0.93 (0.01)†‡	0.85 (0.01)‡	0.94 (0.02)‡	0.95 (0.02)‡
Mark 3	0.83 (0.06)‡	0.82 (0.01)‡	1.00 (0.00)	1.00 (0.00)
Lifesaver	0.81 (0.02)‡	0.70 (0.02)‡	0.99 (0.01)	0.99 (0.01)
Laerdal	0.73 (0.03)‡	0.74 (0.03)‡	1.00 (0.00)	1.00 (0.00)
Hope 4	0.54 (0.02)‡	0.48 (0.01)‡	0.91 (0.02)‡	0.89 (0.01)‡
Disposable Resuscitators				
Code Blue	1.00 (0.00)	0.99 (0.02)	0.97 (0.03)	0.94 (0.00)‡
DMR	0.89 (0.01)‡	0.81 (0.02)‡	0.92 (0.01)‡	0.89 (0.03)
BagEasy	0.66 (0.02)‡	0.64 (0.03)‡	1.00 (0.00)	1.00 (0.00)
CPR Bag	0.53 (0.01)‡	0.51 (0.01)‡	1.00 (0.00)‡	1.00 (0.00)‡
SPUR§	0.00‡	0.00‡	1.00 (0.00)‡	1.00 (0.00)‡

*Tidal volume (mL) × ventilatory rate (cycles/min).
Compliance 0.020 L/cm H_2O [0.20 L/kPa].
Resistance 20 cm $H_2O \cdot s \cdot L^{-1}$ [2 kPa · s · L^{-1}].
Oxygen flow 15 L/min.
†Mean (SD).
‡Significance level $p < 0.01$ when compared to 21°C.
§Bag would not reinflate after first cycle at −18°C.

Table 4. Performance of Ten Resuscitators against Nine Requirements of ASTM F-920 and ISO 8382 at 21°C

	F_{DO_2}		Valve Function			Mechanical Shock		Ventilation	
Resuscitator Tested	With Reservoir	Without Reservoir	With Vomitus	With Flow of 30 L/min	Backward Leakage	Drop Test 1 Meter	Water Immersion	V_T= 600ml	f= 20/min
BagEasy	●	-	●	●	●	●	●	●	●
Code Blue	●	-	●	●	●	●	●	●	●
CPR Bag	●	●	●	●	●	●	●	●	●
DMR	●	-	●	●	●	●	●	●	●
Hope 4	○	●	●	●	●	●	●	●	●
Laerdal	●	●	●	●	●	●	●	●	●
Lifesaver	●	●	●	●	●	●	●	●	●
Mark 3	●	●	●	●	●	●	●	●	●
PMR	●	○	●	●	●	●	●	●	●
SPUR	●	-	●	●	●	●	●	●	●

● Resuscitator met or exceeded standard.
○ Resuscitator did not meet standard.

Table 5. Performance of Ten Resuscitators against Five Requirements of ASTM F-920 and ISO 8382 at -18°C and 50°C

	F_{DO_2}	Valve Function	Mechanical Shock	Ventilation	
Resuscitator	With Reservoir	With Flow of 30 L/min	Drop Test 1 meter	V_T = 600 mL	f = 20/min
Code Blue	● ✓	● ✓	● ✓	● ✓	● ✓
PMR	● ✓	● ✓	● ✓	● ✓	● ✓
DMR	● ✓	● ✓	● ✓	● ✓	● ✓
Mark 3	○ ✓	● ✓	● ✓	● ✓	● ✓
Lifesaver	○ ✓	● ✓	● ✗	● ✓	● ✓
Laerdal	○ ✓	● ✓	● ✓	● ✓	● ✓
BagEasy	○ ✓	● ✓	● ✓	● ✓	● ✓
Hope 4	○ ✓	● ✓	○ ✓	● ✓	● ✓
CPR Bag	○ ✓	● ✓	○ ✓	● ✓	● ✓
SPUR	○ ✓	○ ✓	○ ✓	○ ✓	○ ✓

● Resuscitator met or exceeded standard at -18°C.
○ Resuscitator did not meet standard at -18°C.
✓ Resuscitator met or exceeded standard at 50°C.
✗ Resuscitator did not meet standard at 50°C

and ASTM requirements for resuscitator performance and safety. We found that resuscitator design significantly affects the F_{DO_2} ($p < 0.0001$). The F_{DO_2} of all the resuscitators, except the Code Blue, was significantly affected by extreme cold temperature ($p < 0.01$). Ventilation pattern significantly affected the F_{DO_2} of the Hope 4, PMR, DMR, and SPUR at 21°C ($p < 0.01$) and the PMR, Lifesaver, Hope 4, DMR, and CPR Bag at –18°C ($p < 0.01$); but ventilation pattern did not significantly affect the F_{DO_2} of any of the resuscitators at 50°C. The extreme hot ambient temperature of 50°C affected the F_{DO_2} performance of 6 resuscitators ($p < 0.01$). The F_{DO_2} of the SPUR, Hope 4, CPR Bag, and Laerdal was higher at 50°C than at 21°C; and the F_{DO_2} of the DMR and PMR was lower at 50°C than at 21°C.

All the resuscitators (except the SPUR) were successfully used to ventilate a spontaneously breathing subject for 10 min in the arctic chamber at –18°C. All the resuscitators that were tested for ice formation remained functional after being set aside for 5 min with patient valves that were wet with condensate from exhaled gas. Decreased V_T due to ice formation was encountered with two resuscitators set aside wet for 30 min. The SPUR could not be tested for ice formation because its bag would not expand after being squeezed once.

Code Blue

The Code Blue passed nine ASTM and ISO standards at 21°C. It was the only unit to deliver an F_{DO_2} of 1.00 at both –18°C and 21°C. The Code Blue passed all five ASTM and ISO specifications tested at –18°C and 50°C.

PMR

The PMR passed eight ASTM and ISO standards at 21°C, but failed the requirement for F_{DO_2} without the oxygen reservoir attached. A significant drop in F_{DO_2} occurred at Ventilation Patterns 2 and 3 at 21°C ($p < 0.001$) and at Ventilation Pattern 3 at –18°C and 50°C ($p < 0.05$). The PMR passed all five ASTM and ISO specifications tested at –18°C and 50°C.

DMR

The DMR passed eight ASTM and ISO standards at 21°C. The requirement for F_{DO_2} without the oxygen reservoir was not evaluated because the reservoir is permanently attached. A significant drop in F_{DO_2} occurred at Ventilation Patterns 2 and 3 at 21°C ($p < 0.001$) and Ventilation Pattern 3 when tested at –18°C and 50°C ($p < 0.001$). The DMR passed all five ASTM and ISO specifications tested at –18°C and 50°C.

Mark 3

The Mark 3 passed nine ASTM and ISO standards at 21°C but failed the F_{DO_2} requirement at –18°C. The Mark 3 passed all five ASTM and ISO specifications tested at 50°C.

Lifesaver

The Lifesaver passed nine ASTM and ISO standards at 21°C, but failed the F_{DO_2} requirement at –18°C. A significant further drop in F_{DO_2} occurred at –18°C with Ventilation Pattern 3 ($p < 0.001$). The F_{DO_2} did not vary significantly with changes in the ventilation pattern at 21°C or 50°C. The Lifesaver failed the mechanical shock test at 50°C because the patient-valve assembly separated into three parts, and the gas-intake valve fell into the resuscitator bag when it was dropped from a height of 1 meter.

Laerdal

The Laerdal passed nine ASTM and ISO standards at 21°C, but failed the F_{DO_2} requirement at –18°C. The Laerdal passed all five ASTM and ISO specifications tested at 50°C.

BagEasy

The BagEasy passed eight ASTM and ISO standards at 21°C. The requirement for F_{DO_2} without the oxygen reservoir was not evaluated because the reservoir is permanently attached. The BagEasy failed the F_{DO_2} requirement at –18°C. During the mechanical shock test at –18°C, the PEEP adjustment screw broke off when the unit was dropped from a height of 1 meter. The BagEasy passed all five ASTM and ISO specifications tested at 50°C.

Hope 4

The Hope 4 passed eight ASTM and ISO standards at 21°C, but failed the F_{DO_2} requirement because it delivered an oxygen concentration of only 0.77 at Ventilation Pattern 1. The Hope 4 also failed the F_{DO_2} requirement at –18°C, delivering an F_{DO_2} of only 0.54. The mechanical shock test was failed at –18°C because the oxygen reservoir shattered into pieces when dropped from a height of 1 meter. The Hope 4 passed all five ASTM and ISO specifications tested at 50°C. The F_{DO_2} increased significantly from 0.77 at 21°C to 0.91 at 50°C ($p < 0.001$).

CPR Bag

The CPR Bag passed nine ASTM and ISO standards at 21°C. The resuscitator failed the F_{DO_2} requirement at –18°C because it delivered an oxygen

concentration of only 0.53 at Ventilation Pattern 1. The mechanical shock test was failed at –18°C because the exhalation port where a PEEP device would be attached shattered when dropped from a height of 1 meter. The CPR Bag passed all five ASTM and ISO specifications tested at 50°C. The F_{DO_2} increased significantly from 0.96 at 21°C to 1.00 at 50°C ($p < 0.0001$).

SPUR

The SPUR passed eight ASTM and ISO standards at 21°C. The requirement for F_{DO_2} without the oxygen reservoir was not evaluated because the reservoir is permanently attached. The SPUR failed all five of the ASTM and ISO requirements at –18°C because the bag would not inflate after being squeezed only once. The SPUR passed all five ASTM and ISO specifications tested at 50°C. The F_{DO_2} increased significantly from 0.93 at 21°C to 1.00 at 50°C ($p < 0.01$).

Discussion

There were several clinically important differences in performance and safety among the resuscitators tested. Only the Code Blue, DMR, and PMR passed the ASTM and ISO standards for F_{DO_2} at the cold end of the operational range (–18°C). The Hope 4 (with reservoir) and PMR (without reservoir) failed the F_{DO_2} requirement at 21°C. The SPUR failed five ASTM and ISO specifications at –18°C because it failed to inflate after being squeezed only once. The extreme cold (–18°C) caused the SPUR, CPR Bag, and Hope 4 to fail the shock tolerance test, and extreme heat (50°C) caused the Lifesaver to fail the shock test.

Shock Test

Shock tolerance is an important requirement for resuscitators because time lost to repair or replace a disabled resuscitator may be critical during emergency ventilation. The most likely accident would be dropping the resuscitator onto a hard floor during a resuscitation or transport. Shock tolerance is evaluated based on the premise that a resuscitator should be functional after being dropped from 1 meter, which is the height of an average hospital bed. Previously tested resuscitators have been reported to have clinically important problems with mechanical shock resistance at an ambient temperature of 21°C.[8,9] The ASTM and ISO standards for resuscitators specify an operational range of –18°C to 50°C. Cardiopulmonary resuscitation is provided by rescuers in conditions at the extreme ends of the operating range often with equipment that is also stored at the ambient temperature.

All ten units passed the shock test at 21°C. However, three units failed at –18°C: The SPUR failed to inflate after the bag was squeezed only once, the oxygen reservoir of the Hope 4 shattered into several pieces when dropped from 1 meter, and the patient connection of the CPR Bag separated from the patient-valve assembly when dropped. Although PEEP is not addressed by either the ASTM or ISO standards, loss of ability to attach a PEEP valve or to regulate an integral PEEP mechanism may be critical to maintenance of adequate oxygenation. When dropped at –18°C, the BagEasy resuscitator had the PEEP-regulating screw break off, negating adjustment of PEEP.

The total collapse of the bag of the SPUR at –18°C is the most serious resuscitator failure we have encountered in over 12 years of testing bag-valve devices. The unit becomes immediately nonfunctional as a result of normal use at the cold end of the operating range. Emergency personnel may not be aware of the problem until attempting to use the device during a resuscitation because the bag maintains its shape until squeezed.

The separation of the patient connection of the CPR Bag when dropped at –18°C is a serious problem because the chances of a bag-valve device being subjected to rough treatment is greater in the field under extreme cold-temperature conditions. The loss of the Hope 4's oxygen reservoir when dropped at –18°C was of little consequence because the 10-mil plastic was as hard as glass, and the reservoir was not able to inflate.

The only shock-test failure at 50°C occurred when the patient valve and gas-intake valve of the Lifesaver fell apart when dropped. If all the parts could be located, an experienced user could possibly reassemble the unit; however, an unacceptable delay in emergency ventilation would occur. Further, the average rescuer may not have the skill to identify the missing parts and assemble the unit—especially the gas-intake valve, which fell into the bag when it was dropped. We suspect that the heat caused

the threaded parts to expand beyond the tolerance limit for the components to remain together.

The water-immersion test is part of the ISO standard, but it is not required by the ASTM standard. The rationale for the ISO specification is that resuscitators are often used in areas where the device could be dropped into water during resuscitation.[4] Not surprisingly, all the units floated when dropped in the water-immersion tank, and the small amount of water that entered the units was easily cleared.

F_{DO_2}

Delivery of a minimum oxygen concentration of 0.85 is often necessary for treatment of severely hypoxic patients during resuscitation. This concentration should be obtainable with oxygen flows $\leq$ 15 L/min, because flows $>$ 15 L/min exceed the normal calibration of standard adult flowmeters and could potentially lead to extremely high input flows and valve lockup.[14] Previous studies have reported that interaction between resuscitator design and flowrate significantly affects F_{DO_2} and that each factor independently affects F_{DO_2}.[8,9,11]

The Hope 4 failed the F_{DO_2} test at 21°C and –18°C because of the failure of the oxygen reservoir to inflate fully. The reservoir is manufactured of heavy 10-mil plastic that became extremely brittle and hard at –18°C and was stiff even at 21°C. The Hope 4 had an F_{DO_2} of only 0.54 at –18°C, which can be compared to its F_{DO_2} of 0.42 without a reservoir at 21°C. The extremely low F_{DO_2} of 0.77 at room temperature makes the Hope 4 unacceptable for use during normal hospital CPR. It should be noted that the Hope 4 passed the F_{DO_2} requirement at 50°C because the heat made the oxygen reservoir soft and compliant and consequently fully expandable.

All the resuscitators that had a bag-type oxygen reservoir delivered an unacceptable F_{DO_2} when tested at –18°C (Table 3). We believe this is the result of incomplete expansion of the oxygen reservoir due to the plastic becoming stiff and noncompliant at low temperatures. The combination of small reservoir volume and stiff plastic has previously been reported to affect the F_{DO_2} performance of resuscitators.[8,9] In our study the three units that passed the F_{DO_2} requirement at –18°C all had tube reservoirs. We suspect that the Mark 3, Lifesaver, Laerdal, Hope 4, and CPR Bag might all have had a higher F_{DO_2} at –18°C if they had used a tube reservoir, similar to the type used by the Code Blue.

The PMR had marginal F_{DO_2} performance at the ventilation patterns recommended by the AHA (800 mL × 12)[15] and required by the ASTM and ISO specifications for ventilation (600 mL × 20).[3,4] We believe this was due to an air leak located where the bag attaches to the patient-valve assembly. This leak appears to occur only when a larger force is applied to the bag for delivering a 800-mL tidal volume or when the cycle rate is increased from 12/min to 20/min (Table 2). Another cause of the problem may be that the volume capacity of the PMR and DMR reservoirs may be too small to accommodate a $\dot{V}_E$ above 7.2 L/min. Indeed, we have recently been notified by Puritan-Bennett that they plan to lengthen the oxygen reservoirs of the PMR and DMR to improve F_{DO_2} capability.

The PMR failed the F_{DO_2} test that requires an $F_{DO_2} \geq 0.40$ when an oxygen reservoir is not attached to the resuscitator, but we do not believe this is a clinically important problem. Resuscitators used to deliver a low F_{DO_2} are normally cycled at rates low enough to allow retarding of bag refill, which would easily increase the F_{DO_2} of the PMR to over 0.40.[12]

Valve Performance

A locked patient valve at high supplemental oxygen flow may cause excessive airway pressures.[14] The resuscitators should be capable of functioning normally at high flows of 30 L/min because the adjustment between 15 L/min and the 30 L/min portion of the flood setting is small. The nonrebreathing valves of all units functioned well with high flow of 30 L/min at –18°C, 21°C, and 50°C except those of the SPUR, which were nonfunctional at –18°C. The SPUR might have passed at –18°C if its bag had not collapsed.

Because it is very important to provide adequate ventilation during resuscitation, bag-valve units should be capable of being cleared of vomitus within 20 seconds. Clearing simulated vomitus from the

patient valves was not a problem with the resuscitators tested, and all units were functional in less than 20 seconds.

Exhaled water vapor condensing on the patient-valve assembly was a potential problem at the low end of the required operational temperature range. We found that at –18°C, the valve did not freeze after 10 min of bag-valve-mask ventilation of a volunteer. Further, the valve was still functional after being set aside wet with condensate for 5 min. Two of the units set aside wet for 30 min had enough ice to significantly lower the V_T capability; however, we do not believe this is clinically important because it is unlikely that a bag-valve unit would be set aside wet for more than a few minutes during CPR or transport.

Patient-valve backward leakage was not a problem with the 10 units tested at 21°C. Backward leakage was not tested at –18°C and 50°C because of the constraints of collecting data in the environmental chambers. However, this test should be done when possible because valve seating may be disrupted at low and high temperatures, and exhaled gas may leak back into the bag. Exposure to even low levels of inspired CO_2 may lead to increased arterial CO_2 tension (P_{aCO_2}) and to decreased arterial pH.[16]

Ventilation

The ASTM and ISO tidal-volume requirement of 600 mL is important because it is the maximum volume that typically can be delivered with one-handed use of adult manual resuscitators at the compliances and resistances found in abnormal lungs.[3,4,7] The frequency requirement of 20/min represents the upper limit typically used in adult resuscitation.[3,4,15] We found that the ventilation pattern recommended by the AHA (800 mL × 12) required a two-handed squeeze of the resuscitator bag at the compliances and resistances specified by the ASTM and ISO standards.

Conclusions

Of the 10 units tested, only the Code Blue and DMR met the ASTM and ISO standards for operator-powered adult resuscitators across the operational range of –18°C to 50°C (Tables 4 and 5). We conclude that in extremely cold ambient conditions, manual resuscitators with tube-type oxygen reservoirs deliver a higher F_{DO_2} than that delivered by manual resuscitators with bag-type reservoirs, and the difference is large enough to be clinically important. We recommend that the SPUR not be used at –18°C because the bag fails to inflate after only one cycle. Also, respiratory and emergency care practitioners should be aware that bag-type oxygen reservoirs significantly impair the F_{DO_2} capability of resuscitators used in cold weather conditions.

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PRODUCT SOURCES

Manual resuscitators:
(Note: Disposable units are sold 6 per case; prices are suppliers' list prices per unit as of November 28, 1990.)

BagEasy, Respironics Inc, Murraysville PA, $20.50

Code Blue, Vital Signs Inc, Totowa NJ, $19.85

CPR Bag, Mercury Medical Inc, St Petersburg FL, $22.75

DMR, Puritan-Bennett Corp, Overland Park KS, $19.95

Hope 4, Matrx Medical Inc, Orchard Park NY, $119.00

Laerdal, Laerdal Medical Corp, Armonk NY, $157.25

Lifesaver, Hudson RCI, Temecula CA, $177.90

Mark 3, Ambu Inc, Hanover MD, $192.50

PMR, Puritan-Bennett Corp, Overland Park KS, $125.25

SPUR, Ambu Inc, Hanover MD, $20.95

Test lung:

Model VT-1 Ventilator Tester, Bio-Tek Instruments Inc, Winooski VT

Vent-Aid Training Test Lung (TTL), Michigan Instruments, Grand Rapids MI

Calibration analyzer:
Model RT-200 Calibration Analyzer System, Timeter Instrument Corp, Lancaster PA

Oxygen monitor:
Beckman OM-15 Oxygen Monitor, SensorMedics Corp, Anaheim CA

Ametek S-3A/I Oxygen Analyzer, Ametek Inc, Pittsburgh PA

Respirometer:
Model L-D Panel-Mounted Wright Respirometer, formerly marketed by Fraser Harlake Inc, Orchard Park NY; now marketed by Ferraris, Medical Inc, Holland NY

Flowmeter:
Pressure-Compensated Thorpe-Tube Flowmeter, Puritan-Bennett Corp, Overland Park KS

Statistical software:
Exstatix, version 1.0.1, (1988), Select Micro Systems Inc, Yorktown Heights NY

Data Monitor:
Macintosh IIX computer and Two Page Monitor, Apple Corp, Cupertino CA

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Total Opacification of the Left Hemithorax after Relief of Endobronchial Obstruction by Foreign Body—A Case Report

Leland Hanowell MD, David Thurston MD, Sulpicio Soriano MD, and Walter R Martin MD

We report the case of a 2-year-old boy who aspirated a fragment of a walnut and shell. Two days after the incident, the child was in respiratory distress and was referred to our medical center. Rigid bronchoscopy was performed, and the foreign body was removed from the left main-stem bronchus; however, the left chest failed to expand fully with positive-pressure ventilation. An intraoperative radiograph revealed complete opacification of the left lung, suggestive of unilateral pulmonary edema; however, leftward mediastinal shift was suggestive of massive atelectasis and lung collapse. Repeat bronchoscopy failed to reveal additional foreign bodies; however, airway edema was observed, and a copious amount of clear serosanguinous fluid was suctioned from the left main-stem bronchus. Reexpansion of the left lung occurred following administration of nebulized racemic epinephrine. An understanding of the physiologic changes associated with endobronchial obstruction and relief of such obstruction is prerequisite to providing appropriate therapy based on radiologic and clinical monitoring during perioperative management of endobronchial foreign-body obstruction. (Respir Care 1991;36: 173-177.)

Introduction

Airway obstruction can be a life-threatening disorder in the pediatric patient. We describe a complication, detected by intraoperative radiographs, that followed removal of an endobronchial foreign body. The case illustrates appropriate anesthetic, surgical, and intensive-care management of airway obstruction, based on radiologic and clinical monitoring.

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Case Summary

History

A 2-year-old Caucasian boy was referred from a community hospital for management of respiratory distress associated with suspected aspiration of a foreign-body into the airway. According to the mother, the child had exhibited paroxysmal cough after ingesting walnuts 2 days prior to admission. A chest radiograph at the referring hospital had demonstrated air trapping in the left hemithorax. The child, who had a history of good health prior to this episode, was brought to the university medical center for further evaluation.

Physical Examination

The well-developed, anxious 14-kg child was in moderate respiratory distress—respiratory rate 40/min, heart rate 150/min, blood pressure 100/45, and temperature 38.6°C. There was no cyanosis. The child's eyes, ears, nose, and throat appeared normal,

Abbreviations Used in this Paper

IMV	—Intermittent mandatory ventilation
$P_{(A-a)O_2}$	—Alveolar-arterial oxygen-tension gradient
PCV	—Pressure-controlled ventilation
PIP	—Peak inspiratory pressure
PEEP	—Positive end-expiratory pressure

A Guide to the Use of SI in this Paper*

The SI unit for pressure is the kilopascal (kPa).
(cm H_2O)(0.098 06) = kPa.

*For further information on SI (le Systeme International d'Unites), see Respir Care 1988;33:861-873 (October 1988) and Respir Care 1989;34:145 (February 1989—Correction).

but coarse breath sounds were audible without a stethoscope. Decreased excursion of the left chest was noted. Auscultation revealed diminished breath sounds in the left chest and coarse wheezes in the right chest. Suprasternal and intercostal retractions were noted. Cardiac and other physical examinations were normal. No intravenous access was present. Figure 1 shows the radiographic appearance of the chest immediately prior to surgical intervention.

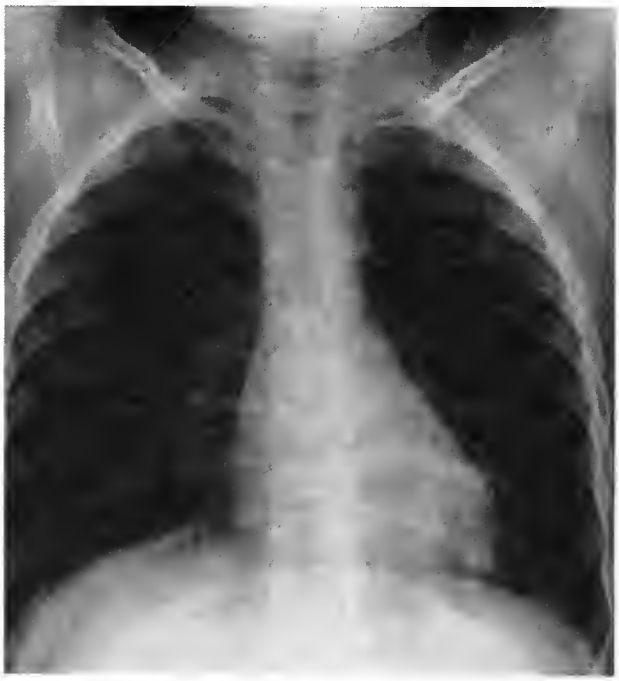

Fig. 1. Chest radiograph of a 2-year-old boy in respiratory distress associated with suspected foreign-body aspiration, taken immediately prior to bronchoscopic intervention.

Intraoperative Course

The child was taken to the operating room where 100% oxygen was administered via a standard face mask and anesthetic circle system. Halothane was delivered during spontaneous ventilation until loss of consciousness, at which time a peripheral venous catheter was inserted. While a rigid bronchoscope was prepared for insertion, atropine 0.2 mg and atracurium 5.0 mg were administered intravenously, and isoflurane was substituted for halothane to maintain general anesthesia. After the onset of neuromuscular relaxation, documented by loss of twitch response to peripheral nerve stimulation, the rigid bronchoscope was inserted and a large, hard fragment of a walnut and shell was removed from the left main-stem bronchus. Controlled ventilation was initiated, and bronchoscopic inspection of the airway was carried out. Edema and inflammation of the mucosa surrounding the carina and generalized tracheal irritation were observed.

After bronchoscopic removal of the foreign body, the left chest failed to expand fully with positive-pressure ventilation. Breath sounds in the left chest continued to be diminished despite use of peak inflation pressures of 35 cm H_2O [3.4 kPa]. An intraoperative radiograph was taken that revealed complete opacification of the left lung (Fig. 2). Bronchoscopy was repeated. No additional foreign bodies were disclosed, but a copious amount of clear, serosanguinous fluid was suctioned from the left main-stem bronchus and sent for bacteriologic culture. A nasogastric tube was inserted and attached to suction, and the bronchoscope was replaced with a cuffless endotracheal tube. Arterial oxygen saturation was 90% despite delivery of 100% oxygen. Nebulized racemic epinephrine was delivered via the inspiratory limb of the anesthetic circuit, yielding improved ventilation of the left lung and increased excursion of the left chest wall.

Postoperative Course

A radiograph taken in the recovery room showed partial resolution of the left-lung opacification. Arterial blood analysis, performed while the child was mechanically ventilated with 100% oxygen in the intermittent mandatory ventilation (IMV) mode,

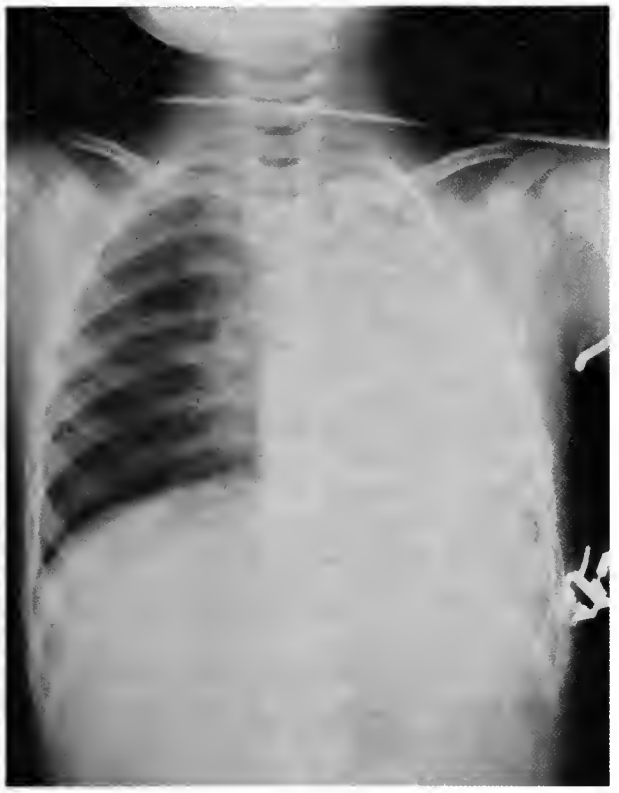

Fig. 2. Chest radiograph of a 2-year-old boy in respiratory distress, taken immediately following bronchoscopic removal of a large, hard fragment of a walnut and shell from the left-main-stem bronchus.

revealed P_{aO_2} 203 torr [27.1 kPa], P_{aCO_2} 37 torr [4.9 kPa], and pH 7.29.

