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RESPIRATORY CARE

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Professionalism, Respiratory Care Practice, and
Physician Acceptance of a Respiratory Consult Service

ORIGINAL CONTRIBUTIONS

Medical House Staff Impressions of a
Respiratory Therapy Consult Service

Two-Tiered Response for Emergency Airway
Management by Respiratory Therapists and
Anesthesiologists

Acute Pulmonary Effects of Toxic
Nitrogen Dioxide Fume Inhalation

CASE REPORTS

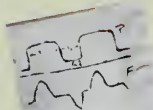
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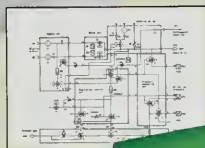
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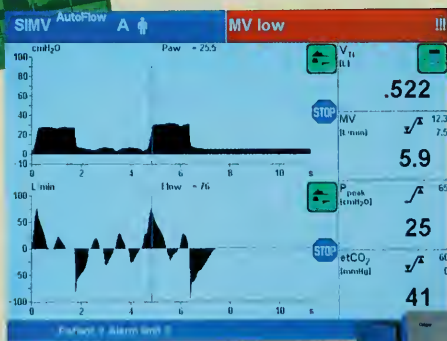


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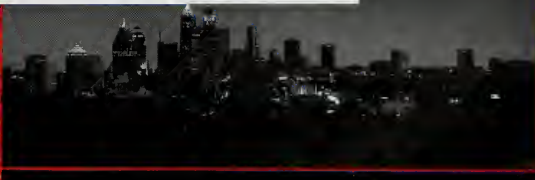
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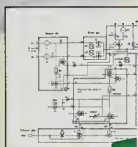
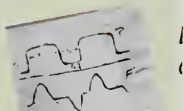
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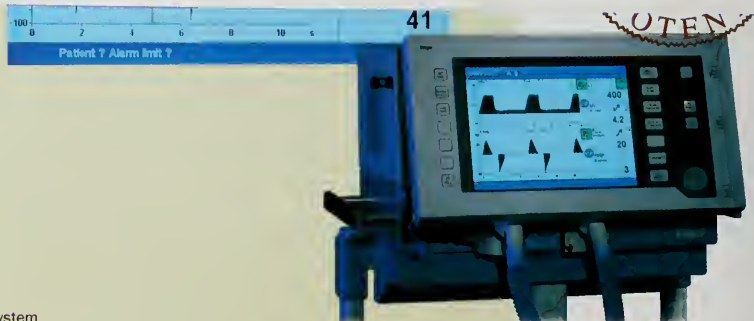
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Editorials, Commentaries, & Reviews To Note

Life-Support System Benefits from Noise—Suki B, Alencar AM, Sujeer MK, Lutchene KR, Collins JJ, Andrade JS Jr, et al. *Nature* 1998;393(6681):127-128.

The End-of-Life Sequence (Review)—Waisel DB, Truog RD. *Anesthesiology* 1997;87(3):676-686.

Case 30-1997: Pulmonary Interstitial Emphysema in Infancy (Letter)—Fox RB, Wright AM. *N Engl J Med* 1998;338(10):688.

Case 30-1997: Pulmonary Interstitial Emphysema in Infancy (Letter)—Lefebvre F. *N Engl J Med* 1998;338(10):688-689.

Non-Steroidal Anti-Inflammatory Therapy for Asthma (Letter)—Kon OM, Robinson DS. *Lancet* 1998;351(9103):672.

Prognosis of ARDS Patients: Light at the End of the Tunnel? (Editorial)—Steltzer H, Krafft P. *Intensive Care Med* 1997;23(8):803-805.

Staffing ICUs: The Good News and the Not-So-Good News (Editorial)—Barash PG, Rosenbaum SH. *Chest* 1998;113(3):569-570.

Multiple-Breath Nitrogen Washout Techniques: Including Measurements with Patients on Ventilators—Newth CJ, Enright P, Johnson RL. *Eur Respir J* 1997;10(9):2174-2185.

Roentgen, Tourette, Angstrom, Bechterew, and Other Misspelled Names (Letter)—Navarro FA. *Lancet* 1998;351(9103):682.

Inhaled Nitric Oxide: A Tenth Anniversary Observation—Truog WE. *Pediatrics* 1998;101(4 Pt 1):696-697.

Noninvasive Ventilatory Support—Saving a Life without Intubation—Birnhauser DM. *West J Med* 1998;168(3):182-183.

Predictors of Quality of Life and Adjustment after Lung Transplantation—Cohen L, Littlefield C, Kelly P, Maurer J, Abbey S. *Chest* 1998;113(3):633.

OBJECTIVE: Few studies have examined predictors of quality of life and adjustment after lung transplantation. This study determined whether pretransplant psychological measures predicted physical health, quality of life, and overall adjustment post-transplant. Cross-sectional analyses also examined differences in adjustment and quality of life for lung transplant candidates and recipients. **DESIGN & PARTICIPANTS:** Seventeen transplant candidates and 60 transplant recipients completed questionnaires measuring adjustment and quality of life. In addition, we examined archival data on 107 transplant candidates who had received pretransplant psychological assessments, and post-transplant physical health status

data were collected on these patients. Of the 107 patients who provided a pretransplant psychological assessment, 32 completed the questionnaires measuring post-transplant adjustment and quality of life. **SETTING:** University medical center transplant service. **RESULTS:** Cross-sectional analyses indicated significantly better adjustment and quality of life post-transplant. Pretransplant psychological variables were not associated with measures of post-transplant physical health. Hierarchical multiple regression analyses found that pretransplant anxiety and psychopathology predicted post-transplant adjustment (β s ranging from 0.32 to 0.68) and greater pretransplant anxiety also predicted worse post-transplant quality of life (β s ranging from 0.29 to 0.62). Subjective sleep disturbances were associated with poorer adjustment and quality of life (β s ranging from 0.36 to 0.75), and were found to mediate the relationship between pre-surgical anxiety and post-transplant

adjustment and quality of life. **CONCLUSIONS:** This study found that psychological status pre-transplant predicted adjustment and quality of life post-transplant. Moreover, increased anxiety levels pretransplant predicted subsequent subjective sleep disturbances, which were, in turn, associated with poorer adjustment and quality of life. The benefits of pretransplant stress management interventions are discussed.

Compliance with National Asthma Management Guidelines and Specialty Care: A Health Maintenance Organization Experience—Legorreta AP, Christian-Herman J, O'Connor RD, Hasan MM, Evans R, Leung KM. *Arch Intern Med* 1998;158(5):457.

BACKGROUND: To improve asthma disease management, the National Asthma Education Program (NAEP) Expert Panel published *Guidelines*



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for the Diagnosis and Management of Asthma in 1991. OBJECTIVES: To compare the current status of asthma disease management among patients in a large health maintenance organization with the NAEP guidelines and to identify the factors that may be associated with medical care (eg, emergency department visits and hospital admissions) and adherence to the guidelines. METHODS: Analyses of 1996 survey data from 5,580 members with asthma (age range, 14 to 65 years) covered by a major health maintenance organization in California (Health Net). RESULTS: In general, adherence to NAEP guidelines was poor. Seventy-two percent of respondents with severe asthma reported having a steroid inhaler, and of those, only 54% used it daily. Only 26% of respondents reported having a peak flowmeter, and of those, only 16% used it daily. Age (older), duration of asthma (longer), increasing current severity of disease, and treatment by an asthma specialist correlated with daily use of inhaled steroids. Ethnicity (African-American and Hispanic) correlated negatively with inhaled steroid use but positively with emergency department visits and hospital admissions for asthma. Increasing age and treatment by an asthma specialist were also identified as common factors significantly related to the daily use of a peak flowmeter and, interestingly, to overuse of β -2 agonist metered dose inhalers. CONCLUSIONS: Although the NAEP guidelines were published 7 years ago, compliance with the guidelines was low. It was especially poor for use of preventive medication and routine peak flow measurement. Furthermore, the results showed that asthma specialists provided more thorough care than did primary care physicians in treating patients with asthma. Combining the results of the regression analyses revealed that some of the variation in rates of emergency department visits and hospitalizations among some subpopulations can be explained by the underuse of preventive medication. This study serves the goal of documenting the quality of care and services currently provided to patients with asthma through a large health maintenance organization and provides baseline information that can be used to design and assess effective population-based asthma disease management intervention programs.

A Randomized Clinical Trial Comparing an Extended-Use Hygroscopic Condenser Humidifier with Heated-Water Humidification in Mechanically Ventilated Patients—Kollef MH, Shapiro SD, Boyd V, Silver P, Von Harz B, Trovillion E, Prentice D. *Chest* 1998;113(3):759.

OBJECTIVE: To determine the safety and cost-effectiveness of mechanical ventilation with an extended-use hygroscopic condenser humidifier (Duration; Nellor Puritan-Bennett; Eden Prairie, MN) compared with mechanical ventilation with heated-water humidification. **DESIGN:** Prospective randomized clinical trial. **SETTING:** Medi-

cal and surgical ICUs of Barnes-Jewish Hospital, St Louis, a university-affiliated teaching hospital. **PATIENTS:** Three hundred ten consecutive qualified patients undergoing mechanical ventilation. **INTERVENTIONS:** Patients requiring mechanical ventilation were randomly assigned to receive humidification with either an extended-use hygroscopic condenser humidifier (for up to the first 7 days of mechanical ventilation) or heated-water humidification. **MEASUREMENTS:** Occurrence of ventilator-associated pneumonia, endotracheal tube occlusion, duration of mechanical ventilation, lengths of intensive care and hospitalization, acquired multiorgan dysfunction, and hospital mortality. **RESULTS:** One hundred sixty-three patients were randomly assigned to receive humidification with an extended-use hygroscopic condenser humidifier, and 147 patients were randomly assigned to receive heated-water humidification. The two groups were similar at the time of randomization with regard to demographic characteristics, ICU admission diagnoses, and severity of illness. Risk factors for the development of ventilator-associated pneumonia were also similar during the study period for both treatment groups. Ventilator-associated pneumonia was seen in 15 (9.2%) patients receiving humidification with an extended-use hygroscopic condenser humidifier and in 15 (10.2%) patients receiving heated-water humidification (relative risk, 0.90; 95% confidence interval = 0.46 to 1.78; $p = 0.766$). No statistically significant differences for hospital mortality, duration of mechanical ventilation, lengths of stay in the hospital ICU, or acquired organ system derangements were found between the two treatment groups. No episode of endotracheal tube occlusion occurred during the study period in either treatment group. The total cost of providing humidification was \$2,605 for patients receiving a hygroscopic condenser humidifier compared with \$5,625 for patients receiving heated-water humidification. **CONCLUSION:** Our findings suggest that the initial application of an extended-use hygroscopic condenser humidifier is a safe and more cost-effective method of providing humidification to patients requiring mechanical ventilation compared with heated-water humidification.

The Effect of Physiologic and Mechanical Aging on the Performance of Peak Flowmeters—Johns DP, Side E, Kendrick AH, Williams TJ, Walters EH. *Chest* 1998;113(3):774.

PURPOSE: To investigate the effects of physiologic and mechanical aging on peak flowmeters. **MATERIALS & METHODS:** Eight each of Mini-Wright (MW; Clement Clark; Harlow, UK), Personal Best (PB; HealthScan Products; Cedar Grove NJ), Vitalograph (V; Vitalograph Ltd; Buckingham, UK), and Breath-Taker (BT; Medical Development Australia; Melbourne, Australia) peak flowmeters were assessed for accuracy and repeatability before and after aging using a computer-driven syringe to deliver peak flows from

100 to 700 L/min. Four of each type of flowmeter were physiologically aged by normal subjects performing up to 6 peak flows daily for 1 year. The remaining 4 of each flowmeter were mechanically aged using an accelerated aging device to deliver 2,000 exponential waveforms with a peak flow of 600 L/min over a period of 3 h. **RESULTS:** The V and BT flowmeters were linear and accurate over the range 100 to 700 L/min, while the PB overread at high flows. The MW was alinear throughout. The SD of the difference between readings before and after aging ranged from 8.6 to 40.6 L/min (mean, 9.2). Comparing the slopes of the relationship of actual against reference peak expiratory flow (PEF) showed that 16 flowmeters—5 BTs, 6 MWs, 4 PBs, and 1 V had no significant change in slope after aging. Mechanical aging caused a consistent underreading in PEF at high flow rates. Physiologic aging showed a more variable pattern both within and between flowmeter types. The MW was the most affected by physiologic aging, producing overestimates of PEF by as much as 100 L/min at 500 L/min. **CONCLUSIONS:** We conclude that the effects of physiologic and mechanical aging are different, and that while mechanical aging may provide a guide to the effects of aging, studies using physiologic aging would be more appropriate.

Patterns of Dissimilarities among Instrument Models in Measuring P_{O_2} , P_{CO_2} , and pH in Blood Gas Laboratories—Hansen JE, Casaburi R. *Chest* 1998;113(3):780.

OBJECTIVES: To ascertain the degree of dissimilarities among blood gas and pH analyzer models of the same and different manufacturers in measurement of P_{O_2} , P_{CO_2} , and pH using fluorocarbon containing emulsion (FCE) proficiency testing material. **DESIGN:** Statistically and graphically analyze data from 6 recent proficiency testing surveys for the 20 more frequently used models of analyzers. **SETTING & PARTICIPANTS:** Over a 2-year period, approximately 900 participants from blood gas laboratories in the United States analyzed similar ampules from each of 30 lots. **MEASUREMENTS & RESULTS:** Both graphic and statistical comparisons were used to demonstrate differences between manufacturers. For each of the 4 major manufacturers, comparisons revealed statistically significant differences not only for P_{O_2} , but also for P_{CO_2} and pH. Additionally, comparison models within each of the 3 manufacturers (those with multiple models and > 15 instruments/model represented) disclosed statistically significant dissimilarities among models for each analyte in 115 of 153 model pairings. Previously reported tonometered blood differences among analyzer models for P_{O_2} are qualitatively similar to the differences found in these same models in this FCE study. Model differences are important in research studies and may be clinically important in deciding abnormality, selecting oxygen therapy, or the treatment of patients with res-

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piratory failure or severe respiratory alkalosis. CONCLUSIONS: To minimize the likelihood of misleading clinicians, laboratory directors should consider the degree of dissimilarity among blood gas analyzer models in current use and when changing instrumentation.

Rehabilitation of Hypoxemic Patients with COPD at Low Altitude at the Dead Sea, the Lowest Place on Earth—Kramer MR, Springer C, Berkman N, Glazer M, Bubli M, Bar-Yishay E, Godfrey S. *Chest* 1998;113(3):571.

BACKGROUND: In patients with COPD, oxygen therapy has been shown to improve exercise capacity and survival. Increase in barometric pressure at low altitude can serve as a simple way to improve arterial oxygenation in hypoxemic patients. We have tried to evaluate the effect of staying at low altitude on arterial oxygenation and exercise performance in patients with COPD. **PATIENTS & METHOD:** Eleven patients with COPD (9 male, 2 female) aged 38 to 79 years (mean FEV₁, 0.96 L; 36% predicted) with hypoxemia (mean P_{aO₂}, 54.2 ± 8.9 mm Hg) at Jerusalem (altitude 800 m above sea level) were taken down to the Dead Sea area (altitude 402 m below sea level) for 3 weeks. At both locations we tested arterial blood gases, spirometry, progressive exercise, 6-min walking distance, and sleep oxime-

try. The study was repeated 2 weeks after returning to Jerusalem. **RESULTS:** Spirometry results were unchanged. Mean arterial P_{aO₂} rose from 54.2 ± 8.9 mm Hg to 69.5 ± 11 at the first week and to 66.6 ± 11 at the third week of stay ($p < 0.001$). P_{aCO₂} rose from 43.5 ± 9.8 mm Hg to 47.7 ± 9 and 49.5 ± 8.4 ($p < 0.006$). Six-min walking distance rose from 337 ± 107 m to 449 ± 73 and 507 ± 91 in the third week ($p < 0.005$). Maximum oxygen consumption ($\dot{V}_{O_{2max}}$) rose from 901 ± 257 mL/min to 1,099 ± 255 and 1,063 ± 250 mL/min ($p = 0.01$). Sleep oximetry showed an increase in mean sleep arterial oxygen saturation from 86.0 ± 4.3% to 89.9 ± 4.2% and 88.3 ± 3.0 at 1 and 3 weeks, respectively ($p < 0.05$). Following the return to Jerusalem, arterial gases returned to their baseline levels (P_{aO₂}, 52.9 ± 9.4 mm Hg) but 6-min walking distance remained significantly high, 453 ± 47 ($p < 0.02$), and $\dot{V}_{O_{2max}}$ remained high as well (1,102 ± 357 mL/min), although it did not reach statistical significance. **CONCLUSIONS:** Decline to low altitude or staying at high oxygen environment improves arterial oxygenation and exercise capacity in hypoxemic patients residing in moderate or high altitude. Low altitude (or pressurized wards) can improve pulmonary rehabilitation of hypoxemic patients with COPD. See the related article: *What Happens in the Dead Sea?* (editorial)—Minh VD. *Chest* 1998;113(3):566-567.

Validation of a New Dyspnea Measure: The UCSD Shortness of Breath Questionnaire. University of California, San Diego—Eakin EG, Resnikoff PM, Prewitt LM, Ries AL, Kaplan RM. *Chest* 1998;113(3):619.

OBJECTIVE: Evaluate the reliability and validity of a new version of the University of California, San Diego Shortness of Breath Questionnaire (SOBQ), a 24-item measure that assesses self-reported shortness of breath while performing a variety of activities of daily living. **DESIGN:** Patients enrolled in a pulmonary rehabilitation program were asked to complete the SOBQ, the Quality of Well-Being Scale, the Center for Epidemiologic Studies Depression Scale, and a 6-min walk with modified Borg scale ratings of perceived breathlessness following the walk. **SETTING:** University medical center pulmonary rehabilitation program. **Patients:** Thirty-two male subjects and 22 female subjects with a variety of pulmonary diagnoses: COPD ($n = 28$), cystic fibrosis ($n = 9$), and postlung transplant ($n = 17$). **MEASUREMENTS & RESULTS:** The current version of the SOBQ was compared with the previous version, the format of which often resulted in a significant number of "not applicable" answers. The results demonstrated that the SOBQ had excellent internal consistency ($\alpha = 0.96$). The SOBQ was also significantly correlated with all validity criteria.

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CONCLUSIONS: The SOBQ is a valuable assessment tool in both clinical practice and research in patients with moderate-to-severe lung disease.

Evaluation of Dyspnea during Physical and Speech Activities in Patients with Pulmonary Diseases—Lee L, Friesen M, Lambert JR, Loudon RG. *Chest* 1998;113(3):625.