Pressure-controlled ventilation (PCV), with peak inspiratory pressure (PIP) of 20 cm H_2O [2.0 kPa] and positive end-expiratory pressure (PEEP) of 5 cm H_2O [0.5 kPa], was instituted but was successfully discontinued on the first postoperative day. Arterial blood analysis revealed prompt resolution of metabolic acidosis. Reduction in the alveolar-arterial oxygen-tension gradient [$P_{(A-a)O_2}$], during the 24-hour period following bronchoscopy, suggested resolution of pulmonary shunt. Stridor, following extubation, was managed with aerosolized racemic epinephrine and intravenous dexamethasone. Antibiotics were administered, but no specific pathogens were cultured. Fever, respiratory distress, and radiographic abnormalities resolved, and the child was discharged in stable condition on the fourth hospital day.

Discussion

This case illustrates routine management of pediatric foreign-body aspiration. However, the possible cause of the left-lung opacification seen on the intraoperative radiograph was a source of much speculation. The left-lung opacification was striking, suggestive of possible unilateral pulmonary edema; however, the leftward mediastinal shift (evident by the absence of the right heart border) was more suggestive of massive atelectasis and left-lung collapse.

The high concentration of oxygen administered during bronchoscopy and the vigorous suctioning of copious secretions could have contributed to lung collapse. Absorption atelectasis can occur in poorly ventilated alveoli distal to airway obstruction, but gross atelectasis was not apparent in this child's preoperative chest radiograph. Persistently decreased breath sounds and decreased excursion of the left chest wall suggested a retained foreign body.

Repeat bronchoscopy revealed only airway edema. The improvement in ventilation of the left lung after the administration of aerosolized racemic epinephrine suggests that this airway edema may have contributed to the left-chest abnormalities, although the bronchodilatory effects of nebulized racemic epinephrine also may have been therapeutic. Copious fluid aspirated from the left lung after the radiographic detection of unilateral lung opacification, though suggestive of unilateral pulmonary edema, does not confirm that this entity was present.

The prompt clearing of radiographic abnormalities observed in this case is compatible with either atelectasis or pulmonary edema.

Atelectasis or Pulmonary Edema?

Pulmonary edema. Pulmonary edema secondary to airway obstruction has been frequently described. Price and Hecker[1] described bilateral pulmonary edema subsequent to airway obstruction due to mediastinal tumor. Shumaker et al[2] described bilateral pulmonary edema subsequent to strangulation injury in an 11-year-old boy. Lavertu and Gervais[3] reported recurrent episodes of pulmonary edema, each following partial airway obstruction due to incomplete resection of epiglottic tumor.

Surgical excision of residual epiglottic tissue resolved these episodes. Szucs and Floyd[4] described pulmonary edema following relief of laryngospasm occurring after tracheal extubation. Warner et al[5] noted pulmonary edema in an infant after a brief period of endotracheal-tube obstruction. Scherer et al[6] reported a case of pulmonary edema that followed relief of partial upper-airway obstruction (subglottic swelling) subsequent to endotracheal intubation for general anesthesia. Galvis[7] reported 20 cases of bilateral pulmonary edema after relief of upper-airway obstruction in children.

Unilateral pulmonary edema associated with unilateral airway obstruction has been previously reported. Shikhani et al[8] reported a case of unilateral pulmonary edema associated with contralateral bronchial obstruction. The mechanism was unclear; however, the unilateral pulmonary edema they described was thought to have occurred prior to alleviation of contralateral airway obstruction. Kramer et al[9] described unilateral pulmonary edema associated with right-main-stem intubation. Pulmonary edema in their three elderly patients followed resuscitation from cardiac arrest, but preceded relief of left endobronchial obstruction by endotracheal-tube repositioning.

Inspiratory effort against an obstructed airway generates negative interstitial pressure and promotes edema formation. This phenomenon is explained by the Starling equation:[10]

$$Q = K(P_c - P_I) - (\pi_c - \pi_I),$$

where Q is the rate of fluid flow across the capillary membrane; K is the filtration coefficient; P_c is the capillary hydrostatic pressure, P_I is the interstitial hydrostatic pressure, π_c is capillary oncotic pressure, and π_I is interstitial oncotic pressure. If P_I becomes a negative value, movement of fluid out of the capillaries and into the interstitium is facilitated. Stalcup and Mellins[11] measured mean pleural pressures up to –25.5 cm H_2O [–2.50 kPa] and peak inspiratory pressures up to –38.8 cm H_2O [–3.80 kPa] in pediatric asthmatics.

Capillary hydrostatic pressure can rise acutely during hypoxia due to a shift of systemic blood to the lung. Hypoxic pulmonary vasoconstriction in some segments of the lung can also shift blood flow to a reduced portion of the pulmonary vasculature. Subsequent to lung reexpansion, if capillary membrane integrity is impaired, pulmonary edema may ensue.

Atelectasis. Radiographic infiltrates and atelectasis occur in at least 30% of pediatric foreign-body aspirations, although nearly half of the children presenting with this disorder will have normal chest radiographs.[12] Our patient had no atelectasis on his admission radiograph (Fig. 1); however, the intraoperative chest film (Fig. 2) clearly shows volume loss in the left lung with leftward mediastinal shift, indicative of atelectasis or left-lung collapse. Close inspection by a radiologist may discern subtle radiographic abnormalities in the majority of children with foreign-body aspiration. Unilateral emphysema, a common finding after foreign-body aspiration, can be more readily discerned in expiratory films.

Atelectasis occurring during general anesthesia may present in a variety of clinical settings. Generally it is associated with mucus plugging and occasionally may be profound and result in hypoxemia.[13] Inadvertent intubation of the right main-stem bronchus may result in left-lung atelectasis and/or right-upper-lobe atelectasis if the endotracheal tube occludes the right-upper-lobe bronchus.

Atelectasis may be a factor in the development of reexpansion pulmonary edema. Intrapleural pressure may decrease when atelectasis develops. Atelectasis developing under circumstances of reduced intrapleural pressures is associated with impaired hypoxic pulmonary vasoconstriction.[14] Relative hyperperfusion of atelectatic lung may lead to circumstances favoring edema formation.

Summary

Atelectasis after aspiration and subsequent removal of a foreign body was associated with left-lung opacification. Reexpansion or postobstruction pulmonary edema is a possible sequela associated with the relief of airway obstruction, though not confirmed in our patient. Retained foreign body, bronchospasm, airway edema, atelectasis, and pulmonary edema must be considered in the differential diagnosis of unilateral lung opacification

after relief of airway obstruction. Effective management of airway obstruction requires radiographic and clinical monitoring for pulmonary sequelae associated with the relief of airway obstruction.

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CORRECTION

A production error occurred in AJ Beechko's response (Bear Medical) to the Monaco and Goettel letter entitled "Increased Airway Pressures in BEAR 2 and 3 Circuits following Airway-Pressure-Line Disconnection," which appeared in the February issue (Respir Care 1991;36:132-133). The fourth full paragraph should have begun as shown below. We regret the error.

The Low Pressure Alarm is the first activated in the situation described by Monaco and Goettel. This alarm is activated when a positive pressure breath is delivered that is unable to transcend *machine pressure* above and below the threshold established by the alarm setting. Next, within 7-9 seconds of the commencement of compensatory flow of $>$ 22 L/min, the Loss of PEEP Alarm will activate due to the internal-flow-transducer monitoring system.

Oxygen-Conservers, Home Oxygen Prescriptions, and the Role of the Respiratory Care Practitioner

John W Shigeoka MD

Oxygen-conserving devices have been available for several years, offering a solution to the problems of conventional continuous-flow oxygen therapy—namely, wasteful flow, inconvenience, and expense. A 1985 editorial in this journal discussed the status of oxygen-conservers (the transtracheal catheter, reservoir cannula, and demand valve) and indicated that they offered the potential to substantially reduce oxygen costs and improve the quality of life for patients with chronic hypoxemia.[1] However, physicians, vendors, payers, and patients were not anxious to support this new technology. Since that editorial was published, profound changes in home oxygen reimbursement have altered prescribing practices and attitudes toward oxygen-conservers. Further, these changes may affect the role of respiratory care practitioners in home oxygen therapy. This paper reviews these changes.

The Medicare Home Oxygen Reimbursement Problem

In 1985, the Health Care Financing Administration (HCFA-Medicare) announced a new policy of standardized prescription and reimbursement for home oxygen therapy to eliminate unreasonable and unnecessary prescription, simplify administration, and reduce program costs.[2] Health care professionals,[3,4] the National Association of Medical Equipment Suppliers, and others worked closely with HCFA to modify the new policy that became law in 1987 and was implemented in 1989. Because Medicare is the largest single payer,[5] this new policy has had major effects on home oxygen services. Unfortunately, much of the effect has been negative.

Problems with Form 484

The very complicated Certificate of Medical Necessity (CMN) for Home Oxygen Therapy (HCFA Form 484) serves as a prescription and justification.[6] It was not well understood that a physician should complete the CMN *before* a patient is discharged from the hospital on oxygen therapy. Failure to complete the form before discharge created havoc for physicians and vendors unfamiliar with the new policy. Physicians belatedly discovered that they needed the patient's medical record to provide the necessary information, such as blood gas results. Because records are notoriously difficult to obtain immediately after discharge, certification was delayed. To worsen matters, Medicare administrators denied payment if physicians failed to use approved terminology, to conform to strict criteria, and to provide specific justifications. To expedite certification and payment, some vendors entered the necessary laboratory results, approved terminology, and justifications on the form that the physicians later reviewed and signed. Unfortunately, HCFA considered this practice to be too vulnerable to inappropriate prescription and has mandated that only physicians *or their designated employees* complete the CMN. Alarmed vendors and patients began to pester physicians for the delinquent or

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revised CMN, and paperwork rapidly became onerous.

Problems with the New Rates

The new simplified monthly payment for oxygen is based on the average of local costs for liquid, cylinder, and concentrator oxygen. It applies to stationary equipment at flows of 1 to 4 L/min and is modified only for extremes of flow (50% of base if < 1 L/min and 150% if > 4 L/min), for annual inflation, and for a scheduled conversion to regional costs. There is a small supplement for portable equipment. In 1990, our Medicare administrator paid $232 plus a $40 supplement for portable equipment; the total reimbursement was one third lower than in 1987![7] The new rates have discouraged the use of portable equipment and the more convenient liquid-oxygen system.

Additional Factors

Recently the Joint Commission on Accreditation of Healthcare Organizations has required that vendors document infection control, maintenance, repair, safety, patient instruction, and proper operation of durable medical equipment in the home.[8] This documentation and the sudden jump in gasoline prices have increased vendors' operating costs.

The Effect of the New Policies

Poor prescribing practices, complex paperwork, and the new Medicare payment rates have reduced cash flow for vendors who also now have higher operating expenses. One ironic result is that some vendors now advocate less expensive (but less convenient) equipment that gives them a higher profit margin. Thus, some patients are using less convenient equipment and others are tethered to their stationary equipment.

It is clear that oxygen-conservers should receive more attention. If patients had them, vendors could make fewer deliveries and reduce their operating expenses, and patients could ambulate longer with lighter, more convenient equipment. The need is not restricted to Medicare patients; vendors have recently restructured prices, and our Medical Center costs have risen dramatically. Therefore, let us look at what oxygen-conserver technology offers today.

Oxygen-Conserver Technology

Transtracheal Oxygen Catheter

This device reduces dead space, provides a larger anatomic reservoir, reduces expiratory loss of oxygen, eliminates nasal irritation, and is less conspicuous than a nasal cannula.[1]The original simple catheter[9] has been withdrawn from the market because of problems with kinking. A more complex system (SCOOP, Transtracheal Systems, Denver CO) provided an average 55% reduction of oxygen flow in the first 100 patients and had few complications such as mucus plugging, subcutaneous emphysema, cellulitis, malplacement, and hemoptysis.[10] Others reported a higher frequency of complications that may reflect, in part, the problems associated with establishing a new program.[11] SCOOP patients must be motivated to undergo a 7-week program and be able to care for the mini-tracheostomy site and catheter.[10] Our Medicare insurer supports part of the cost of the minor surgical procedure and of the expensive ($100) catheters that are replaced every 90 days. A special low-flow flowmeter (eg, 0-4 L/min full scale) may be required for greatest oxygen savings.

Reservoir Nasal Cannula and Pendant Reservoir Cannula

These store oxygen during expiration and deliver a 20-mL bolus during early inspiration (Oxymizer and Oxymizer Pendant, Chad Therapeutics, Chatsworth CA). A 1985 paper reported that the reservoir nasal cannula reduced oxygen *flow* 75% at rest and 50% during exercise.[12] However, patients do not readily accept the device's mustache appearance. The more attractive pendant device was shown in 1986 to reduce oxygen flow 67% during exercise.[13] In contrast, a study in the home setting, published in 1987, found an average 35% oxygen savings and $71 in cost savings during a month-long evaluation.[14] In this study, discomfort substantially reduced patient compliance in using the pendant, and replacement costs of $6/week offset cost savings. When patients tolerated the pendant, oxygen savings averaged 53% and cost savings $141. This study illustrates the importance of long-

Table 1. A Comparison (for Discussion Only, Not for Patient Care) of Three Oxygen Demand Values

Attribute	Valve X	Valve Y	Valve Z
Size (cm)	9 × 6 × 12	9 × 6 × 20	10.5 × 14 × 5
Weight (g)	370	890	890
Regulator required	special pressure	conventional flow	special flow
Settings (L/min)	1-5	1,1.5,2,2.5,3,3.5,4,5	0.5,1,1.5,2,3,4,5
Ease of setting	excellent	poor to fair	good
Ease of setting for exercise	excellent	fair (both regulator & valve had to be adjusted to higher flow for exercise, then changed back)	good (regulator had to be adjusted to higher flows during exercise, then changed back)
Sensitivity	poor to fair (failed to detect inspiration in sleeping patients—manufacturer now recommends model not be used during sleep)	excellent	excellent
Laboratory savings	83% at 10 bpm* 66% at 20 bpm 32% at 40 bpm	50-60% at 8-50 bpm (computer switches to continuous flow if respiratory rate is out of 8 to 50-bpm range or is chaotic)	60% at 10 bpm, 55% at 15 bpm (valve failed to actuate in phase when respiratory rate > 20 bpm and failed to maintain O_2 saturation)
Battery operation	6-8 h	3-4 h	3-4 h
Solenoid position, if power fails	closed (no oxygen flows; override switch must be changed manually to continuous position)	open	open
Alarm loudness	poor to fair	poor to fair	poor to fair

*bpm = breaths/min.

term field investigations (in contrast to brief laboratory studies), the need for others to verify the initial reports of a device's inventor or manufacturer,[15] and the difference between oxygen savings and cost savings. Unfortunately, our Medicare carrier does not support the cost of the reservoir cannula or pendant.

Demand Oxygen Valve

This device reduces expiratory waste of oxygen by restricting delivery to inspiration, and it may minimize dead-space loss by delivering oxygen in early inspiration.[1] Their noninvasive and relatively inconspicuous nature may explain why demand

oxygen valves are popular—a recent home health care trade journal listed 11 manufacturers![16]

Unfortunately, experience remains limited. Published evaluations have involved small numbers of subjects studied for brief periods under controlled conditions, with oxygen savings reported from 55% to 88%.[17-23] Different operating characteristics of the various models make it difficult to pool information. This can be seen by examining three demand oxygen valves that have the following similarities: cost of $500, built-in rechargeable batteries, and alarms (Table 1). Valve X has advantages for ambulatory patients who are observant and able to switch to continuous mode when the batteries are discharged. Because this model can be used only during waking hours, overall oxygen savings will be less than those seen in laboratory studies. Valve Y has advantages for those less active patients who need a computerized device that switches to continuous flow when breathing rate changes drastically or the batteries are discharged. Valve Z does not seem to offer any advantage. Thus, although the three valves superficially resemble each other, they are quite different.

The lack of long-term field studies of demand valves' performance and reliability remains a major problem. The threat of rapid technologic obsolescence may explain why investigators are reluctant to perform such investigations and why vendors have been cautious about purchasing demand valves.

Because of pressing needs, it would be wise for manufacturers to pool resources, to consider trading (cross-licensing) proprietary technology, and to plan continued refinements with medical advice. Some problems with current valves might be solved by simply adopting technology from the electronics industry (Table 2). Other problems might be solved by cross-licensing; for example, the pulsed-dose concept might be traded for the computer-

Table 2. Problems with Current Demand Valves and Possible Solutions*

Problem	Comments	Solution	Precedent
Internal batteries hold limited charge	short ambulatory time and long recharge time	snap-on power packs	camcorder power packs
Heavy, bulky regulators	traditional design in brass; stationary and portable units use different fittings	compact design in aluminum; quick-connect coupler	compact cameras in alloys and plastic; electronic flash couplers for different cameras
Excess weight and size	ambulatory and stationary needs differ	modular design; simple valve for ambulation with bedside unit for more sophisticated options (see below)	laptop computer with desktop docking station for options (color CRT, modem, printer)
Sensitivity is too low for sleep yet too high for activity	different inspiratory signal strengths require different sensitivity settings	bi-level sensitivity switch; low for exercise and high for sleep	FM radio with distant and local station settings
Alarms are too quiet or dim	the elderly may not hear or see well	extra loud and bright alarms in bedside unit; telemetry for nearby family member	clock radio alarm; radio intercom

*A partial listing of problems with current demand valves (see Table 1) and potential solutions that may be adapted from precedents found in the electronics industry.

monitored-valve concept. Some problems may require additional research—for example, the safe use of demand valves during sleep[24] (Table 2). Furthermore, marketing decisions may not be medically sound; for example, a manufacturer may consider an apnea alarm too costly, yet clinicians recognize how commonly nasal cannulas become dislodged during sleep.

The technical aspects of oxygen-conservers have been reviewed by O'Donohue[25] and by Tiep and Lewis.[26]

Now and in the Future

The new Medicare policy has changed how long-term oxygen therapy is prescribed and provided. Evaluating the need for chronic home oxygen therapy must be part of hospital-discharge planning. Physicians are more objective and altruistic than profit-minded vendors and economy-minded payers when making important therapeutic decisions for their patients. Thus, physicians, not vendors, must select the most appropriate therapy based on patient need and availability of equipment,[7,27] must enter the information supporting that decision on Form 484, and must provide the vendor with enough time before discharge to properly train the patient and family to use the equipment.[6] With this approach, patients will benefit from carefully selected therapy and training, payers will no longer have to support unnecessary or inappropriate therapy, physicians and medical records departments will be spared post-discharge requests, and vendors will have better cash flow.

Respiratory care practitioners should play a larger role in the assessment for chronic home oxygen therapy. Wise physicians will learn that informed respiratory care practitioners can help them select the most appropriate equipment systems and titrate oxygen doses. The latest version (5/90) of the CMN allows designated employees of the physician to enter the necessary information that the physician must later review and sign.

Finally, there is a need to support the cost of oxygen-conservers more consistently. The Medicare half-rate provision for flows < 1 L/min discourages the use of transtracheal catheters and reservoir conservers that operate by reducing oxygen flow. It is unclear why our Medicare insurer supports replacement transtracheal catheters but not reservoir cannulas that have the same annual cost. In turn, the cost of replacing transtracheal catheters for two years would equal the cost of purchasing a demand valve. In this regard, there may be hope. The appearance on the CMN of a check box for oxygen-conserving devices suggests that HCFA might be willing to spend a little money to save money—possibly as a small supplement for disposable conservers or demand-valve rental fees. Clinicians, patients, and the home oxygen industry should be prepared when this happens!

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Long-Term Mechanical Ventilation Revisited

David J Pierson MD and Roger S Goldstein MD

Five years ago this Journal published a special issue devoted to long-term mechanical ventilation (LTMV).[1] That issue's eight articles were developed from a 1985 postgraduate course presented jointly by the American Association for Respiratory Care (AARC) and the American College of Chest Physicians (ACCP). These articles remain a valuable resource for those involved in the care of patients managed with LTMV, whether this involvement is focused in the intensive care unit (ICU), the home, or the regulating agency.

The foreword introducing the 1986 special issue[2] concluded with several comments and predictions about the future of LTMV in the home. It predicted that the number of patients receiving such therapy would increase. This has probably occurred during the intervening 5 years, although we have no more nationwide data than we did in 1985. It pointed out the need for hospital-based and community-based components of the health care delivery system to work together in preparing for and carrying out LTMV in the home, and this need has not changed. It predicted that more sophisticated devices and services would be used in the home, and indeed there has been a marked increase in this area. It emphasized the need for better funding and reimbursement for home LTMV, "as government agencies and private carriers acknowledge the financial advantages of care outside the hospital."[2] Although there is now greater awareness of the problem at several levels,[3] the modest progress in reimbursement that has occurred has been at the local level, and for specific individuals rather than across the board for all patients in need of LTMV.

Finally, the 1986 editorial noted that as the numbers of ventilator-assisted patients increased, they would be increasingly visible as a special-interest group, and might well develop "a 'National Organization of Ventilator-Assisted Persons,' with its own advocates and publications, helping its members to participate more and more successfully in society."[2] In fact, an organization exactly fitting this description—the International Ventilator Users Network*—has been in existence for a number of years. That this excellent organization is not more widely known among health professionals and patients is an example of the need for better communication about all aspects of LTMV.

The articles in the 1986 special issue focused on LTMV in patients for whom this therapy represents life support—that is, individuals unable to be weaned from the ventilator after acute respiratory failure. It was hoped that increased understanding of respiratory muscle function and the work of breathing might permit earlier and more accurate separation of weanable and unweanable patients. Furthermore, although good clinical evidence was lacking, the authors postulated that initiating ventilatory assistance electively in selected individuals might forestall acute respiratory failure and improve both gas exchange and daytime functional level in these patients. In the intervening 5 years, there has been tremendous interest in this approach to LTMV, with numerous groups of investigators studying it and several companies introducing new ventilators and other apparatuses designed for this purpose.

In this and the next issue of RESPIRATORY CARE, six articles revisit the subject of LTMV in the context

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of the 1990s. These articles were developed from a symposium presented at the 36th Annual Convention of the AARC in New Orleans on December 9, 1990. The symposium was structured to emphasize the two distinct settings in which LTMV is now used: life support and elective therapy.

This month the focus is on LTMV as life support, as in the 1986 papers. Stoller[4] first reviews the factors determining the need for ventilatory support and offers guidelines for establishing clinical unweanability. In the second paper, Nochomovitz et al[5] describe the placement options currently available for ventilator-dependent patients who no longer need ICU care. To conclude this month's articles, Gilmartin[6] draws on her vast clinical experience in managing ventilator-dependent patients outside the hospital, and offers specific practical guidelines for selecting appropriate patients and preparing them for management in the home.

Next month the focus is on LTMV as elective therapy. Stoller[7] first discusses the theoretical and physiologic reasons for attempting such therapy in the light of the natural history of chronic respiratory insufficiency in different clinical settings. Spearman[8] then provides an update on ventilators and other equipment available for home use. Finally, Goldstein and Avendano[9] place elective LTMV in a clinical perspective by reviewing what is currently known about its practical application and its effectiveness in altering the course and manifestations of chronic respiratory insufficiency.

Numerous problems remain. We still do not know how many ventilator-dependent patients there are. Such data are important both for research and for improved communication among investigators, caregivers, and patients. Paying for LTMV remains a formidable problem, both for the health care system and for individual patients and their families, and for most patients the logistics of successful home care are far more complicated than they should be. There is still a critical shortage of beds for ventilator-dependent patients in hospitals and in skilled nursing facilities. The equipment available for LTMV is often unnecessarily complicated, and poses other problems for patients and caregivers. One can only hope that a third series of special articles in another 5 years will be able to describe the satisfactory resolution of these problems, and at the same time bring exciting new information that will be of real benefit to those whose health care includes LTMV.

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Establishing Clinical Unweanability

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Introduction

For the respiratory care practitioner working in intensive care, establishing clinical unweanability is a common challenge, which is posed whenever a chronically ventilated patient has recovered enough to no longer require hemodynamic support and intensive care monitoring but not enough to be ventilator-independent.

Because no precise predictors of unweanability are available, this paper examines the available literature to develop a strategy for establishing clinical unweanability. In developing this strategy, the epidemiology of unweanability is considered first. Specifically, What is the prevalence of unweanable patients in reported series, and how many patients are currently estimated to be unweanable and, therefore, are ventilated outside of intensive care unit settings? The next section considers the statistics of negative prediction to illustrate how criteria for establishing unweanability are best judged. The third section considers available weaning predictors, both univariate and multivariate, highlighting their shortcomings in establishing unweanability but proposing a weaning "review of systems" based on approaches described in the literature.

The final section of the paper reviews some strategies that are promising maneuvers (albeit unproven) to enhance weaning success, but that when unsuccessful buttress the conclusion that the patient is unweanable.

Epidemiology of Unweanability

Table 1 reviews several available series that report the frequency of unweanable patients.[1-5] The pooled prevalence of unweanable patients in these five series (total n = 762) is 4.2%, but this frequency must be interpreted cautiously, recognizing that the five component series vary greatly in the types of patients studied. Specifically, whereas Larca and Greenbaum[2] considered unweanability among all patients admitted to a medical intensive care unit, several of the series[1,3,5] examined a different patient population to determine the rate of unweanability (ie, those patients known to be difficult-to-wean by virtue of established longstanding mechanical

Table 1. Studies Reporting the Frequency of Clinical Unweanability

Author (Date)	n	Patient Characteristics	Unweanable
Sivak (1980)[1]	15	Respiratory failure, on MV > 14 d	20%
Larca & Greenbaum (1982)[2]	573	All MICU admissions	1%
Morganroth et al (1984)[3]	11	COPD, on MV > 30 d	18%
Pardee et al (1984)[4]	133	Respiratory failure, medical & surgical ICU	9%
Aldrich et al (1989)[5]	30	Respiratory failure, deemed unweanable	37%
Total (Pooled)	762		4.2%

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ventilation). If only the three series of difficult-to-wean patients are considered, the frequency of clinical unweanability is higher (pooled prevalence 28.6%), though the number of pooled patients considered is smaller (n = 56). Although the prevalence of unweanable patients varies with the profile of patients being weaned, the clinical observation that most patients who are easily weaned are weaned within a week of intubation[6] and that many of those not weaned early will experience prolonged (and perhaps indefinite) durations of ventilatory support underscores the importance of having a systematic approach to establishing unweanability.

Estimates of the total number of ventilator-dependent patients in the United States suggest that between 6,500 and 10,000 patients are currently chronically ventilator-dependent.[6,7] Perhaps the best available estimates come from a survey of facilities caring for chronically ventilated patients in Massachusetts.[7] In polling these institutions, Make and colleagues identified 147 chronic ventilator patients in Massachusetts, of whom 62% were in acute care hospitals, 24% were in chronic care facilities, and 14% were at home. Leading causes of chronic ventilator dependence included chronic obstructive pulmonary disease, or COPD (19%), amyotrophic lateral sclerosis (12%), peripheral neuromuscular disease (12%), and central nervous system disease (8%). Based on this survey, the estimated prevalence of chronic ventilator-dependent patients is 2.8 per 100,000, or 6,573 chronic ventilator patients in the United States.

Abbreviations Used in this Paper

COPD	— Chronic obstructive pulmonary disease
MIP	— Maximal inspiratory pressure
MV	— Mechanical ventilation
MVV	— Maximal voluntary ventilation
O_2COB	— Oxygen cost of breathing
P_{aO_2}/F_{IO_2}	— Ratio of arterial oxygen tension to inspired oxygen concentration
$P_{0.1}$	— Mouth occlusion pressure
TT_{di}	— Tension time index
VC	— Vital capacity
V_D/V_T	— Dead space-tidal volume ratio
$\dot{V}_E$	— Minute ventilation
V_T	— Tidal volume
WOB	— Work of breathing

The Statistics of Negative Prediction

Figure 1 presents a 2 × 2 table, which is a standard tool by which the diagnostic (or predictive) performance of a test (or predictor) is evaluated.[8] When one assembles a 2 × 2 table for evaluating a weaning predictor, patients are placed in one of four cells: those predicted to wean who actually weaned (Cell a), those predicted to wean who could not be weaned (Cell b), those not predicted to wean who did actually wean (Cell c), and those predicted not to wean who did not wean (Cell d). Four summary statistics—sensitivity, specificity, positive predictive value, and negative predictive value—can be derived from the 2 × 2 table. Though widely cited, the sensitivity of the weaning predictor (ie, of those who actually wean, how many were predicted to wean, or a/a + c?) is perhaps the least applicable to clinical decision making. Because the denominator of the sensitivity expression consists of patients who actually weaned (a + c), sensitivity presupposes knowledge of the very outcome (ie, weanability) that the parameter is intended to predict. More clinically germane are the positive and negative predictive values, the latter of which asks the question: Of those patients predicted not to wean on the basis of a weaning parameter, how many actually fail to wean? This is the question that the clinician poses when attempting to establish

		Actually weaned?	
		Yes	No
Predicted to wean?	Yes	a	b
	No	c	d

Fig. 1. 2 × 2 table for predicting weaning status and summary statistics: sensitivity, specificity, positive and negative predictive values. Sensitivity = $\frac{a}{a+c}$, specificity = $\frac{d}{b+d}$, positive predictive value = $\frac{a}{a+b}$, and negative predictive value = $\frac{d}{c+d}$.

Table 2. Proposed Univariate Predictors of Weanability

Lung mechanics and work
Vital capacity > 10 mL/kg
Tidal volume > 300 mL
Respiratory rate < 25/min
$V_D/V_T < 0.60$
Minute ventilation < 10 L/min
Maximal voluntary ventilation (MVV) > 2 $\dot{V}_E$
Dynamic compliance > 25 mL/cm H_2O
Work of breathing < 1.8 kg·m·min^{-1}
Oxygen cost of breathing < 15% of $\dot{V}_{O_2}$
Respiratory muscle strength
Maximal inspiratory pressure (MIP) > 30 cm H_2O
Respiratory drive
$P_{0.1} < 6$ cm H_2O
Gas exchange
$P_{(A-a)O_2} < 350$ torr on F_{IO_2} of 1.0
$P_{aO_2}/F_{IO_2} > 238$
$P_{aO_2}/P_{AO_2} > 0.47$

clinical unweanability, and weaning parameters that help to establish unweanability should have high negative predictive values. Because a statistical property of both positive and negative predictive values is that they may vary with the prevalence of the outcome (ie, weaning success or failure) in the population under study, parameters with high negative predictive values will be more credible to the extent they derive from studies in which the prevalence of unweanability is low. Specifically, in a series or clinical practice where most patients are unweanable, achieving a high negative predictive value is easy because almost any predictor (of unweanability) common to the unweanable majority of patients will show a high negative predictive value (including such nonsensical predictors as having ten fingers). However, because unweanable patients comprise only a minority of patients in available series (≤ 37%, Table 1), criteria achieving a high negative predictive value in these series are better discriminators of unweanability and are therefore deemed more clinically useful.

Predictive Performance of Available Univariate Weaning Predictors

Table 2 summarizes the univariate predictors of weanability that have been proposed. As shown, many predictors reflect aspects of lung mechanics—respiratory muscle strength, respiratory drive, and work of breathing (WOB) and oxygen cost of breathing (O_2COB). Finally, gas exchange parameters and the ratio of arterial oxygen tension to inspired oxygen concentration (P_{aO_2}/F_{IO_2}) have been proposed. Tables 3-5 review the predictive performance of selected univariate weaning predictors: minute ventilation ($\dot{V}_E$) < 10 L/min, vital capacity (VC) > 10-17 mL/kg of body weight, and maximal inspiratory pressure (MIP) > 30 cm H_2O.[9-14] A review of the performance of these three predictors, reported in several studies, suggests:

Table 3. Minute Ventilation ($\dot{V}_E$) < 10 L/min as a Univariate Weaning Predictor

Study (Date)	n	Sensitivity (%)	Specificity (%)	Prediction Performance: Positive Predictive Value (%)	Prediction Performance: Negative Predictive Value (%)
Sahn & Lakshminarayan (1973)[9]*	100	92	100	100	71
Tahvanainen et al (1983)[10]	47	45	78	89	25
Krieger et al (1989)[11]†	269	NS‡	NS	93	15
Yang & Tobin (1989)[12]	41	24	69	55	37

*$\dot{V}_E$ < 10 L/min, MIP > 30 cm H_2O, and MVV > 2 $\dot{V}_E$.
†$\dot{V}_E$ < 10 L/min and MIP > 30 cm H_2O.
‡NS = not stated.

Table 4. Vital Capacity (VC) as a Univariate Weaning Predictor

Study (Date)	Criterion	n	Sensitivity (%)	Specificity (%)	Prediction Performance: Positive Predictive Value (%)	Prediction Performance: Negative Predictive Value (%)
Millbern et al (1978)[13]*	VC > 15 mL/kg	33	25	0	58	0
Tahvanainen et al (1983)[10]	VC > 10 mL/kg	47	97	13	83	50
Pardee et al (1984)[4]	VC > 17 mL/kg	133	90	60	88	NS†

*VC > 15 mL/kg and MIP > 25 cm H_2O.
†NS = not stated.

Table 5. Maximal Inspiratory Pressure (MIP) > 30 cm H_2O as a Univariate Weaning Predictor

Study (Date)	n	Patient Type	Sensitivity (%)	Specificity (%)	Prediction Performance: Positive Predictive Value (%)	Prediction Performance: Negative Predictive Value (%)
Sahn & Lakshminarayan (1973)[9]*	100	mean MV 37 h	92	100	100	71
Millbern et al (1978)[13]†	33	mean MV 37 h	25	0	58	0
Tahvanainen et al (1983)[10]	47	mean MV 5 d	68	0	74	0
DeHaven et al (1986)[14]	48	mean MV 55 h	49	100	100	12
Krieger et al (1989)[11]	269	mean age > 70 y, MV 71 h	NS	NS‡	92	21
Yang & Tobin (1989)[12]	41	NS	76	25	61	40

*MIP > 30 cm H_2O, $\dot{V}_E$ < 10 L/min, and MVV ≥ 2 $\dot{V}_E$.
†MIP > 25 cm H_2O, VC ≥ 15 mL/kg.
‡NS = not stated.

Table 6. How Long To Wean the Difficult-To-Wean Patient?

Study (Date)	No. Weaned (%)	Mean Days on Ventilator (Range)
Larca & Greenbaum (1982)[2]	8 (57%)*	29 (11-78)
Morganroth et al (1984)[3]	9 (82%)	55.8 (30-100)
Aldrich et al (1989)[5]	12 (40%)	52 (21-196)

*Of those deemed difficult to wean.

(1) With the possible exception of the study by Sahn and Lakshminarayan,[9] the negative predictive values for all three parameters in all available studies are low (±50%). (2) Although the higher positive predictive values have appeal as predictors of weanability, the low negative predictive values suggest little usefulness as predictors of unweanability. (3) None of the studies[9-14] considers the difficult-to-wean patient, for whom accurate predictors of unweanability are most needed.

Specifically, in the series by Sahn and Lakshminarayan[9] with the highest available negative

Table 7. Estimates of Pulmonary Component of Resting Work of Breathing

Author (Date)	Mean Work of Breathing			
	Normal (at rest)	Emphysema	Obesity	Kyphoscoliosis
McIlroy & Christie (1954)[15]	0.034 kg · m · L^{-1} 0.29 kg · m · min^{-1}	–	–	–
Bergofsky et al (1959)[16]	0.051 kg · m · L^{-1}	–	–	0.065 kg · m · L^{-1}
Fritts et al (1959)[17]	0.31 kg · m · min^{-1}	0.8 kg · m · min^{-1}	0.64 kg · m · min^{-1}	–
Otis (1964)[18]	< 0.05 kg · m · L^{-1}	–	–	–
Sharp et al (1964)[19]	0.035 kg · m · L^{-1}	–	0.063 kg · m · L^{-1}	–

Table 8. Oxygen Cost of Breathing (O_2COB) in Normal Subjects at Rest and in Subjects with Asthma and Emphysema at Rest and with Hyperventilation

Author (Year)	Mean O_2COB*		
	Normals (rest)	Asthma	Emphysema
Cherniack (1959)[20]	1.16	-	5.96
Campbell et al (1959)[21]	0.35	-	-
McGregor & Becklake (1961)[22]†	2.23	6.68	31.85
Harden et al (1961)[23]	2.96	-	-

*mL O_2/L resting ventilation.
†unobstructed hyperventilation.

Table 9. Transpulmonary Work of Breathing as a Predictor of Nonweanability

Study (Year)	n	Threshold Value	Comment
Peters et al (1972)[24]	55	1.80 kg · m · min^{-1}	—
Proctor et al (1973)[25]	168	1.34 kg · m · min^{-1}	13.8% false positive & negative rate with this cutoff
Henning et al (1977)[26]	28	1.70 kg · m · min^{-1}	Mean value for patients ventilated > 24 h
Fiastro et al (1988)[27]	17	1.60 kg · m · min^{-1}	Better discriminator than VC, V_T, MIP, $\dot{V}_E$
Brochard et al (1989)[28]	8	0.8 kg · m · min^{-1}	—

predictive value (71%), the mean duration of mechanical ventilation was 37 hours, with a maximal duration of 6 days, unlike the prolonged duration of mechanical ventilation seen in most series examining difficult-to-wean patients (Table 6).[2,3,5] Univariate weaning predictors regarding work of breathing (WOB) and the oxygen cost of breathing (O_2COB) have received attention recently because ventilator dependency has come to be recognized as the final common pathway of ventilatory muscle fatigue. As shown in Table 7,[15-19] available estimates of the resting WOB (pulmonary component) cluster around 0.3 kg · m · min^{-1}, with a range from 0.29 kg · m · min^{-1} to 0.33 kg · m · min^{-1}. As also shown in this table, resting estimates of WOB in emphysema and kyphoscoliosis, both conditions that may be associated with difficulty in weaning, are increased approximately 1.5 to 3-fold. The O_2COB is a manifestation of respiratory muscle work, which includes the isometric component of muscle contraction not measured by WOB estimates (which integrate the area under the pressure-volume curve). As such, O_2COB may be a more accurate indicator of muscle energy expenditure and efficiency. As shown in Table 8,[20-23]

available estimates of normal resting O_2COB range from 0.35 to 2.96 mL of oxygen/L of resting $\dot{V}_E$. At a $\dot{V}_E$ of 6 L/min, these estimates translate to 7 to 18 mL of oxygen consumed by the respiratory muscles per minute, or approximately 2% to 7% of the total resting oxygen consumption ($\dot{V}_{O_2}$). As also shown in this table, resting O_2COB is increased as much as 5-fold in emphysema. With hyperventilation (simulated exercise) O_2COB can be increased 15-fold above resting levels in normal subjects. This is evidence of the mechanical disadvantage caused by hyperinflation and the lowered efficiency of respiratory muscles in obstructive lung disease.

Based on these considerations, several investigators have examined the diagnostic value of WOB as a predictor of weanability (Table 9).[24-28] Although the threshold values for weaning cluster between 0.8 kg · m · min^{-1} and 1.8 kg · m · min^{-1} (8 to 18 J/min), the available reports do not permit a close analysis of the negative predictive values associated with these 'cut points.' Only the study by Fiastro et al[27] compares the predictive performance of WOB with other univariate parameters (eg, VC, V_T, MIP). In this study of 17 patients with respiratory failure due to parenchymal disease (only 6 of whom were on mechanical ventilation for longer than 4 days), ultimate weaning success occurred only when transpulmonary work fell below 1.6 kg · m · min^{-1} and 0.14 kg · m · L^{-1} of $\dot{V}_E$. In contrast, none of the more traditional univariate weaning predictors distinguished between weaning success and failure. Despite these tantalizing findings in the study by Fiastro et al,[27] several impediments to adopting transpulmonary WOB as a predictor of unweanability remain: (1) the limited number of available studies, (2) the lack of hypothesis-validating studies, (3) the persisting lack of information regarding the predictive performance in chronically ventilated patients, and (4) the unavailability of WOB estimates in routine clinical intensive care. Although future investigation may overcome these current shortcomings, at present WOB must be considered only a promising predictor of unweanability.

As reviewed in Table 10,[29-35] O_2COB has also been examined as a predictor of weanability in seven available studies, four of which suggest a relationship between O_2COB and weaning duration or success[29,30,32,35] but three do not.[31,33,34] Of the four supportive studies, only that by Shikora and colleagues[35] proposes a criterion that can be used as a cut point to segregate weaning success from failure (ie, $O_2COB \geq 15\%$ of $\dot{V}_{O_2}$ during mechanical ventilation). However, despite initial appeal, closer

Table 10. Oxygen Cost of Breathing (O_2COB) as a Predictor of Weanability

Study (Year)	n	Patient Type	MV Time	COB Useful?/Comment
Nishimura et al (1984)[29]	11	Cardiothoracic surgery	NS*	Yes, O_2COB higher in nonweaners (20.6% vs 7.8%),† $p < 0.05$
Harpin et al (1987)[30]	20	Mixed (COPD in 9)	16.4 d (mean)	Yes, linear correlation with days to wean
Kemper et al (1987)[31]	35	All postoperative	8.1 d (mean)	No, O_2COB did not distinguish nonweaners
McDonald et al (1988)[32]	30	Mixed (COPD in 11)	1-60 d	Yes, nonlinear correlation with days to wean
Hubmayr et al (1988)[33]	10	Mixed (COPD in 2)	1 to > 30 d	No, O_2COB did not distinguish nonweaners
Annat et al (1990)[34]	9	COPD exacerbation	3 d (mean)	No, no correlation with days to wean
Shikora et al (1990)[35]	20	18 Surgical, no COPD	All ≥ 11 d	Yes, $O_2COB \geq 15\%$† predicts unweanability

*NS = not stated.
†Percent of resting O_2 consumption.

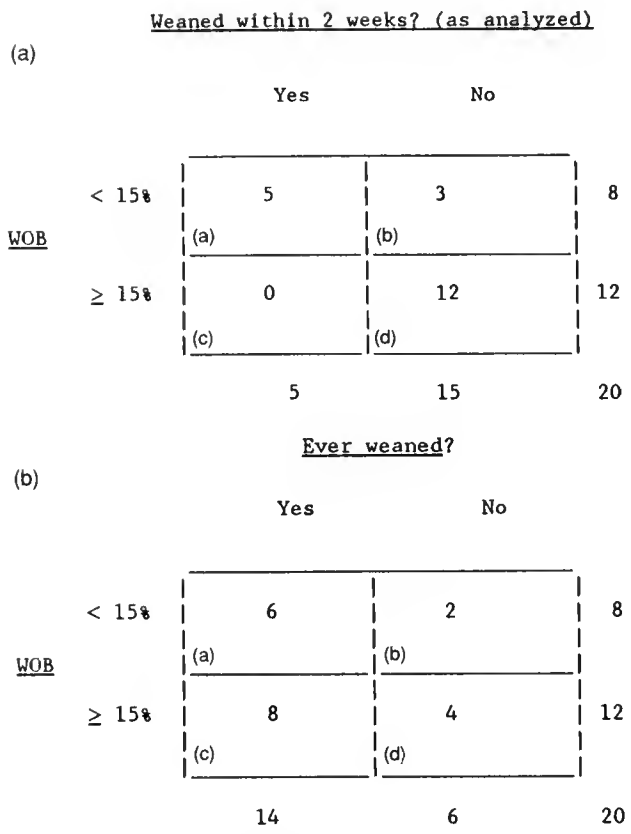

Fig. 2. 2 × 2 tables analyzing data from Reference 35. Figure 2a considers the weaning outcome as weaned within 2 weeks of study onset (as specified in the study), whereas Figure 2b considers the weaning outcome as 'ever weaned.' Note that because 9 patients were eventually weaned later than 2 weeks following the study onset, the sums of Cells a and c differ in these two analyses, with resultant differences in the predictive values. For Fig. 2a negative predictive value = 75%. For Fig. 2b negative predictive value = 33%.

scrutiny of this study suggests shortcomings with O_2COB as a predictor of unweanability. Specifically, Shikora and colleagues[35] examined 20 consecutive, stable, ventilator-dependent (≥11 days) patients, of whom 18 were ventilated postoperatively. O_2COB was determined as the difference between oxygen consumption during total ventilatory support and that during spontaneous breathing. Weaning failure in this trial was defined as the persistence of ventilator dependence 2 weeks after initiating the study, and 15 of the 20 study participants were considered weaning failures by this criterion. Figure 2a presents the study data in a 2 × 2 table to elucidate the predictive performance of the O_2COB. Notably, the negative predictive value of 100% (based on the emptiness of Cell c [patients with an $O_2COB \geq 15\%$ but successfully weaned within 2 weeks]) is appealing, but reconsideration of the data according to whether patients were ever weaned (vs weaned within 2 weeks of study onset) suggests a different conclusion. Specifically, Figure 2b re-analyzes the data using a definition of successful weaning as "ever weaned." With this rendition of the data, Cell c of the 2 × 2 table contains 8 patients who were ultimately successfully weaned, albeit later than the 2-week window proposed by the first criterion of weaning failure. In this second analysis, the negative predictive value of $O_2COB \geq 15\%$ of $\dot{V}_{O_2}$ during mechanical ventilation falls to 33%, suggesting less usefulness of this parameter as a criterion of unweanability. Overall, as with WOB, O_2COB shows several limitations as a weaning predictor:

1. Conclusions from available studies are mixed.
2. The negative predictive value of this criterion is low, even in the most supportive series.
3. A useful cut point for cost of breathing is not currently available.
4. The O_2COB is not routinely available in intensive care units.

In summary, because of low negative predictive values, available studies fail to provide a useful univariate predictor of unweanability. Not surprisingly, strict adherence to available univariate predictors with low negative predictive values can delay weaning. For example, Krieger and colleagues[11] estimated that dependence on MIP and $\dot{V}_E$ as weaning predictors would have delayed weaning in 99 of 241 (41%) successfully weaned patients in their series. The inadequate predictive performance of univariate parameters suggests that the need for mechanical ventilation is a complex phenomenon and is, therefore, more likely to be successfully summarized by accounting for at least several variables rather than just one.[3,36-38]

Available Multivariate Predictors of Weanability

Recognizing the severe limitations of univariate weaning parameters, several investigators (Table 11) have evaluated the predictive performance of multivariate indexes to predict weanabil-

ity.[3,9,11,12,39,40] As shown in Table 11, the complexity of these multivariate indexes varies. The simplest includes just two parameters (MIP and $\dot{V}_E$),[11] and the most complex incorporates a large number of clinical features.[3] Table 12 reviews the predictive performance of these multivariate indexes and

Table 11. Multivariate Indexes To Predict Weanability

Author (Year)	Index	Components (n)	Patients Examined
Sahn & Lakshminarayan (1973)[9]	Satisfy all 3 criteria	MIP, $\dot{V}_E$, MVV (3)*	100 patients, mean time on MV 37 h
Hilberman et al (1976)[39]	Nurses' assessments	NS†	125 open heart surgery patients
Morganroth et al (1984)[3]	Adverse Factor Score‡ Ventilator Score‡	Heart, lung, nutrition (21) F_{IO_2}, f, compliance (6)	11 COPD patients, MV > 30 d
Higgins et al (1988)[40]	Ventilator Dependency Score‡	Compliance, F_{IO_2} heart, bilirubin, creatinine (9)	29 open heart patients, MV ≥ 48 h
Krieger et al (1989)[11]	Satisfy both criteria	MIP and $\dot{V}_E$ (2)	269 patients ≥ 70 y, mean MV 71 h
Yang & Tobin (1989)[12]	'CROP'	Compliance, rate, oxygenation, pressure (4)	41 patients, unspecified

*MIP = maximum inspiratory pressure; $\dot{V}_E$ = minute ventilation; MVV = maximal voluntary ventilation; MV = mechanical ventilation; f = frequency; COPD = chronic obstructive pulmonary disease; CROP = compliance, rate, oxygenation, & pressure.
†NS = not specified.
‡See text.

Table 12. Summary of Available Multivariate Indexes for Weaning Prediction

Study (Year)	n	Index	Patient Type	Predictive Value: Positive Prediction (%)	Predictive Value: Negative Prediction (%)
Sahn & Lakshminarayan (1973)[9]	100	MIP >30 cm H_2O, $\dot{V}_E$ < 10L/min, and MVV ≥ 2 $\dot{V}_E$	mixed	100	71
Hilberman et al (1976)[39]	124	Nurses' assessments	open heart surgery	82	67
Krieger et al (1984)[11]	269	MIP > 30 cm H_2O,* $\dot{V}_E$ < 10 L/min	> 70 y, mean time on MV 71 h	93	15
Morganroth et al (1984)[3]	11	Adverse Factor Score† Ventilator Score†	COPD, MV > 30 d	73	97‡
Higgins et al (1988)[40]	29	Ventilator Dependence Score§	open heart surgery, MV > 48 h	NS	NS‡
Yang & Tobin (1989)[12]	41	'CROP' Score§	NS	87	72

*MIP = maximum inspiratory pressure; $\dot{V}_E$ = minute ventilation; MV = mechanical ventilation; COPD = chronic obstructive pulmonary disease; MVV = maximal voluntary ventilation; NS = not stated.
†See text and Table 13.
‡Hypothesis-generating study not confirmed in separate data set.
§See Table 12.

Table 13. Components of the Adverse Factor and Ventilator Scores of Morganroth et al[3]

Adverse Factor Score (total points assigned = 48)
Hemodynamics & vital signs
Heart rate (0-3)
Blood pressure (0-5)
Temperature (0-3)
Central venous pressure (0-2)
Arrhythmia. by type (1-4)
Vasopressors needed? (0-2)
Presence of infection
Oral antibiotics needed? (0-1)
Parenteral antibiotics needed? (0-2)
Nutrition
Calories/24 h (0-2)
Neurologic/psychiatric state
Level of consciousness (0-4)
Communication (0-4)
Mobility (0-1)
Emotional status (0-2)
Sedatives needed? (0-3)
Pain medications needed? (0-3)
Ventilator Score (total points assigned = 27)
Fraction inspired oxygen (0-4)
Level of PEEP (0-10)
Static complicance (0-4)
Dynamic compliiance (0-3)
Ventilator minute volume (0-5)
Triggered respiratory rate (0-1)

shows that the negative predictive values are generally higher than those reported with univariate predictors.[3,11,12,39,40] That comprehensive clinical assessment can help predict unweanability is suggested in a study by Hilberman et al.[39] Nurses' weaning predictions based on a clinical gestalt of 124 patients following open heart surgery demonstrated a negative predictive value of 67%, somewhat higher than most of the univariate predictors available (Tables 3-5). On the other hand, the impact of this study is limited by the fact that the patients were generally short-term ventilator patients. Similarly, most of the multivariate studies that characterize patients have examined patients mechanically ventilated for a short term, again limiting the generalizability of these study conclusions for predicting unweanability among long-term mechanically ventilated patients. One exception is the study by Morganroth et al,[3] which considered only patients on mechanical ventilators for at least 30 days. Examining 11 patients with COPD, these investigators found that VC and MIP failed to distinguish the 9 successfully weaned patients from the other 2 who remained unweanable. However, in a post-hoc analysis, Morganroth et al did find that better prediction of weaning failure could be made using a multivariate system (the Adverse Factor Score and the Ventilator Score). Components of these scores are listed in Table 13, with points assigned for increasing degrees of physiologic derangement. Recipients of a score exceeding 55 (out of a total 75 points for the sum of the Adverse Factor and Ventilator Score) were less likely to wean successfully. Despite the study's limitations—(1) it considers only a small number of patients, each of whom received multiple score determinations; (2) it is hypothesis-generating only, without validation of the predictive performance of the Adverse Factor and Ventilator Scores in a separate validation data set; and (3) its actual scoring system is cumbersome for routine clinical use—the high negative predictive value (97%) for scores exceeding 55 is compelling. Combined with data on nurses' assessments by Hilberman et al[39] the impact of this study is to emphasize that the decisions regarding unweanability can best be made by assessing multiple clinical features, many of which are cited in the Adverse Factor and Ventilator Scores.

Overall, because of the inadequate negative predictive value of available univariate predictors and the unvalidated performance of the more

Table 14. Review of Systems: Key Factors To Consider Prior to Weaning

Patient Features
Hemodynamic stability?
Control of infection?
Adequate oxygenation?
Optimized control of secretions and bronchospasm?
Preserved drive to breathe?
Psychologically ready to wean?
Optimized nutrition?
Optimized respiratory muscle function and endurance?
Features of the patient-ventilator interface
Minimized ventilator-imposed work of breathing?
Maximized endotracheal-tube size?
Inspiratory flow demands met?
Minimized auto-PEEP?

promising multivariate indexes, current assessment of unweanability cannot depend entirely on available instruments.

Rather, it appears that the best available current strategy to establish unweanability is to review remediable aspects of the patient and the ventilator system and to optimize the likelihood of successful weaning. Unweanability should be deemed to be established only after remediable factors to enhance weaning have been addressed. Key features regarding the patient, the ventilator circuit, and the patient-ventilator interface are summarized in a review of systems in Table 14, which proposes a systematic approach to establishing unweanability.

Specific Strategies To Enhance Weanability

The issues of optimizing weaning status and minimizing the WOB during mechanical ventilation have been recently reviewed extensively.[37,38,41] Table 15 lists several maneuvers to enhance weaning.

Table 15. Maneuvers To Enhance Weanability

Optimize nutrition
Optimize respiratory muscle strength and stamina
with drugs
with inspiratory muscle training
Optimize psychologic readiness
Minimize imposed work of breathing
by maximizing endotracheal-tube caliber
by minimizing auto-PEEP

As in the review of systems in Table 14, maneuvers to enhance weanability can be classified into clinical features of the patient that affect weanability and features of the ventilator-patient interface that affect weanability.

In considering selected maneuvers, accumulating data suggest that nutritional repletion is an important aspect of optimizing weanability.[2,42-44] Several lines of evidence support this concept: (1) Malnutrition causes decreased respiratory muscle strength and endurance,[43] (2) nutritional repletion of malnourished COPD patients can enhance respiratory muscle strength and endurance,[43,44] and (3) in uncontrolled series, a response to nutritional repletion is associated with enhanced weanability.[2,42]

The most optimistic reports available demonstrate the favorable effects of refeeding. Specifically, Whittaker and colleagues[44] have recently conducted a randomized, double-blind, controlled trial in 10 malnourished (< 85% ideal body weight) patients with COPD. Six patients were allocated to a refeeding regimen consisting of > 1,000 kcal above usual intake, given as an enteral formula (Isocal, Mead Johnson, Evansville IN) while four patients in the control group were maintained on their usual diet (>100 kcal above usual intake) over the mean 6-day study period. Refed patients demonstrated greater weight gain (mean 2.4 kg increase) and with significant increases in maximal expiratory pressure and in mean sustained inspiratory pressure, a measure of respiratory muscle endurance.

Although no prospective, randomized trials are available to establish the efficacy of refeeding to enhance weanability, supportive evidence comes from two observational studies. In a retrospective observational cohort study, Bassili and Deitel[42] showed that among patients ventilated for ⩾ 3 days, recipients of nutritional support (8,300-12,600 kJ/day) had a higher rate of weaning (92.8%) than a non-alimented (1,650 kJ/day) comparison group (54.5% weaning success). In a subsequent case-control study, Larca and Greenbaum[2] observed that in a group of ventilator-dependent patients on comparable nutritional regimens, those who failed to wean were less likely to show improvement in nutritional parameters (eg, albumin and transferrin levels) than those successfully weaned. Though neither of these studies establishes the efficacy of nutritional repletion for enhancing unweanability, the weight of clinical evidence suggests that unweanability cannot be established until nutritional repletion has been undertaken. Unfortunately, available studies do not establish nutritional target parameters for establishing unweanability. However, evidence suggests that improvement in respiratory muscle function and endurance can be

Other promising strategies to optimize respiratory muscle strength and endurance and thus to enhance the patient's candidacy to wean include administering drugs to enhance respiratory muscle strength and endurance[45] (eg, methylxanthines[46,47] and sympathomimetics[48,49]), and, in select instances, training the inspiratory muscles by inspiratory resistive exercises.[5,50] In the absence of controlled trials, neither strategy can be strongly endorsed, but favorable experience in uncontrolled series suggests that a trial of aminophylline and/or inspiratory resistive training may occasionally be warranted before considering a patient to be unweanable.

resistive training may occasionally be warranted before considering a patient to be unweanable.

Finally, the patient's psychologic status is an important determinant of weanability. Though infrequently given specific attention (perhaps because psychologic readiness is difficult to measure and modify), behavioral and psychologic maneuvers can enhance weanability and should be exercised before unweanability is considered established. Following scattered reports suggesting enhanced weaning by hypnosis[51,52] and biofeedback,[52] a recent randomized trial suggests that biofeedback can accelerate weaning in difficult-to-wean patients.[53] Forty awake ICU patients ventilated for $\geq$ 7 days were randomly allocated to receive biofeedback (ie, reassurance plus reinforcement for adequate tidal volume and maximizing relaxation) or control (reassurance alone) maneuvers. Though the compared groups were similar at baseline for APACHE II scores and MIP, biofeedback recipients weaned significantly faster than control patients (20.6 $\pm$ 8.9 days vs 32.6 $\pm$ 17.6 days, $p < 0.01$) and showed greater and more efficient tidal volumes during weaning. These results establish biofeedback and reassurance as additional useful maneuvers for enhancing weanability. Psychologic readiness should be optimized before unweanability can be considered established.

In addition to optimizing patient features to enhance weanability, minimizing WOB imposed by the ventilator circuit and ventilatory strategy can also aid weaning. Remediable aspects of the ventilator-patient interface include maximizing the caliber of the endotracheal tube, assuring that the patient's inspiratory flow demands are met by the ventilator, and, in the setting of obstructive airways disease, assuring that auto-PEEP is minimized.