OBJECTIVES: Dyspnea is most commonly assessed by questioning patients about their subjective perception of shortness of breath during physical exertion. Although speech production is altered by pulmonary disease, it has not been included in current dyspnea assessment tools. A questionnaire was developed to address reports of dyspnea during (1) physical activity, (2) speech activity, and (3) simultaneous speech and physical activity. **DESIGN:** An equal number of self- and experimenter-administered 30-item questionnaires was given to 203 patients with restrictive and obstructive pulmonary diseases. Their responses were analyzed statistically. **RESULTS:** The questionnaire had high internal consistency for individual items within each of the 3 sections. The sections were highly correlated but provided separate and distinct information. Factors extracted from each section were related to severity of dyspnea. Pairwise *t* tests demonstrated highly significant differences in subject responses to the 3 sections. The least dyspnea was experienced during speech activities, more during physical activities, and the most when speech and physical activities were combined. **CONCLUSIONS:** The questionnaire proved to be a quickly administered tool for providing information about the effect of dyspnea on activities of daily living. Because of the emphasis on dyspnea during speech production, it may be particularly useful for assessing patients who rely extensively on speaking ability for their livelihood.

Rate of FEV₁ Change following Lung Volume Reduction Surgery—Brenner M, McKenna RJ Jr, Gelb AF, Fischel RJ, Wilson AF. *Chest* 1998;113(3):652.

INTRODUCTION: Lung volume reduction surgery (LVRS) improves pulmonary function and dyspnea symptoms acutely in selected patients with heterogeneous emphysema. Limited data are available regarding long-term function following LVRS. We analyzed short-term (< 6 months) and long-term rate of change of pulmonary function in 376 patients who underwent unilateral or bilateral LVRS using thoracoscopic or median sternotomy, staple, laser, or combined techniques. We hypothesized that the long-term rate of deterioration in lung function would be dependent on the surgical procedure used and would be greatest in those with the largest short-term postoperative improvement. **METHODS:** Pulmonary function was assessed preoperatively and at repeated intervals following LVRS. The change

in pulmonary function over time was assessed for each patient by determining the individual change in FEV₁ using linear regression analysis short and long term. Overall rate of change in pulmonary function was calculated for the composite group of patients and subgrouped by operative procedure. **RESULTS:** Lung function appears to improve in the first few months following LVRS in most patients, maximizing at approximately 3 to 6 months and declining thereafter. The short-term incremental improvement following staple procedures is superior to improvements following laser procedures or unilateral surgery: FEV₁ increase (mean \pm SD) of 0.39 \pm 0.03 L for bilateral staple, 0.25 \pm 0.03 L for unilateral staple, 0.10 \pm 0.03 L for unilateral laser, and 0.22 \pm 0.1 L for mixed unilateral staple/laser procedures. However, the long-term rate of decline in FEV₁ was greatest for bilateral staple LVRS procedures as well: 0.255 \pm 0.057 L/y for bilateral staple, 0.107 \pm 0.068 L/y for unilateral staple, 0.074 \pm 0.034 L/y for unilateral laser, and 0.209 \pm 0.12 L/y for mixed staple laser procedures. There was a general correlation between the magnitude of short-term incremental improvement and the rate of deterioration in FEV₁ ($r = 0.292$, $p = 0.003$). **CONCLUSIONS:** While bilateral staple LVRS procedures lead to greater short-term improvement in FEV₁, the more rapid rate of FEV₁ decline in these patients and the general association between greater short-term incremental improvement and higher rates of deterioration raise questions regarding optimal long-term procedures. Further studies will be needed to answer these important questions.

Transcutaneous P_{CO2} To Monitor Noninvasive Mechanical Ventilation In Adults: Assessment of a New Transcutaneous P_{CO2} Device—Janssens JP, Howarth-Frey C, Chevrolat JC, Abajo B, Rochat T. *Chest* 1998;113(3):768.

The present study was designed to analyze the usability of a commercially available, transcutaneous P_{CO2} (TcP_{CO2}) sensor for monitoring noninvasive positive pressure ventilation (NPPV). Twenty-six hemodynamically stable patients with intra-arterial radial catheters were assessed. After stabilization of TcP_{CO2}, arterial blood was analyzed and results were compared with TcP_{CO2} at time of sampling. To evaluate the drift of the signal, samples were taken hourly in 5 patients for 4 h while continuously recording TcP_{CO2}. Finally, to assess for the response of the sensor to changes in P_{aCO2}, 6 patients underwent continuous TcP_{CO2} recording while initiating or interrupting NPPV; arterial samples were analyzed before the event, and 1, 3, 5, 7, 9, and 20 min afterwards. **RESULTS:** TcP_{CO2} and P_{aCO2} were tested over a range of 26 to 71 mm Hg and were found to be closely correlated ($r = 0.968$, $p < 0.0001$); mean bias was 0.75 mm Hg. There was no significant drift of TcP_{CO2} as compared with P_{aCO2} over 4 h. The time of response of TcP_{CO2} to initiation or

interruption of NPPV was < 60 s. An estimation of the lag time averaged 5 \pm 3 min (range, 1 to 7 min). **CONCLUSION:** TcP_{CO2} in hemodynamically stable adults was in excellent agreement with arterial measurements. The time of response to a change in ventilation was compatible with the aim of clinical monitoring of patients under NPPV.

Patient-Induced Complications of a Heimlich Flutter Valve—Crocker HL, Ruffin RE. *Chest* 1998;113(3):838.

Heimlich flutter valves have gained widespread acceptance in the treatment of pneumothorax. However, some features of their design may predispose them to inadvertent misuse. A case of tension pneumothorax is described which resulted from the insertion of a drinking straw into the Heimlich flutter valve assembly.

Use of Conventional and Self-Adjusting Nasal Continuous Positive Airway Pressure for Treatment of Severe Obstructive Sleep Apnea Syndrome: A Comparative Study—Konermann M, Sanner BM, Vyleta M, Laschewski F, Groetz J, Sturm A, Zidek W. *Chest* 1998;113(3):714.

OBJECTIVES: To compare conventional and self-adjusting nasal continuous positive airway pressure (nCPAP) therapy in patients with severe obstructive sleep apnea syndrome with respect to suppression of respiratory disturbances, quality of sleep, mean mask pressure, and patient compliance. **DESIGN:** Cohort study of consecutive patients with obstructive sleep apnea syndrome, single-blinded. **SETTING:** Clinical sleep laboratory in Germany. **PATIENTS:** Fifty patients (44 men, 6 women) who ranged in age from 35 to 71 years) with polysomnographically confirmed severe obstructive sleep apnea syndrome (respiratory disturbance index [RDI], $> 20/h$). **MEASUREMENTS & INTERVENTIONS:** After baseline polysomnography, patients were randomly treated with nCPAP either in conventional (Group 1) or in automatically adjusting (Group 2) mode. Three to 6 months after adjustment, all patients underwent polysomnography again. They also were examined with a portable monitoring device and received a questionnaire on subjective well-being and device evaluation. **RESULTS:** Anthropometric and respiratory data were comparable in both groups; body mass index had not changed significantly in the follow-up. RDI dropped by 91.5% (from 38.3 \pm 13.9/h to 3.6 \pm 4.4/h) in conventional and by 93.6% (from 35.5 \pm 9.6/h to 2.4 \pm 1.6/h) in self-adjusting mode (statistically not significant [NS]). Sleep efficiency decreased by 4.0% in conventional and increased by 2.0% in self-adjusting mode (NS). In both groups, normal sleep structure was largely restored. Mean mask pressure was 8.1 \pm 2.5 cm H₂O in Group 1 and 6.5 \pm 1.7 cm H₂O in Group 2 ($p < 0.01$). Patient compliance in terms of nights per week of mask appliance was better in the self-adjusting mode

(5.7 \pm 0.7 to 6.5 \pm 0.4; $p < 0.01$). **CONCLUSION:** Self-adjusting nCPAP demonstrates the same reliability in suppression of respiratory disturbances as fixed-mask pressure therapy. Sleep quality is slightly superior, patient compliance is highly significantly better.

Hour-to-Hour Variability of Oxygen Saturation in Sleep Apnea—Chaudhary B, Dasti S, Park Y, Brown T, Davis H, Akhtar B. Chest 1998;113(3):719.

OBJECTIVES: Methods used to express the severity of oxygen desaturation during polysomnography include the average oxygen saturation (AO_2), lowest oxygen saturation (LO_2), and the percent of the total time with oxygen saturation level lower than 90% ($t < 90\%$). We wanted to determine which one of these methods is least variable during different hours of monitoring. **DESIGN:** Prospective, observational study. **SETTING:** Sleep center at a medical university. **PATIENTS:** One hundred fifty patients with apnea-hypopnea index from 5 to 130. **MEASUREMENTS:** AO_2 , LO_2 , and $t < 90\%$ were calculated during each of the 8 h of polysomnography. Data for each hour were compared and the

Cronbach α coefficients were calculated. **RESULTS:** There was a high degree of correlation among the three methods as well as between each method and the severity of sleep apnea. The mean \pm SD values for each method were as follows: AO_2 , 92.7 ± 5.6 ; LO_2 , 68.5 ± 19.3 ; and $t < 90\%$, 15.7 ± 24.2 . The α coefficients for these methods were AO_2 , 0.98; LO_2 , 0.88; and $t < 90\%$, 0.98. In all methods, the data of the first hour were significantly different from the data of the subsequent hours. **CONCLUSION:** Both AO_2 and $t < 90\%$ methods show less hour-to-hour variability compared with LO_2 , and there is more variability in the first hour. Since the AO_2 values $> 90\%$ may not convey the severity of O_2 desaturation, $t < 90\%$ may be the best method of expressing oxygen saturation changes during polysomnography.

The Effect of a Mandibular Advancement Device on Apneas and Sleep in Patients with Obstructive Sleep Apnea—Marklund M, Franklin KA, Sahlin C, Lundgren R. Chest 1998;113(3):707.

OBJECTIVE: To evaluate the effects of a mandibular advancement device on apneas and sleep in mild, moderate, and severe obstructive sleep

apnea. **DESIGN:** Prospective study. **SUBJECTS:** Forty-four of 47 patients included. **INTERVENTION:** Individually adjusted mandibular advancement devices. **MEASUREMENTS:** Polysomnographic sleep recordings for 1 night without the device and 1 night with it, with a median of 1 day and no changes in weight, medication, or sleep position between the recordings. **RESULTS:** The device reduced the median obstructive apnea-hypopnea index from 11 (range, 7 to 19) to 5 (range, 0 to 17) ($p < 0.001$) in 21 patients with mild sleep apnea, from 27 (range, 20 to 38) to 7 (range, 1 to 19) ($p < 0.001$) in 15 patients with moderate sleep apnea, and from 53 (range, 44 to 66) to 14 (range, 2 to 32) ($p < 0.05$) in 8 patients with severe sleep apnea. The arousal index decreased and the sleep stage patterns improved in all severity groups. Twenty-eight of 44 patients were successfully treated with an obstructive apnea-hypopnea index of below 10 and a subjective reduction in snoring. Nine of 16 patients with treatment failure still reported a reduction in snoring. The success rate correlated inversely to the disease severity ($r = -0.41$; $p < 0.01$). **CONCLUSIONS:** A mandibular advancement device reduces apneas and improves sleep quality in patients with obstructive sleep apnea.

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especially in those with mild and moderate disease. A follow-up sleep recording during treatment is necessary because of the risk of silent obstructive apnea without subjective snoring with the device.

Determinants of Effective Ventilation during Nasal Intermittent Positive Pressure Ventilation—Parreira VF, Jounieaux V, Delguste P, Aubert G, Dury M, Rodenstein DO. *Eur Respir J* 1997;10(9):1975.

Our aim was to verify in healthy subjects submitted to nasal intermittent positive pressure ventilation (nIPPV) with a volumetric ventilator on controlled mode, whether changes in ventilator settings (delivered tidal volume (\dot{V}_T), respiratory frequency (f) and inspiratory flow (\dot{V}_I)) could influence effective minute ventilation (\dot{V}_E), thus allowing identification of the settings resulting in the highest \dot{V}_E during nIPPV. We then compared these experimentally obtained "best" settings to those obtained retrospectively in a group of patients submitted to long-term nIPPV for clinical reasons. We studied 10 healthy subjects awake and asleep, and 33 patients with restrictive ventilatory disorders. Changes in delivered \dot{V}_I (for a constant delivered \dot{V}_T and f) led to significant changes in \dot{V}_E . \dot{V}_E was significantly higher when a given delivered \dot{V}_I was obtained using higher

f and lower \dot{V}_I than when it was obtained using lower delivered f and higher \dot{V}_I . Increases in f generally resulted in increases in \dot{V}_E . The "best" settings derived from these results were: \dot{V}_I : 13 mL \cdot kg $^{-1}$ of body weight; f: 20 breaths/min $^{-1}$ and \dot{V}_E : 0.56-0.85 L \cdot s $^{-1}$. The corresponding average values found in the patient group were: delivered \dot{V}_I : 14 mL \cdot kg $^{-1}$; f: 23 breaths/min $^{-1}$ and delivered \dot{V}_E : 0.51 L \cdot s $^{-1}$. Changes in minute ventilation resulting from modifications in ventilator settings can be attributed to the glottic response to mechanical influences. This leads to "ideal" settings quite different from the standard ones in incubated patients. Values derived from nasal intermittent positive pressure ventilation in healthy subjects seem to apply to patients submitted to long-term nasal intermittent positive pressure ventilation.

Lung Deposition from the Turbuhaler in Children with Cystic Fibrosis—Devadason SG, Everard ML, MacEarlan C, Roller C, Summers QA, Swift P, et al. *Eur Respir J* 1997;10(9):2023.

Drug delivery to patients using dry powder inhalers, such as the Turbuhaler, is believed to be influenced by the inspiratory flow used. Clinical studies have indicated that this delivery system can be used effectively by children. However, it is not known how the total and weight-corrected dose delivered to the airways varies with age. A

deposition study using technetium-99m (99mTc)-labeled budesonide was performed in order to determine the effect of age on delivery. Twenty one children with cystic fibrosis, aged 4-16 y, were recruited. They were clinically stable with normal lung function. Initially, a γ camera scan was taken in front of a flood source containing 37 MBq of 99mTc. Subsequently, subjects inhaled through a low resistance inspiratory filter connected to a commercially available Turbuhaler. Immediately afterwards they inhaled from a noncommercial Turbuhaler containing budesonide labeled with 99mTc, and then underwent anterior and posterior γ camera scans. Both Turbuhaler inhalers were attached to a portable spirometer and the peak inspiratory flow through the Turbuhaler was recorded for each inhalation. The total body dose was calculated from the dose deposited on the inspiratory filter connected to the commercial Turbuhaler. Analysis of the γ camera images provided information on the proportion of the radiolabel delivered to the lungs compared to that deposited in the upper airway and stomach. As expected, a highly significant positive correlation was noted between the peak inspiratory flow generated by the patient through the Turbuhaler and the dose delivered to the lung. Similarly, there was a highly significant positive correlation between age and "total lung dose." However, when total lung dose was corrected for body weight, there was a non-

significant negative correlation with age. This study suggests that the "weight-corrected lung dose" achieved when children aged > 6 yrs use the Turbuhaler, is largely independent of age. It would appear that the flow-dependent properties of this device are such that the reduced peak inspiratory flow generated by younger children results in a lower dose to the lungs, but that this is off-set by their lower body weight. This is unlikely to be a property of other devices with different flow/drug delivery characteristics.

The Effect of Low-Dose Inhalation of Nitric Oxide in Patients with Pulmonary Fibrosis—Yoshida M, Taguchi O, Gabazza EC, Yasui H, Kobayashi T, Kobayashi H, et al. *Eur Respir J* 1997;10(9):2051.

The aim of this study was to determine whether low-dose inhalation of nitric oxide (NO) improves pulmonary haemodynamics and gas exchange in patients with stable idiopathic pulmonary fibrosis (IPF). The investigation included 10 IPF patients breathing spontaneously. Haemodynamic and blood gas parameters were measured under the following conditions: (1) breathing room air; (2) during inhalation of 2 parts per million (ppm) NO with room air; (3) whilst breathing O₂ alone (1 L · min⁻¹); and (4) during combined inhalation of 2 ppm NO and O₂ (1 L · min⁻¹). During inhalation of 2 ppm NO with room air the mean pulmonary arterial pressure (P_{pa} 25 ± 3 vs 30 ± 4 mm Hg) and the pulmonary vascular resistance (PVR 529 ± 80 vs 699 ± 110 dyn · s · cm⁻⁵) were significantly ($p < 0.01$) lower than levels measured whilst breathing room air alone. However the arterial oxygen tension (P_{aO_2}) did not improve. The combined inhalation of NO and O₂ produced not only a significant ($p < 0.01$) decrease of P_{pa} (23 ± 2 vs 28 ± 3 mm Hg) but also, a remarkable improvement ($p < 0.05$) in P_{aO_2} (14.2 ± 1.2 vs 11.7 ± 1.0 kPa) (107 ± 9 vs 88 ± 7 mm Hg) as compared with the values observed during the inhalation of O₂ alone. These findings suggest that the combined use of nitric oxide and oxygen might constitute an alternative therapeutic approach for treating idiopathic pulmonary fibrosis patients with pulmonary hypertension. However, further studies must first be carried out to demonstrate the beneficial effect of oxygen therapy on pulmonary haemodynamics and prognosis in patients with idiopathic pulmonary fibrosis and to rule out the potential toxicity of inhaled nitric oxide, particularly when used in combination with oxygen.

Use of Conventional and Self-Adjusting Nasal Continuous Positive Airway Pressure for Treatment of Severe Obstructive Sleep Apnea Syndrome: A Comparative Study—Konermann M, Sanner BM, Vyleta M, Laschewski F, Groetz J, Sturm A, Zidek W. *Chest* 1998;113(3):714.

OBJECTIVES: To compare conventional and self-adjusting nasal continuous positive airway

pressure (nCPAP) therapy in patients with severe obstructive sleep apnea syndrome with respect to suppression of respiratory disturbances, quality of sleep, mean mask pressure, and patient compliance. **DESIGN:** Cohort study of consecutive patients with obstructive sleep apnea syndrome, single-blinded. **SETTING:** Clinical sleep laboratory in Germany. **PATIENTS:** Fifty patients (44 men, 6 women who ranged in age from 35 to 71 years) with polysomnographically confirmed severe obstructive sleep apnea syndrome (respiratory disturbance index [RDI], > 20/h). **MEASUREMENTS & INTERVENTIONS:** After baseline polysomnography, patients were randomly treated with nCPAP either in conventional (Group 1) or in automatically adjusting (Group 2) mode. Three to 6 months after adjustment, all patients underwent polysomnography again. They also were examined with a portable monitoring device and received a questionnaire on subjective well-being and device evaluation. **RESULTS:** Anthropometric and respiratory data were comparable in both groups; body mass index had not changed significantly in the follow-up. RDI dropped by 91.5% (from 38.3 ± 13.9/h to 3.6 ± 4.4/h) in conventional and by 93.6% (from 35.5 ± 9.6/h to 2.4 ± 1.6/h) in self-adjusting mode (statistically not significant [NS]). Sleep efficiency decreased by 4.0% in conventional and increased by 2.0% in self-adjusting mode (NS). In both groups, normal sleep structure was largely restored. Mean mask pressure was 8.1 ± 2.5 cm H₂O in Group 1 and 6.5 ± 1.7 cm H₂O in Group 2 ($p < 0.01$). Patient compliance in terms of nights/wk of mask appliance was better in the self-adjusting mode (5.7 ± 0.7 to 6.5 ± 0.4; $p < 0.01$). **CONCLUSION:** Self-adjusting nCPAP demonstrates the same reliability in suppression of respiratory disturbances as fixed-mask pressure therapy. Sleep quality is slightly superior, patient compliance is highly significantly better.

Partial Liquid Ventilation with Small Volumes of FC 3280 Increases Survival Time in Experimental ARDS—Kaisers U, Max M, Walter J, Kuhlén R, Pappert D, Falke K, Rossaint R. *Eur Respir J* 1997;10(9):1955.

The aim of this study was to determine the prolonged effects of sequential doses of a highly purified perfluorocarbon (FC 3280) on gas exchange and survival time in experimental acute respiratory distress syndrome (ARDS). The study was prospective, randomized, and controlled. Twelve pigs (body weight 30 ± 5 (mean ± SD) kg) were surfactant-depleted by repetitive lung lavages, reducing arterial oxygen tension (P_{aO_2}) to 6.9 ± 1.6 kPa (52 ± 12 mm Hg) (mean ± SD) at an inspired oxygen fraction (F_{IO_2}) of 1.0. They were then randomized to receive partial liquid ventilation by sequential intratracheal application of 7.5 mL · kg⁻¹ FC 3280 at 30 min intervals to a cumulative dose of 15 mL · kg⁻¹ (treatment group), or to receive no further treatment (control group).

Haemodynamics and gas exchange were assessed at 30 min intervals after instillation, and hourly afterwards in both groups until death. In the treatment group, P_{aO_2} was 8.9 ± 4.4 kPa (67 ± 33 mm Hg) after 7.5 mL · kg⁻¹ FC 3280 and 14.1 ± 9.9 kPa (106 ± 74 mm Hg) after 15 mL · kg⁻¹ FC 3280 (NS). In the control group, gas exchange remained unchanged. Haemodynamics were stable in the treatment group and deteriorated in the control group. Peak airway pressures and dynamic compliance were not significantly affected in the treatment group. Mean survival time was 8.2 ± 4.5 h in the treatment group and 1.8 ± 1.4 h in the control group ($p < 0.05$). Upon histological examination, both study groups were not significantly different in total lung injury scores. We conclude that partial liquid ventilation with small volumes of FC 3280 provides improvement in gas exchange and increases survival time in experimental acute respiratory distress syndrome. See the related article: *Partial Liquid Ventilation (editorial)*—Verbrugghe SJ, Lachmann B. *Eur Respir J* 1997;10(9):1937-1939.

Autoset Nasal CPAP Titration: Constancy of Pressure, Compliance and Effectiveness at 8 Month Follow-Up—Teschler H, Farhat AA, Exner V, Konietzko N, Berthoin-Jones M. *Eur Respir J* 1997;10(9):2073.

We have previously shown that AutoSet satisfactorily improves sleep-disordered breathing and sleep architecture in subjects with obstructive sleep apnoea (OSA) syndrome. The aim of this study was to determine, in subjects treated with long-term conventional fixed pressure continuous positive airway pressure (CPAP) at the AutoSet recommended pressure, whether: the long-term compliance is satisfactory; the improvement persists once initial rebound is over; the titration pressure is stable with time; and the titration pressure is comparable with manual titration pressure using a similar end point. Twenty males with OSA, previously studied with full polysomnography on their diagnostic night, at manual and AutoSet titration, and at the AutoSet recommended fixed pressure, were re-studied after a mean of 3 and 8 months of treatment at the recommended fixed pressure. Re-study included home respiratory monitoring (Nellcor EdenTrace), and repeated manual and AutoSet titration with polysomnography. Compliance was assessed with hour-mean readings. Mean (± SEM) usage was 5.7 ± 0.1 h/night at 3 and 8 months. The arousal index remained normalized. Diagnostic respiratory disturbance index (RDI) was 60.3 ± 5.7 events/h⁻¹. On AutoSet at fixed CPAP, RDI was initially 2.6 ± 0.7 events/h⁻¹, then rose slightly ($p < 0.001$) to 4.3 ± 0.6 events/h⁻¹ at 3 months, and was 3.6 ± 0.5 events/h⁻¹ at 8 months. AutoSet titration pressure was: 9.9 ± 0.4 cm H₂O initially, 10.6 ± 0.4 cm H₂O at 3 months, and 9.7 ± 0.5 cm H₂O at 8 months (NS). Manual titration pressure at 8 months was 10.4 ± 0.4 cm H₂O. The standard deviation of the discrepancy

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with AutoSet was 0.84 cm H₂O. In conclusion, the AutoSet recommended pressure varies little with time, and closely predicts the final manual titration pressure; the improvement in respiratory disturbance index was largely maintained, and compliance was good, although probably enhanced by close supervision.

Water Vapour and Carbon Dioxide Decrease Nitric Oxide Readings—van der Mark TW, Kort E, Meijer RJ, Postma DS, Koeter GH. *Eur Respir J* 1997;10(9):2120.

Measurement of nitric oxide levels in exhaled air is commonly performed using a chemiluminescence detector. However, water vapour and carbon dioxide affect the chemiluminescence process. The influence of these gases at the concentrations present in exhaled air, has not yet been studied. For this in vitro study, mixtures of 50, 100, and 200 parts per billion (ppb) NO in air were prepared and fed into the NO analyser either directly or bubbled through water. Mixtures with CO₂ were prepared by adding 0-10% CO₂ to the

diluent air. We found a significant decrease in NO readings in the water-saturated samples compared to the dry gas ($p < 0.001$), strongly dependent on the partial pressure of water. NO levels in exhaled air (mean 10 ± 2 ppb) showed a decrease of $17 \pm 3\%$ when water vapour was not absorbed. From the experiments with CO₂ we found a decrease in NO reading of $1.04 \pm 0.07\%$ /volume CO₂(%). Presence of water vapour, thus, leads to a systematic underestimation of NO levels. Insertion of a water absorber might, therefore, be advantageous. The influence of CO₂ concentrations in the normal respiratory range is negligible. With high expiratory CO₂ levels as applied in permissive hypercapnia, the effects may be substantial.

Airway Deposition and Airway Effects of Anti-asthma Drugs Delivered from Metered-Dose Inhalers—Pauwels R, Newman S, Borgstrom L. *Eur Respir J* 1997;10(9):2127.

Many different metered dose inhalation devices are becoming available for the treatment of airway diseases. Each of these inhalers differs in its

delivery characteristics. An assessment of the efficacy of drug delivery by these inhalers is essential, in view of their therapeutic use. A review of the literature on the relationship between airway deposition and airway effects of drugs delivered from metered dose inhalers is presented. Nebulizers or spacers are not discussed. The effect of an inhaler depends on the characteristics of the inhaler and the inhalation manoeuvre performed by the patient. This review focuses on the influence of inhaler characteristics on the airway deposition and the airway effects of the drug. Studies on the relationship between airway deposition and airway effect have been troubled by methodological problems, such as the absence of multiple dose comparisons and the difficulty in obtaining steep dose-response curves. The techniques for measuring airway deposition of inhaled drugs, namely the scintigraphic and the pharmacokinetic method, are discussed and compared. The appropriate use of these techniques can help to define

and compare the drug delivery characteristics of different devices, thus enabling inhaled therapy to be optimized.

Intensive Care 1980-1995: Change in Patient Characteristics, Nursing Workload and Outcome—Jakob SM, Rothen HU. *Intensive Care Med* 1997;23(11):1165.

OBJECTIVE: To assess temporal changes in patient characteristics, nursing workload and outcome of the patients and to compare the actual amount of available nursing staff with the estimated needs in a medical-surgical intensive care unit (ICU). **DESIGN:** Retrospective analysis of prospectively collected data. **SETTING:** A medical-surgical adult ICU in a Swiss university hospital. **PATIENTS:** Data of all patients staying in the ICU between January 1980 and December 1995 were included. **INTERVENTIONS:** None. **MEASUREMENTS & RESULTS:** The estimated number of nurses needed was defined according to the Swiss Society of Intensive Care Medicine (SGI) grading system: Category I = 1 nurse/patient/shift (= 8 h). Category II = 1 nurse/2 patients/shift. Category III = 1 nurse/3 patients/shift. An intervention score (IS) was obtained, based on a number of specific activities in the ICU.

There was a total of 35,327 patients (32% medical and 68% postoperative/trauma patients). Over time, the number of patients/year increased (1980/1995: 1,825/2,305, $p < 0.001$) and the length of ICU stay (LOS) decreased (4.1/3.8 days, $p < 0.013$). There was an increase in the number of patients aged > 70 years (19%/28%, $p < 0.001$), and a decrease in the number of patients < 60 years (58%/41%, $p < 0.001$). During the same time period, the IS increased 2-fold. Measurement of nursing workload showed an increase over time. The number of nursing days/year increased (1980/1995: 7454/8681, $p < 0.019$), as did the relative amount of patients in Category I (49%/71%, $p < 0.001$), whereas the portion of patients in Category II (41%/28%, $p < 0.019$) and Category III (10%/0%) decreased. During the same time period, mortality at ICU discharge decreased (9.0%/7.0%, $p < 0.002$). **CONCLUSIONS:** During the last 16 years, there has been a marked increase in workload at this medical-surgical ICU. Despite an increase in the number of severely sick patients (as defined by the nursing grading system) and patient age, ICU mortality and LOS declined from 1980 to 1995. This may be ascribed to improved patient treatment or care. Whether an increasingly liberal discharge policy (transfer to newly opened intermediate care units, trans-

fer of patients expected to die to the ward) or a more rigorous triage (denying admission to patients with a very poor prognosis) are confounding factors cannot be answered by this investigation. The present data provide support for the tenet that there is a trend toward more complex therapies in increasingly older patients in tertiary care ICUs. Calculations for the number of nurses needed in an ICU should take into account the increased turnover of patients and the changing patient characteristics.

High Survival Rate in 122 ARDS Patients Managed According to a Clinical Algorithm Including Extracorporeal Membrane Oxygenation—Lewandowski K, Rossaint R, Pappert D, Gerlach H, Slama KJ, Weidemann H, et al. *Intensive Care Med* 1997;23(8):819.

OBJECTIVE: We investigated whether a treatment according to a clinical algorithm could improve the low survival rates in acute respiratory distress syndrome (ARDS). **DESIGN:** Uncontrolled prospective trial. **SETTING:** One university hospital intensive care department. **PATIENTS & PARTICIPANTS:** 122 patients with ARDS, consecutively admitted to the ICU. **INTERVENTIONS:** ARDS was treated according to a criteria-defined clinical algorithm. The algorithm



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- A. Appreciate the cost of care
- B. Understand the scope and limitations of the practitioners role in patient assessment
- C. Understand the principle of patient self-determination
- D. Appreciate the psychological, social, and physical characteristics of wellness/illness
- E. Recognize the patient's right to confidentiality
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2. Perform a Comprehensive Health Assessment

- A. Review patient records
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- C. Conduct an interview
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- A. Identify sites of care and caregivers
- B. Interview the caregivers
- C. Assess the abilities of caregivers to manage physical, technological, and emergency situations
- D. Assess home health resources

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- A. Review the physician's order with the patient
- B. Assess the patient's understanding of technique and components

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- C. Assess patient ability to administer treatments, use equipment, monitor changes, interpret and respond to those changes
D. Document assessment findings

5. Assess the Patient's Environment as It Relates to Health Status and Supporting Physician's Orders

- A. Identify barriers to wellness

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- B. Inspect the patient care environment
C. Determine the resources available to the patient and family
D. Document assessment findings

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6. Assess Continued Appropriateness of Physician Orders and Care Plan

- A. Review physician orders
B. Evaluate the patient's response to physician orders
C. Evaluate the appropriateness of the orders and the care plan
D. Recommend modification of the treatment plan
E. Document assessment findings

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distinguished 2 main treatment groups: The AT-sine-ECMO (advanced treatment without extracorporeal membrane oxygenation) groups ($n = 73$) received a treatment consisting of a set of advanced noninvasive treatment options, the ECMO treatment group ($n = 49$) received additional extracorporeal membrane oxygenation (ECMO) using heparin-coated systems. **MEASUREMENTS & RESULTS:** The groups differed in both APACHE II (16 ± 5 vs 18 ± 5 points, $p = 0.01$) and Murray scores (3.2 ± 0.3 vs 3.4 ± 0.3 points, $p = 0.0001$), the duration of mechanical ventilation prior to admission (10 ± 9 vs 13 ± 9 days, $p = 0.0151$), and length of ICU stay in Berlin (31 ± 17 vs 50 ± 36 days, $p = 0.0016$). Initial P_{aO_2}/F_{iO_2} was 86 ± 27 mm Hg in AT-sine-ECMO patients that improved to 165 ± 107 mm Hg on ICU day 1, while ECMO patients showed an initial P_{aO_2}/F_{iO_2} of 67 ± 28 mm Hg and improvement to 160 ± 102 mm Hg was not reached until ICU day 13. $\dot{Q}_{sp}/\dot{Q}_{O_2}$ was significantly higher in the ECMO-treated group and exceeded 50% during the first 14 ICU days. The overall survival rate in our 122 ARDS patients was 75%. Survival rates were 89% in the AT-sine-ECMO group and 55% in the ECMO treatment group ($p = 0.0000$). **CONCLUSIONS:** We conclude that patients with ARDS can be successfully treated with the clinical algorithm and high survival rates can be achieved.

Orbital Herniation Associated with Noninvasive Positive Pressure Ventilation—Lazowick D, Meyer TJ, Pressman M, Peterson D. Chest 1998;113(3):841.

A diagnosis of severe obstructive sleep apnea was made after a 52-year-old hypertensive man developed a large intracranial hemorrhage. Therapeutic noninvasive positive pressure ventilation (NPPV) for obstructive sleep apnea and hypoventilation was complicated by transient unilateral orbital herniation. As best as can be determined, this represents a new, potentially deleterious side effect of NPPV.

IPEEP-Induced Tricuspid Regurgitation—Artucio H, Hurtado J, Zimet L, de Paula J, Beron M. Intensive Care Med 1997;23(8):836.

OBJECTIVE: To determine the presence of tricuspid regurgitation (TR) in patients affected by acute lung injury (ALI) and the adult respiratory distress syndrome (ARDS) during mechanical ventilation with positive end-expiratory pressure (PEEP). **DESIGN:** A prospective clinical study. **SETTING:** 10-bed general intensive care unit in a University Hospital. **PATIENTS:** 7 consecutive patients an age 44.7 ± 8.6 years with a diagnosis of ALI or ARDS were studied. All were on mechanical ventilation with PEEP. **INTERVENTIONS:** PEEP was increased in steps of 5 cm H₂O until the appearance of TR or up to a limit of 20 cm H₂O. **MEASUREMENTS & RESULTS:** Right atrial pressure, pulmonary artery pressure,

and wedge pressure were measured and cardiac output was determined by thermodilution. TR was graded from 0 to 3. Standard 2-D echocardiographic and pulsed-wave images were obtained at each level of PEEP. PEEP was increased from 4 ± 3 to 17 ± 2 cm H₂O. Mean PAP increased from 27.7 ± 2.9 to 36.7 ± 3.5 mm Hg ($p < 0.02$) when PEEP was increased. Five patients had competent valves and 2 had mild TR at baseline. In 6 out of the 7, TR either developed or increased when PEEP was increased. **CONCLUSIONS:** Our study demonstrated the development of TR after the use of PEEP in patients with ALI and ARDS as a consequence of pulmonary hypertension and right ventricular overloading. Since TR may randomly affect cardiac output values and derived parameters, the assessment of cardiac performance by some techniques such as thermodilution should be used with caution.

Respiratory Comfort of Automatic Tube Compensation and Inspiratory Pressure Support in Conscious Humans—Guttman J, Bernhard H, Mols G, Benzing A, Hofmann P, Haberthür C, et al. Intensive Care Med 1997;23(11):1119.

OBJECTIVE: To compare the new mode of ventilatory support, which we call automatic tube compensation (ATC), with inspiratory pressure support (IPS) with respect to perception of respiratory comfort. ATC unloads the resistance of the endotracheal tube (ETT) in inspiration by increasing the airway pressure, and in expiration by decreasing the airway pressure according to the nonlinear pressure-flow relationship of the ETT. **DESIGN:** Prospective randomized single blind cross-over study. **SETTING:** Laboratory of the Section of Experimental Anaesthesiology (Clinic of Anaesthesiology; University of Freiburg). **SUBJECTS:** Ten healthy volunteers. **INTERVENTIONS:** The subjects breathed spontaneously through an ETT of 7.5 mm ID. Three different ventilatory modes, each with a PEEP of 5 cm H₂O, were presented in random order using the Dräger Evita 2 ventilator with prototype software: (1) IPS (10 cm H₂O, 1 s ramp), (2) inspiratory ATC (ATC-in), (3) inspiratory and expiratory ATC (ATC-in-ex). **MEASUREMENTS & MAIN RESULTS:** Immediately following a mode transition, the volunteers answered with a hand sign to show how they perceived the new mode compared with the preceding mode in terms of gain or loss in subjective respiratory comfort: "better," "unchanged," or "worse." Inspiration and expiration were investigated separately analyzing 60 mode transitions each. Flow rates were continuously measured. The transition from IPS to either type of ATC was perceived positively, ie as increased comfort, whereas the opposite transition from ATC to IPS was perceived negatively, ie as decreased comfort. The transition from ATC-in to ATC-in-ex was perceived positively whereas the opposite mode transition was perceived negatively in expiration only. Tidal volume was 1220

± 404 mL during IPS and 1017 ± 362 mL during ATC. The inspiratory peak flow rate was 959 ± 78 mL/s during IPS and 1048 ± 197 mL/s during ATC. **CONCLUSIONS:** ATC provides an increase in respiratory comfort compared with IPS. The predominant cause for respiratory discomfort in the IPS mode seems to be lung over-inflation.