To the extent that the endotracheal tube may impose increased WOB[54] and that imposed WOB falls as the diameter of the endotracheal tube increases, provision of an endotracheal tube of the maximum feasible diameter is advised before unweanability can be considered established.[55] The detrimental impact of small endotracheal tube caliber has been demonstrated by Shapiro et al in a lung model system.[55] As shown in Figure 3, the tension-time index (TT_{di}) approaches the fatiguing threshold of 0.15 when $\dot{V}_E$ reaches high levels through small (eg, 6-mm) endotracheal tubes. The observation that imposed WOB in vivo exceeds the WOB ascribed to the endotracheal tube using a lung model underscores the importance of maximizing the endotracheal-tube caliber in clinical care.[56] Tubes with inner diameters $\geq$ 8 mm should be used whenever technically possible.

Another source of imposed WOB is auto-PEEP.[57] Realization that auto-PEEP can be an inspiratory pressure load has fostered interest in using explicit PEEP (ie, PEEP that is 'dialed in') to offset the inspiratory pressure loading. Two recent studies demonstrate that explicit PEEP can diminish the WOB imposed by auto-PEEP in patients with dynamic hyperinflation and expiratory flow limitation.[58,59] Studying 10 ventilated patients with COPD, Smith and Marini[58] showed concomitant decreases in auto-PEEP and inspiratory WOB as applied PEEP increased from 0 to 10 cm H_2O. Although the magnitude of the decline in WOB varied greatly, features associated with the greatest diminution of WOB with applied PEEP were high

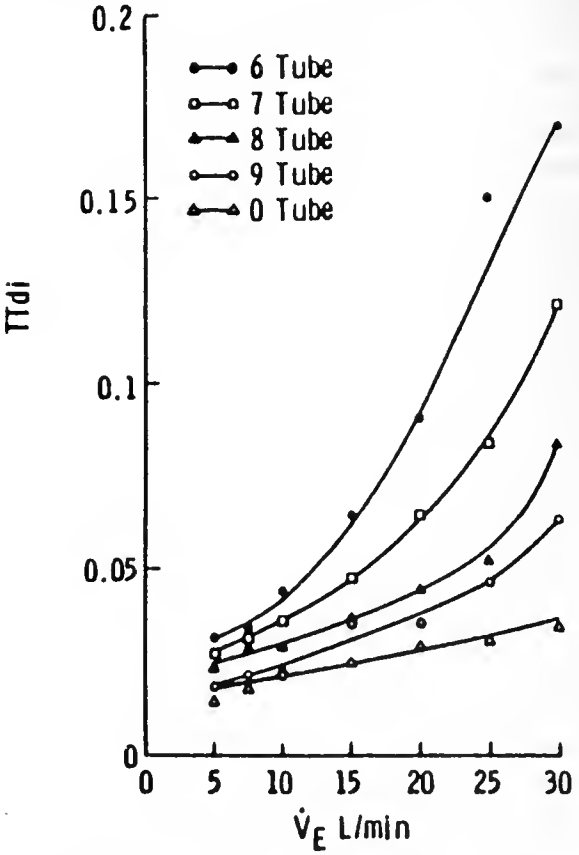

Fig. 3. Relation between tension-time index (TT_{di}) and increasing minute ventilation ($\dot{V}_E$) through endotracheal tubes of different sizes. With high $\dot{V}_E$ through small caliber endotracheal tubes (6-& 7-mm), TT_{di} approaches the fatiguing threshold of 0.15. From Reference 52, with permission.

initial WOB and stability of static peak pressure with rising levels of applied PEEP. Similar observations by Petrof et al[59] underscore considering auto-PEEP as a remediable source of imposed WOB that should be addressed before considering a COPD patient to be unweanable.

Conclusions

To the extent that weaning represents a complex interaction between the patient and the ventilator system, establishing unweanability is not easily summarized by a simple rule of thumb. Rather, unweanability can only be considered established once the patient fails to wean despite optimal management to facilitate weaning.

The foregoing discussion suggests the following conclusions:

- Available parameters are poor predictors of unweanability.
- For long-term ventilated patients, the predictive performance of available predictors is even more limited.
- Multivariate indices are more promising, but no valid predictor of unweanability is currently available.
- In view of an inability to predict unweanability, establishing unweanability is empiric (ie, requires showing that the patient cannot wean despite optimized candidacy to wean).
- Until better negative prediction is available, optimizing clinical features summarized in the patient-ventilator review of systems is a sensible approach.

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Placement Alternatives for Ventilator-Dependent Patients Outside the Intensive Care Unit

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Patient Management and Financial Consequences of Prolonged ICU Care

Advances in medical practice and the increased availability of new technology have increased the scope of care delivered to critically ill patients in the modern intensive care unit (ICU). Munoz et al suggest that critical care medicine accounts for approximately 15% of hospital costs.[1] A corollary of our ability to prolong life in the ICU, for both medical and surgical conditions, is a large number of patients dependent on mechanical ventilators for a prolonged period of time.

The introduction of the diagnosis-related-group, or DRG, system of reimbursement for hospitalized Medicare patients has resulted in institutions' often incurring substantial losses from such hospitalizations. This reality has resulted in a focus on the efficient utilization of these resources and innovative experiments for the saving of ICU resources for patients most requiring this intensity of care and this particular environment for their treatment.

The Ventilator-Dependent Patient

The nature of current practice patterns and their implication for resource utilization have resulted in the refinement of the concept of the ventilator-dependent patient. The characterization of the ventilator-dependent patients has been extended beyond the patient destined for lifelong ventilatory support. The experience with more than 737 patients in the medical intensive care unit at the University Hospitals of Cleveland suggests that only one third of these patients remain intubated for more than 1 week (Fig. 1).

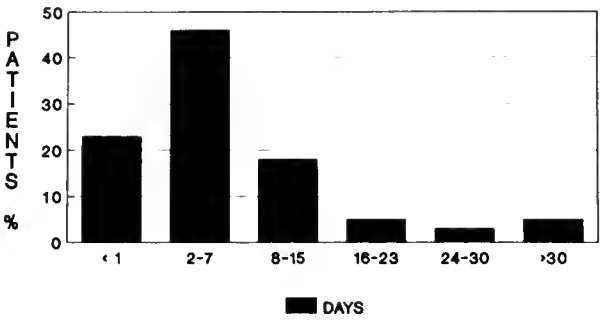

Fig. 1. Length of mechanical ventilation for 757 consecutive mechanically ventilated patients in the medical intensive care unit of University Hospitals of Cleveland.

Of the patients requiring intubation and mechanical ventilation, most are weaned and extubated rapidly; however, some require prolonged hospitalization before being weaned from the ventilator and discharged from hospital; and a still smaller number require chronic ventilation following discharge from hospital.

Chronic ventilator care may take the form of full-time mechanical ventilation or more frequently only part-time or nighttime assistance. An increasing number of patients are utilizing noninvasive ventilatory assistance in the form of negative-pressure support (cuirass, body suit) or positive-pressure nasal ventilation.[2-6] Although these patients

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are not dependent on ventilatory assistance for life support, patient and family education and financial resources are required to institute and maintain these forms of therapy.

The group requiring permanent tracheostomy and positive-pressure ventilation tend to be ventilator dependent for life support. They are often the most difficult challenges for discharge planning because of the financial and social support systems required. In addition, these patients may, despite all efforts, be unable to return to a home environment and require discharge to a chronic care facility, nursing home, or alternate community location.[7,8]

Patients in chronic respiratory failure often may have other organ-system diseases that further complicate their discharge planning and long-term management. Their care necessitates the use of often scarce intensive care beds at extremely high cost to the third-party payer, the patient, or the institution.

Managing those ventilator patients who are not speedily weaned and extubated requires specific skills that are different from those traditionally found among intensivists. The patients often require a lesser intensity of care with less reliance on high technology. The therapeutic intervention is of a more rehabilitative nature, and the family or other caregivers at home play an important role in supporting and encouraging the patient being weaned from the ventilator prior to discharge.

The realization that this process is unique and requires a special expertise and environment has been bolstered by the financial pressures in health care to study our practice patterns and modify our strategy. In addition, the paucity of alternatives for placement of ventilator-dependent patients has resulted in a reevaluation of current traditional sites, home ventilation, and consideration of innovative alternatives (eg, group residential and foster care).

Hospital Alternatives to the Intensive Care Unit

A number of attempts have been made to establish intermediate care units to facilitate the management and weaning of patients who require less intense care but are still ventilator dependent. These have been documented to varying degrees since the initial description of a respiratory care unit by Petty et al in 1971.[9]

The original respiratory care unit sought to differentiate patients who had ventilatory insufficiency from other critically ill patients. However, the evolution of critical care has been such that this appears to be a flawed separation. Although patients in surgical, cardiac, neurosurgical, and medical intensive care units may normally require ventilatory support for short periods of time during the acute phase of their illness, a number of these patients will enter a more subacute or chronic phase of recovery, with care requirements different from those available in the conventional ICU setting.

The appropriateness of caring for such chronic patients in the traditional ICU setting has also been questioned. A rehabilitative environment rather than intensive care is appropriate for many of these patients and their critical care beds are needed to accommodate patients requiring intensive care.

Prolonged weaning may also be better accomplished in a setting in which the patient can be reoriented to a conventional sleep cycle and can become a participant in the evolution of his care. Such an environment may also facilitate family participation in the patient's care and recovery. A number of models have been proposed, although the data supporting their cost-effectiveness or superiority. over the traditional environment is not yet available.

Models of Intermediate Care Units

Noninvasive Monitoring Unit

The model described by Bone and Balk[10] emphasizes the use of noninvasive technology for monitoring patients and a team approach to patient management and weaning. The unit is located on a general medical floor, with consequent lower costs. The medical director supervises the development and execution of the care plan. Medical housestaff are assigned to the unit and work with nurses assigned in a ratio of 1:3 or 1:4. Noninvasive monitoring includes pulse oximetry, capnography, and inductive plethysmography. Formal comparative data relating to the cost-effectiveness of this unit have not yet been published.

Prolonged Respiratory Care Unit

Indihar and Walker[11] described a unit for prolonged respiratory care staffed by a medical director and a team drawn from nursing, respiratory

therapy, social work, and the nutrition services. No medical house officers are assigned to this unit and the reimbursement is arranged with local third-party payers to conform to one of four levels of care intensity. This reimbursement excludes physician and pharmacy costs. The unit is apparently capable of managing patients for an indefinite period and goes beyond the intermediate care concept; however, the identification of patients who could be discharged home is emphasized. No formal study data are available for evaluation from this unit.

Special Care Unit

The special care unit (SCU) model has been developed at the University Hospitals of Cleveland and is currently being studied within the context of a grant to the Frances Payne Bolton School of Nursing and the Nursing Department of the University Hospitals of Cleveland.[12]

The unit was opened in May 1989 and is structured around an experienced group of nurse case managers and pulmonary physicians. The multidisciplinary team includes a respiratory therapist, nutritionist, and social worker. No house officers work in the SCU, but the various medical and surgical consultants frequently interact via the nurse case managers.

Patients are housed in single patient rooms in the dedicated physical plant. The daily schedule is designed to foster recovery of normal orientation and sleep patterns and family involvement. High technology is restricted, and noninvasive techniques (eg, oximetry, inductive plethysmography, and bedside echocardiography) are routinely utilized.

Following a week in one of the conventional units (medical or surgical), ventilator patients are randomized to the study. Patients may be controls (ie, remain in ICU receiving conventional therapy) or study subjects (ie, be transferred to the special care unit). Severity of illness is controlled for across the two groups by APACHE and TISS scores. No hemodynamic monitoring is employed.

Preliminary study suggests that outcome is no worse for the SCU patients, with a tendency for fewer tests to be performed. Some evidence suggests that this model will be cost-effective from the hospital standpoint, though this remains to be statistically validated.[12]

Chronic Ventilation Outside the Hospital

Home Ventilation

The majority of patients on chronic mechanical ventilation in the United States reside in their homes. Although this is clearly advantageous for many patients, it also reflects the lack of alternate sites.[13]

The decision to discharge a patient home depends on the presence of:

- a diagnosis amenable to home ventilation,
- clinical stability and a manageable care routine,
- an appropriate physical environment,
- qualified caregivers,
- financial resources necessary to meet recurring expenses, and
- informed consent.

Home ventilation, a technically feasible treatment modality for many patients, has been utilized extensively in some centers in the U.S. over the last 10 years. The general experience suggests the most success with chronic respiratory failure secondary to chest-wall deformities (eg, kyphoscoliosis) and neuromuscular disease (eg, Duchennes muscular dystrophy).[2] Such patients may achieve substantial vocational or occupational independence.[14] The results in patients with chronic obstructive pulmonary disease have been less satisfactory, although individual patients may do quite well. Persistent sensations of severe dyspnea and copious secretions have been discouraging to many mechanically ventilated COPD patients. COPD candidates for chronic mechanical ventilation should be carefully selected.

Patients should not be discharged on ventilators to nonhospital environments before they are clinically stable. The challenges of caring for ventilator-dependent patients are sufficiently great without having to contend with a fluctuating clinical course because of other complicating conditions.[15] A patient's need for a second complex technology (eg, home dialysis) usually precludes care at home. However, situations do exist in which additional complexities can be successfully introduced. An example is phrenic nerve pacing in high quadriplegia. Such patients require the presence of a backup ventilator in addition to the pacemaker. Patient, family, and caregivers are required to be well trained in the manipulation of both devices. Outside of a

handful of centers with experience managing these patients, successful application worldwide has been limited.[16]

The home environment needs to be suitable to accommodate the patient and the necessary equipment. A poorly maintained home precludes discharge of the patient to the home, and an alternative location for placement should be sought. A patient usually should not be discharged home unless a caregiver will always be available. Coverage for aides and nurses vary according to the payer. In the case of Medicare, only intermittent nursing visits are usually covered. Private payers may reimburse for continuous care, but policies are usually capped at a level designated by the individual policy. Health maintenance organizations (HMOs) and other managed care programs have a range of benefits that require individual clarification. Appropriate patient and caregiver education must be provided before the patient's discharge. The durable medical equipment required for home ventilation is usually covered by third-party payers (including Medicare and Medicaid). Disposable supplies (eg, tracheostomy tubes and suction catheters) are often not covered by third-party payers, and the patient needs to be able to cover these expenses. Patients are sometimes expected to make co-payments on the order of 20% even when insurance coverage is available, and the presence of this requirement must be clarified at the outset.

If clinical, social, and financial conditions lend themselves to consideration of home ventilation, the patient's informed consent must be obtained before the option is pursued further. The patient must be made aware of the implications of home ventilation in terms of the course of the illness, quality of life, and social and financial impact on the family. At our institution, we make it clear that choosing mechanical ventilation is not an irrevocable decision and that long-term mechanical ventilation can under appropriate circumstances be discontinued. However, in our experience, it is the rare patient who will elect to discontinue ventilatory support.

Respite Care

Although home care for the patient with appropriate resources is clearly ideal, such an arrangement results in severe stress on the extended family. Social and recreational activities are always limited by considerations for the ventilator-dependent loved one. Some hospitals and intermediate care facilities make provisions to allow families to take vacations or just have physical and emotional respite from the burden of chronic care. Such respite may be particularly needed during intercurrent illness, should be viewed as an integral part of health care, and should clearly be recognized under specific guidelines by private and public payers.

Foster Care

It is possible for persons who are not family members to provide home care for ventilator-dependent patients. The importance of pursuing the possibility of community, rather than institutional, care for long-term ventilator patients cannot be overemphasized. Precedents for foster care of such patients have been established, and this could become a more frequently utilized alternative, with formal screening of interested parties and regular follow up by the funding agency and the attending physician caring for the patient.

Nursing-Home Care

Stable ventilator-dependent patients for whom home care is not feasible may require nursing-home placement. However, this alternative is limited nationwide by the small number of nursing-home beds available for ventilator patients. In many states, reimbursement for such care is limited, making the care of ventilator patients a financially nonviable option for many nursing homes.[17]

Reimbursement from private payers is often capped, thereby limiting long-term coverage. Coverage from public sources (eg, Medicare, Medicaid) is usually insufficient to adequately cover the institution's costs—hence the small number of beds available nationwide for this purpose. Although states are mandated by the federal government to provide nursing-home care for the chronically disabled, coverage varies from state to state. In some states (eg, California and Delaware) additional reimbursement is made to nursing homes for ventilator-dependent patients thereby facilitating this alternative. However, in some states the nursing-

home options are so limited for Medicaid patients that cases of out-of-state placement to accommodate these patients have been reported.

An informal survey of acute care hospitals conducted in Ohio in August 1990, revealed that at least 50 ventilator-dependent patients were waiting for nursing-home placement. These patients accounted for 4,345 patient days while waiting for placement (personal communication, S Parran, 1990). The economic implications of this situation for acute care facilities are obvious.

Even though funds allocated for nursing-home placement are limited and often inadequate, they do exist; but such funds are not usually made available to cover the provision of caregivers in the home or group residential facility. The patient who wishes to live outside a nursing home cannot usually obtain public funds to cover the cost of caregivers and a suitable home environment. However, notable exceptions to this generalization do exist. For example, Larry Mcafee, a 34-year-old civil engineer, sustained a permanent high-cervical-cord injury in a motor vehicle accident in 1985. The accident left him ventilator dependent. State benefits for a home attendant were denied, but Mcafee was reluctant to be placed in a nursing home. He successfully petitioned the courts in Georgia for the right to be disconnected from the ventilator. Consequently, state funds were allocated to establish a group residence for ventilator-dependent patients in Augusta, Georgia. Mcafee currently resides in that residence with state support.

Group Community Facilities

In recent years, alternatives to placement of ventilator-dependent patients at home or in nursing homes have been considered—the concept of group accommodation in a noninstitutional setting. Such an approach for clinically appropriate patients offers a number of potential advantages:

- shared attendants,
- shared cost,
- a community residential setting (vs an institution),
- patient independence encouraged, and
- vocational and occupational activity facilitated.

The limited experience with this approach in the United States has not been related to any concern about the validity and favorability of the concept but rather to the lack of public-payer support, legal restrictions on persons attempting to care for chronically ill patients (health codes), and consequent liabilities for prospective private investors.

The best example of the noninstitutional group facility is the product of the work of Mary Williams RN who directs such a private-sector effort (New Start Homes Inc, Chatworth CA). This pilot project in group residential living was initiated in 1982. Through tireless work by Ms Williams, a special waiver program was introduced by the California legislature in 1987. The Congregate Living Health Facilities Law was an amendment to the Health and Safety Code and allows for public funds to be allocated for qualifying patients requiring this care. At the end of 1990 this venture owned three houses in residential areas with a total of 14 ventilator-dependent patients. An additional small number of disabled nonventilator patients with neuromuscular disease are also resident in these homes.

This private initiative serves as an outstanding example of a humane and efficient method of providing long-term care that allows the patient to maintain a place within a community without the institutionalization of a nursing home or the severe family disruption associated with home care. Current initiatives are aimed at obtaining appropriate Medicare certification that would allow for reimbursement from federal sources for group residential care.

The International Experience

Studies have not yet documented prolonged ventilatory care in intensive care units and the need for modulation of the care milieu. Less data are available about cost—perhaps because socialized systems as yet do not have the methodology or the incentive to monitor and 'fine tune' the cost of maintaining these more chronic critically ill patients.

I have observed that home care in general is less accessible and less accepted by the medical community outside the United States. The full spectrum of home care (and respiratory care is no exception) as it has developed in the United States is not available outside this country. Availability is limited and acceptance by physicians seems to be

constrained by the lack of appropriate resources and service industries to meet patient needs.

In most parts of the world, chronic ventilatory support is not a well-developed field of expertise. Physicians in other countries may be more likely to make unilateral decisions regarding chronic or prolonged life support than would physicians in the United States. Chronic ventilator care outside the U.S. has focused on postpoliomyelitis patients, high quadriplegics, and patients with neuromuscular disease. However, two programs for chronic ventilator care in England and France are excellent models.[18,19]

In France, a national system for care of patients with chronic respiratory insufficiency is in place. A central organization, the National Home Care Association for Chronic Respiratory Insufficiency (ANTADIR), is the national organization responsible for a decentralized publicly funded program to provide home oxygen and ventilator care. In 1986, approximately 1,200 patients were reported to be on full-time or part-time ventilatory support in that system. A breakdown of patient diagnoses and the number of patients on positive- and negative-pressure ventilation is not available.

In Great Britain, a program has been reported that involves intermediate care for patients requiring ultimate discharge from hospital. A limited framework for group residential living also exists, and facilities for respite care have been developed. The program is geographically limited but open to patients from anywhere in the country. The program was developed originally to service the needs of the Intensive Care Unit at St Thomas Hospital in London. This combined effort involving private and public funding is limited in its scope by its size and location but has provided service to several hundred patients since its inception.

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Long-Term Mechanical Ventilation: Patient Selection and Discharge Planning

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Introduction

Over the past decade, an increasing number of patients have been maintained in the home on life-support equipment. This population of patients has consisted of (1) those unable to be weaned from ventilatory support after an acute episode of respiratory failure and (2) those who are being electively ventilated with intermittent noninvasive modes of ventilatory support.[1-17]

Successful outcome for ventilator-assisted patients depends on many factors: appropriate selection of patients for care outside the hospital, stability of the patient, patient and family motivation, and their ability to learn. Comprehensive discharge planning and coordination of hospital and community services are major components of care, and play a key role in the success of home care.

Patient Selection Based on the Underlying Disease Process

If the goals of home mechanical ventilation are to extend life, enhance quality of life, provide an environment to enhance individual potential, reduce morbidity, improve physical and physiologic function, and be cost-effective,[1] proper selection of patients is extremely important. (Not every patient who is unable to be weaned from mechanical support is candidate for home care, and for some the home ventilation option should not even be considered or offered.)

Appropriate Candidates for Home Ventilation

Most of the reported experiences with home ventilator care have shown that patients with neuromuscular or skeletal disorders are the best candidates for long-term ventilatory support.[2,13,18-24] Table 1 lists the medical conditions that may necessitate long-term mechanical ventilation, classified according to neuromuscular, chest-wall and diaphragmatic disorders, and primary pulmonary disease.

Why are patients with neuromuscular or skeletal disorders the best candidates? Many patients with these disorders can be maintained on noninvasive ventilatory support. They do not have problems with airflow obstruction, and their ventilation and oxygen requirements do not change very much over time. Some patients (such as those with kyphoscoliosis, poliomyelitis, thoracoplasty, and Ondine's curse) may only need ventilation at night, thus decreasing the need for backup ventilators and allowing them much more mobility. Because some of the neuromuscular disorders are slowly progressive, a decision about long-term ventilation can be made on an elective rather than emergency basis, which lessens complications and shortens the hospital stay. Lastly, many patients with neuromuscular or skeletal disorders seem to adjust better to the increasing disability associated with their disease than those with chronic lung disease. Many patients with neuromuscular or skeletal disease have had to deal with their disability throughout their lives but

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patients with lung disease are affected later in life and therefore may feel particularly limited by their dyspnea (physically and psychologically).

Although, in general, those with neuromuscular disorders are the best candidates for long-term ventilation in the home, exceptions do exist. If a child with a neuromuscular disorder develops chronic lung or airway involvement because of multiple infections related to mechanical ventilation and tracheostomy, he may not be medically stable; or if he has had inadequate ventilation and periods of hypoxemia, he may develop cor pulmonale and pulmonary hypertension.[1] The medical caretakers need to be aware of these problems and be sure that the child is stable before discharge to home.

Inappropriate Candidates for Home Ventilation

Of the many patients with neuromuscular disease, those with amyotrophic lateral sclerosis (ALS), in general, do not adjust well to their progressive disability. These patients tend to have a much more rapid progression, with loss of swallowing and speech and total dependency on their caretakers. The loss of independence related to this disease causes depression and feelings of hopelessness, which makes it very difficult to provide successful home care.

Other disorders that stimulate much controversy related to the use of long-term support fall into the category of primary pulmonary disease (Table 1). Adults with severe pulmonary disease who need ventilatory support usually have concomitant pulmonary hypertension and cor pulmonale; may also have rapid deterioration secondary to changes in airflow obstruction, infections, and heart failure; and are more difficult to manage at home because of the potential for frequent changes in ventilation and oxygen requirements. Children and adults with cystic fibrosis are not considered good candidates; placing them on a ventilator does not diminish the problems with copious secretions and recurrent infections, and the ventilator and artificial airway may actually increase the chance of infection.

Patients who have pulmonary fibrosis generally are not considered good candidates for ventilatory support, even in an acute situation, because of their need for very high ventilatory pressures and oxygen. Their hypoxemia and dyspnea may be refractory to medical management, and the mechanical ventilator may actually increase their work of breathing.[1]

Infants with bronchopulmonary dysplasia may not be clinically stable until they are 1-2 years of age because of the immaturity of the lungs and airways. This instability may necessitate that they remain

Table 1. Conditions that may Necessitate Long-Term Mechanical Ventilation

Neuromuscular Disorders
- Central Nervous System
  - Central hypoventilation syndromes
  - Ondine's Curse
  - Arnold-Chiari malformation
- Spinal Cord
  - Traumatic injuries
  - Thoracic myelomeningocele
  - Syringomyelia
- Anterior Horn Cell (Lower Motor Neuron)
  - Poliomyelitis
  - Spinal muscle atrophy (Werdnig-Hoffman)
  - Amyotrophic lateral sclerosis*
- Muscle
  - Muscular Dystrophy (Duchenne's, limb girdle, myotonic dystrophy)
  - Congenital myopathies
- Peripheral Nerve
  - Phrenic neuropathies
  - Diaphragmatic paralysis
    - Idiopathic
    - Postsurgical
  - Guillain-Barre syndrome

Chest-Wall and Diaphragmatic Defects
- Kyphoscoliosis
- Postsurgical (thoracoplasty)
- Diaphragmatic hernia

Primary Pulmonary Disorders
- Tracheomalacia
- Bronchiectasis
- Bronchopulmonary dysplasia
- Chronic aspiration
- Chronic bronchitis, emphysema*
- Cystic fibrosis*
- Interstitial lung disease (multiple causes)*
- Adult respiratory distress syndrome

*Less appropriate disorders for home mechanical ventilation

in the hospital until sufficient maturation occurs to assure a safe transition to the home without adverse consequences.

Clinical and Physiologic Stability

Co-Existing Disease

Many patients, especially the elderly population, may have other medical diseases that will interfere with successful discharge and home care. A cardiac evaluation may be appropriate, especially in the elderly, to rule out underlying cardiac dysfunction. When these patients are immobilized in the bed or chair, cardiac dysfunction may not be apparent; but, when a program of rehabilitation is started, the underlying disease interferes with their physical progress.

Patients with terminal cancer who are on ventilatory support are not appropriate candidates for home care because of the time and expense needed to prepare the patient and family for the transition to home. Even with full-time professional caretakers in the home, the family may need to be responsible for some of the care. The equipment and supplies needed by these patients will also be very expensive and probably not justified from a cost-containment point of view.

Other patients who may not be candidates for home care are those with unstable psychiatric illness. Their potential inability to make rational decisions or judgments about their care puts them at high risk for major physical and mechanical problems. They would need to have a family willing to take total responsibility for their care.

Medical Readiness for Discharge

If home care is to be successful and cost-effective, the patient must meet certain criteria of clinical and physiologic stability (Table 2). The patient should be stable for at least 2 to 4 weeks prior to discharge. You cannot write a home care plan for a moving target (personal communication, A Goldberg, presentation at annual ACCP meeting, Toronto, Canada, 1990). We may all have anecdotal reports of patients who have not met these criteria and survived at home, but, in general, this places a tremendous burden on the patient, family, and caregivers.

The critically ill patient (one who needs frequent changes in ventilator settings, high oxygen concentrations, and positive end-expiratory pressure [PEEP]) is still in need of intense medical care and is not ready for discharge.[1,8,25] The ventilators used in the home, even though sophisticated, would require modifications to provide higher oxygen concentrations, PEEP, and adequate flow for synchronized intermittent mandatory ventilation (SIMV),[1,8,15] which would greatly increase the potential for malfunction and error.

The patient who requires frequent laboratory work because of unstable metabolic and acid-base status, which in turn necessitate changes in medications, is not ready to be discharged. The same applies to the patient who is having multiple episodes of infection and requires frequent coverage with antibiotics.

In infants and children, nutritional status is very important not only for growth and development but for increasing their weaning potential.[16] Failure to thrive in an infant may be indicative of irreversible damage, and caring for this infant in the home may not be an option. Nutritional status needs frequent

Table 2. Patient Stability and Medical Readiness for Discharge

Control or absence of sustained dyspnea
Acceptable arterial blood gases, with $F_{IO_2} < 0.40$
Stable ventilator settings
$F_{IO_2} < 0.40$
Assist/control or pressure-limited mode (pediatrics)
Limited use of PEEP
Minimal fluctuations in airway resistance and compliance
Stable 'free time' periods
Optimal metabolic and acid-base status
Absence of acute infectious processes
Absence of life-threatening cardiac dysfunction or arrhythmias
Other organ systems stable
Ability to clear secretions and protect airway
Adequate nutrition
Progression of growth and development (in children)
If artificial airway, a tracheostomy, not an oral or nasal airway
Able to handle the daily stressors
Management at home expected to be stable, without the need for readmission within at least 1 month

monitoring to be sure that the patient is either maintaining or gaining weight and that the method of providing nutrition is appropriate for the patient.