Interpretation of the Pulmonary Artery Occlusion Pressure in Mechanically Ventilated Patients with Large Respiratory Excursions in Intrathoracic Pressure—Hoyt JD, Leatherman JW. Intensive Care Med 1997;23(11):1125.

OBJECTIVE: To assess the reliability of the pulmonary artery occlusion pressure (P_{pao}) when respiratory excursions in intrathoracic pressure are prominent. **DESIGN:** We studied 24 critically ill patients who had 15 mm Hg or more of respiratory excursion in their P_{pao} tracing. Large respiratory excursions resulted from respiratory muscle activity that persisted despite sedation and mechanical ventilation in the assist-control mode. From the P_{pao} tracing, the end-expiratory and mid-point values were recorded; the latter was measured halfway between end-expiration and the nadir due to inspiratory triggering. The P_{pao} was then re-measured after administration of a non-depolarizing muscle relaxant. **SETTING:** Medical intensive care unit of a university-affiliated teaching hospital. **MEASUREMENTS & RESULTS:** The difference between the pre-relaxation end-expiratory P_{pao} and the relaxed P_{pao} was larger than the difference between the pre-relaxation mid-point P_{pao} and the relaxed P_{pao} (11 ± 5 vs 3 ± 3 mm Hg, $p < 0.01$). In 21 of 24 (88%) cases, the relaxed P_{pao} was more closely approximated by the mid-point P_{pao} than by the end-expiratory P_{pao} . The difference between the end-expiratory P_{pao} and the relaxed P_{pao} increased as the amount of respiratory excursion increased ($r = 0.51$; $p < 0.01$). **CONCLUSIONS:** In mechanically ventilated patients whose respiratory muscles produce large excursions in the P_{pao} , the end-expiratory P_{pao} is often much higher than the P_{pao} measured after muscle relaxation. The pre-relaxation mid-point P_{pao} and the relaxed P_{pao} are usually similar, but this may not be true in individual patients. In this setting, the P_{pao} measured after muscle relaxation probably provides the most clinically reliable estimate of left heart filling pressure.

Functional Magnetic Stimulation of Expiratory Muscles: A Noninvasive and New Method for Restoring Cough—Lin VWH, Hsieh C, Hsiao IN, Canfield J. J Appl Physiol 1998;84(4):1144.

The purpose of this study was to assess the effectiveness of functional magnetic stimulation (FMS) for producing expiratory function in normal human subjects. Twelve able-bodied normal subjects were recruited for this study. FMS of the expiratory muscles was performed by using a

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magnetic stimulator and placing the magnetic coil along the lower thoracic spine. Results showed that peak expired pressure, volume, and flow rate generated by FMS at the end of normal inspiration (102.5 ± 13.62 cm H₂O, 1.6 ± 0.16 L, and 4.8 ± 0.35 L/s, respectively) were comparable to their voluntary maximal levels ($p > 0.1$). The optimal coil placement was between T7 and T11, and the optimal stimulation parameters were a frequency of 25 Hz and 70-80% of maximal intensity. We conclude that (1) FMS of the lower thoracic nerves in normal subjects resulted in a significant expiratory function comparable to their voluntary maximum; (2) FMS was noninvasive and was well tolerated by all subjects; and (3) FMS may be useful to produce cough in patients in critical care or perioperative settings, or in patients with neurological disorders.

Distribution of Inhaled Nitric Oxide during Sequential and Continuous Administration into the Inspiratory Limb of the Ventilator—Mourgeon E, Gallart L, Rao GS, Lu Q, Law-Koune JD, Puybasset L, et al. *Intensive Care Med* 1997;23(8):849.

OBJECTIVES: The concentrations of nitric oxide (NO) in the ventilatory circuits and the patient's airways were compared between sequential (SQA)

and continuous (CTA) administration during inspiratory limb delivery. **DESIGN:** Prospective controlled study. **SETTING:** Fourteen-bed Surgical Intensive Care Unit of a teaching University hospital. **PATIENTS & PARTICIPANTS:** Eleven patients with acute lung injury on mechanical ventilation and 2 healthy volunteers. **INTERVENTIONS:** A prototype NO delivery device (Opti-NO) and Cesar ventilator were set up in order to deliver 1, 3, and 6 parts per million (ppm) of NO into the bellows of a lung model in SQA and CTA. Using identical ventilatory and Opti-NO settings, NO was administered to the patients with acute lung injury. **MEASUREMENTS & RESULTS:** NO concentrations measured from the inspiratory limb [INSP-NO_{Mech}] and the trachea [TRACH-NO_{Mech}] using fast response chemiluminescence were compared between the lung model and the patients using controlled mechanical ventilation with a constant inspiratory flow. INSP-NO_{Mech} were stable during SQA and fluctuated widely during CTA (fluctuation at 6 ppm = 61% in the lung model and $58 \pm 3\%$ in patients). In patients, [TRACH-NO_{Mech}] fluctuated widely during both modes (fluctuation at 6 ppm = $55 \pm 3\%$ during SQA and $54 \pm 5\%$ during CTA). The NO flow requirement was significantly lower during SQA than during CTA (74 ± 0.5 vs 158 ± 2.2 mL/min⁻¹ to attain 6 ppm, $p = 0.0001$). INSP-NO_{Mech} were

close to the values predicted using a classical formula only during SQA (bias = -0.1 ppm, precision = ± 1 ppm during SQA; bias = 2.93 ppm and precision = ± 3.54 ppm during CTA). During SQA, INSP-NO_{Mech} varied widely in healthy volunteers on pressure support ventilation. **CONCLUSIONS:** CTA did not provide homogenous mixing of NO with the tidal volume and resulted in fluctuating INSP-NO_{Mech}. In contrast, SQA delivered stable and predictable NO concentrations during controlled mechanical ventilation with a constant inspiratory flow and was economical compared to CTA. However, SQA did not provide stable and predictable NO concentrations during pressure support ventilation.

Predictors of Smoking Cessation after Percutaneous Coronary Revascularization—Hasdai D, Garratt KN, Grill DE, Mathew V, Lerman A, Gau GT, Holmes DR Jr. *Mayo Clin Proc* 1998;73(3):205.

OBJECTIVE: To identify factors predictive of smoking cessation after successful percutaneous coronary revascularization. **MATERIAL & METHODS:** We undertook a case-control study of the smoking status of all patients at Mayo Clinic Rochester from September 1979 through December 1995 who were smokers at the time of an index



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percutaneous coronary revascularization procedure in the nonperi-infarction setting (no myocardial infarction within 24 hours). Maximal duration of prospective follow-up was 16 years. Patients were classified into those who permanently quit smoking immediately after the procedure ($n = 435$; mean follow-up, 5.1 ± 3.7 years) or those who continued to smoke at some time during follow-up ($n = 734$; mean follow-up, 5.3 ± 3.7 years). Logistic regression models were formulated to determine independent predictors of smoking cessation. RESULTS: Predictors of continued smoking were greater prior cigarette consumption (odds ratio [OR] = 1.009 for each pack-year; 95% confidence interval [CI] = 1.004 to 1.014) and having one or more risk factors for coronary artery disease other than cigarette smoking (OR = 1.49; 95% CI = 1.15 to 1.93). Older age (OR = 0.98 for each additional year; 95% CI = 0.97 to 0.99) and unstable angina at time of initial assessment (OR = 0.69; 95% CI = 0.52 to 0.91) were associated with less likelihood of continued smoking. CONCLUSION: Younger patients with a worse risk profile and greater prior cigarette consumption were more likely than other patients to continue smoking after percutaneous coronary revascularization in the nonperi-infarction setting. Patients who had unstable angina were more likely to quit smoking than those who had stable angina. Despite the proven benefits of smoking cessation after percutaneous coronary revascularization, a substantial proportion of smokers (63%) continue to smoke; thus, smoking-cessation counseling should be addressed in this population.

Antibiotic Prescribing for Children with Colds, Upper Respiratory Tract Infections, and Bronchitis—Nyquist AC, Gonzales R, Steiner JF, Sande MA. JAMA 1998;279(11):875.

CONTEXT: The spread of antibiotic-resistant bacteria is associated with antibiotic use. Children receive a significant proportion of the antibiotics prescribed each year and represent an important target group for efforts aimed at reducing unnecessary antibiotic use. OBJECTIVE: To evaluate antibiotic-prescribing practices for children younger than 18 years who had received a diagnosis of cold, upper respiratory tract infection (URI), or bronchitis in the United States. DESIGN: Representative national survey of practicing physicians participating in the National Ambulatory Medical Care Survey conducted in 1992 with a response rate of 73%. SETTING: Office-based physician practices. PARTICIPANTS: Physicians completing patient record forms for patients younger than 18 years. MAIN OUTCOME MEASURES: Principal diagnoses and antibiotic prescriptions. RESULTS: A total of 531 pediatric office visits were recorded that included a principal diagnosis of cold, URI, or bronchitis. Antibiotics were prescribed to 44% of patients with common colds, 46% with URIs, and 75% with bronchitis. Extrapolating to the United States, 6.5

million prescriptions (12% of all prescriptions for children) were written for children diagnosed as having a URI or nasopharyngitis (common cold), and 4.7 million (9% of all prescriptions for children) were written for children diagnosed as having bronchitis. After controlling for confounding factors, antibiotics were prescribed more often for children aged 5 to 11 years than for younger children (odds ratio [OR], 1.94; 95% confidence interval [CI], 1.13-3.33) and rates were lower for pediatricians than for nonpediatricians (OR, 0.57; 95% CI, 0.35-0.92). Children aged 0 to 4 years received 53% of all antibiotic prescriptions, and otitis media was the most frequent diagnosis for which antibiotics were prescribed (30% of all prescriptions). CONCLUSIONS: Antibiotic prescribing for children diagnosed as having colds, URIs, and bronchitis, conditions that typically do not benefit from antibiotics, represents a substantial proportion of total antibiotic prescriptions to children in the United States each year. See the related article: *Why Do Physicians Prescribe Antibiotics for Children with Upper Respiratory Tract Infections?* (editorial)—Schwartz B, Mainous AG 3rd, March SM. JAMA 1998;279(11):881-882.

Comparative Efficacy of a Two-Hour Regimen of Streptokinase versus Alteplase in Acute Massive Pulmonary Embolism: Immediate Clinical and Hemodynamic Outcome and One-Year Follow-Up—Meneveau N, Schiele F, Metz D, Valette B, Aitali P, Vuilleminot A, et al. J Am Coll Cardiol 1998;31(5):1057.

OBJECTIVES: This study sought to compare the efficacy of 2-h regimens of alteplase and streptokinase in acute massive pulmonary embolism. The primary end point was immediate hemodynamic improvement, and secondary end points included early clinical efficacy and safety, as well as 1-year clinical outcome. BACKGROUND: Several thrombolytic regimens have been compared for the past 10 years in randomized studies, showing that 2-h infusion regimens of alteplase or urokinase lead to faster hemodynamic improvement than former 12- to 24-h administration protocols in acute massive pulmonary embolism. Many trials have focused on immediate hemodynamic and angiographic outcomes, but none has addressed long-term follow-up after thrombolysis. METHODS: Sixty-six patients with acute massive pulmonary embolism (Miller score > 17 and mean pulmonary artery pressure > 20 mm Hg) were randomly assigned to receive either a 100-mg 2-h infusion of alteplase ($n = 23$) or 1.5 million IU of streptokinase over 2 h ($n = 43$). In both groups, heparin infusion was started at the end of thrombolytic infusion and adapted thereafter. Total pulmonary resistance was monitored over a 12-h period. Pulmonary vascular obstruction was assessed 36 to 48 h after thrombolytic therapy. One-year follow-up information included death, cause of death, recurrent pulmonary embolism, chronic thromboembolic pulmonary hypertension, stroke, and

bleeding. RESULTS: Both groups had similar baseline angiographic and hemodynamic characteristics of severity, with maintained cardiac output in 64 (97%) of 66 patients. The results (mean \pm SD) demonstrated that despite a faster total pulmonary resistance improvement observed at 1 h in the alteplase group compared with the streptokinase group ($33 \pm 16\%$ vs $19 \pm 16\%$, $p = 0.006$), a similar hemodynamic efficacy was obtained at 2 h when both thrombolytic regimens were completed ($38 \pm 18\%$ vs $31 \pm 19\%$). There was no significant difference in either pulmonary vascular obstruction at 36 to 48 h or bleeding complication rates. One-year event-free survival was similar in both groups, as most events were related to concomitant diseases. CONCLUSIONS: These results suggest that a 2-h regimen of streptokinase can be routinely used in patients with massive pulmonary embolism and maintained cardiac output without obviously compromising efficacy or safety.

High-Frequency Oscillatory Ventilation in Neonatal RDS: Initial Volume Optimization and Respiratory Mechanics—Kalenga M, Battisti O, Francois A, Langhendries JP, Gerstmann DR, Bertrand JM. J Appl Physiol 1998;84(4):1174.

To determine whether initial lung volume optimization influences respiratory mechanics, which could indicate the achievement of optimal volume, we studied 17 premature infants with respiratory distress syndrome (RDS) assisted by high-frequency oscillatory ventilation. The continuous distending pressure (CDP) was increased stepwise from 6-8 cm H₂O up to optimal CDP (OCDP), ie, that allowing good oxygenation with the lowest inspired O₂ fraction. Respiratory system compliance (C_{rs}) and resistance were concomitantly measured. Mean OCPD was 16.5 ± 1.2 cm H₂O. $F_{I(O_2)}$ could be reduced from an initial level of 0.73 ± 0.17 to 0.33 ± 0.07 . However, C_{rs} (0.45 ± 0.14 mL \cdot cm H₂O⁻¹ \cdot kg⁻¹ at starting CDP point) remained unchanged through lung volume optimization but appeared inversely related to OCPD. Similarly, respiratory system resistance was not affected. We conclude that there is a marked dissociation between oxygenation improvement and C_{rs} profile during the initial phase of lung recruitment by infants with high-frequency oscillatory ventilation in early RDS. Thus optimal lung volume cannot be defined by serial C_{rs} measurement. At the most, low initial C_{rs} suggests that higher CDP will be needed.

Immunizations for International Travelers—Thanassi WT. West J Med 1998;168(3):197.

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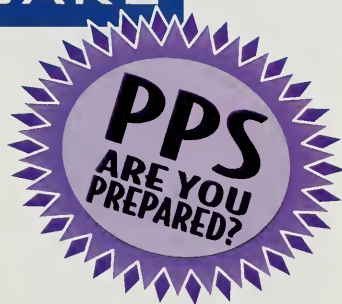
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to 75% chance of becoming ill, although only 1% will require hospitalization. The 2 most common illnesses that affect travelers, which do have immunizations and are often overlooked by physicians, are influenza and hepatitis A. The risk of illness to the traveler varies by health and age status, by the region to be visited, by the time of year, and by the length of the journey. Immunization advice for the traveler, therefore, is complicated and is best approached in a systematic manner. This article outlines 6 steps to sound immunization advice. These steps include ascertaining the traveler's special individual needs, routine immunization status, and routine travel immunization status, as well as the entry requirements for the country to be visited, geographically indicated vaccines, and immunizations as indicated for extended stays abroad.

Effects of Inhaled Nitric Oxide in Patients with Acute Respiratory Distress Syndrome: Results of a Randomized Phase II Trial. Inhaled Nitric Oxide in ARDS Study Group—Dellinger RP, Zimmerman JL, Taylor RW, Straube RC, Hauser DL, Criner GJ, et al. Crit Care Med 1998;26(1):15.

OBJECTIVES: To evaluate the safety and physiologic response of inhaled nitric oxide (NO) in patients with acute respiratory distress syndrome (ARDS). In addition, the effect of various doses of inhaled NO on clinical outcome parameters was assessed. **DESIGN:** Prospective, multicenter, randomized, double-blind, placebo-controlled study. **SETTING:** Intensive care units of 30 academic, teaching, and community hospitals in the United States. **PATIENTS:** Patients with ARDS, as defined by the American-European Consensus Conference, were enrolled into the study if the onset of disease was within 72 hrs of randomization. **INTERVENTIONS:** Patients were randomized to receive placebo (nitrogen gas) or inhaled NO at concentrations of 1.25, 5, 20, 40, or 80 ppm. **MEASUREMENTS & MAIN RESULTS:** Acute increases in P_{aO_2} , decreases in mean pulmonary arterial pressure, intensity of mechanical ventilation, and oxygenation index were examined. Clinical outcomes examined were the dose effects of inhaled NO on mortality, the number of days alive and off mechanical ventilation, and the number of days alive after meeting oxygenation criteria for extubation. A total of 177 patients were enrolled over a 14-month period. An acute response to treatment gas, defined as a P_{aO_2} increase $\geq 20\%$, was seen in 60% of the patients receiving inhaled NO with no significant differences between dose groups. Twenty-four percent of placebo patients also had an acute response to treatment gas during the first 4 hrs. The initial increase in oxygenation translated into a reduction in the F_{iO_2} over the first day and in the intensity of mechanical ventilation over the first 4 days of treatment, as measured by the oxygenation index. There were no differences among the pooled inhaled NO groups and placebo with respect to mortality rate, the number of days alive and off

mechanical ventilation, or the number of days alive after meeting oxygenation criteria for extubation. However, patients receiving 5 ppm inhaled NO showed an improvement in these parameters. In this dose group, the percentage of patients alive and off mechanical ventilation at Day 28 (a post hoc analysis) was higher (62% vs 44%) than the placebo group. There was no apparent difference in the number or type of adverse events reported among those patients receiving inhaled NO compared with placebo. Four patients had methemoglobin concentrations $>5\%$. The mean inspired nitrogen dioxide concentration in inhaled NO patients was 1.5 ppm. **CONCLUSIONS:** From this placebo-controlled study, inhaled NO appears to be well tolerated in the population of ARDS patients studied. With mechanical ventilation held constant, inhaled NO is associated with a significant improvement in oxygenation compared with placebo over the first 4 hrs of treatment. An improvement in oxygenation index was observed over the first 4 days. Larger phase III studies are needed to ascertain if these acute physiologic improvements can lead to altered clinical outcome. See the related editorials: *Just Say NO to Inhaled Nitric Oxide for the Acute Respiratory Distress Syndrome*, Matthay MA, Pittet JF, Jayr C. *Crit Care Med* 1998;26(1):1; *Nitric Oxide Inhalation in Acute Respiratory Distress Syndrome: It Works, But Can We Prove It?* Zapol WM. *Crit Care Med* 1998;26(1):2.

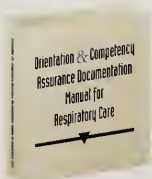
"Postpolio" Sequelae and Sleep-Related Disordered Breathing—Hsu AA, Staats BA. Mayo Clin Proc 1998;73(3):216.