If the patient is being ventilated through an artificial airway, it must be a tracheostomy, not an oral or nasotracheal tube. The patient must also be able to clear secretions either through suctioning or adequate coughing. Lastly, the patient and family must be able to deal with the stress involved in home ventilator care.

Nonmedical Aspects of Patient Selection

The medical diagnosis and stability of the patient are only part of the process related to successful home care. Each patient with whom we deal in the hospital comes to us with his own individual strengths and weaknesses; it is these aspects and other individual characteristics that play a major role in successful outcomes.

Internal Factors—Coping Skills

The patient may have the 'best' diagnosis, but have no coping skills or other resources that would allow for successful home care. Because these nonmedical aspects of patient selection may play a significant role in home care, it is important to assess factors that would make a patient an 'ideal' candidate. This profile of an ideal candidate can serve as a basis for choosing the acceptable candidate (Table 3).[25] A determination of the

Table 3. Patient Characteristics That May Determine Success in Home Ventilator Care

Ideal	Acceptable	Unacceptable
Individual Coping Style		
Optimistic	Optimistic	None
Motivated	Motivated	
Resourceful	Sense of humor	
Flexible		
Adaptable		
Sense of humor		
Directive		
Support Systems		
Close family & social supports	Social supports	Lack of family & social supports
Education		
College degree	Ability to learn	Altered mental status
Ability to learn	Mechanically astute	Unable to learn
Financial Resources		
Adequate personal assets	Adequate health insurance	Lack of personal assets
Optimal health insurance		Lack of health insurance
Medical Condition		
Stable neuromuscular disease	Stable neuromuscular or obstructive disease	Medically unstable
Adequate free time off ventilator	Limited or no time off ventilator	
No other illnesses		
Self-Care Ability	Able to provide self-care	Unable to care for self or direct others
Able to provide self-care and/or direct others		

patient's individual coping style can be elicited through a psychological evaluation and the patient's past performance during times of stress.[7,25] If the patient and family don't feel optimistic about their future and lack the motivation needed to get through the hospitalization and discharge-planning process, then home care may not be successful. The patient must take an active part in planning his care and in the decision-making process; without this participation, the risk of failure is greatly increased.[25] The resourceful, flexible, and adaptable person will have an easier time dealing with the many potential problems related to changes in physical condition, caregiver support, and equipment function. A sense of humor is one asset that I have found to be important in successful home care; being able to relieve stresses through humor is extremely helpful to patients, families, caregivers, and multidisciplinary team members.

External Factors

Support systems. Close family and social supports are integral components of the ideal patient profile. Many patients who cannot provide their own care will need to direct others in the provision of daily care. Maintaining the family identity, with the patient assuming much of his prior role in the family structure and continuing with certain responsibilities, contributes a great deal to successful home care. When the patient is treated as the 'sick' person, his identity and role in the family will change ,which will contribute to loss of self-esteem and self-worth.

Education. The college-educated patient, unless factors such as cognitive impairment, depression or anxiety interfere with learning, will be able to understand and assimilate all of the concepts of medical and physical care as well as the functioning of the equipment.[25,26] For many patients and families, an educational background is not important as long as they can learn about their care and utilize the knowledge to function safely in the home environment.

Financial considerations. The patient's financial situation should not be a contributing factor in successful home care; but in this era of cost-containment and reimbursement issues, the patient who has his own personal assets and an excellent insurance policy will have fewer worries. The issues of equipment, supplies, home modifications, and caretaker support will not be major obstacles to discharge planning and home care for those with adequate finances.

The Ideal vs the Acceptable Candidate

The person with stable neuromuscular disease, no other medical problems, and the ability to be off the ventilator for significant periods of time is an ideal candidate for home ventilator care. When a patient can breath spontaneously for long periods of time, equipment needs are less and there are fewer problems associated with equipment malfunction and power interruptions. Also, the patient who can perform his own care or direct others in providing his care is ideal.

Regretfully, few people fit this profile of the ideal candidate, but by using it we can decide on the criteria that make a patient 'acceptable' for home care. These patients must still be motivated and optimistic, otherwise discharge planning and home care will be frustrating, unrewarding, and likely to fail. A sense of humor is a key asset with these patients, because they may have to deal with many frustrations related to obtaining supplies, insurance woes, and lack of understanding by other people. Home care ventilator patients may not adapt well to change and may be rigid in their thinking and daily routine, which will create anxiety when problems arise. Caregivers need to be aware of these personality traits and be somewhat flexible in their routines.

These patients may live alone but have helpful friends and/or may live in a supportive community. Many small communities will rally to the support of one of their members by providing meals, transportation, social activities, or even some personal care. Patients will need to be able to ask for help, to accept the support offered to them, and to learn how to take care of themselves and understand the functions of the equipment, even though they may not have any formal education. Patients who have some mechanical aptitude will feel less threatened about the equipment.[25]

Adequate third party coverage is necessary, even if it is a state Medicaid program. If these patients do not have any personal funding, they or their families will need to provide the necessary care, supplemented only by intermittent nursing care covered by the health insurance policy.

The Unacceptable Candidate

Patients 'unacceptable' for home care are those patients who do not have any of the personal, educational, or financial assets important for its success. These patients are either unlikely to be able to deal with the daily stressors or their cognitive functioning is limited or altered, which makes home an unsafe environment for them. They have no family or community support and somehow 'fall through the cracks' for adequate health insurance or state-funded medical coverage. Because of unstable disease and lack of other assets, successful home care is unlikely and if placed in the home, morbidity and mortality may be greatly increased.

If we look not only at the medical condition of the patients but at their own individuality, the decision for placement in the home can be made somewhat more easily. These patient assets must be looked at early in the hospital course prior to making any decisions about home care and, if possible, before elective ventilatory support is initiated.

Comprehensive Discharge Planning

Multidisciplinary Team

Discharge planning requires the support and interest of the physician, nurses, and other allied health professionals. If the driving force is only to get the patient out of the hospital as quickly as possible, the preparation for discharge and home care will not be smooth and the patient and family will get mixed messages. The team must work together to make the transition from hospital to home a safe and satisfactory one (Fig. 1).

The physician is the leader of the team because he makes the judgment calls about the stability of the patient and the feasibility for home care. Because he has this responsibility, it is extremely important that he has an interest and some expertise in rehabilitation and home care. Further, he needs to understand the intricacies involved in the discharge-planning process and realize that it is not accomplished overnight.

Many centers that routinely discharge ventilator patients will have a discharge-planning coordinator who will oversee the day-to-day process of preparing the patient for home. This person will work directly with the physician, be the link between the hospital and home services, and work directly with the patient and family.

Patient and family are also key members of the team. Without their motivation, desire, and participation in the rehabilitation and discharge-planning process, home care will not be successful. Careful attention must be paid to their progress and willingness to participate throughout the discharge preparation and ultimately when home

The amount of involvement of other members of the team (except for nursing, respiratory therapy, and the durable medical equipment [DME] company) depends on the patient's condition and self-care needs. Because the hospital program should be individualized for each patient, each of the services may have some involvement with the patient even on a limited basis. The home care services need to be involved early in the discharge-planning process in order for a smooth transition to take place. The therapist who will be involved with the patient after discharge should meet with the patient and family, and may also participate in teaching the patient and family, which will make the patient feel comfortable with the therapist and ease the transition to home.

Communication among all members of the team is mandatory for successful discharge planning. The

Fig. 1. Physician authorized services coordinated by the discharge planner.

multidisciplinary team should meet on a regular basis to discuss progress, set new goals for the ultimate discharge to home, and discuss any problems that have developed.

Patient and Family Education

Education is a major component of the discharge-planning process and needs to begin early in the patient's hospital stay. Initially, this education may consist of explaining the procedures and the reasons for doing them. The patient and family will also learn by observation, and this makes it critical that good technique be used when performing procedures.[26] It's not uncommon for an observant patient or family member to comment that what they are being taught is different from what they see performed by others.

A basic checklist of skills that the patient and family will need to know should be developed; skills that will be needed for individual situations can be added to the list. Table 4 lists some of the skills the patient and family must learn prior to discharge; they not only need to learn these skills, but they must become proficient at them. Documentation is necessary to determine when the task was taught and when it was performed independently by the patient and family.

The team needs to decide who will be doing the teaching and how it will progress; the patient should not be inundated with all of the material at one time. The patient should learn in a timely manner and progress from simple to more complex tasks. As each new task is learned, it should be reinforced by all team members and the patient given positive feedback about progress. The patient and family need to understand the importance of practice; many times they think that if the procedure is done correctly once, they do not need to do it again.

Particular attention needs to be paid to emergency measures (such as those to be used when the ventilator fails, the airway obstructs, or bleeding occurs), and these measures should be taught in a stepwise fashion. The patient and family need to be prepared for all emergencies (Table 4), even though they may never happen; for that reason, some of the potential emergency situations should be simulated. Both patient and family caregivers will function better in a stressful situation if there is a step-by-step plan of action to follow.

In one recent study, patients and families felt that learning skills through demonstration at the bedside with only a few people present was the most beneficial.[9] They also felt that the sessions should be short and that learning skills was more beneficial than learning about their lung disease. Teaching methods should be geared to the patient's learning capabilities. Some patients will need to have the procedures written on large cue cards to refer to as they practice a procedure. Many times

Table 4. Skills Needed by Patient and/or Caregivers prior to Discharge

Self-Care Techniques
Airway Management
Tracheostomy and stoma care
Cuff care
Tracheal suctioning
Changing the tracheostomy tube
Changing the tracheostomy ties
Chest physical therapy techniques
Percussion
Vibration
Coughing
Medication administration
Oral
Inhaled
Bed-to-chair transfers
Feeding-tube care
Indwelling-catheter care
Implantable-I.V.-line care
Bowel care
Switching from the ventilator to weaning device
Equipment Maintenance
Ventilator
Humidifier
Suction machines
Battery and charger
Oxygen administration
Manual resuscitator
Troubleshooting for problems
Cleaning and disinfection
Emergency Measures
Ventilator failure
Power failure
Dislodged tracheostomy tube
Obstructed airway
Cuff leaks
Shortness of breath
Ventilator circuit problems
Infection
Falls
Bleeding
Cardiac arrest

the psychosocial evaluation will help decide which teaching method will be appropriate for the patient (eg, some patients may not be able to read but are embarrassed to admit it). Special needs should be determined early so that time is not lost in educating the patient or his family.

Selection of Caregivers

Some patients will provide all of their own care and only need minimal support from family members. In other situations, the family will be the primary caregiver. Many times, it will depend on the patient's condition and what the insurance company will cover—skilled vs unskilled care, specific activities requiring care, hours per day of coverage needed. If the patient only needs unskilled care for personal hygiene, dressing, and meal preparation, a home health aide can be used. In many states, there are centers for independent living that assist patients to continue to live in a home environment by helping them find accessible housing and providing funding for personal care attendants. Because these personal care attendants are hired and trained by the patient or family, there are no restrictions on the complex care that they perform. These programs have allowed many patients with neuromuscular disease to live at home, at a much lower cost than using professional caretakers—even though they are ventilator-dependent. However, it is important to note that these patients need to possess the patient characteristics discussed under patient selection (Table 3).

Rehabilitation

An individualized program must be developed for each patient, depending on the patient's primary problem. For patients with COPD, many of the principles of pulmonary rehabilitation can be utilized in their care plan. For patients with a neuromuscular disorder, the goal may be to prevent further loss of function and to maintain joint mobility. The use of assistive devices for mobility and physical functioning will increase patient independence and improve quality of life.

In any rehabilitation program, realistic short- and long-term goals are necessary. All members of the team need to work together to prevent fragmentation in care, to assist the patient in meeting goals, and to help the patient progress from the simple to the more complex tasks. For many patients, the ability to move from the bedside by having the ventilator mounted on a wheelchair or cart gives them a sense of freedom. Patients who require a wheelchair for long-term use will need a prescription and professional guidance in the selection process.[27] Having specific wheelchair accessories and adaptive equipment to meet the patient's needs is critical, as is anticipating and planning for needs that may arise as the patient's physical condition changes.

A rehabilitation program for children may be very extensive and require many more services than for adults. Children need frequent evaluation of their physical, emotional, and intellectual development. If they are progressing well, as they grow, their equipment will need modification or replacement.

Home Care Equipment

Once the feasibility of home care is established, a durable medical equipment (DME) company should be contacted. The DME selected should be one that can provide all of the necessary equipment and supplies, has experienced home care respiratory therapists, and has previous experience with home ventilator care. An equipment and supply list should be developed and individualized for each patient. (If the patient has an artificial airway, some equipment is mandatory, as listed in Table 5.)

Table 5. Accessory Equipment for Patients with Artificial Airways in the Home

Essential	According to Need
Manual resuscitator bag	Oxygen
Humidifier	Hygroscopic condenser humidifier
Suction machine	Water traps
electric	Compressors
battery	medication delivery
Backup ventilator*	bland aerosol
Battery*	Generator
Battery charger*	Remote alarms
Ventilator cable to battery*	
Secondary ventilator alarms	

*Needed for any patient who cannot maintain spontaneous ventilation for 4 consecutive hours or who lives in a rural area.

It is important that the patient use the ventilator that will be used in the home for at least two weeks prior to discharge. He will also need to get accustomed to using other equipment such as the suction machine, air compressors, or oxygen because these are operationally different from the

piped-in compressed gases and vacuum available in the hospital. Everything that will be available in the home should be used in the hospital, and the hospital room should be set up as the patient's room will be in the home.

Assessment of the Home Environment

An assessment of the patient's home should be made by a hospital team member and the DME early enough in advance of discharge to allow time for modifications. Other team members may need to visit the home, depending on the patient's functional status and mobility. Table 6 lists the more common aspects of the home environment that need to be observed.

Table 6. Assessment of the Ventilator-Dependent Patient's Home

Accessibility
In and out of home
Bathroom
Kitchen
Between rooms
Wheelchair mobility
Doorway width
Thresholds
Stairways
Carpeting
Equipment
Space
Electrical power supply
Amperage
Grounded outlets
Environment
Temperature
Lighting
Living space

Patients who will be using quite a bit of electrical equipment may need additional wiring and placement of grounded outlets. I like to use an electrical power strip with a circuit breaker for the patient's ventilator and accessory equipment because it can be mounted on a table or cart with the ventilator and be clearly visible.

Ideally, space should be adequate to allow for the patient's comfort and privacy and equipment and supply placement and storage, but realistically this may not always be possible. Home modifications are out-of-pocket costs and not always affordable by the patient. It is also important not to have the home look like a hospital. This can be avoided by taking care to keep as many personal items in the room as possible (desk, pictures, book case, favorite furniture).

Nursing Agencies and Community Resources

The choice of a nursing agency may depend on the patient's need for continuous or intermittent care, the need for other allied health professionals, and each agency's experience with home ventilator care. Other factors to be considered in the selection process are whether they (1) are certified Medicare providers, (2) accept assignment for state-funded Medicaid patients, and (3) as with any home care agency, are accredited by the Joint Commission of Accreditation for Healthcare Organizations (JCAHO). The nursing agency should be contacted well in advance of discharge to give the staff ample time to meet with the patient and family, participate in discharge-planning conferences, and obtain the necessary information on reimbursement.

Other community resources (such as housekeeping, meal preparation, transportation, and school services) should be contacted depending on the needs of the patient.

Transition from Hospital to Home

Whenever possible, trials away from the hospital should be part of the discharge-planning process. However, this should only be initiated when the patient and family have learned all of the skills necessary for successful home care. Generally, overnight trials in the home are done just prior to discharge. These trials will help the patient and family gain confidence in their own abilities, help both the patient and management team address unanticipated problems, and ensure continued teaching of aspects of care in which the patient and family are weak.

Medical Follow-Up

Prior to discharge, the physician who will be providing medical care should be identified. This may be a physician caring for the patient in the hospital or it may be the patient's local medical doctor. The physician will need to be updated frequently about the patient's condition, understand the home care plan, and have a backup consultant for ventilatory problems (if he's not a pulmonologist). All facets of care should be considered—eg,

if this physician will be involved with tracheostomy tube changes, has he had prior experience? All follow-up care must be organized in advance of discharge to decrease the stress for the patient and family.

Specific Issues Related to Long-Term Ventilator Patients

Airway Management

Each patient with a tracheostomy should have his airway assessed via bronchoscopy for tube placement, airway obstruction by granulation tissue, tracheomalacia, and tracheal stenosis. The tracheal tube should be positioned comfortably in the stoma with the flange resting against the neck without any pressure, and the curve of the tube should be appropriate for the patient's anatomy. The inner diameter should be appropriate to the patient's anatomy for comfort and to decrease airway resistance. The length of the tube may need to be customized depending on tracheal problems.[28]

If the patient will be progressing to a fenestrated tracheostomy tube, the position of the fenestration within the airway should be assessed. If the fenestration is not positioned fully in the airway, irritation of tracheal tissue can result during repeated placement of the inner cannula with resultant bleeding, granulation tissue formation, or intrusion of tissue into the fenestration. An intrusion of tissue can result in the inability to place the inner cannula, so proper positioning is important.

The vocal cords and upper airway should also be assessed for edema, granulation tissue, and vocal cord movement—especially if the patient can be off the ventilator for long periods of time and will have the ability to phonate normally. Vocal-cord dysfunction will interfere with swallowing and will need early intervention to prevent aspiration into the lungs.

Evaluation and Treatment of Swallowing Dysfunction

Reports are scarce on the incidence of dysphagia in patients requiring ventilatory support through an artificial airway.[29] The mechanisms of swallowing disorders may be related to the rigidity of the tracheostomy tube and anchoring of the trachea to the strap muscles, decreased laryngeal sensation related to the diversion of air through the stoma, and overinflation of the tracheal tube cuff.[29]

All patients with an artificial airway should be evaluated for swallowing disorders; this can be done at the bedside by the speech pathologist, using foods and liquids of varying consistencies with food coloring added to the food. The patient is suctioned and positioned upright, small amounts of liquids and solids are given to him, and then he is suctioned with the cuff inflated and then deflated. Aspiration into the airway may not always be immediately noted because of pooling in the valleculae and pyriform sinuses, which may cause spillover into the airway at a later time.

If there is any suspicion of aspiration, this bedside evaluation should be followed by a radiographic evaluation. A video fluoroscopy with barium is the best method to determine swallowing disorders, but this technology may not be available in all medical centers. With this technique, the patient is given small boluses of barium in different consistencies while the video fluoroscopy is being done. The video is extremely helpful because the swallow may be so fast that small amounts of aspiration may not be noted on the fluoroscopy alone. The radiologist and speech pathologist can review the video and assess each aspect of the swallowing mechanism.

Once diagnosed, the treatment for swallowing disorders will depend on the cause. For some patients, positioning upright with the head tilted forward may be all that is necessary. For others, teaching compensatory mechanisms and tongue exercises may help. Some patients may require foods of only one consistency to prevent aspiration, and others will not be able to have oral feedings at all; these patients will need a nasal or gastric feeding tube placed for nutrition. Patients who cannot tolerate oral or nasogastric feedings will need intravenous hyperalimentation.

Communication Strategies

When a patient cannot communicate normally because of an artificial airway and/or problems with phonation secondary to bulbar weakness, an alternative means of communication should be available. As with swallowing disorders, the speech therapist is invaluable in the assessment and treatment of communication problems. Table 7 shows many of the communication alternatives available for the ventilator-assisted patient.

If the patient is able to speak with his normal voice either by cuff deflation or an uncuffed

Table 7. Communication Alternatives for Ventilator-Assisted Patients

Cuff deflation (or uncuffed tracheostomy tube)
'Talking' tracheostomy tube
Electrolarynx neck placement intra-oral placement
Pneumatic voice device
Lip speaking
Writing
Sign language
Communication boards
Alphabet keyboards with printed messages
Preprogrammed computers with voice synthesizers

tracheostomy tube and augmented tidal volumes on the ventilator, his sense of self-esteem and feelings of self-control will be greatly increased. For other patients, a 'talking' tracheostomy tube will allow normal speech. Many simple devices such as the electrolarynx or sophisticated, highly technical devices such as computers with voice synthesizers are available to augment speech. The choice of a device needs to be individualized, although some patients may refuse to use the electronic devices because they are self-conscious about the projected sound of their voice. The patient's, family's, and caretaker's frustration is greatly increased when the patient cannot be understood, so it is important that this aspect be considered.

'Free' Time from the Ventilator (Weaning)

Prior to the patient's discharge, the amount of free time he can tolerate off the ventilator should be determined. If weaning is possible, the technique should be simple and able to be carried out in the home. Weaning with a critical care ventilator on CPAP and/or a pressure support mode cannot be continued in the home, and the amount of free time with these methods may differ greatly when weaning with a T-piece. Portable ventilators are not adequate for SIMV without modification as discussed earlier.

The amount of free time should be consistent from day to day, and the patient should be stable within predetermined limits. As the patient progresses to longer weaning periods, ability to perform functional activities while weaning needs to be assessed. If the patient can wean for long periods of time and be active, he will have much more freedom and mobility.

Psychosocial Issues

For any patient who has been hospitalized for a long period of time, the transition to home may provoke much anxiety. For patients who are on life-support equipment, the impending discharge to home may stir up many feelings. As much as the patient wants to be at home, the change from a protected environment and experienced caretakers to the home with only family or self as caretaker will increase anxiety and ambivalence about going home.[7,25] Patients and families react to this in many ways; they may become increasingly dependent and angry, and have many somatic complaints. The family may miss teaching appointments or not visit as often, or they may fail to make necessary home arrangements. Frequent family meetings, realistic discussions about home care, and meetings between the patient, his family, and other ventilator-assisted patients and their families will help.

After discharge, the patient and family will need to establish routines and gain confidence in themselves. They will need frequent reassurance and review of the skills that they have learned. If they are providing all of the care, they may feel overburdened and overwhelmed by the care requirements, especially until they establish routines. Respite care can help with some of these issues, but a mechanism needs to be in place to provide it (neighbors or relatives who are trained to relieve the primary caregiver for short periods of time).

Conclusion

The process of discharging a ventilator-assisted patient has many intricate facets and requires the coordination of many services and people. Transition to home ventilator care is easier when the criteria for patient selection is realistic; trying to prepare an unstable patient or one without support systems for home is an exercise in futility. The frustration resulting from this futility only leads to anger and a feeling of impotence among the team. When this happens, many of the team members will not be interested in or have the energy to deal

with another patient deemed to be a candidate for home care. We need to ensure safe, quality care that is cost-effective, and at the same time we need to feel a sense of accomplishment for a job well done.

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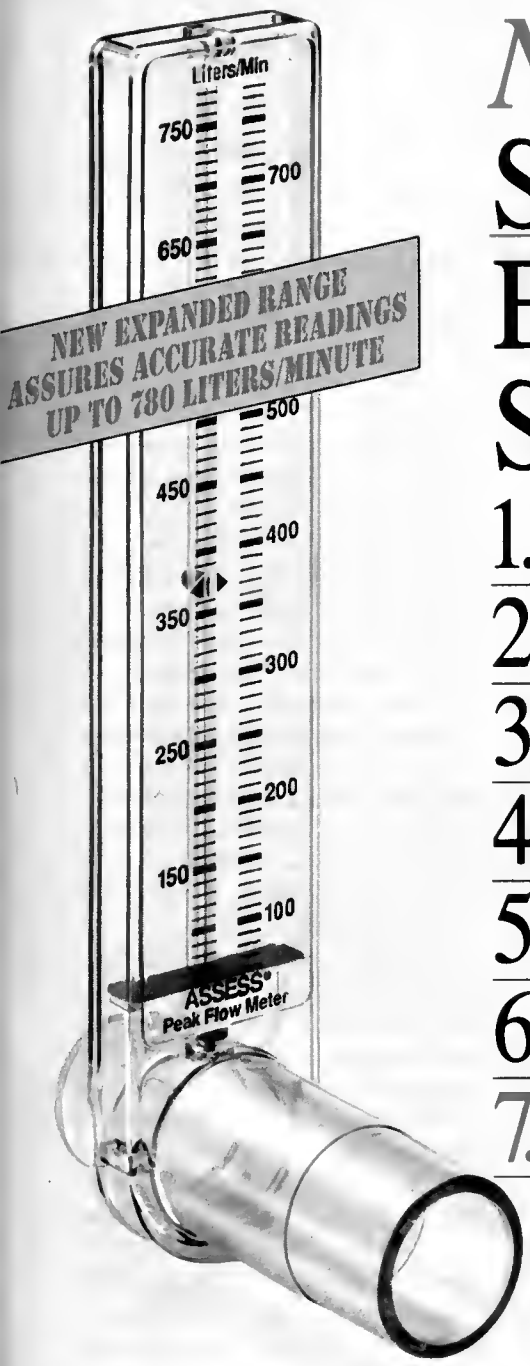

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Theophylline—A Continuing Enigma

Hugh S Mathewson MD

Since strong coffee was recommended for the treatment of asthma over a century ago,[1] the methylxanthines (caffeine, theobromine, theophylline) have been used for both prophylaxis and therapy of bronchospasm. Caffeine is still used as a respiratory stimulant for neonatal apnea, whereas theobromine has practically disappeared. However, an estimated 10 million Americans take theophylline, according to industry officials, about half of them for asthma, the rest for chronic lung disease.[2] Even this is a modest figure compared to the number of people who took the drug 25 years ago.

Theophylline once had clinical stature as a cardiac stimulant, a peripheral vasodilator, and a diuretic. It was often prescribed for congestive heart failure and for angina pectoris. In these therapeutic applications, it has been superseded by more effective drugs. Now its status as a bronchodilator is subject to challenge, particularly when it is administered intravenously for treatment of acute asthmatic episodes. Although monitoring of plasma concentrations has become routine,[3] some clinicians have expressed doubt whether theophylline can be given with predictable safety, and whether its contribution to the treatment of asthma is sufficient to justify its continued use.

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A recent paper by Lam and Newhouse[4] summarizes in detail the shortcomings of theophylline. In an accompanying editorial, Newhouse[5] states that it has been a first line antiasthmatic drug for 50 years despite its relatively weak bronchodilator effect, narrow therapeutic window, numerous drug interactions, and low therapeutic index. Its recent surge of popularity, which peaked about 1988, is attributable to the introduction of sustained-release preparations, which bridged the nocturnal gap of 6 hours that could not be covered by beta-adrenergic aerosols.[6] However, new long-acting beta-adrenoceptor agonists such as salmeterol[7] and formoterol[8] appear to provide continuous bronchodilator efficacy for periods of 10 to 12 hours. Also, aerosol administration has been markedly improved by the use of metered dose inhalers supplemented by mist-confining devices (so-called spacers). This is of especial benefit to children and elderly adults, for whom the use of conventional nebulizers may be grossly inefficient.

A more flexible and adaptable regimen for control of asthma may be provided by inhaled beta-adrenergic agents, perhaps supplemented by the anticholinergic aerosol ipratropium bromide thus avoiding the use of oral bronchodilator preparations entirely.[9] One reason that heavy maintenance dosage of theophylline persists is probably that beta-adrenoceptor agonists are not given in adequate doses.[10] The two-puff self-administration recommended in some package inserts does not deliver enough drug to accomplish an optimal response.

The potential for dose-related toxic side actions of theophylline is well documented.[11–14] The therapeutic plasma level range is about 10 to 20 μg/mL. Toxic side actions in the form of emesis, precordial pain, tachyarrhythmias, and central-nervous-system hyperirritability are likely to appear at concentrations above 20 μg/mL. Convulsions and death have occurred at concentrations of 25 μg/mL, although seizures are relatively rare at values below 40 μg/mL[14]. Sustained-release preparations create the problem of continued absorption in the face of toxic manifestations. Although activated charcoal administered orally will accelerate clearance of theophylline, levels above 100 μg/mL will require invasive measures, including hemoperfusion through charcoal cartridges.[14]

Most cases of severe toxicity occur in patients receiving repeated oral or parenteral theophylline.[15] Long-term intoxication appears to render patients more prone to seizures than do short-term overdoses,[16] which compounds the difficulty in establishing a relationship between plasma concentration and severity of side actions.[17] Rapid intravenous administration of aminophylline in 500-mg doses can result in sudden death from cardiac arrhythmia.[18] The drug should be injected slowly over a period of 20 to 40 minutes, and should not be given to a patient already taking theophylline until a plasma concentration value has been obtained.