OBJECTIVE: To analyze the clinical manifestations and various types of sleep-related disordered breathing (SRDB) in patients with a history of poliomyelitis and with current "postpolio" sequelae (PPS). **MATERIAL & METHOD:** We retrospectively reviewed the medical records of 108 consecutive patients with PPS and sleep disturbances encountered during an 11-yr period at Mayo Clinic Rochester and abstracted the features of acute polio, PPS, and results of sleep evaluation (overnight oximetry or polysomnography). Only those patients who were not receiving ventilatory support were included in the study. **RESULTS:** The features of PPS were dyspnea, fatigue, new weakness, and musculoskeletal pain. Of the 108 patients, 35 fulfilled the inclusion criteria. Sleep evaluations revealed 3 general types of disturbances: obstructive sleep apnea (Group O, $n = 19$); hypoventilation (Group H, $n = 7$); and both (Group OH, $n = 9$). The mean apnea/hypopnea index was 37, 4, and 16/hr in patients in Groups O, H, and OH, respectively ($p < 0.05$), and the mean arterial carbon dioxide tension was 39, 60, and 55 mm Hg in these respective study groups ($p < 0.05$). The overall mean age at onset of symptoms of SRDB was 47 years, and the mean latent period after acute polio was 37 years. Hypersomnolence was the commonest SRDB symptom,

present in 32 of the 35 patients. Snoring was noted in 100% of patients in Group O, 0% in Group H, and 67% in Group OH. Patients in Group O were obese and had normal lung function. Patients in Group H tended to have normal weights and a history of diffuse neurologic deficits involving the trunk during the acute episode of polio. Scoliosis, restricted lung function, cor pulmonale, and decreased maximal respiratory pressures were common in patients in Group H. Patients in Group OH had overlapping features of those in Groups O and H. **CONCLUSION:** In patients with PPS, we identified 3 patterns of sleep disturbances—obstructive sleep apnea, hypoventilation, and a combination of both. These groups are characterized by clinical features and by results of arterial blood gas determinations, overnight oximetry, and polysomnography. SRDB is a late sequela of poliomyelitis, and clinical evaluation should include information about sleep.

Declining Morbidity and Mortality among Patients with Advanced Human Immunodeficiency Virus Infection. HIV Outpatient Study Investigators—Palella FJ Jr, Delaney KM, Moorman AC, Loveless MO, Fuhrer J, Satten GA, et al. N Engl J Med 1998;338(13):853.

BACKGROUND & METHODS: National surveillance data show recent, marked reductions in morbidity and mortality associated with the acquired immunodeficiency syndrome (AIDS). To evaluate these declines, we analyzed data on 1255 patients, each of whom had at least one CD4+ count below 100 cells/mm³ who were seen at 9 clinics specializing in the treatment of human immunodeficiency virus (HIV) infection in 8 U.S. cities from January 1994 through June 1997. **RESULTS:** Mortality among the patients declined from 29.4/100 person-yrs in the first quarter of 1995 to 8.8/100 in the second quarter of 1997. There were reductions in mortality regardless of sex, race, age, and risk factors for transmission of HIV. The incidence of any of 3 major opportunistic infections (*Pneumocystis carinii* pneumonia, *Mycobacterium avium* complex disease, and cytomegalovirus retinitis) declined from 21.9/100 person-yrs in 1994 to 3.7/100 person-yrs by mid-1997. In a failure-rate model, increases in the intensity of antiretroviral therapy (classified as none, monotherapy, combination therapy without a protease inhibitor, and combination therapy with a protease inhibitor) were associated with stepwise reductions in morbidity and mortality. Combination antiretroviral therapy was associated with the most benefit; the inclusion of protease inhibitors in such regimens conferred additional benefit. Patients with private insurance were more often prescribed protease inhibitors and had lower mortality rates than those insured by Medicare or Medicaid. **CONCLUSIONS:** The recent declines in morbidity and mortality due to AIDS are attributable to the use of more intensive antiretroviral therapies.



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The traditional role of the respiratory therapist was task oriented; in this role the therapist skillfully implemented the procedures prescribed by the physician. This model functioned well for many years and was seldom questioned—particularly in a fee-for-service environment in which respiratory care procedures were lucrative for hospitals. It has been known for

improved and the diminishing need for intensive respiratory care (or perhaps any respiratory care) was overlooked by the prescribing physician.

SEE THE ORIGINAL STUDY ON PAGE 549

many years that a pattern of over-ordering of respiratory therapy occurs in many hospitals and that significant reductions in the volume of respiratory therapy procedures could be achieved with no adverse effect on patient outcomes.¹ Similarly, a pattern of misallocation of respiratory care was described in which patients received either therapy that was not indicated or indicated therapy was not prescribed.² Concurrent with the increasing recognition of the common misallocation of respiratory care, there has been increasing pressure to control health care costs. There has also been increased training of respiratory therapists in patient assessment and a desire of therapists to evolve beyond a task-oriented role to an increased professional role at the bedside.

The Traditional Model Results in Misallocation

The traditional model (ie, the respiratory therapist performs procedures as specifically prescribed by a physician) results in misallocation for several reasons.³ First, there may be insufficient understanding of respiratory care by those who prescribe the therapy. Although any physician can prescribe respiratory care, many never receive any specific instruction in ordering it. Second, the respiratory care needs of patients are variable and the prescribing physician may not be available to change the prescription when the patient's condition changes. The respiratory care prescription may be overlooked as the physician pays attention to other important needs of the patient. I am certain that respiratory care veterans remember many orders that were written at the time of patient admission and remained unchanged for days while the patient's condition

Respiratory Therapists Can Improve Allocation

In the past 10 years, there has been increasing use of respiratory therapists as consultants. This has been achieved by use of therapist-driven protocols, also known as patient-driven protocols, evaluate-and-treat programs, and respiratory therapy consult services.³ With these approaches, the physician orders the protocol rather than the specific therapy. Within the boundaries set by these medical-staff-approved protocols, a respiratory therapist assesses the patient, develops a respiratory care plan, implements the therapy, assesses the patient's response, and modifies the care plan as appropriate. There have been several reports of the effectiveness of such programs,⁴⁻⁷ and arguably the most sophisticated may be that at the Cleveland Clinic.^{8,9}

There are at least two issues of concern to physicians related to respiratory care protocols. First is a concern related to physician autonomy, in which the prescribing physician desires absolute control over all aspects of the patient's care. In many cases, this relates to a lack of confidence by the physician in the skills of the respiratory therapist. A second issue relates to the potential impact of respiratory care protocols on the training of house officers in academic centers. There is a concern among some physician educators that a skillfully implemented respiratory care protocol will decrease the opportunity for house officers to learn how to order respiratory care. I find this argument a paradox, given that the available evidence of the misallocation of respiratory care suggests that physicians may not learn how to order respiratory care very well. In this issue of the *Journal*, Stoller and Michnicki¹⁰ report the results of a survey of house officers' attitudes regarding the impact of their respiratory therapy consult service. They report that almost all house staff surveyed (97%) regarded the respiratory therapy consult service as helpful to the care of their patients. The majority, although only 56%, felt that the consult service enhanced the house staff's

knowledge of respiratory care ordering.

Partnership, Professionalism, & Protocols

What can be done to improve physician acceptance of respiratory care protocols? I believe that this can occur only when respiratory therapists develop partnerships with physicians and when respiratory therapists conduct themselves as professionals at the bedside. An environment must be developed that values the therapist as a consultant. This begins with communication. The therapist-as-consultant role does not happen simply because a respiratory therapy protocol is implemented. In fact, the successful implementation of protocols may depend upon a prior respect of physicians for respiratory therapists based upon professional interactions at the bedside. One might argue that respiratory therapy protocols are unnecessary in an environment where there is free and open communication between physicians and respiratory therapists. In fact, the benefit of forced interactions between respiratory therapists and physicians has been demonstrated.^{11,12} At the Massachusetts General Hospital, we have no structured respiratory care protocols. However, our respiratory therapists communicate freely and frequently with physicians, the opinions of the respiratory therapists are often consulted and respected, and the respiratory care plans of patients often reflect the suggestions of the respiratory therapists.

A mistake that I have noted with the implementation of respiratory therapy protocols is a tendency for therapists to view the protocol as a task rather than a consultation. This can damn the success of the protocol. Strict adherence to protocol instructions with little thought regarding the decisions that are made is task oriented and unprofessional. This will not engender the confidence of physicians in either the protocol or the therapist. I cannot stress enough the importance of the therapist acting as a consultant—whether or not protocols are used. Respiratory therapists can be consultants without protocols and protocols do not assure a consultative role. Carefully reasoned suggestions by a respiratory therapist with the needs of the patient in mind will elevate the respect of the physician for the therapist—even if the physician does not agree with the therapist's suggestions (physicians do not always agree with their physician consultants, either). Protocols can provide a framework to assist with the proper allocation of respiratory care, but the protocol should not be a substitute for thinking and communication.

I do not believe that respiratory care protocols and physician education are incompatible. An important role of respiratory therapists is to teach others—including physicians. Respiratory therapists are the experts on respiratory care and should be intimately involved in teaching physicians. This can occur through formal classroom teaching or, more importantly, at the bedside. A protocol should not be implemented in a vacuum. The therapist's assessment and care plan should

be openly communicated to the prescribing physician. The physician should understand the rationale for the care plan that is chosen and agree with that plan. In this way the decisions of the respiratory therapist become a model for the physician-in-training. An environment should exist in which the physician is a partner with the respiratory therapist in selecting the best care for the patient.

Therapist Responsibility

The responsibility for successful implementation of respiratory care protocols rests primarily with the therapist at the bedside. The medical director who champions the role of respiratory therapists and who promotes the protocol among the medical staff is important. A progressive and receptive medical staff is important. The support of nurses and other health professionals is important. However, the professional respiratory therapist at the bedside assessing the patient, making the correct decisions, and communicating with other members of the health care team adds value—that's what makes the protocol successful.

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Medical House Staff Impressions Regarding the Impact of a Respiratory Therapy Consult Service

James K Stoller MD and Irene Michnicki RRT

BACKGROUND: Although the Respiratory Therapy Consult Service (RTCS) has been shown to be effective in enhancing the allocation of respiratory care services, the criticism has been lodged that allocating algorithm-based decision-making to respiratory care practitioners detracts from physicians' in-training knowledge of respiratory care. To assess whether medical house staff regarded a well-established RTCS to be clinically and educationally helpful, we undertook a survey of house staff attitudes regarding the impact of this service on both patient care and house staff skill in ordering respiratory care. **METHODS:** A single-page, 6-question instrument was distributed at a regularly scheduled meeting of the Cleveland Clinic Foundation medical house staff. The questions posed were: (1) Do you know what the RTCS is? (2) Do you feel the RTCS is helpful or detrimental to the care of your patients? (3) Do you feel it has enhanced or detracted from your knowledge of respiratory care ordering? (4) Do you have a Respiratory Therapy Consult handbook? (5) Have you read it? (6) Have you found it helpful? **RESULTS:** Of a total of 95 available members of the medical house staff, 62 attended the meeting (65%), and 41 submitted responses (66% of attendees). Of the 41 respondents, 95% were aware of the RTCS. Similarly, 97% regarded the RTCS as being helpful to the care of their patients. Of 34 respondents, 56% felt that the RTCS enhanced the house staff's knowledge of respiratory care ordering, 32% felt that the Consult Service detracted from the house staff's knowledge, and 12% indicated that the RTCS had neither effect. Twenty-seven percent (11/41) indicated that they had an RTCS handbook, 19% (7/36) had read the handbook, and 33% (5/15) found it helpful. **CONCLUSIONS:** We conclude that the majority of responding house staff at the Cleveland Clinic Foundation were aware of the RTCS, suggesting successful institutional notification about the service. Similarly, almost all house staff regarded the Consult Service as being helpful in caring for their patients (97%), while 56% felt that the Respiratory Therapy Consult Service enhanced the house staff's knowledge of respiratory care ordering. Further study is required to examine whether these impressions about the impact of the RTCS are supported by direct measure of the house staff's knowledge of respiratory care ordering. [*Respir Care* 1998;43(7):549-551] *Key words:* Respiratory therapy consult services, house staff, protocols.

Introduction

Respiratory care protocols have gained wide acceptance based on emerging evidence that they improve the allocation of respiratory care services, enhance medical outcomes, and

save costs.¹⁻³ However, the use of respiratory care protocols (in which respiratory care practitioners prescribe care based on approved guidelines and/or algorithms) has engendered some concern by medical educators that respiratory care protocols and use of a respiratory therapy consult service will detract from house officers' education regarding the appropriate use of respiratory care therapies for their patients.

James K Stoller MD and Irene Michnicki RRT; Department of Pulmonary and Critical Care Medicine, Section of Respiratory Therapy, Cleveland Clinic Foundation, Cleveland, Ohio

A version of this paper was presented by Ms Michnicki at the Respiratory Care Open Forum during the American Association for Respiratory Care's International Respiratory Congress, December 6-9, 1997 in New Orleans, Louisiana.

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SEE THE RELATED EDITORIAL ON PAGE 546

As part of an ongoing assessment of the efficacy and impact of respiratory care protocols in our institution, the current study was undertaken to determine medical house officers' impressions of a long-standing Respiratory Therapy Consult Service (RTCS). To assess whether or not medical house staff regarded

a well-established respiratory therapy consult service to be clinically and educationally helpful, we undertook a survey of house staff attitudes regarding the impact of this service on both patient care and house staff skill in ordering respiratory care.

Methods

We used a single page, 6-question survey instrument that was administered at a regularly scheduled didactic meeting of the Cleveland Clinic medical house staff. Plans to administer the survey were not announced before the meeting, at which the study investigators distributed the questionnaires with introductory remarks emphasizing our desire for candid feedback on the RTCS. The questions posed were: (1) Do you know what the RTCS is? (2) Do you feel that the RTCS is helpful or detrimental to the care of your patients? (3) Do you feel the RTCS has enhanced or detracted from your knowledge of respiratory care ordering? (4) Do you have an RTCS handbook? (5) Have you read the handbook? (6) Have you found the handbook helpful?

As part of routine house staff orientation in July of each year, each house officer had received a copy of the handbook that describes the RTCS and contains the algorithms that guide respiratory care ordering in our hospital. As previously described,^{4,5} the algorithms are branched logic diagrams that are indexed to specific respiratory signs and symptoms and are based on American Association for Respiratory Care Clinical Practice Guidelines.⁶ Because the RTCS has been mandated for use on most adult, non-ICU inpatients in the Cleveland Clinical Hospital, house officers frequently interact with respiratory care practitioners as they apply the RTCS algorithms.

Results

Of a total of 95 available members of the medical house staff, 62 attended this regularly scheduled meeting (65%) and 41 submitted responses (66% of attendees, 43% of all medical house officers). Of the 41 respondents, 95% were aware of the RTCS (Fig. 1). Similarly, 97% regarded the RTCS as being helpful to the care of their patients. Of the 34 respondents to Question 3 (Do you feel that the RTCS has enhanced or detracted from your knowledge of respiratory care ordering?), 56% felt that the RTCS enhanced the house staff's knowledge of respiratory care ordering, 32% felt that the RTCS detracted from the house staff's knowledge, and 12% indicated that the RTCS had neither effect. Twenty-seven percent (11/41) indicated that they had the handbook, 19% (7/36) had read the handbook, and 33% (5/15) found it helpful.

Discussion

Although contrary and perhaps surprising to critics' expectations, our results show that most participating medical house

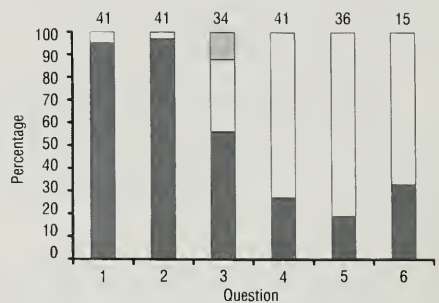


Fig. 1. Histogram distribution of medical house officers' responses to the 6-question instrument: Question 1. Do you know what the Respiratory Therapy Consult Service (RTCS) is? Question 2. Do you feel the RTCS is helpful (yes) or detrimental (no) to the care of your patients? Question 3. Do you feel the RTCS has enhanced (yes) or detracted from (no) your knowledge of respiratory care? Question 4. Do you have an RTCS handbook? Question 5. Have you read the RTCS handbook? Question 6. Have you found the RTCS handbook helpful? ■ = yes responses; □ = no responses; □ = neither or neutral responses. The total number of respondents to each question is indicated above each bar.

officers regarded the RTCS as enhancing the respiratory care their patients received and enhancing their own knowledge of respiratory care ordering. At the same time, almost one third of respondents felt that the RTCS detracted from their knowledge of respiratory care—an issue that raises concerns about the possible undesirable effects of a respiratory therapy protocol program.

Although data about the impact of protocols on medical trainees' education are sparse, our results are consistent with findings from a preliminary report by Messenger.⁷ Specifically, in a survey of 194 physicians to which 26 of 54 respondents (48%) were house officers, Messenger reported that many responding physicians regarded a respiratory care protocol program as increasing the timeliness (48%), quality (50%), and appropriateness (56%) of respiratory care delivered in the hospital. To our knowledge, no other study has examined the impact of respiratory care protocols on house officers' knowledge of respiratory care.

In the context that this is the first study to examine this issue, several shortcomings of this research are noteworthy. First, as with all survey-based research in which response rates are incomplete, we cannot exclude the possibility that respondents comprised a biased sample and, therefore, that the perceptions we recorded were not representative of all medical house officers. Specifically, our findings are based on responses from 66% of the attendees at the meeting, who comprised 43% of all medical house officers at the Cleveland Clinic. At the same time, because the survey was not announced beforehand, except for the unlikely possibility that conscientious attendance at

house staff meetings is somehow tied to favoring the RTCS, we do not suspect that this respondent sample was systematically biased in favor of respiratory care protocols. Also, despite our explicit request for candid responses, even if unflattering, we also cannot discount the possibility that respondents were inclined to provide favorable impressions.

Another important limitation of this initial study is that we elicited only house officers' impressions about the impact of the RTCS but did not actually assess house officers' expertise in ordering respiratory care services. As such, it is possible that the generally favorable impression about the impact of the RTCS on house staff knowledge of respiratory care is discordant with actual knowledge-based performance in ordering appropriate respiratory care. Indeed, addressing this issue requires comparing the actual knowledge base of respiratory care ordering by two groups of house officers: a group that has experienced a respiratory care protocol service versus a group that has not. Because the RTCS has been available in our institution since 1992 and medical house officers have been exposed continuously to this service since that time, an intramural comparison is not possible. Rather, we have initiated a multi-institutional study to compare performance in ordering respiratory care by two matched groups of house officers, one at our institution and the comparative group at an academic institution in which a respiratory care protocol program has not been initiated to date.