Dose scheduling in children is particularly difficult, and an adequate therapeutic response may not be obtained unless plasma values are close to toxic levels.[19] It has been contended

that behavioral abnormalities and poor school performance can be attributed to oral theophylline use,[20,21] although this remains unproven.[22]

The use of theophylline as a neonatal respiratory stimulant has recently been questioned. Its central action in preterm infants is partly attributable to the fact that up to 25% of the drug is converted in vivo to caffeine.[23,24] However, theophylline inhibits erythropoietin production; it has been employed to prevent erythrocytosis in patients after renal transplantation.[25] There is current concern that the drug may have a similar depressant effect on erythropoietin synthesis in the neonate, and therefore could be responsible for causing some cases of anemia of prematurity.[26]

The Association of Trial Lawyers of America (ATLA) held a news conference October 30, 1990, warning of the hazards of theophylline.[2] They reported 26 cases in which Association members provided legal representation for alleged injuries and preventable deaths involving theophylline use. The ATLA has urged the Food and Drug Administration (FDA) to ban over-the-counter theophylline preparations, and to require that package inserts be revised to warn of potential hazards. The FDA did not comment, but there were spirited replies from the American College of Allergy and Immunology and from the Schering-Plough Corporation, which has nearly 50% of the prescription theophylline market in the U.S. Dale B Sparks MD, president of the College, was quoted as saying: "We've used theophylline for 50 years, and physicians are well aware of the complications." A spokesman for Schering-Plough stated that about 60 million prescriptions have been written for the company's theophylline products since 1985. The incidence of serious adverse effects reported to the FDA during that period was 1.3/100,000 prescriptions.[2]

The status of theophylline would probably be improved if more were known about its mechanism of action. Whether the drug produces bronchodilatation primarily by relaxing smooth muscle or by inhibiting the action of spasmogens is unclear. There is current focus on the adenosine-receptor-blocking property of methylxanthines,[27] but the role of adenosine in the pathogenesis of asthma is still undefined. Theophylline can improve exercise capacity in some COPD patients by mechanisms that are probably unrelated to bronchodilatation.[28-30] The therapeutic response of the patient with asthma, itself a disease with multifactorial etiology, can at this time be assessed only empirically. In a recent editorial,[31] Niewoehner pointed out that theophylline is a drug ill-suited for widespread administration. In a retrospective review at his institution (a Veterans Administration Medical Center), the risk of a fatal or life-threatening complication of theophylline therapy was about 0.5% per year, in a population largely composed of elderly patients with COPD and other medical problems. Extrapolation of this figure to the total U.S. population receiving theophylline suggests that several thousand patients experience severe adverse reactions each year. Theophylline continues to have staunch advocates, however. The latest edition of *Goodman and Gilman's Pharmacological Basis of Therapeutics* (1990), arguably the most authoritative source book in the U.S., offers the following statement:

> The oral administration of theophylline-containing preparations has been used to produce bronchodilatation for over 50 years. The efficacy of theophylline is unquestioned, and, with supplemental inhalation of beta$_2$-adrenergic agonists, successful treatment of most patients with moderately severe chronic asthma has been a reality for nearly 20 years.

This is followed by admonitions concerning its narrow margin of safety, the augmented susceptibility to seizures brought on by long-term administration, and the necessity to titrate the drug in successive stages until maximal efficacy is reached. Each upward adjustment of dosage is to be preceded by a determination of plasma concentration. Also, it is noted that, over the long term, theophylline appears to have little effect on bronchial hyperresponsiveness.

The primary emphasis in the treatment of asthma and COPD has now shifted toward control of the chronic inflammatory process responsible for bronchial hyperreactivity, with less importance attached to providing symptomatic relief of bronchospasm.[32] Potent topical steroids[33] and cromolyn congeners[34] can provide good long-term control, with occasional supplementation by beta-adrenoceptor agonists or anticholinergic agents. Large doses of steroids are advocated for acute treatment of asthma attacks.[35,36] It has been stated that theophylline preparations contribute but little when added to combined beta-agonist and steroid therapy.[4,12,37]

In the maintenance of asthma control, Lam and Newhouse[4] place dose-optimized inhaled steroids as first-line therapy. Inhaled adrenoceptor agonists are second-line medications, anticholinergic aerosols third-line, and theophylline, if needed at all, can provide a minor steroid-sparing function in severe asthmatics.

The question whether theophylline should be relegated to obsolescence can probably not be answered unless placebo-controlled trials are carried out.[38] This kind of study would be expensive, cumbersome, and time-consuming, and is not likely to be undertaken because some COPD patients respond poorly to steroids and others cannot tolerate beta-adrenoceptor agonists. It is likely that the use of theophylline for asthma will decline in the foreseeable future, but its place in the treatment of selected patients will remain secure until longer acting and more potent agents whose effects are safer and more predictable are introduced.

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Charles G Durbin Jr MD and
Douglas B Eden BS RRT, Section Editors

A Possible Complication of Central Venous Catheterization

Curt M Morey CPFT RRT, David C Lain PhD RRT, Bjorn Thorarinsson MD,
Arthur A Taft MHS RRT, and Shelley C Mishoe MEd RRT

A 51-year-old woman presented to our emergency room complaining of nausea, vomiting, fever, night sweats, and a productive cough (1 cup of green sputum/day for the previous 4 days). She denied pleuritic chest pain, shortness of breath, abdominal pain, diarrhea, headaches, and neck stiffness.

Physical examination revealed her temperature to be 41°C [314°K], heart rate 132/min, blood pressure 125/45, and respiratory rate 22/min. Chest auscultation revealed diminished breath sounds in the middle posterior portion of the left lung and inspiratory crackles, which were more pronounced in the left lung than in the right. A chest radiograph was taken (Fig. 1).

The white-blood-cell count was 14,700/μL with a left shift. The hemoglobin was 12.2 g/dL with a hematocrit of 35.1%. SMA-10 was normal. Analysis of arterial blood, with the patient breathing room air, revealed pH 7.51, P_{aO_2} 60 torr [8.0 kPa], P_{aCO_2} 30 torr [4.0 kPa], calculated HCO_3^- 24.2 mEq/L [24.2 mmol/L], and measured S_{aO_2} 90.9%.

A sputum gram stain revealed gram-positive cocci, gram-negative rods and cocci, and a few white blood cells. She was admitted to our hospital with a diagnosis of community-acquired pneumococcal pneumonia and was started on intravenous (I.V.) cefuroxime. During the first 48 hours after admission, the patient continued to be febrile, and exhibited increasing respiratory distress. Chest radiographs revealed diffuse bilateral infiltrates. Administration of erythromycin was initiated, and the patient was moved to the intensive care unit.

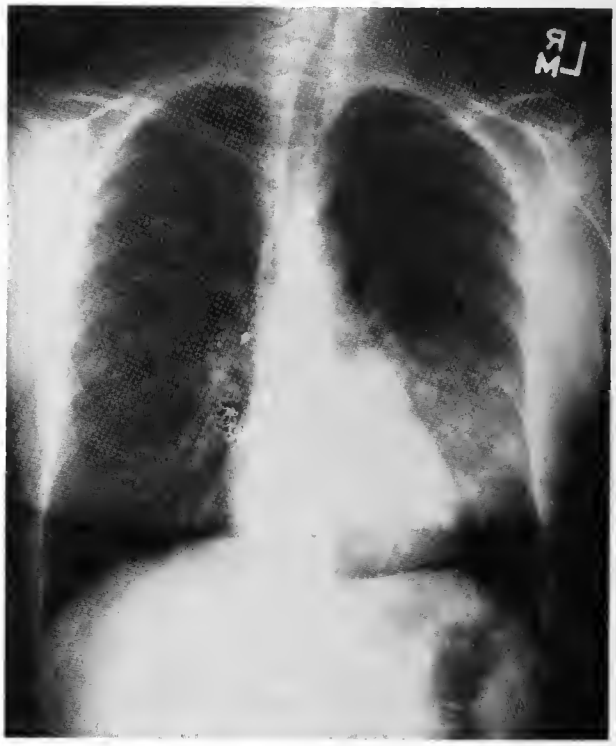

Fig. 1. Admission chest radiograph of a 51-year-old woman complaining of nausea, vomiting, fever, night sweats, and productive cough.

The patient continued to deteriorate, and on the third hospital day required intubation and mechanical ventilation. Later that morning, a bronchoscopy was performed and bronchoalveolar lavage fluid was sent for microbiologic evaluation including direct fluorescent antibody for legionella. Later that day, a pulmonary artery catheter was placed that revealed a normal pulmonary capillary wedge pressure.

Mr Morey was a student in the Respiratory Therapy Program, Dr Lain was Clinical Instructor and Coordinator of Research and Education, Dr Thorarinsson is Assistant Professor of Medicine and Director of the Medical Intensive Care Unit, Mr Taft is Assistant Professor of Respiratory Therapy and Director of Clinical Education, and Ms Mishoe is Associate Professor of Respiratory Therapy and Chairman of the Respiratory Therapy Program—Medical College of Georgia, Augusta, Georgia. Mr Morey is now a staff therapist at Phoebe Putney Memorial Hospital, Albany, Georgia. Dr Lain is now Product Specialist, Ohmeda, Baltimore, Maryland.

On Day 6 and Day 11 of hospitalization, central venous lines were placed. (On Day 10, an unsuccessful attempt to place a central venous line resulted in a right pneumothorax, which was successfully treated with tube thoracostomy.) On Day 20, the patient was extubated, following an uneventful course of weaning from the ventilator. On Day 21, she was moved to the general ward. A few days later, a chest radiograph was taken (Fig. 2).

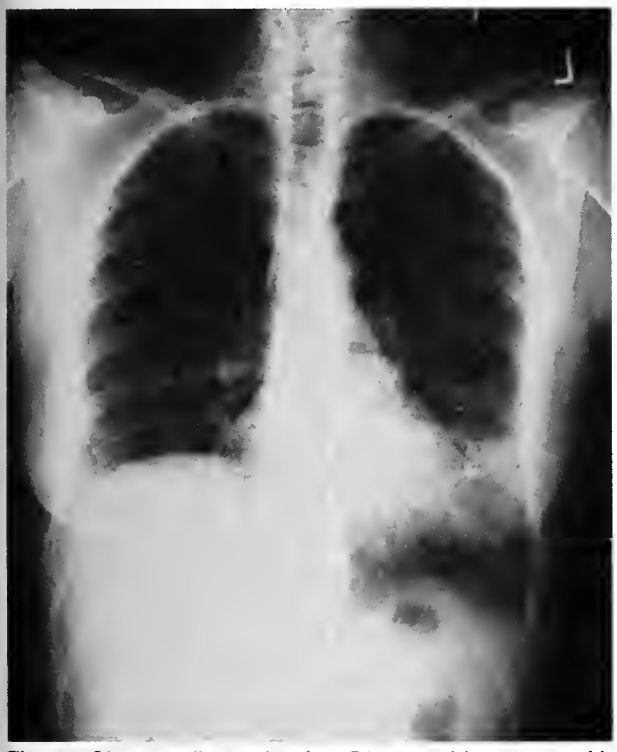

Fig. 2. Chest radiograph of a 51-year-old woman with resolving pneumococcal pneumonia, taken after extubation and subsequent to 17 days of mechanical ventilation.

Questions

Radiographic Findings on Admission: What does the chest radiograph in Figure 1 show? What anatomic areas (lobes, segments) are affected?
Radiographic Findings after Extubation: What does the chest radiograph in Figure 2 suggest?
Further Testing: What test(s) might help confirm or deny your diagnosis?

Answers and Discussion on Next Page

Answers

Radiographic Findings on Admission: The chest radiograph in Figure 1 reveals alveolar infiltrates in the superior segment of the left lower lobe. The clear diaphragmatic border makes basilar involvement unlikely. The clear left-heart border rules out lingular involvement; therefore, the superior segment of the left lower lobe is the most likely anatomic location. This was further supported by a retrocardiac shadow, and was confirmed by a lateral chest radiograph.

Radiographic Findings after Extubation: Figure 2 suggests resolving left-lower-lobe infiltrates and an elevated right hemidiaphragm, possibly due to unilateral diaphragmatic paralysis.

Further Testing: A sniff test was performed under fluoroscopy that revealed paradoxical motion of the right hemidiaphragm, thus confirming the diagnosis of right hemidiaphragm paralysis.

Discussion

Central venous catheterization is indicated when I.V. access is needed for high volume intravenous fluid administration and/or peripheral I.V. access is difficult to obtain.[1,2] The two phrenic nerves, one for each hemidiaphragm, arise from the third, fourth, and fifth cervical nerves. As the phrenic nerves descend from their point of origin, they curve around the lateral borders and obliquely cross the anterior surfaces of the anterior scalene muscles. They pass posterior and lateral to the internal jugular veins and underneath the sternocleidomastoid muscles as they enter the thorax. At this point, they pass posterior to the subclavian veins and anterior to the subclavian arteries descending into the anterior mediastinum, parallel to the lateral surfaces of the brachiocephalic veins.[3-5]

Important complications and potentially lethal hazards associated with insertion of central venous lines and pulmonary artery catheters have been reported: pneumothorax,[1,2,4,6-10] bleeding and/or hemothorax,[1,2,4,6-8,10-12] infection,[1,2,4,6-8,10-12] venous air embolism,[2,4,7,8,11] carotid or subclavian artery puncture,[4,8] cardiac dysrhythmias,[7-10,13-15] perforation of the pulmonary artery,[10,13,14,16] hemoptysis,[12,16] intrapulmonary hemorrhage,[9,12,15] pulmonary infarction,[14,15] pulmonic valve injury,[13,17] tricuspid valve injury,[13,18] catheter embolus,[7,10,19] thoracic duct laceration on the left side,[6] pleural effusion,[6] pulmonary embolus,[7] brachial plexus damage,[8] ventricular tachycardia,[9] vascular occlusion,[13] intracardiac knotting of the pulmonary artery catheter,[14] thrombosis,[14] complete heart block in a patient who already had left-bundle-branch blockage,[20] and injury to the phrenic nerve.[2,3,4,6,21] We suspect that in the case we report, central venous-line placement resulted in injury to the phrenic nerve and consequent unilateral diaphragmatic paralysis.

Unilateral diaphragmatic paralysis can be suspected if one of the hemidiaphragms is observed to be elevated in the chest radiograph.[2] However, this is not observed often in the mechanically ventilated patient, and only becomes obvious when the patient is off the ventilator and breathing spontaneously as in the case of our patient. In addition to radiographic evidence, electromyographic[2,7] and/or fluoroscopic[7] evidence is needed to confirm diagnosis of unilateral diaphragmatic paralysis. Having the patient sniff (creating a brief but intense contraction of the diaphragm and other respiratory muscles) during fluoroscopy is referred to as the sniff test. If unilateral diaphragmatic paralysis is present, when the sniff test is performed the healthy hemidiaphragm will descend sharply (move caudad) and the paralyzed hemidiaphragm will rise (move cephalad).[22,23]

The recovery of the injured diaphragm depends upon the extent of the injury. If a blunt injury or partial laceration of the phrenic nerve has occurred, recovery is usually within several months.[3] If the nerve has been severed, end-to-end anastomosis and sural nerve grafts may be successful in treating the injured nerve.[3] If the nerve has been paralyzed with medication, such as lidocaine, recovery is usually prompt.[4] The case presented in this report is too recent to assess whether permanent damage to the phrenic nerve has occurred.

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In a small town
outside of Chicago. good news
travels fast. In this
particular case a
family physician was the
messenger. A little girl was
the recipient.
Little Nikki was only
9 months old. And her prognosis
for a rich and rewarding
life is excellent.

EACH YEAR, IT IS ESTIMATED THAT 5,000 CHILDREN DIE FROM RSV COMPLICATED INFECTIONS.[1]

Consider treatment with ribavirin aerosol.

For infants hospitalized with lower respiratory tract disease caused by RSV at high risk for **severe or complicated RSV infection.** This includes infants with **congenital heart disease, bronchopulmonary dysplasia** and other **chronic lung conditions,** and certain **premature infants.** In addition, children with **immunodeficiency,** especially those with severe **combined immunodeficiency disease, recent transplant recipients,** and those **undergoing chemotherapy for malignancy,** should also be considered to be at high risk for complicated RSV infection. **Infants hospitalized with RSV** lower respiratory tract disease **who are severely ill.** Since severity of illness is often difficult to judge clinically in infants with RSV infection, determination of the blood gases is often necessary. Infants with **PaO_2 levels of less than 65 mmHg** and those with **increasing $PaCO_2$ levels** should be considered as candidates for ribavirin therapy. Oximetry may be used as a non-invasive means of determining the arterial oxygen saturation. Infants who might be considered for treatment are those hospitalized with lower respiratory tract disease which is not initially severe, but **who may be at some increased risk of progressing** to a more complicated course **by virtue of young age** (<6 weeks), or in whom prolonged illness might be particularly **detrimental to an underlying condition,** such as those with multiple congenital anomalies, neurologic or metabolic diseases.[2]

RSVirazole®
(ribavirin)
...inistration

...ecovery.

Kathy Foltz, R.N.
Marseilles, Illinois 61341

November 19, 1990

ICN Pharmaceuticals, Inc.
ICN Plaza
3300 Hyland Ave.
Costa Mesa, CA 92626

Dear Sirs:

I am not in the habit of writing letters to drug companies but this time I felt a true need. I want to thank you for how your drug helped my daughter recover from RSV.

I am an O.R. supervisor in a surgery department. When my daughter, Nikki, was 9 months old, she developed what we thought was a cold; wheezing, congestion and fever. One night her wheezing worsened and her breathing became extremely rapid. She got progressively worse and at 4 a.m., I took her to the Emergency room. There, they told me I was doing all the right things and sent us home with antibiotics. At home, Nikki's condition deteriorated--faster respirations and significant retraction. I tried steam, gave the antibiotics and felt absolutely helpless. Years of nursing experience had not taught me what was happening to my daughter. I called our family physician a few hours later and explained Nikki's symptoms. He met us at the Emergency room. Nikki was admitted that night with a diagnosis of pneumonia. Blood cultures were ordered, IV antibiotics given and a test was done for something called RSV.

I had never heard of RSV, but when the test returned positive, our physician immediately ordered a "mist treatment". I have since come to know this mist treatment as Virazole. Nikki was so sick at the initiation of treatment, I was afraid she would give up trying to breathe.

Prayers do get answered. By the next afternoon, her breathing normalized and her color improved--she started smiling and playing again. Nikki remained on Virazole for three days and then we went home.

In a small town, 100 miles south of Chicago, my physician knew about Virazole and was able to treat my daughter close to home. Had he not known, Nikki would have been shipped to a PICU in a large Chicago hospital. The physician, the staff and the drug saved my daughter.

Thank you,

Kathy Foltz R.N.

Kathy Foltz, R.N.

Letter composed from actual experience and printed with permission of Kathy Foltz.

Virazole®
(ribavirin)
lyophilized for aerosol administration

Rapid response. Rapid recovery.

PRESCRIBING INFORMATION

WARNING: RIBAVIRIN AEROSOL SHOULD NOT BE USED FOR INFANTS REQUIRING ASSISTED VENTILATION BECAUSE PRECIPITATION OF THE DRUG IN THE RESPIRATORY EQUIPMENT MAY INTERFERE WITH SAFE AND EFFECTIVE VENTILATION OF THE PATIENT. Conditions for safe use with a ventilator are still in development.

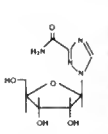
Deterioration of respiratory function has been associated with ribavirin use in infants, and in adults with chronic obstructive lung disease or asthma. Respiratory function should be carefully monitored during treatment. If initiation of ribavirin aerosol treatment appears to produce sudden deterioration of respiratory function, treatment should be stopped and re-instituted only with extreme caution and continuous monitoring.

Although ribavirin is not indicated in adults, the physician should be aware that it is teratogenic in animals (see CONTRAINDICATIONS).

DESCRIPTION:

Virazole® (ribavirin) Aerosol, an antiviral drug, is a sterile, lyophilized powder to be reconstituted for aerosol administration. Each 100 ml glass vial contains 6 grams of ribavirin, and when reconstituted to the recommended volume of 300 ml with sterile water for injection or sterile water for inhalation (no preservatives added), will contain 20 mg/ml ribavirin, pH approximately 5.5. Aerosolization is to be carried out in a SPAG-2 nebulizer only.

Ribavirin is 1-beta-D-ribofuranosyl-1,2,4-triazole-3-carboxamide, with the following structural formula:



Ribavirin, a synthetic nucleoside, is a stable, white, crystalline compound with a maximum solubility in water of 142 mg/ml at 25°C and with only a slight solubility in ethanol. The empirical formula is $C_8H_{12}N_4O_5$ and the molecular weight is 244.2 Daltons.

CLINICAL PHARMACOLOGY:

Antiviral effects:

Ribavirin has antiviral inhibitory activity *in vitro* against respiratory syncytial virus,[1] influenza virus, and herpes simplex virus. Ribavirin is also active against respiratory syncytial virus (RSV) in experimentally infected cotton rats.[2]

In cell cultures, the inhibitory activity of ribavirin for RSV is selective. The mechanism of action is unknown. Reversal of the *in vitro* antiviral activity by guanosine or xanthosine suggests ribavirin may act as an analogue of these cellular metabolites.

Immunologic effects:

Neutralizing antibody responses to RSV were decreased in ribavirin treated compared to placebo treated infants.[3] The clinical significance of this observation is unknown. In rats, ribavirin resulted in lymphoid atrophy of thymus, spleen, and lymph nodes. Humoral immunity was reduced in guinea pigs and ferrets. Cellular immunity was also mildly depressed in animal studies.

Microbiology:

Several clinical isolates of RSV were evaluated for ribavirin susceptibility by plaque reduction in tissue culture. Plaques were reduced 85-98% by 16 μg/ml; however, plaque reduction varies with the test system. The clinical significance of these data is unknown.

Pharmacokinetics:

Assay for ribavirin in human materials is by a radioimmunoassay which detects ribavirin and at least one metabolite.

Ribavirin administrered by aerosol is absorbed systemically. Four pediatric patients inhaling ribavirin aerosol administered by face mask for 2.5 hours each day for 3 days had plasma concentrations ranging from 0.44 to 1.55 μM, with a mean concentration of 0.76 μM. The plasma half-life was reported to be 9.5 hours. Three pediatric patients inhaling ribavirin aerosol administered by face mask or mist tent for 20 hours each day for 5 days had plasma concentrations ranging from 1.5 to 14.3 μM, with a mean concentration of 6.8 μM.

It is likely that the concentration of ribavirin in respiratory tract secretions is much higher than plasma concentrations in view of the route of administration.

The bioavailability of ribavirin aerosol is unknown and may depend on the mode of aerosol delivery. After aerosol treatment, peak plasma concentrations are less than the concentration that reduced RSV plaque formation in tissue culture by 85 to 98%. After aerosol treatment, respiratory tract secretions are likely to contain ribavirin in concentrations many fold higher than those required to reduce plaque formation. However, RSV is an intracellular virus and serum concentrations may better reflect intracellular concentrations in the respiratory tract than respiratory secretion concentrations.

In man, rats, and rhesus monkeys, accumulation of ribavirin and/or metabolites in the red blood cells has been noted, plateauing in red cells in man in about 4 days and gradually declining with an apparent half-life of 40 days. The extent of accumulation of ribavirin following inhalation therapy is not well defined.

INDICATIONS AND USAGE:

Ribavirin aerosol is indicated in the treatment of hospitalized infants and young children with severe lower respiratory tract infections due to respiratory syncytial virus (RSV). In two placebo-controlled trials in infants hospitalized with RSV lower respiratory tract infection, ribavirin aerosol treatment had a therapeutic effect, as judged by the reduction by treatment day 3 of severity of clinical manifestations of disease.[3,4] Virus titers in respiratory secretions were also significantly reduced with ribavirin in one of these studies.[4]

Only severe RSV lower respiratory tract infection is to be treated with ribavirin aerosol. The vast majority of infants and children with RSV infection have no lower respiratory tract disease or have disease that is mild, self-limited, and does not require hospitalization or antiviral treatment. Many children with mild lower respiratory tract involvement will require shorter hospitalization than would be required for a full course of ribavirin aerosol (3 to 7 days) and should not be treated with the drug. Thus the decision to treat with ribavirin aerosol should be based on the severity of the RSV infection.

The presence of an underlying condition such as prematurity or cardiopulmonary disease may increase the severity of the infection and its risk to the patient. High risk infants and young children with these underlying conditions may benefit from ribavirin treatment, although efficacy has been evaluated in only a small number of such patients.

Ribavirin aerosol treatment must be accompanied by and does not replace standard supportive respiratory and fluid management for infants and children with severe respiratory tract infection.

Diagnosis:

RSV infection should be documented by a rapid diagnostic method such as demonstration of viral antigen in respiratory tract secretions by immunofluorescence[3,4] or ELISA[5] before or during the first 24 hours of treatment. Ribavirin aerosol is indicated only for lower respiratory tract infection due to RSV. Treatment may be initiated while awaiting rapid diagnostic test results. However, treatment should not be continued without documentation of RSV infection.

CONTRAINDICATIONS:

Ribavirin is contraindicated in women or girls who are or may become pregnant during exposure to the drug. Ribavirin may cause fetal harm and respiratory syncytial virus infection is self-limited in this population. Ribavirin is not completely cleared from human blood even four weeks after administration. Although there are no pertinent human data, ribavirin has been found to be teratogenic and/or embryolethal in nearly all species in which it has been tested. Teratogenicity was evident after a single oral dose of 2.5 mg/kg in the hamster and after daily oral doses of 10 mg/kg in the rat. Malformations of skull, palate, eye, jaw, skeleton, and gastrointestinal tract were noted in animal studies. Survival of fetuses and offspring was reduced. The drug causes embryolethality in the rabbit at daily oral dose levels as low as 1 mg/kg.

WARNINGS:

Ribavirin administered by aerosol produced cardiac lesions in mice and rats after 30 and 36 mg/kg, respectively, for 4 weeks, and after oral administration in monkeys at 120 and rats at 154 to 200 mg/kg for 1 to 6 months. Ribavirin aerosol administered to developing ferrets at 60 mg/kg for 10 or 30 days resulted in inflammatory and possible emphysematous changes in the lungs. Proliferative changes were seen at 131 mg/kg for 30 days. The significance of these findings to human administration is unknown.

Ribavirin lyophilized in 6 gram vials is intended for use as an aerosol only.

PRECAUTIONS:

General:

Patients with lower respiratory tract infection due to respiratory syncytial virus require optimum monitoring and attention to respiratory and fluid status.

Drug Interactions:

Interactions of ribavirin with other drugs such as digoxin, bronchodilators, other antiviral agents, antibiotics, or antimetabolites has not been evaluated. Interference by ribavirin with laboratory tests has not been evaluated.

Carcinogenesis, mutagenesis, impairment of fertility:

Ribavirin induces cell transformation in an *in vitro* mammalian system (Balb/C3T3 cell line). However, *in vivo* carcinogenicity studies are incomplete. Results thus far, though inconclusive, suggest that chronic feeding of ribavirin to rats at dose levels in the range of 16-60 mg/kg body weight can induce benign mammary, pancreatic, pituitary and adrenal tumors.

Ribavirin is mutagenic to mammalian (L5178Y) cells in culture. Results of microbial mutagenicity assays and a dominant lethal assay (mouse) were negative.

Ribavirin causes testicular lesions (tubular atrophy) in adult rats at oral dose levels as low as 16 mg/kg/day (lower doses not tested), but fertility of ribavirin-treated animals (male or female) has not been adequately investigated.

Pregnancy:

Teratogenic Effects: Pregnancy Category X. See "Contraindications" section.

Nursing Mothers: Use of ribavirin aerosol in nursing mothers is not indicated because RSV infection is self-limited in this population. Ribavirin is toxic to lactating animals and their offspring. It is not known whether the drug is excreted in human milk.

ADVERSE REACTIONS:

Approximately 200 patients have been treated with ribavirin aerosol in controlled or uncontrolled clinical studies.

Pulmonary function significantly deteriorated during ribavirin aerosol treatment in six of six adults with chronic obstructive lung disease and in four of six asthmatic adults. Dyspnea and chest soreness were also reported in the latter group. Minor abnormalities in pulmonary function were also seen in healthy adult volunteers.

Several serious adverse events occurred in severely ill infants with life-threatening underlying diseases, many of whom required assisted ventilation. The role of ribavirin aerosol in these events is indeterminate. The following events were associated with ribavirin use:

Pulmonary: Worsening of respiratory status, bacterial pneumonia, pneumothorax, apnea, and ventilator dependence.

Cardiovascular: Cardiac arrest, hypotension, and digitalis toxicity.

There were 7 deaths during or shortly after treatment with ribavirin aerosol. No death was attributed to ribavirin aerosol by the investigators.

Some subjects requiring assisted ventilation have experienced serious difficulties, which may jeopardize adequate ventilation and gas exchange. Precipitation of drug within the ventilatory apparatus, including the endotracheal tube, has resulted in increased positive end expiratory pressure and increased positive inspiratory pressure. Accumulation of fluid in tubing ("rain out") has also been noted.

Although anemia has not been reported with use of the aerosol, it occurs frequently with oral and intravenous ribavirin, and most infants treated with the aerosol have not been evaluated 1 to 2 weeks post-treatment when anemia is likely to occur. Reticulocytosis has been reported with aerosol use.

Rash and conjunctivitis have been associated with the use of ribavirin aerosol.

Overdosage:

No overdosage with ribavirin by aerosol administration has been reported in the human. The LD_{50} in mice is 2 gm orally. Hypoactivity and gastrointestinal symptoms occurred. In man, ribavirin is sequestered in red blood cells for weeks after dosing.

DOSAGE AND ADMINISTRATION

Before use, read thoroughly the Viratek Small Particle Aerosol Generator (SPAG) Model SPAG-2 Operator's Manual for small particle aerosol generator operating instructions.

Treatment was effective when instituted within the first 3 days of respiratory syncytial virus lower respiratory tract infection.[3] Treatment early in the course of severe lower respiratory tract infection may be necessary to achieve efficacy.

Treatment is carried out for 12-18 hours per day for at least 3 and no more than 7 days, and is part of a total treatment program. The aerosol is delivered to an infant oxygen hood from the SPAG-2 aerosol generator. Administration by face mask or oxygen tent may be necessary if a hood cannot be employed (see SPAG-2 manual). However, the volume of distribution and condensation area are larger in a tent and efficacy of this method of administering the drug has been evaluated in only a small number of patients. Ribavirin aerosol is not to be administered with any other aerosol generating device or together with other aerosolized medications. Ribavirin aerosol should not be used for patients requiring simultaneous assisted ventilation (see Boxed Warnings).

Virazole is supplied as 6 grams of lyophilized drug per 100 ml vial for aerosol administration only. By sterile technique, solubilize drug with sterile USP water for injection or inhalation in the 100 ml vial. Transfer to the clean, sterilized 500 ml widemouth Erlenmeyer flask (SPAG-2 Reservoir) and further dilute to a final volume of 300 ml with sterile USP water for injection or inhalation. The final concentration should be 20 mg/ml. **Important:** This water should not have had any antimicrobial agent or other substance added. The solution should be inspected visually for particulate matter and discoloration prior to administration. Solutions that have been placed in the SPAG-2 unit should be discarded at least every 24 hours and when the liquid level is low before adding newly reconstituted solution.

Using the recommended drug concentration of 20 mg/ml ribavirin as the starting solution in the drug reservoir of the SPAG unit, the average aerosol concentration for a 12 hour period would be 190 micrograms/liter (0.19 mg/l) of air.

HOW SUPPLIED:

Virazole® (ribavirin) Aerosol is supplied in 100 ml glass vials with 6 grams of sterile, lyophilized drug which is to be reconstituted with 300 ml sterile water for injection or sterile water for inhalation (no preservatives added) and administered only by a small particle aerosol generator (SPAG-2). Vials containing the lyophilized drug powder should be stored in a dry place at 15-25°C (59-78°F). Reconstituted solutions may be stored, under sterile conditions, at room temperature (20-30°C, 68-86°F) for 24 hours. Solutions which have been placed in the SPAG-2 unit should be discarded at least every 24 hours.

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The appearance of the name American Academy of Pediatrics does not constitute a guarantee or endorsement of the product advertised or the claims made.

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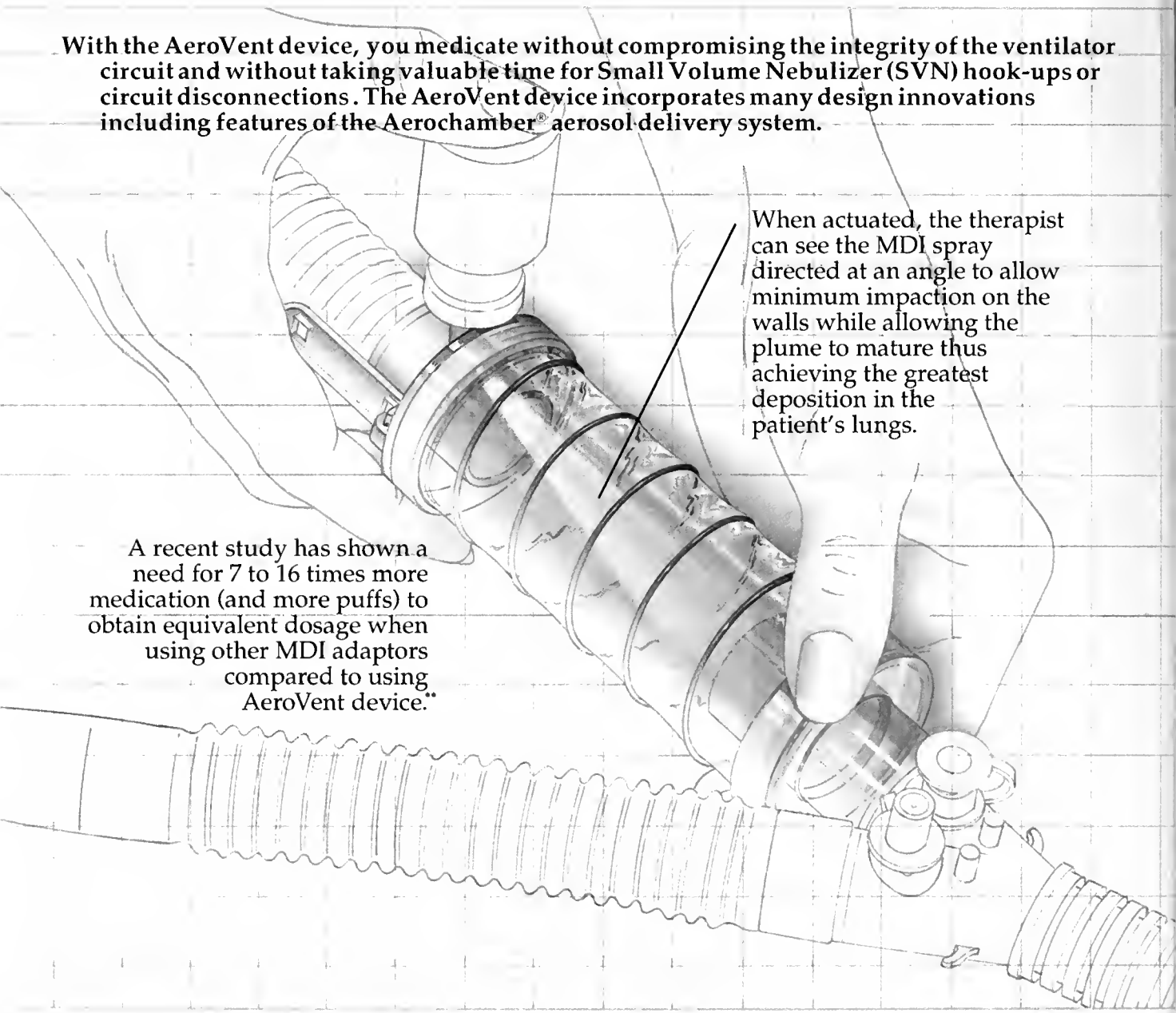

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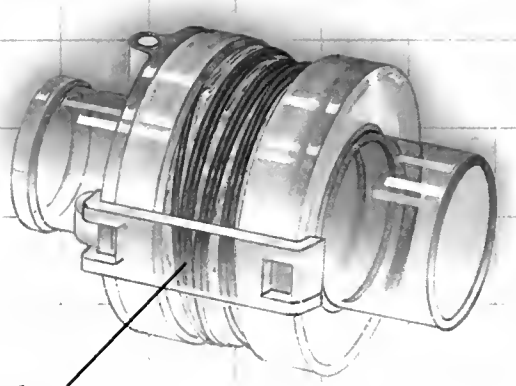

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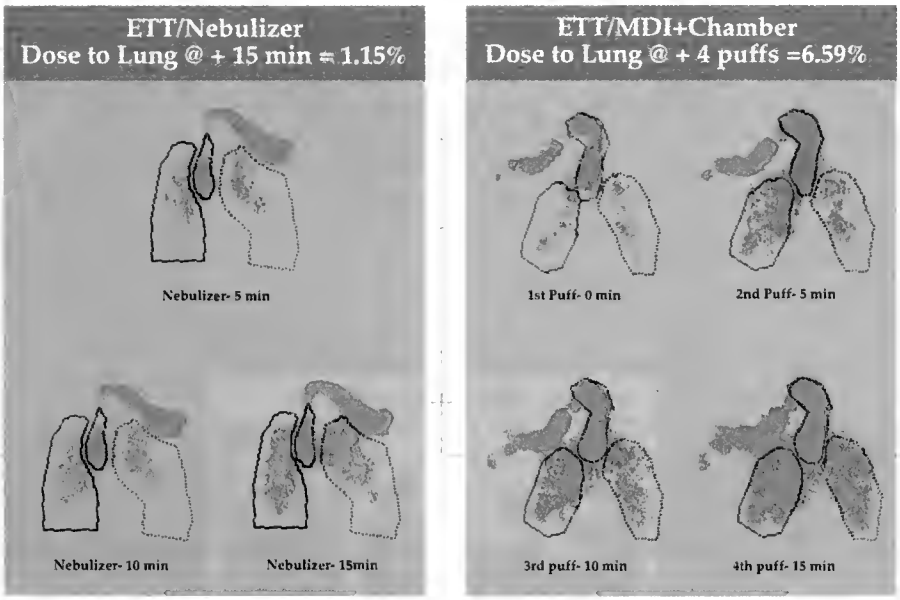

Graphic Representation of Actual Lung Deposition Data.

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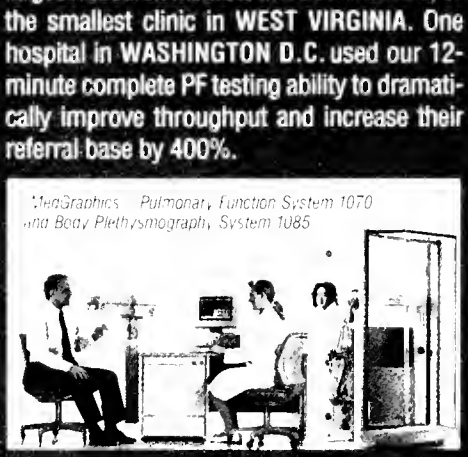

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Books, Films, Tapes, & Software

Asthma Resources Directory, by Carol Rudoff MA. Soft cover, 320 pages. Allergy Publications, PO Box 640, Menlo Park CA 94026. $29.95 plus $2.00 shipping (U.S. dollars).

In her introduction, the author states that this book provides a wide variety of resources and products, with 2,586 listings. I do not think that I have ever seen such a collection of information in one resource book.

The book is organized into four sections: Section I—Asthma Triggers, Section II—Patient Support, Section III—Medical Care, and Section IV—Information Resources.

In Asthma Triggers, the author begins with lists of months of the year, types of pollen, and states in which the pollens are found. A stronger beginning might have been a good introduction to what asthma is, how it is described medically (reversible airways obstruction), what that means, and what the various triggers are known to be. We have noted with patients in our hospital that asthmatic patients have a problem with the inferences that everyone with asthma has allergies and that everyone with allergies has asthma.

The information on filters, humidifiers, and similar devices is extensive. A diagram showing how to calculate the volume (number of cubic feet) in a room might be helpful to one planning to purchase a home filtering system. Placing the description of a device immediately before specific product information would also make the information clearer to the reader. The information contained in the chapters Living with the Air in Your Home and Controlling Your Home Environment could have been combined into one better-organized section. Regardless of the shortcomings in organization and the confusion it caused me, there is still an incredible amount of good information in these chapters.

The chapter on traveling with asthma has valuable information for anyone who has a chronic disease that requires preplanning a trip. Information listed under In an Emergency should be categorized. Putting Epi-Pen information in the same area as Emergency Prescription Service (provides a toll-free number to get prescriptions filled) just didn't make sense to me.

It has been our experience that patients with asthma want to read everything they can get their hands on. The Asthma Resources Book contains names, addresses, and telephone numbers for hundreds of groups that produce written, audio, and video materials related to this disease. For clinicians looking for patient education material, take this advice Don't 'reinvent the wheel.' Get this book, send off for all the free (or cheap) literature, and choose the best for use with your patients.

Medical Care contains information on pulmonary function equipment that may be useful to the technician but confusing to the layman. It might be more helpful to know the Whats and Whys about testing and how that information is used to determine the individualized treatment plan. Much, much more information could be given about self-monitoring. This whole book is talking about a proactive health model, and many clinicians believe that daily measurement of peak flow is one of the most important things that the patient can do to help gain control, instead of letting the disease be in control.

Pictures and diagrams are always helpful when they are simple and directly tied to written descriptions. Pictures of a typical air compressor and a disposable nebulizer would have been good additions to the section on adapters and respiratory devices.

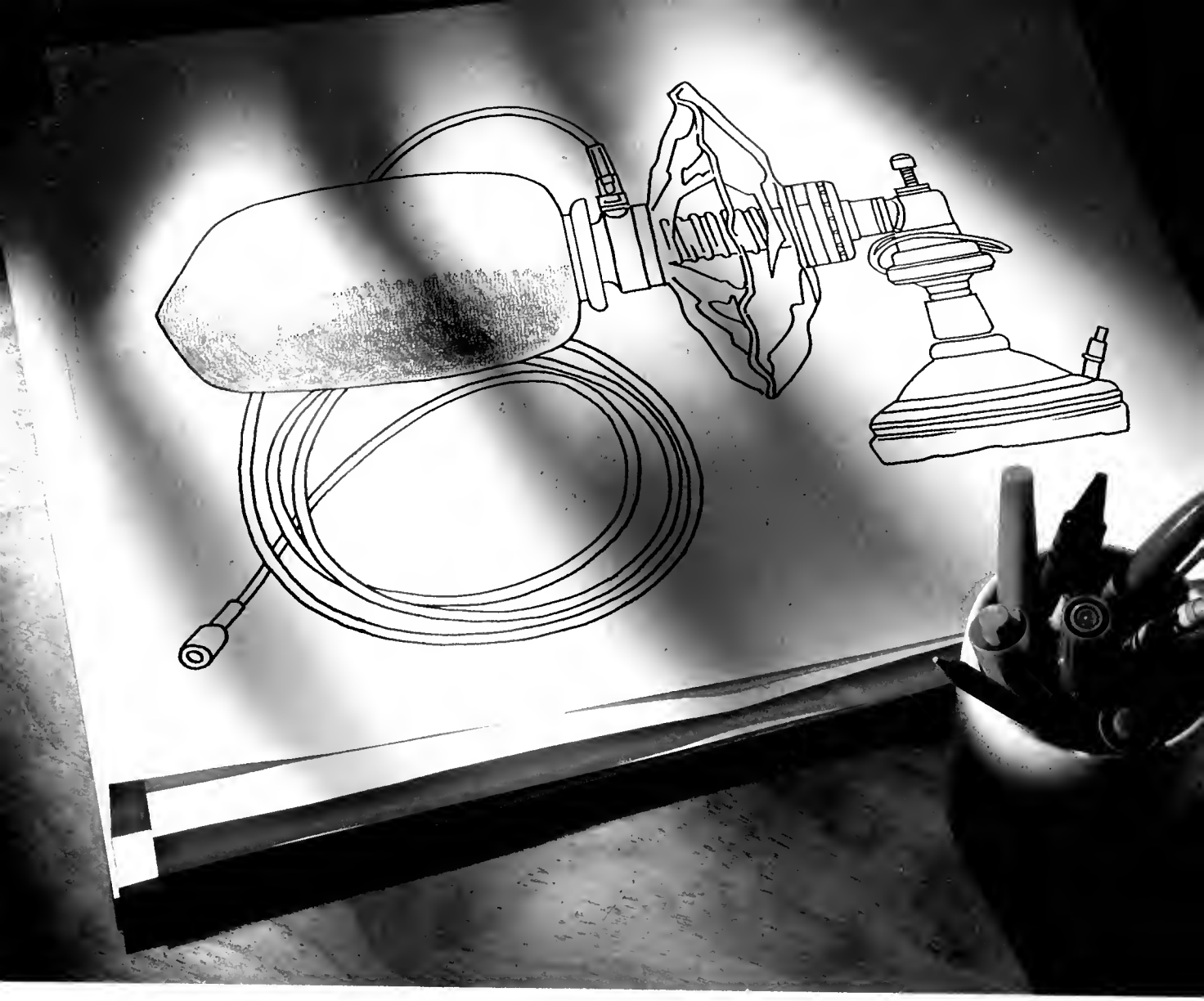
Information Sources provides some of the most valuable information in the book. The resources are categorized—organizations, health lines, directories, newsletters, and adult and childrens' reading materials. Under Libraries, a statement about what to ask for when you call your public library might suffice. Almost every community has a library and a mechanism within that library, no matter how small, to provide requested information. The Database listing was unclear to me. I am not computer literate and would need more information to make use of it.

Insurance was one of the best chapters and contained excellent information related to the very issues that persons with asthma and other so-called pre-existing conditions are struggling with. Remembering the differences among PPOs, HMOs, and IPAs is a challenge for anyone!

Undoubtedly, equipment manufacturers and pharmaceutical companies were asked to provide product information for this book, and, certainly, a book like this could not be compiled without their help. But, regardless of disclaimers, it was hard to miss the advertising contained in these 320 pages. There were reminders on almost every other page for the reader to let companies know they had been referred by the Asthma Resources Directory. That was distracting and tiresome.

The author is to be congratulated for putting together a complete resource. This book can be of great benefit to the clinician who is looking for just the right information to share with patients and their parents.

Gretchen Lawrence RRT
Manager
Asthma & Pulmonary
Rehabilitation Center
Baylor University Medical Center
Dallas, Texas



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Letters

PCIRV: Panacea or Auto-PEEP—A Response Based on Clinical Experience

We were disappointed when we read the editorial by Kacmarek and Hess in the October 1990 issue of RESPIRATORY CARE.[1] The editorial builds its case with what we believe to be some misrepresentation of previously published data. Although there is reference to one of the authors having experience with PCIRV in a laboratory model, there is no mention of clinical experience with the mode to substantiate the authors' point of view. We would like to clarify several points in the editorial that easily could be misconstrued.

The editorial stated that no control group was used during any of the studies. In the study conducted at our institution by Abraham and Yoshihara,[2] the patients themselves served as their own control. We believe that a comparison of the effects of two modes of mechanical ventilation is best achieved on the same patient, whereby the number of variables (eg, the disease state and APACHE score) are minimized. To use a separate group of patients as a control would have introduced additional variables, diminishing the significance of any correlations.

The authors of the editorial stated, "Further, we believe that the same effect might have occurred if the authors used higher levels of PEEP with conventional volume-control ventilation!" This comment may be true, but one must remember that the goal of PCIRV is not only to improve oxygenation, but to do so by using lower peak inspiratory pressures and lower inspired oxygen concentration. The respiratory rate, inspiratory pressure, and I-E ratios are manipulated to intentionally generate auto-PEEP. The auto-PEEP created is then used to replace the high levels of applied (set) PEEP. Thus, the set PEEP can be greatly reduced or eliminated. In addition, the change in pressure (ΔP) is reduced, which theoretically reduces the negative effect of shear forces.[3]

In PCIRV the set pressure and inspiratory flow are reached almost immediately. The inspiratory pressure is maintained for the duration of the set inspiratory time exposing pathologic alveoli to the peak inspiratory pressure (which is lower than conventional) for a longer period of time. This process promotes recruitment based on a time factor (ie, more time is dedicated to a pressure level that exceeds critical pressure). As the inspiratory flow encounters back pressure, the flow decelerates, which not only assists in maintaining the peak inspiratory pressure at a constant level, but also, in conjunction with the sustained inspiratory pressure, aids in the recruitment of unstable lung units and may provide better distribution of gas.[4-6] Therefore, auto-PEEP or high levels of set PEEP may not be the only alternatives to increased F_{IO_2} for improving oxygenation. PCIRV may provide a multifactoral alternative to improve oxygenation and ventilation in patients who are refractory to conventional therapy.

Kacmarek and Hess stated that auto-PEEP was not recognized or measured in the studies cited. Furthermore, they criticized that all of the papers on PCIRV failed to acknowledge that short expiratory times produce air trapping and auto-PEEP. The studies by Tharratt et al[7] and Abraham and Yoshihara[2] make reference to the fact that expiratory flow was monitored with an oscilloscope or strip recorder. The monitors were utilized to ensure that the end expiratory flow did not reach zero prior to triggering of the next breath. Although auto-PEEP is not mentioned as being recorded in any of the articles, the monitoring of the end-expiratory flow indicated that auto-PEEP was being monitored and intentionally generated to replace the applied PEEP. In the article by Abraham and Yoshihara,[2] end-expiratory pressure was measured and adjusted to maintain the same level as the PEEP utilized during volume-control ventilation. End-expiratory pressure is a more applicable term to use because it represents the total PEEP to which the patient is exposed.

Another criticism was "the presence of auto-PEEP also requires increased patient effort to trigger assisted breaths, which might be part of the reason that sedation and paralysis are necessary during PCIRV." The patient is sedated and paralyzed on PCIRV because the inspiratory-to-expiratory ratio is reversed, presenting an unnatural (and uncomfortable) pattern of breathing. To receive full benefit of the PCIRV mode, patients must be prevented from disrupting the preset I-E ratio. Paralysis prevents the patient from 'bucking,' assisting, or, in any other way, disrupting the preset I-E ratio. Because Kacmarek and Hess are well-respected, many readers may regard their editorials as being accurate, well-researched, and, therefore, applicable in the clinical setting. Because many readers may be influenced by these two authors, two unfortunate situations could result (1) patients who are appropriate candidates for PCIRV could be denied the benefit of the mode, and (2) studies with PCIRV could be deterred and the subsequent value of the knowledge and clinical experience lost.

Although we agree with the authors' suggestion that the mode be used with caution, we are not only surprised but also disappointed that they wrote such an adamant editorial without properly citing published data and without citing personal clinical experience with PCIRV to substantiate their views.

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Kacmarek and Hess respond:

Abraham et al imply in their response to our editorial that we developed our concerns regarding PCIRV purely on theoretical grounds. Although we chose to argue our point based on published data, our concern initially resulted from our anecdotal clinical experience. It is true that we may be less capable of applying this ventilatory technique than Abraham et al, but our results have been much less promising than theirs. The frequency of hemodynamic compromise and the development of barotrauma as well as the magnitude of auto-PEEP developed in our experience far exceeds that noted in the literature. We have anecdotally noted a far better response in our patients with conventional ventilation and PEEP than with PCIRV.

We indicated in our editorial[1] that no control group was used by Abraham and Yoshihara (1989);[2] however, they indicate in their letter that patients themselves were used as controls. We must question these authors' understanding of a controlled clinical study. This study[3] did not control for a single variable between the two treatment modalities except for end-expiratory pressure. In addition, no stated protocol for the setup of conventional ventilation with PEEP is presented. We ask the authors to compare these methods in the 1989 study[2] to those in their recently published study on pressure control ventilation.[3] In their 1990 study, the application of volume-limited and pressure-controlled ventilation was controlled adequately enough to allow the authors' results (an increase in PO_2) to be attributed to the use of pressure control.

Abraham et al state that auto-PEEP was used to replace the high levels of applied PEEP. This we believe is exactly what occurred in their study; however, no measure of the auto-PEEP that developed as the I:E was reversed is provided. They do point out in their introduction that PCIRV is associated with auto-PEEP, but make no further reference to its development. Abraham et al argue that they monitored expiratory flow and terminated expiration before expiratory flow fell to zero. We agree that this implies the presence of auto-PEEP. However, in no way does this indicate the magnitude of auto-PEEP. The use of the term end-expiratory pressure and applied PEEP by Abraham et al are confusing to us. They imply that end-expiratory pressure represents total PEEP. We are unaware of the use of this term to represent auto- plus applied PEEP nor do Abraham et al define how total PEEP can be determined without the direct measurement of auto-PEEP. In addition, as we previously indicated, equal levels of auto-PEEP and applied PEEP may not produce the same physiologic response.

The fact that the inspiratory-to-expiratory pressure differential that alveoli are exposed to during volume-limited ventilation with normal I:E is higher than that occurring during PCIRV has never been demonstrated. However, we agree with Abraham et al that the use of pressure control ventilation at normal ratios may result in improved distribution of inspired gas and better gas exchange.[3] However, we believe that additional research is necessary before any conclusion can be drawn.

Two issues confuse the discussions of the efficiency of PCIRV. One is I:E, about which we believe no data exist supporting the use of inversed ratios over conventional ratios. The other is the use of pressure control ventilation at conventional ratios. These two issues need to be separated and studied independently in order to resolve the questions raised regarding PCIRV. We do encourage controlled systematic study of PCIRV, but we must still caution practitioners about the use of inverse-ratio ventilation until more complete data are available.

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3. Abraham E, Yoshihara G. Cardiorespiratory effects of pressure control ventilation in severe respiratory failure. Chest 1990;98:1445-1449.

Notices

Notices of competitions, scholarships, fellowships, examination dates, new education programs, and the like will be listed here free of charge. Items for the Notices section must reach the Journal 60 days before the desired month of publication (January 1 for the March issue, February 1 for the April issue, etc). Include all pertinent information and mail notice to RESPIRATORY CARE Notices Dept, 11030 Ables Lane, Dallas TX 75229.

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March 14-15 in Newport, Rhode Island. The RISRC presents "The Newport Challenge: Sailing into the 90s." Topics include transtracheal oxygenation, ventilator weaning, humor as therapy, microphysiology of asbestosis, and cystic fibrosis. Workshops, social activities and a large exhibit complete the program. Contact Skip Bangley RRT, St Joseph Hospital, 21 Peace St, Providence RI 02907 or call (401) 456-4174.

March 25-27 in Philadelphia, Pennsylvania. The PSRC presents its 26th Annual Conference and Exhibition at the Adam's Mark Hotel. This year's theme is "The Next Generation," featuring a space-age trek into the realm of respiratory care and cardiovascular technology. Contact Betsy Schneck, (215) 829-3578 or Kathy Yandle, (215) 453-4517.

April 3-5 in Nashville, Tennessee. The TSRC presents its Annual Convention and Exhibition at the Vanderbilt Plaza Hotel. This year's theme is Everything Under The Sun in '91, and focuses on current trends and all phases of cardiopulmonary care. For registration information contact Collen Schabacker at (615) 384-1569. For exhibit information contact Candy Partee at (615) 449-0500.

April 10-12 in Bismarck, North Dakota. A New Decade of Strength in Respiratory Care is the theme for NDSRC's annual convention. Topics include nutrition studies, adult and neonatal critical care issues, and management strategies. For more information, call (701) 224-7870.

April 17-19 in Osage Beach, Missouri. The Missouri Society for Respiratory Care presents its 20th Annual Meeting at the Tan-Tar-A resort, Lake of the Ozarks. Speakers include Neil McIntypre MD and Sheldon Braun MD Contact Jim Pattinson RRT, at (417) 885-2800.

May 1-3 in Rapid City, South Dakota. Rapid City Regional Hospital hosts the annual convention of the SDSRC. Featured speakers (including WJ O'Donohue Jr MD, Robert Kacmarek PhD RRT, and Anthony Talbert MD) discuss neonatal, pediatric, and adult critical care issues. Contact Terry Anderson RRT, Respiratory Care Department, 1-800-232-9287.

May 16-17 in Wichita, Kansas. The KRCS presents its 14th Annual Education Seminar at the Airport Hilton. Topics range from future directions of the profession to PFT, asthma, and critical care issues. Scheduling allows for golf, exhibits and a great time for everyone. Contact Don Richards, MS RRT, VA Medical Center, (316) 685-2221 for more information.

May 28-31 in Jekyll Island, Georgia. The Georgia/ South Carolina Region VI presents its 15th Annual Conference and Assembly at the Holiday Inn, Jekyll Island. Contact Mike Payne RRT, 730 South Pleasantburg Dr, Suite 525, Greenville SC 29607. (803) 879-0130.

June 12-14 in Vail, Colorado. The CSRC State Convention's theme "Bringing It All Together" focuses on the crossover between hospital and home care. A golf tournament is featured for the morning of the 12th, followed by a barbecue picnic that evening. Contact Jim Bowman, Vencor Hosn, 1920 High St, Denver CO 80218, (303) 320-5871.

July 10-12 in Houston, Texas. The TSRC meeting features AARC President Patrick Dunne, Kevin Shrake, Diane Lewis, Connie Podesta, and Ulf Borg. For information contact TSRC Executive Office, PO Box 515239, Dallas TX 75251, (214) 239-8772 or FAX (214) 239-6418.

OTHER MEETINGS

March 10-13 in Denver, Colorado. The National Jewish Center for Immunology and Respiratory Medicine, in conjunction with the American College of Chest Physicians, presents the 3rd International Conference on Pulmonary Rehabilitation and Home Mechanical Ventilation, with concurrent workshops on home ventilator care and pulmonary rehabilitation, at the Denver Hyatt. Contact Adele Gelfand, Conference Coordinator, (303) 398-1359.

April 12-14, Miami to Nassau. Another "Floating Seminar" for Health Care Professionals on "Quality Assurance — How To Accomplish It." A hands-on program. Deadline for reservations — March 15, 1991. Contact Dave Robbins at (305) 441-6819.

May 17-18 in Las Vegas, Nevada. The American Lung Association of Nevada sponsors the 6th Annual Southern Nevada Respiratory Health Conference at the Sahara Hotel. Contact American Lung Association of Nevada, PO Box 44137, Las Vegas NV 89116. (702) 454-2500.

August 25-September 1, Caribbean Cruise. Cruise the Western Caribbean aboard the SS Sea Breeze while earning 8 CRCE credits. Topic is "Aid for AIDS." $895 prepaid includes airfare, cruise, transfers, food, and entertainment. Friends and family welcome. Call or write Dream Cruises, 10882 LaDona Ave, Garden Grove CA 92640. 1-800-462-3628.