Finally, it will be important to more fully understand the specific reasons that 32% of the respondents felt that the RTCS detracted from their educational experience and whether this impression is supported by shortcomings of actual respiratory care knowledge.

Conclusions

In summary, our results show that most responding medical house officers in our institution regarded the RTCS favorably, with 97% regarding the RTCS as enhancing the clinical care provided to their patients and 56% reporting that the RTCS enhanced their knowledge of respiratory care ordering. To our knowledge, this is the first study to address this important educational question. Although our overall results challenge the view that a respiratory care protocol service detracts from house officers' education, this study clearly rep-

resents only an initial analysis of this issue, and additional study is needed in order to confirm and extend these findings. Specifically, before house staff acceptance of respiratory care protocols can be considered firm, reproducibly favorable results from studies of other house staff groups are needed. Even more broadly, demonstrating physician acceptance of respiratory care protocols will require sampling a fuller spectrum of physicians with whom respiratory care practitioners interact, namely attending physicians, fellow physicians, and both private and academic physicians.

It is important to note that our finding that support for the educational benefits of the RTCS was not unanimous raises concern and underscores the need to better understand potential detrimental effects of a respiratory care protocol service.

To address the shortcomings of the current research, a larger study with a more complete response rate is needed, ideally surveying house officers in multiple disciplines and in multiple institutions. Furthermore, to extend the analysis beyond opinion alone, the actual impact of a respiratory care protocol service on house officers' education should be assessed in studies that test house officers' knowledge of respiratory care ordering.

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Two-Tiered Response for Emergency Airway Management by Respiratory Therapists and Anesthesiologists

John D Hussey MBA RRT, Michael J Bishop MD, Lewis Massey, S Lakshminarayan MD, James Joy MD, and Jennifer Finley MD

INTRODUCTION: Out-of-operating-room intubations carry the potential for serious morbidity and mortality and require the around-the-clock presence of personnel trained in emergency airway management. **METHODS:** We established a hybrid model for response to airway problems of an acutely ill patient population; in this model, respiratory therapists handled emergent intubations with telephone back up from anesthesiologists whereas the anesthesiologist came in to the hospital to handle urgent intubations. Emergent intubations are cases in which the patient was expected to require intubation in < 30 min. The model is based on an assessment that demonstrated that the most complex intubations are urgent rather than emergent. Following the training described, the model was put in place, and the results of the first 18 months are reported. **RESULTS:** One hundred sixty four out-of-operating-room intubations were required, including 89 cardiac arrest patients and 75 noncardiac arrest patients. Respiratory therapists intubated the majority of cardiac arrest patients whereas the majority of nonarrest situations were intubated by anesthesiologists. Intubation was successful in 162 of 164 patients; 2 failures occurred in cardiac arrest situations with a physician present but no anesthesiologist immediately available. However in both cases, the anesthesiologist who arrived also could not intubate. **CONCLUSIONS:** This model permits the anesthesiologist to be available for the majority of intubations in the noncardiac arrest situation in which medical management and pharmacologic therapy are often required; whereas respiratory therapists perform the majority of intubations during cardiac arrest. This model is successful and resulted in economic savings. [Respir Care 1998;43(7):552-556] *Key words: Intubation, algorithms, role of respiratory therapist.*

Introduction

Because of the potential for airway emergencies to result in serious morbidity and mortality, an acute care hospital requires personnel trained in emergency airway management to be available at all times. In the acute care facilities with anesthesiology training programs, a physician trained in anesthesiology or emergency medicine is present at the hospital and responsible for emergency airway management in more than 90% of institutions surveyed.¹ Although respiratory therapists all receive some training in endotracheal intubation,² the extent of ongoing practice is variable and therapists do not routinely perform intubations in many hospitals—only 1% of hospitals with anesthesiology training programs used therapists for floor intubations.¹ A major impediment to the more widespread practice of intubation by respiratory therapists is that many patients requiring tracheal intubation have complex medical problems that may require pharmacologic therapy during the intubation.

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Although hospitals with anesthesiology training programs mostly use physicians for emergency airway management, the vast majority of hospitals do not have training programs. The number of anesthesiology training programs in the United States has declined in each of the last three years and will decline again this July (1998) to approximately 140, only a small minority of hospitals in the country.³ Many of the existing programs have cut back on the number of residents being trained, limiting staffing. Reductions in available anesthesia staffing at our 280-bed medical center led us to evaluate possible models to ensure safe and timely care of airway emergencies without requiring around-the-clock presence of a physician trained in airway management. Our review of possible models in the literature suggested that properly trained non-physicians could intubate the trachea with a success rate equal to or better than most physicians.^{4,6} These data came from experience both with paramedics in the out-of-hospital setting and respiratory care practitioners in the hospital.^{4,7} However, much of the literature refers to intubations for cardiac arrests, where no pharmacologic intervention to produce muscle relaxation or sedation is necessary. In reviewing our out-of-operating-room intubations, we found that many were in unstable but not arrested patients with high risk of deterioration during intubation. These patients included postcardiac surgery patients, bone marrow transplant patients, post-aortic surgery patients, post-thoracotomy patients, and patients with chronic obstructive pulmonary disease (COPD) or cardiac failure.

We hypothesized that a hybrid two-level model of emergency airway care would result in safe management of airways with acceptable outcomes. This hybrid model was designed to have the respiratory therapist intubate the trachea during cardiac arrest situations with an anesthesiologist available via telephone for consultation. Patients requiring intubation for respiratory failure, airway maintenance, or other progressive conditions were deemed to be more complex situations and an anesthesiologist was routinely present for such cases.

We describe the structure of the program and report the results of the first 18 months of the program.

Methods

Airway management and tracheal intubation training were initiated through a program within the operating room (O.R.) under the supervision of the Department of Anesthesiology. All therapists on staff rotated through the O.R. and were required to demonstrate a series of cognitive and procedural milestones (Table 1). Prior to patient contact, each therapist reviewed basic head positioning, blade placement, and tube positioning using a mannequin. Every O.R. intubation was evaluated by the attending anesthesiologist using standardized criteria. Criteria included head positioning, placement of the tube, and assessment of tube placement. Strengths

Table 1. Required Skills for Certification

Cognitive	Procedural
Access airway for difficult intubation	Bag-and-mask ventilation
Describe views of larynx	Application of cricoid pressure
Understand indications and contraindications for muscle relaxants	Intubation using MacIntosh and Miller blades
Plan for failed intubation	Intubation over a gum elastic bougie
Identification of successful intubation 1. Using bulb syringe 2. Using end-tidal CO ₂	Placement of laryngeal mask airway

and weaknesses were reviewed immediately to support improvement and a written assessment produced. In-service sessions, lectures, and written resource materials were used to support the training. A second level of educational support was accomplished through anesthesia-supervised elective and emergent intubations outside the O.R. Final certification included passing a written test.

Although we had initially established 10 as the minimum number of intubations required in the O.R. based in part on a literature report using 12 as a minimum,⁶ the minimum number actually performed by any therapist prior to certification ranged from 15 to 46, with a mean (SD) 31(17). The variability in the number of intubations required resulted from differing levels of prior training and experience as well as varying rates of acquisition of the required knowledge and skills.

Our model consisted of defining airway care as either urgent

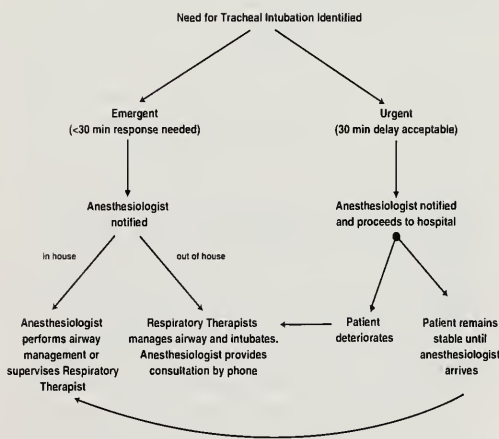


Fig. 1. The algorithm for tracheal intubations out of the operating room.

or emergent based on whether the patient was expected to require endotracheal intubation within 30 min. The decision tree for intubation is depicted in Figure 1. For emergent intubations, the respiratory therapist could proceed immediately whether an anesthesiologist was immediately available. Simultaneously, the anesthesiologist was paged and alerted to the situation. The anesthesiologist was thus available for phone consultation in case of intubation difficulty and could proceed to the hospital if further assistance was needed. The anesthesiologist could also consult with the medical house officer at the bedside via cell phone.

For urgent intubations, the anesthesiologist was called to the hospital and the therapist remained with the patient until the anesthesiologist arrived. Intubation was then performed either by the therapist under the direction of the anesthesiologist or by the anesthesiologist. The decision was based on the assessment by the anesthesiologist. The anesthesiologist directed all pharmacologic interventions.

Training also included providing the therapists with options in case of initial failure to intubate. The therapists were trained to use both MacIntosh and Miller laryngoscopic blades, instructed to use a smaller diameter tube if visualization was a problem, or to place a gum bougie and to feed a tube over the bougie. Finally, therapists were shown how to place a laryngeal mask airway; they then performed the procedure so they could use the mask in case of failure to intubate. Therapists were also instructed that if, after initial attempts, intubation could not be achieved, they should persist with bag-and-mask support pending the arrival of the anesthesiologist. We believed this would allow the therapists to fall back on a skill with which they were generally comfortable and would minimize the risk of traumatizing the airway and creating a situation where the anesthesiologist would be unable to intubate.

Results

Our results over the first 18 months are presented in Figure 2. Of the nonarrest cases, 65 of 75 had an anesthesiologist present. An anesthesiologist supervised 39 of 89 cardiac arrest intubations. Of the 104 supervised intubations, supervised either because of daytime hours or because they were urgent rather than emergent, all were successfully intubated. Anesthesiologist were not initially present for 60 intubations; and, of these, 2 were never intubated and both patients died. Three patients were intubated by a surgeon who was present after an initial attempt by a respiratory therapist was not successful, and one patient was supported with a bag and mask after initial unsuccessful attempts by a respiratory therapist and a surgeon, at which time the on-call anesthesiologist arrived and intubated the patient. This resulted in 54 successful unsupervised intubations by respiratory therapists. The unsuccessful intubations were in patients with difficult airways—one had massive facial swelling and the other was in a halo following transoral cervical spine surgery. Neither patient had been

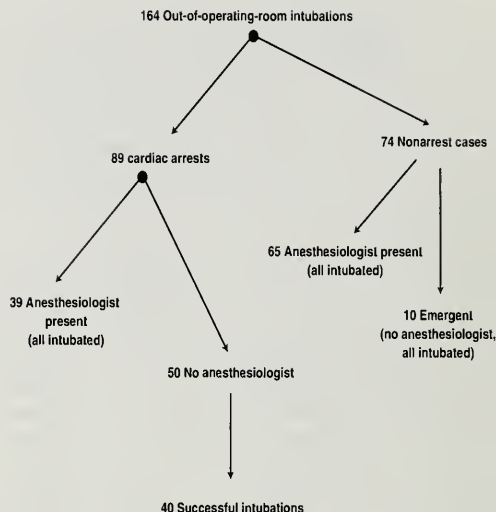


Fig. 2. Results of the 164 consecutive out-of-operating-room intubations performed during the first 18 months, using this model.

expected to survive; and upon their arrival the anesthesiologists were also unable to intubate either patient. Our model resulted in 162 of 164 patients successfully intubated.

Discussion

This retrospective 18-month review of our intubation model demonstrates that an airway management algorithm used in a tertiary care medical center does not necessarily require an in-house anesthesiologist or emergency physician. The 1.2% failure rate is within reported rates for out-of-operating-room intubations (Table 2).⁷⁻⁹ Meaningful statistical comparisons with other reports are difficult because of variations in the patient populations described, the circumstances described, and definitions of complications and failure rates. Some reports include only out-of-hospital intubation while others are limited to intensive care unit intubations only. Other reports do not note a failure rate but include intubations taking longer than 10 minutes or requiring extensive attempts. Our training discouraged repeated attempts by the therapist, substituting bag-and-mask support until the anesthesiologist arrived and established a plan. Table 2 summarizes existing studies and the circumstances and outcomes described. The low incidence of complications and the variation in definitions makes statistical comparison to other models virtually impossible. Rather, we judged our results to be acceptable based on outcome and based on failure rates within the range described

Table 2. Reported Results for Out-of-Operating-Room Intubations

Author	Intubators	Setting	Experience (% failure)
Mascia ¹⁰	Anesthesiologists	In-hospital	13/613 failed (2)
McLaughlin & Scott ⁶	Therapists	In-hospital	50/50 intubated, but 10% required longer than 5 min
Conley & Smith ⁵	Therapists Anesthesiologists Certified nurse anesthetists Other physician	In-hospital	3/74 failed (4) 0/30 failed 0/5 failed 4/34 failed (12)
Schwartz et al ¹¹	Critical care physician	Intensive care unit	0/297, 13 required > 4 attempts, 2 required 10 attempts
Thalman et al ¹²	Therapists	In-hospital	42/833 (5)
Zyla & Carlson ¹³	Therapists	In-hospital	18/178 (10)
Stauffer et al ⁹	House physician or anesthesiologist	In-hospital	1/97 failed (1), 20% required > 4 attempts
Stewart et al ⁴	Paramedics	Field cardiac arrest	70/779 (8)
DeLeo ¹⁴	Paramedics	Field	74/785 failed (9)

from other institutions. To perform a study demonstrating that this model was better would require numbers of patients that would be impossible to achieve.

Given the difficulty with statistical comparisons, we reviewed each case to consider whether we believed the outcome would have been different under our prior system of 24-hour in-house anesthesiologist presence. Under this model for 57% of the intubations, an anesthesiologist was still present, and in no case could we identify any morbidity from a delay in intubation pending the arrival of an anesthesiologist. Among the remaining intubations, the airway was generally established promptly by the respiratory therapist. In three cases a surgeon intervened after an initial failed laryngoscopy by a therapist. The surgeons had all completed anesthesia rotations and the interventions took place while the airway was controlled and before the therapists had begun to pursue alternative strategies. The two patients who were never intubated did not have a change in their expected outcomes because both were judged to have terminal illnesses prior to their arrests.

This model was established based on prior experience at our institution. We had noted that the majority of intubations actually occurred in patients who exhibited signs of progressive deterioration over a period longer than 30 min. Fur-

ther, we had observed that the more complex airway problems (requiring anesthesiologist involvement) were in patients who were deteriorating but had not yet arrested. Patients requiring emergent intubation generally had already suffered cardiac arrest. Following cardiac arrest, there is no need to consider the issues of sedation, muscle relaxation, or hemodynamic instability with intubation. One prior study of emergency room intubations found that 9 of 22 intubations for respiratory failure required prolonged attempts versus only 3 of 21 in patients who had suffered cardiac arrest,⁹ corroborating our experience that intubations for cardiac arrest tended to be less complicated.

The impetus for establishing this program was the loss of an anesthesia resident position. To continue in-house coverage by a physician skilled in airway management would have required an additional 12 hours per weekday night of physician time to cover a 6 pm-6 am shift plus 48 hours per weekend, for a total of 5,628 additional hours of coverage. The cost for an anesthesiologist or emergency physician in our area was \$50-75/h. We also considered the possibility of a nurse anesthetist but at a cost of \$40/h, the additional expense would have been formidable. We thus estimated a cost of well over \$200,000/yr to provide emergency airway coverage during night and weekend hours. The costs of our training program were minimal, and we expect the costs for recertification each year to remain low. Given the excellent initial outcomes, the program seems both clinically effective and cost-effective.

Conclusions

Although a recent survey found that only 1% of hospitals with anesthesiology training programs had respiratory therapists perform out-of-operating-room intubations, our model establishes that involving therapists in intubations can be cost-effective and provide an excellent level of care.

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Acute Pulmonary Effects of Toxic Nitrogen Dioxide Fume Inhalation

Dheeraj Gupta MD DM, Ashutosh Nath Aggarwal MD DM, Sanjay Jain MD DM, Digamber Behera MD, and Surinder Kumar Jindal MD

OBJECTIVES: To study pulmonary function in subjects accidentally exposed to nitrogen dioxide fumes. **DESIGN:** Case series. **SETTING:** Medical emergency service and respiratory laboratory at a tertiary care hospital in North India. **PARTICIPANTS:** Sixty-three male workers exposed to toxic fumes following an accidental explosion in a tunnel at their workplace. **MEASUREMENTS & RESULTS:** Six severely ill subjects were hospitalized; of them, 2 patients with acute respiratory distress syndrome needed assisted ventilation, one of whom died. Spirometry performed on the other 57 mildly affected subjects revealed restrictive defect in 42 (74%) and obstructive defect in 2 (4%) subjects. Arterial blood gas (ABG) analysis showed hypoxemia in 9 (16%) and respiratory alkalosis in 34 (60%) of these subjects. **CONCLUSIONS:** Acute toxic exposure to nitrogen dioxide fumes is potentially fatal. Even mildly affected and asymptomatic individuals may show abnormalities on pulmonary function evaluation. [Respir Care 1998;43(7):557-561] *Key words: Pulmonary function, nitrogen dioxide, toxic exposure, arterial blood gases, spirometry, acute inhalation injury.*

Introduction

Concern about the toxicity of the oxides of nitrogen has been frequently expressed in clinical and toxicological literature. The earliest evidence of human toxicity was recorded in 1804 when a merchant and his dog died after breathing nitric acid fumes.¹ Since then, occupational exposures to the oxides of nitrogen as a by-product in the process of silage fermentation, with acetylene oxygen flame in the field of welding, by improper detonation of explosives in mining activities, and, rarely, as a complication during anesthesia in medicine have been recorded.²⁻⁶

Of all the oxides of nitrogen, nitrogen dioxide is probably responsible for most of the pulmonary effects. Molecular nitrogen and oxygen combine to form nitric oxide (NO)

if heated to more than 2000° C. By cooling to approximately 600° C, nitrogen dioxide (NO₂) forms, which is a heavy red-brown pungent gas.⁴ Workers engaged in mining or underground tunnel activities are exposed to these gases either after the use of explosives or from diesel exhaust. Long-term exposures may lead to chronic lung damage characterized by hyperinflation.⁷ Massive exposure over a short period of time may result in acute lung toxicity, especially with exposures in excess of 200 parts per million (ppm).^{8,9}

A few investigators have studied pulmonary function in volunteers exposed to low levels of NO₂ (around 4 ppm or less) under controlled experimental conditions.¹⁰⁻¹⁴ Some data are also available regarding pulmonary function in hospitalized patients who presented with acute lung toxicity.^{4,15-18} We recently evaluated pulmonary function in a group of workers engaged in underground tunnel activities. The subjects were referred to us for respiratory assessment shortly after exposure to toxic fumes following an accidental explosion in a carriage carrying explosives.