1991 Call for Abstracts

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 papers at the Open Forum during the AARC Annual Meeting in Atlanta, Georgia, December 7-10, 1991. Accepted abstracts will be published in the November 1991 issue of RESPIRATORY CARE. Membership in the AARC is not necessary for participation.

Specifications

An abstract may report (1) an **original study**, (2) the **evaluation of a method or device**, or (3) a **case or case series**. Topics may be aspects of adult acute care, continuing care/rehabilitation, perinatology/pediatrics, cardiopulmonary technology, health occupations education, or management of personnel and 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 will decide whether the author should be invited to present a paper 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

An **original study** abstract ***must*** include (1) Introduction: 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.

A **method/device evaluation** abstract ***must*** include (1) Introduction: identification of the method or device 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 notation of lack of experience; (5) Conclusions: interpretation of the evaluation and experience. Cost comparisons should be included where possible and appropriate.

A **case report** abstract ***must*** report a case that is uncommon or of exceptional teaching/learning value and must include: (1) case summary and (2) significance of case. 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.

Abstract Format and Typing Instructions

An optical scanner will be used to process abstracts. First line of abstract should be the title. Title should explain content. Type or electronically print the abstract ***double-spaced*** on ***plain white bond paper***, on ***one page*** only (copier bond is excellent). Do not underline or boldface and insert only one letter space between sentences. Provide a 1-inch margin top and bottom, a ½-inch left margin, and an approximate ½-inch ragged-right margin. Text may be submitted on diskette but must be accompanied by a hard copy.

No identification of authors or institutions is to appear on the abstract sheet or in the abstract itself. Make the abstract all one paragraph. Data may be submitted in table form ***provided the table width is limited to 60 letter spaces*** (ie, letters or numbers plus necessary blank spaces = 60). ***No*** figures or illustrations are to be attached to the abstract.

Type all information required to complete the author information form on the other side of this page. A photocopy of good quality may be used.

Standard abbreviations may be employed without explanation. A new or infrequently used abbreviation should be preceded by the spelled-out term the first time it is used. 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; (3) conformance to these specifications. An abstract not prepared as requested may not be reviewed.

Questions about abstract preparation may be telephoned to the editorial staff of RESPIRATORY CARE at (214) 243-2272.

Deadlines

The mandatory Final Deadline is June 5 (postmark). Authors will be notified of acceptance or rejection ***by letter only***—to be mailed by August 15.

Authors may choose to submit abstracts early. Abstracts received by March 20 will be reviewed and the authors notified by April 26. 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 (June 5).

Mailing Instructions

Mail (Do not fax!) 1 copy of the abstract, 1 author information sheet, and a stamped, self-addressed postcard (for notice of receipt) to:

RESPIRATORY CARE
11030 Ables Lane
Dallas TX 75229

1991 OPEN FORUM
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Mail, with abstract and stamped self-addressed postcard, to RESPIRATORY CARE Open Forum
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RESPIRATORY CARE

Instructions for Authors and Typists

These Instructions are meant to guide authors and typists, including veterans in those roles, in the production of quality manuscripts. Perfection is not expected, but the well-prepared manuscript has the best chance for prompt review and early publication.

General Requirements

Submissions should (1) be related to respiratory care, (2) be planned for one of the publication categories below, and (3) be prepared as indicated in these Instructions. A letter accompanying the manuscript must specify the intended publication category, be signed by all the authors, and, when there are two or more authors, state that "We, the undersigned, have all participated in the work reported, read the accompanying manuscript, and approved its submission for publication."

Publication Categories

Research Article (Study): A report of an original investigation.
Evaluation of a Device/Method/Technique: A description and evaluation of an old or new device, method, technique, or modification.
Case Report: A report of a clinical case that is uncommon or of exceptional teaching value. The author(s) must have been associated with the case. A case-managing physician must be one of the authors or, if not an author, must supply a letter approving the manuscript.
Case Series: Like a Case Report but including a number of cases.
Review Article: A comprehensive, critical review of the literature and state of the art of a pertinent topic that has been the subject of 40 or more published research papers.
Overview: A critical review of a pertinent topic about which not enough research has been published to merit a Review Article.
Update: A report of subsequent developments in a topic that has been critically reviewed (not necessarily in this journal).
Point of View: A paper expressing the author's personal opinions on a pertinent topic.
Special Article: If a paper does not fit one of the foregoing categories but is pertinent, the editors may consider it as a Special Article.
Editorial: A paper that draws attention to a pertinent concern.
Letter: A signed communication about material published in this journal or on topics of interest or value to readers.
Blood Gas Corner: A brief, instructive case report (real or fictional) involving invasively or noninvasively obtained respiratory care blood data, followed by questions for readers—with answers and discussion.
PFT Corner: Like Blood Gas Corner but involving pulmonary function testing.
Test Your Radiologic Skill: Like Blood Gas Corner and PFT Corner but involving pulmonary-medicine radiography and including one or two 4 × 5 or 5 × 7 inch prints of radiographs. The case must be real.
Review of Book, Film, Tape, or Software: Anyone interested in writing a review can discuss it with an editor.

Editorial Consultation and Author's & Typist's Kit

To discuss a writing project, write to RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229 or call 214/243-2272.

Authors are urged to obtain the RESPIRATORY CARE Author's & Typist's Kit. The Kit provides *authors* with specific guidance about writing a research paper, writing a case report, converting to and from SI units, and in-house manuscript review. *Typists* can use the Kit's Model Manuscript, a list of journal name abbreviations, and a copy of these Instructions. The Kit is free from the Journal office.

Preparing the Manuscript

General Concerns—*Typist*

- Double-space ALL lines, including those in references, figure legends, and tables. Do not justify right margins.
- Number pages in upper right corner and leave margins of 1½″ or more on all four sides of the page.
- For research articles, follow format of Model Manuscript, Respir Care 1984;29:182 (Feb 1984).
- Meticulously follow instructions for typing references.

General Concerns—*Author:*

- Structure manuscript as specified hereafter.
- Provide all requested information on title page as specified hereafter.
- Proofread manuscript for completeness, clarity, grammar, spelling; be sure all references, figures, and tables are cited in the text.
- Consider having paper reviewed in-house before submission.
- Have all co-authors proofread and approve manuscript and sign submission letter.

Manuscript Structure

Most kinds of papers have standard parts in a standard order. However, papers can vary individually, and not every paper will have all the parts listed here.

Research Article: Title page, abstract page, continuous text (Introduction, Materials & Methods, Results, Discussion), Product Sources page, Acknowledgments page, references, tables, figure legends. Please consult "Writing a Research Paper," Respir Care 1985;30:1057 (Dec 1985) and Model Manuscript, Respir Care 1984;29:182 (Feb 1984).
Evaluation of Device/Method/Technique: Title page, abstract page, continuous text (Introduction, Description of Device/Method/Technique, Methods of Evaluation, Results of Evaluation, Discussion), Product Sources page, Acknowledgments page, references, tables, figure legends.
Case Report or **Case Series:** Title page, abstract page, continuous text (Introduction, Case Summary, Discussion), Acknowledgments page, references, tables, figure legends. Also see "How To Write a Better Case Report," Respir Care 1982;27:29 (Jan 1982).
Review Article: Title page, Table of Contents page, continuous text (Introduction, History, Review of Literature, State of the Art, Discussion, Summary), references. May include figures & tables. No abstract. Table of Contents optional. Other formats may be appropriate.
Overview, Update, Point of View, or Special Article: Title page, text (introduction, message), references, tables, figure legends. No abstract.
Letter: Title page (provide a title), text, writer's name & affiliation, references. Tables & figures may be included. Double-space everything. Write "For Publication" on title page.

Structure: Important Details

Title Page: List title of paper, all authors' full names, degrees, credential letters, professional positions, and affiliations. List correspondence address, telephone number, and reprint address if desired. Name sources of grants or other support. Identify any author's consulting or commercial relationships that pertain to the paper's topic.

Abstract Page: Number this Page 1. List paper's title but omit authors' names. Abstract should be 200 words or less and must be informative, briefly specifying main points of paper, such as methods, results, and conclusions drawn.

Statistical Analysis: In research articles, identify statistical tests and chosen level of significance in the Methods section. In Results section, report actual P values.

Figures (illustrations): All photographs, diagrams, & graphs must be numbered as Figure 1, Figure 2, etc, according to the order in which each is first mentioned in the text. Photographs must be glossy prints 5 × 7 to 8 × 10 inches and should be black & white unless color is essential. Letters and numerals must be neat and large enough to remain legible if figure is reduced in size for publication. Final figures must be of professional quality, but 'rough' sketches may accompany the submitted manuscript, with final figures to be prepared after review. Identify each figure on back with a stick-on label showing figure number and arrow indicating top; omit author's name. Cover label with clear tape so ink will not smudge other prints. Supply three sets of unmounted figures. If figure has been published before, include copyright-holder's written permission to use it.

Figure Legends: List figure legends on a separate page, not on figures. If a figure has been published before, list the source in the legend.

Tables: Type each table on a separate page. Avoid more than 8 columns across. Continue a deep table on following pages. Give each table a number and descriptive title, placed above the table. Double-space ALL lines in tables, including column headings and footnotes.

Drugs: Brand names may be given, but always also show generic names.

Units of Measurement: In addition to conventional units of measure, show SI values and units in brackets after conventional expressions: ie, "PEEP, 10 cm H_2O [0.981 kPa]." For conversion to SI, see RESPIRATORY CARE 1988;33:861-873 (Oct 1988).

Commercial Products: If three or fewer commercial products are named in the text, list the manufacturer's name and location in parentheses the first time each is mentioned. If four or more products are named, do not list manufacturers in the text; instead, name the products and manufacturers in a Products Sources list at the end of the text. Provide model numbers when available.

Abbreviations: Use an abbreviation only if the term occurs several times in the paper. Write out the full term the first time it appears, followed by the abbreviation in parentheses. Thereafter, employ the abbreviation alone. Never use an abbreviation without defining it. Do not create new abbreviations unless absolutely necessary.

References:

- Use references to support statements of fact, indicate sources of information, or guide readers to further pertinent literature.
- Cite only published works—or works accepted for publication. When listing an accepted but still unpublished work, designate the accepting journal's name, followed by "(in press)."
- In the text, cite references by superscript numerals (half space above text), not in parentheses. The first reference cited in the text is number 1, the next is number 2, etc.
- In the reference list, place the cited works in numerical order.
- For the reference list, obtain author names, article and book titles, dates, volume and page numbers from the *original* cited articles and books, not from secondary sources such as other articles' reference lists, which often are inaccurate.
- Type references in medical-journal style. Examples appear at the end of these Instructions. Abbreviate journal names as in *Index Medicus*. A list of many journal-name abbreviations was published in Respir Care 1988;33:1050 (Nov 1988).
- DOUBLE-SPACE the lines of references.
- List ALL authors' names. Do not use "et al" to substitute for names.
- Identify abstracts, editorials, and letters as such. See examples.

Personal Communications, Unpublished Papers, and Unpublished Observations: List unpublished items in parentheses in the text, not in the reference list.

Examples of How To Type References

Notes: Although the examples here are printed with single-spaced lines, please double-space references in manuscripts. Also, note that words in article and book titles are not capitalized—except proper names.

Standard Journal Article:

1. Shepherd KE, Johnson DC. Bronchodilator testing: An analysis of paradoxical responses. Respir Care 1988;33:667-671.

Corporate Author Journal Article:

2. American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. Respir Care 1988;33:1044-1046.

Article in Journal Supplement:

(Journals differ in their methods of numbering and identifying supplements. Supply sufficient information to allow retrieval.)

3. Reynolds HY. Idiopathic interstitial pulmonary fibrosis. Chest 1986;89(3, suppl):139s-143s.

Abstract in Journal:

(Abstracts are not strong references; when possible, full papers should be cited. When cited, abstracts should be identified as such.)

4. Lippard DL, Myers TF, Kahn SE. Accuracy of pulse oximetry in severely hypoxic infants (abstract). Respir Care 1988;33:886.

Editorial in Journal:

5. Rochester DF. Does respiratory muscle rest relieve fatigue or incipent fatigue? (editorial). Am Rev Respir Dis 1988;138:516-517.

Letter in Journal:

6. Smith DE, Herd D, Gazzard BG. Reversible bronchoconstriction with nebulised pentamidine (letter). Lancet 1988;2:905.

Personal Author Book:

7. Nunn JF. Applied respiratory physiology. New York: Appleton-Century-Crofts, 1969.

Note: To specify pages cited in a book, place a colon after the year and then list the page(s). Examples: 1969:85 (one page), 1963:85-95 (series of contiguous pages), 1963:85,95 (separated pages).

Corporate Author Book:

8. American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group, 1977.

Book with Editor, Compiler, or Chairman as 'Author':

9. Guenter CA, Welch MH, eds. Pulmonary medicine. Philadelphia: JB Lippincott, 1977.

Chapter in Book:

10. Pierce AK. Acute respiratory failure. In: Guenter CA, Welch MH, eds. Pulmonary medicine. Philadelphia: JB Lippincott, 1977:171-223.

Submitting the Manuscript

After preparing the manuscript according to these Instructions, perform a final proofreading and check for accuracy and completeness. Then mail three copies of the manuscript and three sets of figures to RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229 (or Federal Express to RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229). Manuscript copy on IBM-compatible or Macintosh disks in addition to the requisite three hard copies will facilitate processing (Macintosh preferred). Enclose a letter as specified under **General Requirements** at the beginning of these Instructions. Do not submit material that has been published or is being considered elsewhere.

Author's Checklist

1. Is paper for a listed publication category?
2. Does cover letter meet specifications?
3. Is title page complete?
4. Are all pages double-spaced and numbered?
5. Are all references, figures, and tables cited in the text?
6. Are references typed in requested style?
7. Have SI values been provided?
8. Has all arithmetic been checked?
9. Has manuscript been proofread by all authors?

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I. Type of Instn/Practice
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2. ☐ Hosp. 300 to 500 beds
3. ☐ Hosp. 200 to 300 beds
4. ☐ Hosp. 100 to 200 beds
5. ☐ Hosp. 100 or less beds
6. ☐ Clinic/Group Practice
7. ☐ Independent RT Provider
8. ☐ Industry (Mfgr/Sales)

II. Department
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B. ☐ Cardiopulmonary
C. ☐ Anesthesia Service
D. ☐ Emergency Dept.

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1. ☐ Clinical Practice
2. ☐ Perinatal Pediatrics
3. ☐ Critical Care
4. ☐ Clinical Research
5. ☐ Pulmonary Func Lab
6. ☐ Home Care/Rehab
7. ☐ Education
8. ☐ Management

IV. Position
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B. ☐ Chief Therapist
C. ☐ Supervisor
D. ☐ Staff Technician
E. ☐ Staff Therapist
F. ☐ Educator
G. ☐ Medical Director
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V. Are you a member of the AARC?
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I. Type of Instn/Practice
1. ☐ Hosp. 500 or more beds
2. ☐ Hosp. 300 to 500 beds
3. ☐ Hosp. 200 to 300 beds
4. ☐ Hosp. 100 to 200 beds
5. ☐ Hosp. 100 or less beds
6. ☐ Clinic/Group Practice
7. ☐ Independent RT Provider
8. ☐ Industry (Mfgr/Sales)

II. Department
A. ☐ Respiratory Ther.
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III. Specialty
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4. ☐ Clinical Research
5. ☐ Pulmonary Func Lab
6. ☐ Home Care/Rehab
7. ☐ Education
8. ☐ Management

IV. Position
A. ☐ Dept. Head
B. ☐ Chief Therapist
C. ☐ Supervisor
D. ☐ Staff Technician
E. ☐ Staff Therapist
F. ☐ Educator
G. ☐ Medical Director
H. ☐ Anesthesiologist
I. ☐ Other MD
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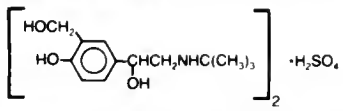
DESCRIPTION PROVENTIL, brand of albuterol sulfate, Solution for Inhalation, is a relatively selective $beta_2$-adrenergic bronchodilator (see **CLINICAL PHARMACOLOGY** section below). Albuterol sulfate has the chemical name α^1-[(*tert*-Butylamino) methyl]-4-hydroxy-*m*-xylene-α,α'-diol sulfate (2:1) (salt), and the following chemical structure:



Albuterol sulfate has a molecular weight of 576.7 and the empirical formula $(C_{13}H_{21}NO_3)_2H_2SO_4$. Albuterol sulfate is a white crystalline powder, soluble in water and slightly soluble in ethanol.

The international generic name for albuterol base is salbutamol.

PROVENTIL Solution for Inhalation is available in two concentrations. The 0.5% solution is in concentrated form. Dilute 0.5 mL of the solution to 3 mL with normal saline solution prior to administration. The 0.083% solution requires no dilution prior to administration.

Each mL of PROVENTIL Solution for Inhalation (0.5%) contains 5 mg of albuterol (as 6.0 mg of albuterol sulfate) in an aqueous solution containing benzalkonium chloride; sulfuric acid is used to adjust the pH between 3 and 5. PROVENTIL Solution for Inhalation (0.5%) contains no sulfiting agents. It is supplied in 20 mL bottles.

Each mL of PROVENTIL Solution for Inhalation (0.083%) contains 0.83 mg of albuterol (as 1.0 mg of albuterol sulfate) in an isotonic aqueous solution containing sodium chloride and benzalkonium chloride; sulfuric acid is used to adjust the pH between 3 and 5. PROVENTIL Solution for Inhalation (0.083%) contains no sulfiting agents. It is supplied in 3 mL bottles for unit-dose dispensing.

PROVENTIL Solution for Inhalation is a clear, colorless to light yellow solution.

CLINICAL PHARMACOLOGY The prime action of beta-adrenergic drugs is to stimulate adenyl cyclase, the enzyme which catalyzes the formation of cyclic-3',5'-adenosine monophosphate (cyclic AMP) from adenosine triphosphate (ATP). The cyclic AMP thus formed mediates the cellular responses. *In vitro* studies and *in vivo* pharmacologic studies have demonstrated that albuterol has a preferential effect on $beta_2$-adrenergic receptors compared with isoproterenol. While it is recognized that $beta_2$-adrenergic receptors are the predominant receptors in bronchial smooth muscle, recent data indicate that 10 to 50% of the beta receptors in the human heart may be $beta_2$ receptors. The precise function of these receptors, however, is not yet established. Albuterol has been shown in most controlled clinical trials to have more effect on the respiratory tract, in the form of bronchial smooth muscle relaxation, than isoproterenol at comparable doses while producing fewer cardiovascular effects. Controlled clinical studies and other clinical experience have shown that inhaled albuterol, like other beta-adrenergic agonist drugs, can produce a significant cardiovascular effect in some patients, as measured by pulse rate, blood pressure, symptoms, and/or ECG changes.

Albuterol is longer acting than isoproterenol in most patients by any route of administration because it is not a substrate for the cellular uptake processes for catecholamines nor for catechol-*O*-methyl transferase.

Studies in asthmatic patients have shown that less than 20% of a single albuterol dose was absorbed following either IPPB or nebulizer administration; the remaining amount was recovered from the nebulizer and apparatus and expired air. Most of the absorbed dose was recovered in the urine 24 hours after drug administration. Following a 3.0 mg dose of nebulized albuterol, the maximum albuterol plasma level at 0.5 hour was 2.1 ng/mL (range 1.4 to 3.2 ng/mL). There was a significant dose-related response in FEV_1 and peak flow rate (PFR). It has been demonstrated that following oral administration of 4 mg albuterol, the elimination half-life was 5 to 6 hours.

Animal studies show that albuterol does not pass the blood-brain barrier. Recent studies in laboratory animals (minipigs, rodents, and dogs) recorded the occurrence of cardiac arrhythmias and sudden death (with histologic evidence of myocardial necrosis) when beta-agonists and methylxanthines were administered concurrently. The significance of these findings when applied to humans is currently unknown.

In controlled clinical trials, most patients exhibited an onset of improvement in pulmonary function within 5 minutes as determined by FEV_1. FEV_1 measurements also showed that the maximum average improvement in pulmonary function usually occurred at approximately 1 hour following inhalation of 2.5 mg of albuterol by compressor-nebulizer, and remained close to peak for 2 hours. Clinically significant improvement in pulmonary function (defined as maintenance of a 15% or more increase in FEV_1 over baseline values) continued for 3 to 4 hours in most patients and in some patients continued up to 6 hours.

In repetitive dose studies, continued effectiveness was demonstrated throughout the 3-month period of treatment in some patients.

INDICATIONS AND USAGE PROVENTIL Solution for Inhalation is indicated for the relief of bronchospasm in patients with reversible obstructive airway disease and acute attacks of bronchospasm.

CONTRAINDICATIONS PROVENTIL Solution for Inhalation is contraindicated in patients with a history of hypersensitivity to any of its components.

WARNINGS As with other inhaled beta-adrenergic agonists, PROVENTIL Solution for Inhalation can produce paradoxical bronchospasm, which can be life threatening. If it occurs, the preparation should be discontinued immediately and alternative therapy instituted.

Fatalities have been reported in association with excessive use of inhaled sympathomimetic drugs and with the home use of sympathomimetic nebulizers. It is, therefore, essential that the physician instruct the patient in the need for further evaluation if his/her asthma becomes worse. In individual patients, any $beta_2$-adrenergic agonist, including albuterol solution for inhalation, may have a clinically significant cardiac effect.

Immediate hypersensitivity reactions may occur after administration of albuterol as demonstrated by rare cases of urticaria, angioedema, rash, bronchospasm, and oropharyngeal edema.

PRECAUTIONS General: Albuterol, as with all sympathomimetic amines, should be used with caution in patients with cardiovascular disorders, especially coronary insufficiency, cardiac arrhythmias and hypertension, in patients with convulsive disorders, hyperthyroidism or diabetes mellitus, and in patients who are unusually responsive to sympathomimetic amines.

Large doses of intravenous albuterol have been reported to aggravate preexisting diabetes mellitus and ketoacidosis. Additionally, beta-agonists, including albuterol, when given intravenously may cause a decrease in serum potassium, possibly through intracellular shunting. The decrease is usually transient, not requiring supplementation. The relevance of these observations to the use of PROVENTIL Solution for Inhalation is unknown.

Information For Patients: The action of PROVENTIL Solution for Inhalation may last up to 6 hours and therefore it should not be used more frequently than recommended. Do not increase the dose or frequency of medication without medical consultation. If symptoms get worse, medical consultation should be sought promptly. While taking PROVENTIL Solution for Inhalation, other antiasthma medicines should not be used unless prescribed.

Drug Interactions: Other sympathomimetic aerosol bronchodilators or epinephrine should not be used concomitantly with albuterol.

Albuterol should be administered with extreme caution to patients being treated with monoamine oxidase inhibitors or tricyclic antidepressants, since the action of albuterol on the vascular system may be potentiated.

Beta-receptor blocking agents and albuterol inhibit the effect of each other.

Carcinogenesis, Mutagenesis, and Impairment of Fertility: Albuterol sulfate, like other agents in its class, caused a significant dose-related increase in the incidence of benign leiomyomas of the mesovarium in a 2-year study in the rat, at oral doses corresponding to 10, 50, and 250 times the maximum human nebulizer dose. In another study, this effect was blocked by the coadministration of propranolol. The relevance of these findings to humans is not known. An 18-month study in mice and a lifetime study in hamsters revealed no evidence of tumorigenicity. Studies with albuterol revealed no evidence of mutagenesis. Reproduction studies in rats revealed no evidence of impaired fertility.

Teratogenic Effects–Pregnancy Category C: Albuterol has been shown to be teratogenic in mice when given subcutaneously in doses corresponding to the human nebulization dose. There are no adequate and well-controlled studies in pregnant women. Albuterol should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus. A reproduction study in CD-1 mice with albuterol (0.025, 0.25, and 2.5 mg/kg subcutaneously, corresponding to 0.1, 1, and 12.5 times the maximum human nebulization dose, respectively) showed cleft palate formation in 5 of 111 (4.5%) of fetuses at 0.25 mg/kg and in 10 of 108 (9.3%) of fetuses at 2.5 mg/kg. None were observed at 0.025 mg/kg. Cleft palate also occurred in 22 of 72 (30.5%) of fetuses treated with 2.5 mg/kg isoproterenol (positive control). A reproduction study in Stride Dutch rabbits revealed cranioschisis in 7 of 19 (37%) of fetuses at 50 mg/kg, corresponding to 250 times the maximum human nebulization dose.

Labor and Delivery: Oral albuterol has been shown to delay preterm labor in some reports. There are presently no well-controlled studies which demonstrate that it will stop preterm labor or prevent labor at term. Therefore, cautious use of PROVENTIL Solution for Inhalation is required in pregnant patients when given for relief of bronchospasm so as to avoid interference with uterine contractibility.

Nursing Mothers: It is not known whether this drug is excreted in human milk. Because of the potential for tumorigenicity shown for albuterol in some animal studies, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.

Pediatric Use: Safety and effectiveness of albuterol solution for inhalation in children below the age of 12 years have not been established.

ADVERSE REACTIONS The results of clinical trials with PROVENTIL Solution for Inhalation in 135 patients showed the following side effects which were considered probably or possibly drug related:

Central Nervous System: tremors (20%), dizziness (7%), nervousness (4%), headache (3%), insomnia (1%).
Gastrointestinal: nausea (4%), dyspepsia (1%).
Ear, Nose and Throat: pharyngitis (<1%), nasal congestion (1%).
Cardiovascular: tachycardia (1%), hypertension (1%).
Respiratory: bronchospasm (8%), cough (4%), bronchitis (4%), wheezing (1%).

No clinically relevant laboratory abnormalities related to PROVENTIL Solution for Inhalation administration were determined in these studies.

In comparing the adverse reactions reported for patients treated with PROVENTIL Solution for Inhalation with those of patients treated with isoproterenol during clinical trials of 3 months, the following moderate to severe reactions, as judged by the investigators, were reported. This table does not include mild reactions.

Percent Incidence of Moderate To Severe Adverse Reactions

Reaction	Albuterol N = 65	Isoproterenol N = 65
Central Nervous System		
Tremors	10.7%	13.8%
Headache	3.1%	1.5%
Insomnia	3.1%	1.5%
Cardiovascular		
Hypertension	3.1%	3.1%
Arrhythmias	0%	3.0%
**Palpitation	0%	22.0%
Respiratory		
*Bronchospasm	15.4%	18%
Cough	3.1%	5%
Bronchitis	1.5%	5%
Wheeze	1.5%	1.5%
Sputum Increase	1.5%	1.5%
Dyspnea	1.5%	1.5%
Gastrointestinal		
Nausea	3.1%	0
Dyspepsia	1.5%	0
Systemic		
Malaise	1.5%	0

*In most cases of bronchospasm, this term was generally used to describe exacerbations in the underlying pulmonary disease.

**The finding of no arrhythmias and no palpitations after albuterol administration in this clinical study should not be interpreted as indicating that these adverse effects cannot occur after the administration of inhaled albuterol.

Rare cases of urticaria, angioedema, rash, bronchospasm, and oropharyngeal edema have been reported after the use of inhaled albuterol.

OVERDOSAGE Manifestations of overdosage may include anginal pain, hypertension, hypokalemia, and exaggeration of the pharmacological effects listed in **ADVERSE REACTIONS.**

The oral LD_{50} in rats and mice was greater than 2,000 mg/kg. The inhalational LD_{50} could not be determined.

There is insufficient evidence to determine if dialysis is beneficial for overdosage of PROVENTIL Solution for Inhalation.

DOSAGE AND ADMINISTRATION The usual dosage for adults and children 12 years and older is 2.5 mg of albuterol administered 3 to 4 times daily by nebulization. More frequent administration or higher doses is not recommended. To administer 2.5 mg of albuterol, either dilute 0.5 mL of the 0.5% solution for inhalation to a total volume of 3 mL with normal saline solution, or administer the contents of one unit-dose bottle (3 mL of 0.083% nebulizer solution) by nebulization. The flow rate is regulated to suit the particular nebulizer so that the PROVENTIL Solution for Inhalation will be delivered over approximately 5 to 15 minutes.

The use of PROVENTIL Solution for Inhalation can be continued as medically indicated to control recurring bouts of bronchospasm. During treatment, most patients gain optimum benefit from regular use of the nebulizer solution.

If a previously effective dosage regimen fails to provide the usual relief, medical advice should be sought immediately, as this is often a sign of seriously worsening asthma which would require reassessment of therapy.

HOW SUPPLIED PROVENTIL Solution for Inhalation, 0.5%, is a clear, colorless to light yellow solution, and is supplied in bottles of 20 mL (NDC-0085-0208-02) with accompanying calibrated dropper; boxes of one. **Store between 2° and 25°C (36° and 77°F).**

PROVENTIL Solution for Inhalation, 0.083%, is a clear, colorless to light yellow solution, and is supplied in unit-dose bottles of 3 mL each, boxes of 25 (NDC-0085-0209-01.) **Store between 2° and 25°C (36° and 77°F).**

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