Subjects & Methods

Sixty-three workers from an upcoming hydro-electric power project located more than 250 kilometers from this institute

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in the neighboring state of Himachal Pradesh were referred following exposure to toxic fumes after an accidental explosion in an underground tunnel. All of the subjects were working in the underground tunnel at the time of explosion. We saw the affected individuals approximately 48 hours after the mishap occurred. We obtained a detailed history regarding symptoms and smoking habits from all subjects and performed relevant physical examinations. We admitted severely affected patients for indoor management and followed them until discharge or death. We gave only symptomatic therapy to the remainder and observed them for up to 24 hours before they were discharged home.

After obtaining informed consent, we performed spirometry on all subjects capable of performing pulmonary function tests, using a Morgan Spiroflow spirometer (P K Morgan Ltd, Kent UK). We recorded forced vital capacity (FVC), forced expiratory volume in the first second (FEV₁), and peak expiratory flow (PEF) for all subjects as per standard guidelines, and expressed results at body temperature and pressure saturated with water vapor (BTPS).¹⁹ We calculated the predicted normal values for different parameters using regression equations previously derived by us.²⁰ We also expressed FVC, FEV₁, and PEF recordings as a percentage of normal predicted values (FVC%, FEV₁%, and PEF%, respectively). We interpreted spirometric data using FVC, FEV₁, and FEV₁/FVC ratios as the primary variables.²¹ We defined restrictive defect as FVC < 80% of predicted with a normal FEV₁/FVC ratio, and obstructive defect as an FEV₁/FVC ratio < 70%. We classified severity of restriction (or obstruction) as mild for FVC (or FEV₁) between 61-80%, moderate for 41-60%, and severe for < 40% of the predicted values. Estimation of total lung capacity (TLC) and lung compliance could not be carried out due to logistic reasons; hence, these variables were not included for the confirmation of restrictive defects identified on spirometry.

We obtained blood samples from the radial artery using a heparinized syringe, with the subject breathing room air (or supplemental oxygen in severely affected individuals) for arterial blood gas (ABG) analysis. We defined normoxemia as P_{O₂} ≥ 80 mm Hg [10.6 kPa] and hypoxemia as P_{O₂} 60-79 mm Hg [8.0 - 10.5 kPa] (mild); P_{O₂} 45-59 mm Hg [6.0-7.9 kPa] (moderate); and P_{O₂} < 45 mm Hg [6.0 kPa] (severe).²² We defined normocapnia as P_{CO₂} 35-45 mm Hg [4.7 - 6.0 kPa], hypocapnia as P_{CO₂} less than this range, and hypercapnia as more than this range. We defined normal pH as 7.35 to 7.45.

Results

All the subjects seen in the Emergency Service were men with a mean age of 28.4 (± 6.9) years (range 20-53 yrs). Fifty-two of the 63 subjects (83%) were asymptomatic; others complained of dyspnea, chest discomfort, burning in eyes, giddiness, vomiting, and headache. Two had severe respiratory distress, altered sensorium, and bilateral crepitations on aus-

cultation. ABG analysis showed severe hypoxemia, and their chest radiographs revealed bilateral extensive alveolar infiltrates. Neither had any known pre-existing cardiac disease; however, pulmonary capillary wedge pressure measurements were not available. Considering these findings, we diagnosed acute respiratory distress syndrome (ARDS) in both the patients in accordance with standard criteria.²³ Four more patients had severe dyspnea and/or chest discomfort. We admitted these 6 patients and discharged the remaining 57 subjects after observation and symptomatic treatment. The 2 patients with ARDS required assisted ventilation, one of whom had refractory hypoxemia and died within 12 hours of admission. The second patient improved after 7 days of mechanical ventilation. The other 4 severely affected patients improved with supplemental oxygen therapy and were discharged after 2 (3 patients) or 3 days (1 patient). These 6 patients had moderate to severe hypoxemia on room air at the time of admission (Table 1).

Table 1. Clinical Details of 6 Severely Affected Subjects

Subject, Age (years)	ABG at Admission (Room Air)		Chest Radiograph*	Outcome
	P _{O₂} (mm Hg)	P _{CO₂} (mm Hg)		
1, 26	21	39	Bilateral alveolar infiltrates suggestive of ARDS†	Ventilated & recovered
2, 26	32	34	Bilateral alveolar infiltrates suggestive of ARDS	Ventilated but died
3, 37	52	28	Normal	Recovered
4, 24	56	28	Normal	Recovered
5, 30	52	32	Normal	Recovered
6, 24	37	35	Normal	Recovered

* Chest radiographs of the other 57 mildly affected individuals were normal

† ARDS = acute respiratory distress syndrome

Table 2. Classification of Spirometry Data in 57 Mildly Affected Subjects

	Smokers	Nonsmokers	Total (%)
Number of subjects	18	39	57
Normal study	3	10	13 (23)
Mild obstruction	1	1	2 (3)
Mild restriction	10	22	32 (56)
Moderate restriction	4	5	9 (16)
Severe restriction	0	1	1 (2)

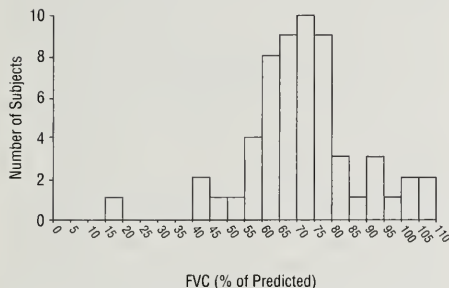


Fig 1. Distribution of forced vital capacity (FVC) (expressed as a percentage of normal predicted) in 57 mildly affected subjects.

Study of ABG analyses and spirometry performed on the 57 less severely affected subjects showed interesting findings (Tables 2 & 3). Spirometry revealed a restrictive defect in most of the subjects (Table 2 & Fig. 1). Of the 2 subjects with obstructive defects, one had an FEV₁/FVC ratio of 68% with an FEV₁ value of 63% of predicted; the other had an FEV₁/FVC ratio of 65% with an FEV₁ value of 61% of predicted. Only one of them was a smoker, with a 7 pack-year smoking history. The only subject with severe restriction, revealed with spirometry, had mild hypoxemia and did not

complain of any symptoms. The presence or severity of restriction on spirometry was not associated with any particular symptomatology or the degree of hypoxemia on ABG analysis. Chest radiographs done in all subjects with abnormalities on spirometry and/or ABG analysis were within normal limits.

We compared the variables studied in the mildly affected individuals between the smoking and nonsmoking subjects. There were 18 (32%) smokers of 1 to 40 pack-years. Although FEV₁%, FVC%, and PEF% were all lower among smokers in comparison to nonsmokers, the differences were not statistically significant (Table 3). Only the FEV₁/FVC ratio was significantly reduced in smokers ($p < 0.05$). Diagnoses obtained on analyzing spirometry data were also not significantly different for the two groups (Table 2).

ABG analysis showed mild hypoxemia in 6 (11%) and moderate hypoxemia in 3 (5%) subjects; P_{CO₂} was normal in 23 (40%) and low in 34 (60%) subjects; the latter group was diagnosed to have respiratory alkalosis. pH was normal in 48 (84%) and alkalotic in 9 (16%) subjects.

Among the mildly affected subjects, we did not find any significant difference in the ABG values or spirometry data between the symptomatic and asymptomatic subjects.

On follow-up, the patient who had recovered after mechanical ventilation was completely asymptomatic and had normal spirometry. Further evaluation for estimation of lung compliance or TLC was therefore not done. Other subjects have not reported for subsequent follow-up despite repeated reminders to the concerned authorities, presumably because the laborers in such projects usually consist of migrant population.

Table 3 Pulmonary Functions in 57 Mildly Affected Smoker and Nonsmoker Subjects (Mean, Standard Deviation, and 95% Confidence Intervals)

	Nonsmoker (n = 39)	Smoker (n = 18)	Total (n = 57)
FEV ₁ , *L	2.76 ± 0.70 (2.55 - 2.97)	2.52 ± 0.53 (2.28 - 2.76)	2.69 ± 0.66 (2.51 - 2.87)
FEV ₁ , % of predicted	82 ± 19 (76 - 88)	76 ± 16 (68 - 84)	80 ± 18 (75 - 85)
FVC, L	2.97 ± 0.81 (2.72 - 3.22)	2.80 ± 0.52 (2.28 - 2.76)	2.91 ± 0.73 (2.71 - 3.01)
FVC, % of predicted	73 ± 17 (67 - 78)	70 ± 13 (64 - 76)	72 ± 16 (68 - 76)
FEV ₁ /FVC, %†	94 ± 6 (92 - 96)	90 ± 7 (86 - 93)	93 ± 7 (91 - 95)
PEF, L/min	442 ± 105 (409 - 475)	395 ± 90 (314 - 476)	427 ± 102 (401 - 454)
PEF, % of predicted	93 ± 20 (86 - 100)	85 ± 18 (77 - 93)	91 ± 20 (86 - 96)
P _{O₂} , mm Hg	84.7 ± 15.0 (79.3 - 90.1)	82.6 ± 9.4 (76.1 - 89.1)	85.3 ± 14.3 (80.8 - 89.8)
P _{CO₂} , mm Hg	33.9 ± 3.5 (32.6 - 35.2)	33.8 ± 3.0 (33.7 - 35.9)	33.8 ± 3.3 (32.8 - 34.8)
pH	7.42 ± 0.03 (7.40 - 7.44)	7.40 ± 0.05 (7.36 - 7.44)	7.42 ± 0.04 (7.40 - 7.44)

* FEV₁ = forced expiratory volume in the first second; FVC = forced vital capacity; FEV₁/FVC = ratio of forced expiratory volume in the first second to forced vital capacity; PEF = peak expiratory flow.

† $p < 0.05$

Discussion

Acute toxic pneumonitis following an exposure to high doses of NO₂ may present as acute respiratory distress syndrome shortly after exposure,⁵ progressive breathlessness—which is generally related to an obliterative bronchiolitis²⁴—starting a week or two after exposure, or a combination of the two. NO₂ reacts with water in the respiratory tract to form nitric acid, which then dissociates into nitrates and nitrites.²⁵ These substances damage the cells of the respiratory tract by various mechanisms such as direct toxic damage, peroxidation of cell membrane lipids by free oxygen radicals,²⁶ inhibition of surfactant,²⁷ and collagen degradation.²⁸ Methemoglobinemia may also contribute to hypoxia.⁶ Because of its low solubility, NO₂ causes damage deep in the respiratory tract at the level of conducting airways and adjacent respiratory bronchioles, resulting in a noncardiogenic pulmonary edema. Patients usually present with cough (dry or productive), wheeze, dyspnea, central chest pain, sweating, and weakness a few hours after exposure.^{2,25} Examination may reveal fever, tachypnea, wheeze, and/or hypotension in severely affected individuals. Although mildly affected patients usually improve with supportive treatment and supplemental oxygen, severely affected cases may need mechanical ventilation.

Acute respiratory distress syndrome is a well-recognized complication of acute NO₂ exposure, and exposed patients usually require mechanical ventilation during the time the lung injury recovers. Despite timely intervention, the damage may still prove fatal. It is well known that both the concentration of the gas and the exposure time dictate the severity of damage.⁴ Incidentally, we did not have the actual levels of the toxic gases in the environment at the time of the accident. It was, therefore, not possible to quantify the severity of exposure in the cases reported.

The available data on pulmonary functions following acute severe exposure to NO₂ fumes is sparse. Most of this information is limited to severely ill patients requiring hospitalization, in whom both restrictive and obstructive defects have been identified.^{4,15-18} Reduction in the diffusing capacity and compliance of the lung have also been noted.¹⁵⁻¹⁷ Arterial blood gas analysis shows varying degrees of hypoxemia and respiratory alkalosis, with a widened alveolo-arterial gradient, even in less severely affected individuals.^{2,15,18} Experiments have also been conducted on healthy volunteers breathing low concentrations of NO₂ (up to 4 ppm) under controlled conditions for varying lengths of time. Most of these studies have not shown any statistically significant alteration in pulmonary functions.¹⁰⁻¹⁴

Our results indicate that both restrictive and obstructive defects may be seen in mildly affected individuals. Previous investigators have reported obstructive defects during the acute phase following severe exposures and often in nonsmokers and patients with no other underlying pulmonary disease.^{4,18} Obstructive defects and increase in airway resistance have been

demonstrated in a few studies on subjects exposed to low levels of NO₂ under controlled conditions.¹⁴ Obstructive defects in our study were observed even in nonsmokers and light smokers, implying that the results were probably independent of the smoking habit. However, the majority of patients had restrictive defects on spirometry. This could be attributed to a possible subclinical alveolitis that was reflected in the spirometry results. Estimation of TLC, or lung compliance, was, however, not carried out for the confirmation of these findings. Although the final diagnosis of a restrictive lung abnormality is based on reduction in TLC, vital capacity may be used to suspect and assess severity of restriction in situations where TLC measurements are not available.²¹ Vital capacity measurements correlate well, though not absolutely, with TLC.

Except for 3 subjects, all other mildly affected subjects (95%) were able to maintain adequate oxygenation (P_{O₂} > 60 mm Hg) on room air. We, therefore, believe that the damage at the alveolar level, if any, was little and not important enough to impair gas exchange. The hypocapnia and respiratory alkalosis may result from tachypnea and hyperventilation related to this damage.

There were no significant differences in the ABG and spirometry results between symptomatic and asymptomatic individuals. It is possible that the symptomatic individuals did not have a more severe exposure. These differences were probably only related to individual differences in symptom perception (only 5 of 57 subjects were symptomatic and the only subject with a severe restrictive defect on spirometry was asymptomatic).

A proportion of patients with severe exposure may have residual lung damage, mainly in the form of obliterative bronchiolitis, even after complete recovery from the acute event.²⁴ In the absence of adequate follow-up data, we cannot at this time predict if some of these subjects will ultimately develop a similar complication.

Conclusion

In summary, we have studied 63 patients accidentally exposed to NO₂ fumes. One patient of the 6 severely affected died. In some of the remaining 57 less affected individuals, we could demonstrate abnormalities on spirometry and ABG analysis, possibly due to subclinical damage to the lungs.

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Intractable Wheezing Due to an Obstructing Tracheal Neuroendocrine Tumor in an Adolescent with HIV Infection

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and Frederick P Ognibene MD

We describe a 13-year-old boy with human immunodeficiency virus (HIV) infection who presented with wheezing, fever, and dyspnea. Because he had a history of wheezing with a previous episode of viral pneumonia, he underwent diagnostic procedures for possible pulmonary infections and was treated for exacerbation of reactive airway disease. When he failed to respond to aggressive anti-inflammatory and bronchodilator therapy, further workup revealed an endobronchial neuroendocrine tumor occluding 75% of the trachea at the level of the carina. We describe our patient's treatment and review the literature on endobronchial lesions in HIV-infected patients. [Respir Care 1998;43(7):562-566] Key words: Human immunodeficiency virus, bronchial obstruction, reactive airways disease, endobronchial neuroendocrine tumor, pediatrics.

Introduction

The relationship between wheezing, airflow obstruction, and human immunodeficiency virus (HIV) infection is unclear. The published literature includes studies that found an association between HIV infection and evidence of wheezing or airflow obstruction¹⁻³ and studies where no increased frequency of wheezing was found in this population.⁴ Both reactive airway disease and airflow obstruction from endobronchial lesions of the airways have been described.

In immunodeficient children, reactive airway disease is felt to be common.⁵ It is postulated that frequent or recurrent infection may induce inflammation, resulting in airway hyper-responsiveness. It is also possible that altered immunity and circulating immunoglobulins contribute to airflow limitations. Ellaurie and colleagues⁶ noted that approximately 30% of

infants and children with HIV infection develop wheezing at some point during the course of their disease, particularly with episodes of *Pneumocystis carinii* pneumonia or lymphocytic interstitial pneumonitis.

We describe a case of severe wheezing and dyspnea in an adolescent with HIV infection that was due to airway obstruction caused by an undifferentiated neuroendocrine tumor. We also review the literature describing endobronchial lesions in HIV-infected patients.

Case Summary

A 13-year-old boy with transfusion-related acquired immunodeficiency syndrome (AIDS), *Mycobacterium avium-intracellulare* infection, and failure to thrive presented with wheezing, respiratory distress, and fever. He had a history of mild asthma following a viral pneumonia three years prior, that was treated with aerosolized albuterol sulfate for about one month. Until the onset of acute symptoms, the patient had not required any additional asthma therapy.

The patient was admitted to the hospital and treated for reactive airway disease with albuterol sulfate, hydration, intravenous (I.V.) methylprednisolone, and I.V. antibiotics. He responded to this treatment and was discharged within five days. Sputum was negative for *P. carinii*, respiratory syncytial

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virus, and other viral pathogens. Pulmonary function testing revealed severe obstructive disease with forced expiratory volume in the first second (FEV₁) 27% of predicted, FEV₁/forced vital capacity ratio of 29%, reduced lung volumes and diffusion capacity, and no response to bronchodilators, despite the clinical improvement observed.

The patient continued to improve until 13 days after discharge, when he presented with wheezing and emesis. Symptoms persisted despite emergency room treatment, and he was transferred to our critical care unit.

Physical examination upon admission revealed a thin male adolescent in moderate respiratory distress with prolonged expiration, suprasternal and intercostal retractions, and intermittent wheezing. The patient was afebrile. Chest radiograph demonstrated lung hyperinflation but no infiltrates. Direct laryngoscopy revealed chronic oral candidiasis but was otherwise unremarkable.

After three days of aggressive therapy in the intensive care unit with I.V. and aerosolized bronchodilators and high dose I.V. corticosteroids, blood gas analysis no longer showed evidence of hypercarbia. However, the patient continued with intermittent periods of severe respiratory distress with wheezing, retractions, and nasal flaring.

Given the lack of clinical improvement, despite intensive treatment for reactive airway disease, diagnostic options were considered. However, the patient suddenly complained of severe dyspnea, developed hypercarbia, and required urgent endotracheal intubation. After intubation the patient's wheezing resolved. Subglottic stenosis was observed during laryngoscopy for the intubation, and the etiology of the patient's wheezing was presumed to be mechanical obstruction rather than bronchospasm.

Computerized tomography (CT) of the chest and neck was

obtained (Fig. 1). Approximately 1.5 cm above the carina, a 12-mm polypoid mass was obstructing about 75% of the trachea. Patchy infiltrates were noted in the upper lobes, while the left lower lobe was completely collapsed.

The patient underwent rigid bronchoscopy and laser excision of the mass. Follow-up CT revealed residual tracheal narrowing of about 10%, resolved left lower lobe collapse, and a small amount of persistent atelectasis. Preliminary histopathologic assessment revealed superficial fungal elements. A final pathological diagnosis was not made due to tissue necrosis from the laser. The patient was discharged with the plan to closely follow the mass with serial CT and magnetic resonance imaging. Over the subsequent six weeks, the mass increased in size and repeat laser excision via bronchoscopy was required for tissue diagnosis. The final histopathologic diagnosis based on a chromogranin stain was an undifferentiated neuroendocrine tumor (Fig. 2). A total of 4180 cGy radiotherapy was administered over five days and, in conjunction with the laser excision, resulted in a considerable increase in the tracheal lumen, although some residual mass remained (Fig. 3).

Discussion

Neuroendocrine tumors are a diverse group of malignancies, such as small-cell lung carcinoma, neuroblastoma, and carcinoid tumors, that share common structural features upon examination with light microscopy.⁷ Although similar in composition, clinically these tumors grow and respond to therapy very differently. The incidence of this tumor is unknown; in fact, Lequaglie and colleagues⁸ at the National Cancer Institute of Milan reported that neuroendocrine carcinoma of the lung may be misdiagnosed or unrecognized. In 10 years of experience with 19 patients at this center, surgery was cura-

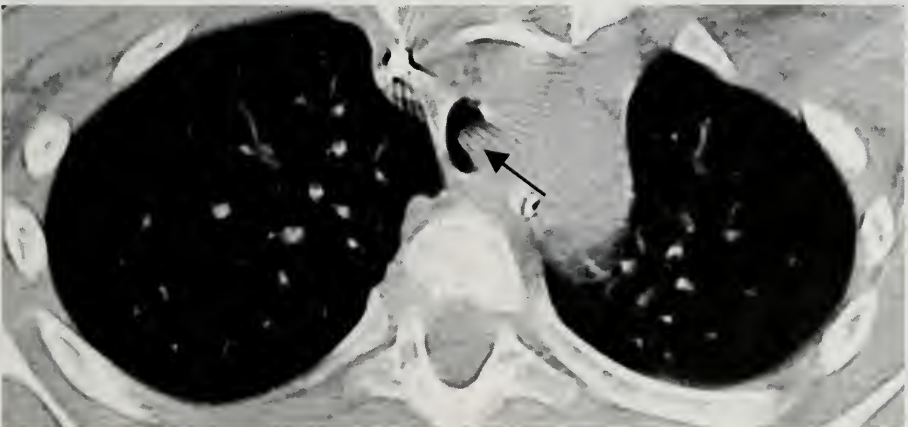


Fig. 1. Computerized tomography of the chest of the 13-year-old boy, revealing an intratracheal mass lesion (arrow) obstructing approximately 75% of the tracheal lumen.

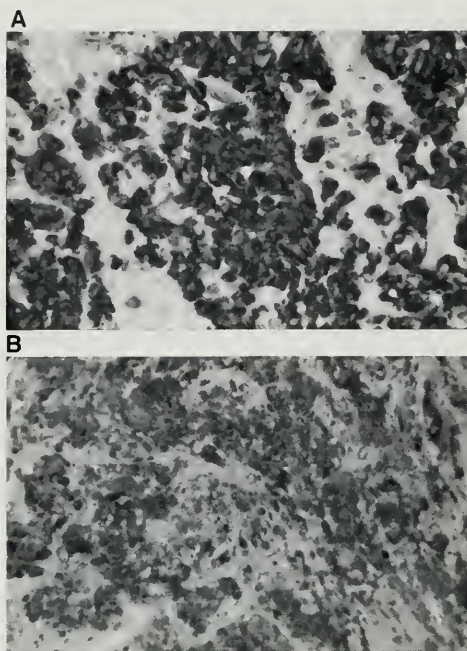


Fig. 2. A. Photomicrograph of the tracheal mass using the AE1/AE3 monoclonal antibodies stain for cytokeratin demonstrates evidence of epithelial carcinoma ($20\times \times 10$, 200-fold magnification). B. Photomicrograph of the tracheal mass using the chromogranin stain demonstrates reactive uptake of granules characteristic of neuroendocrine tumors ($20\times \times 10$, 200-fold magnification).

tive for more than half of the patients with localized disease. Some patients were treated with adjuvant chemotherapy, based on analysis of their tumors. Tumors recurred in 10 of the 19 patients, with seven of these metastases occurring in the brain. Although surgery and chemotherapy are recommended for this tumor, our patient was so clinically and nutritionally debilitated, that his oncology team did not recommend chemotherapy. Surgery was similarly ruled out because of the same clinical concerns and the questionable benefit of further tumor reduction. Radiation was therefore given as palliative therapy. Six months following completion of radiation therapy, this patient developed metastatic recurrence of his disease that was treated successfully with chemotherapy.

Endobronchial lesions are an uncommon pulmonary complication of HIV infection.^{9,10} Because patients can present with symptoms mimicking other, more frequently occurring, pulmonary complications of HIV infection, it is important for the respiratory care clinician to be aware of them. The most common endobronchial lesion associated with HIV infection is Kaposi sarcoma (KS).

KS is an AIDS-defining illness.¹¹ It typically presents with pink or red lesions of the skin or mucous membranes. The exact frequency of pulmonary KS is not known, although it is estimated to affect 8-14% of HIV-infected patients with respiratory symptoms, and up to 49% of HIV-infected patients with mucocutaneous KS.^{12,13} In pediatric patients with HIV infection, KS is rare.⁵ Although pulmonary KS in the absence of skin, mucous membrane, or lymphoid lesions has been reported, it typically occurs in the setting of widespread disease. Typical symptoms of pulmonary KS include cough and dyspnea, making it potentially difficult to distinguish from other pul-



Fig. 3. Computerized tomography of the chest of the 13-year-old boy after laser excision of the mass and five weeks of radiotherapy. Arrow indicates residual intratracheal mass.

monary manifestations of HIV infection. Furthermore, up to half of patients with pulmonary KS can have concurrent infections.¹⁴ Airway obstruction due to compression of the upper or lower airways can occur, resulting in wheezing or stridor. The prognosis of pulmonary KS is poor, and treatment with chemotherapy or radiation is palliative.

Both *Mycobacterium tuberculosis*^{15,18} and atypical *Mycobacterium* species^{19,21} with endobronchial manifestations have been reported to occur in up to 18-25% of HIV-infected patients with tuberculosis. Fever, cough, and dyspnea are common presenting symptoms. Clinically significant airflow obstruction can develop, either from compression of the airway by the endobronchial mass or swollen lymph nodes or possibly from a hypersensitivity reaction to released tubercular antigens during antituberculosis therapy.²² Treatment of the hypersensitivity reaction with corticosteroids has been successful. Endobronchial mycobacterial infections usually respond to typical antituberculosis therapy, albeit slowly. Residual airway stenosis can persist after resolution of the disease.

Endobronchial non-Hodgkin's lymphoma has been documented in two case reports.^{23,24} The exact incidence of thoracic involvement with non-Hodgkin's lymphoma is unknown, but it is thought to be rare, usually less than 10% of the HIV-infected patients who have non-Hodgkin's lymphoma.²⁴ However, the incidence of non-Hodgkin's lymphoma is thought to be increasing among HIV-infected patients, so endobronchial lymphomas may be seen more frequently in the future. Patients with endobronchial non-Hodgkin's lymphoma typically present with severe dyspnea, dysphagia, or wheezing. The prognosis of endobronchial lymphoma is poor, and treatment with chemotherapy or radiation therapy is palliative in nature.

Adenocarcinoma of the lung is another cause of endobronchial lesions. There is no strong evidence that HIV-infected individuals have an increased incidence of lung carcinoma compared to HIV seronegative patients. However, some clinicians have noticed an altered clinical presentation in HIV-infected patients, including younger age at diagnosis, more advanced disease stage at presentation, and shortened survival.^{25,27}

Pulmonary aspergillosis,²⁸ bacillary angiomatosis,²⁹ bacterial and cytomegalovirus tracheitis,¹⁰ actinomycosis,³⁰ *Rhodococcus equi*,³¹ *Pneumocystis carinii*,³² pediatric smooth muscle tumors,³³ *Nocardia asteroides*,³⁴ and granular cell myoblastoma³⁵ have also been noted to be rare causes of endobronchial lesions in some HIV-infected patients. The small number of reported patients with these atypical endobronchial lesions precludes generalizations about their association with HIV infection or their prognoses. However, many of the reported patients died within a short time of diagnosis. The patients with bacterial tracheitis and nocardiosis responded to therapy and survived to hospital discharge. The reported cases of smooth muscle tumor, bacillary angiomatosis, endobronchial *P. carinii*, pulmonary actinomycosis, and two of four cases of *Aspergillus tracheobronchitis* all failed to respond

to therapy and subsequently died. Outcomes of endobronchial *Rhodococcus equi* were not reported.

Our patient's clinical course is instructive. First, final histopathologic diagnosis revealed a very unusual etiology of an endobronchial lesion in an HIV-infected patient. The incidence of malignancy in children with HIV infection is much less than that for adults. Lymphomas and leiomyosarcomas are the two most frequently reported pediatric malignancies associated with HIV infection.³⁶ Another interesting feature of our patient's illness was his initial improvement in clinical symptoms following his first hospitalization for reactive airway disease, despite his lack of response to bronchodilators on pulmonary functions tests. The role that corticosteroids played in his recovery from this first episode is unclear. His improvement, coupled with his history of asthma led to delay in correct diagnosis and definitive therapy. Finally, unlike many other HIV-infected patients with endobronchial lesions, our patient had a good outcome, despite his metastatic recurrence. As noted, his metastases responded to chemotherapy, and he remains alive and functioning well.

Conclusion

In summary, we have presented a case of life-threatening airway obstruction caused by a neuroendocrine tumor in an adolescent with HIV infection. To our knowledge, this is a previously undescribed phenomenon. Although rare, health care providers should consider the possibility of endobronchial lesions as a cause of airflow obstruction in HIV-infected patients, particularly if there is no improvement with bronchodilator and anti-inflammatory therapy.

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Jack Emerson: Notes on His Life and Contributions to Respiratory Care

Richard D Branson RRT

If you have had the pleasure of attending the annual meeting anytime in the last couple of decades, you undoubtedly caught a glimpse of a tall, aristocratic figure hovering around the Emerson booth. That distinctive, New England, Ichabod Crane-like frame belonged to John Haven Emerson (Fig. 1). Better known as "Jack," Emerson was a pioneer in biomedical device development, with a particular emphasis on respiratory equipment. His death in February 1997 at the age of 91 brought to close a remarkable, storied, yet surprisingly quiet career in ingenious innovation.

Emerson was born February 5, 1906, in New York City to a scholarly family. He was educated in private schools. As a youngster he attended the Ethical Culture School and graduated despite lacking a few of the required studies. His son George remembers that Jack would often quip, "I never graduated from high school." Attending an Ivy League school was a tradition for Emerson children. Jack's father, Haven Emerson, was a Professor of Public Health at Columbia University in New York. In fact, I came across a manuscript by Haven Emerson, regarding artificial respiration for resuscitation.* Apparently the apple did not fall far from the tree. Jack, however, was not interested in a higher education and preferred to tinker and attempt to solve problems with the materials ready at hand.

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*Emerson HA. Artificial respiration in the treatment of edema of the lungs: a suggestion based on animal experimentation. Arch Intern Med 1909;3:368-371. Reprinted on Pages 583-584 in this issue, with permission.

Despite protests from his family, at the age of 22, Emerson purchased the rudiments of a machine shop from the estate of a local inventor. His father refused to aid in this folly, but his mother arranged for the \$1,600 necessary for the purchase. He moved this equipment to a small warehouse at 15 Brattle Street in Harvard Square and set up shop. There Emerson built research apparatus to order for professors and researchers at the prestigious schools of medicine and physiology in the

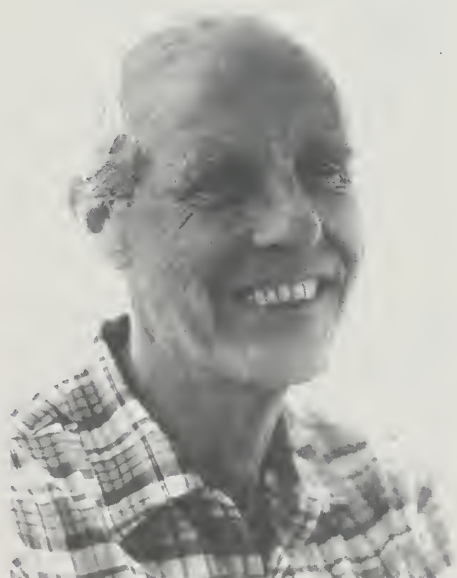


Fig. 1. John Haven "Jack" Emerson, February 5, 1906 — February 4, 1997

Boston area. It is interesting that many of his early clients were relatives who had more closely followed the Emerson family traditions.

In 1928 Emerson designed a Barcroft-Warburg apparatus for tissue respiration studies. These devices were used for studies of photosynthesis and in later years for cancer research. Essentially, this device used tissue cultures in small flasks that were agitated in a constant temperature waterbath. Gases generated during growth were measured in U-shaped manometers.

Emerson designed, in 1930, a micromanipulator for the precise movement of instruments under a microscope. This device proved invaluable in early physiology research including cell manipulation and injection. Decades later the same device would find use in the assembly of electronic parts. Emerson designed this device for his older brother Robert, who was a faculty member of the Botany Department at Harvard.

In 1931 Emerson built an oxygen tent that included an improved system for cooling the patient's environment. Antibiotic therapy was not yet available and oxygen therapy was the mainstay of treatment. Previous devices were prone to rust and failure.

Emerson is best known for the development of the iron lung during the polio epidemic of the 1930s. According to David Garrison (one of Jack's relatives and a co-worker), Emerson's father, then Health Commissioner of New York City, began to notice the escalating number of polio cases. Haven took his son aside and suggested, "If you are ever going to make an artificial respirator, now is the time." Evidently, the two had previously discussed the possibility, unbeknown to others.

At the time Drinker was the leading manufacturer of iron lungs, but Emerson's improvements to the design were classic examples of his work to come—simple, functional, and cost effective. The Drinker iron lung was developed by Philip Drinker, Louis Agassiz Shaw, and James Wilson at Harvard. Upon hearing of Emerson's dalliance into making respirators, Drinker warned Jack that he owned the patents on these devices and to expect litigation if Emerson continued to pursue this technology. What followed represents the spirit of Jack Emerson, perhaps as well as any other event. He ignored Drinker's warning and went about producing iron lungs that were quieter, more reliable, and cheaper than Drinker's. When the lawsuit arrived, Emerson and his colleagues began an exhaustive search of the engineering and medical literature from Europe and the United States. The result of that research is contained in a bright yellow pamphlet¹ available from the J H Emerson Company, containing photographs and drawings of negative pressure ventilation devices preceding Drinker's by decades. Drinker's patents were declared invalid, for want of an invention. The resourcefulness of Emerson and company produced prior artwork that proved that although Drinker may have had some excellent ideas, others had them earlier.

The Emerson design replaced blowers and valves with a flexible diaphragm. The flexible diaphragm was fashioned from elk hide in a dual layer. In this fashion, if one layer became

torn, the second redundant layer would continue operation. Emerson also improved the shape and size of the chamber by having it manufactured by a boiler company in Boston (Market Forge). The result was a quieter, simpler, lighter, and less expensive device. Although difficult to source, the Emerson device was said to cost half the price of the Drinker device. Emerson's first device was used for a polio patient at Chapin Hospital in Providence, Rhode Island. The patient had been given last rites, but survived the illness. This iron lung, affectionately referred to as "Old No. 1," now resides in the Smithsonian Institution. Emerson's improvements to the iron lung continued, and he added a quick opening and closing function, an improved pressure gauge, and emergency hand operation. Emerson's design was innovative and yet so simple, it was copied by others. His final development of the iron lung was the creation of a transparent positive pressure dome to allow ventilation when the body compartment was opened for patient care.

Like other ventilator entrepreneurs of his time (Forrest Bird and V Ray Bennett), Emerson was involved in the development of demand valves for high altitude flight and SCUBA (self-contained underwater breathing apparatus) for the Navy. In 1942 Emerson developed an automatic resuscitator, which provided alternating positive and negative pressure along with delivering oxygen. His interest in resuscitation techniques led to his formulation of the Emerson method of artificial respiration for drowning victims. This technique placed the patient in the prone position and alternately raised and lowered the patient's hips. The hipbones were grasped and lifted upward to create inspiration and drainage of fluid out of the lungs. When the hips were lowered, exhalation occurred. This method was widely used, replacing the Shafer prone pressure technique, until the introduction of mouth-to-mouth resuscitation.

In 1949 Emerson turned his attention to positive pressure devices and created a mechanical assistor for anesthesia. Using the bag-in-the-box technique, Emerson's device was triggered by patient effort, and the ventilator compressed the bag, ventilating the patient. This device was developed in concert with the anesthesia department at Harvard.

Emerson developed equipment for intermittent positive pressure breathing (IPPB), cardiopulmonary bypass equipment, hospital beds, negative pressure ventilators (pneumo-wrap), and body positioning devices. In 1955 Emerson introduced a pleural suction pump that provided continuous low-pressure suction for thoracostomy tubes in postoperative thoracic surgery. These devices were on wheels, utilized a large glass jar and a quiet, effective pump. These devices continue to be widely used and are well known as Emerson Postop Pumps in surgery departments around the world.

In 1957 Emerson built a volume plethysmograph for Dr Jere Mead at Massachusetts General Hospital for the measurement of residual lung volume. Mead was a preeminent pulmonary physiologist of his time. This device was later adapted for the measurement of other lung volumes. At Mead's

encouragement, Emerson also developed the first "deep breath" modification for negative pressure ventilation, a predecessor of the sigh breath. Mead's idea was first suggested by Vissscher, who believed a periodic deep breath would restore lung compliance.

In 1964 Emerson built one of the early volume ventilators. This simple device resembled a green washing machine and used a piston to deliver precise volumes. Oxygen was added into a 'trombone-shaped' accumulator connected to the intake of the piston for delivery of elevated F_{IO_2} . The tidal volume was changed by a crank on the front of the machine, which controlled the stroke of the piston. Respiratory rate and inspiratory-to-expiratory-time ratio (I:E) were adjustable. The humidifier was a modified pressure cooker and was known as the Emerson Hot Pot. A belt, connected to a DC motor and pulley wheel, served to move the piston. In case of failure of the existing belt, a spare was hung inside each cabinet. The belts were similar to those used to circulate air in forced air gas furnaces in homes. On numerous occasions I have heard the story of the belt becoming loose or breaking and the spare found to be missing. Under these circumstances, the resourceful respiratory therapist would run to the parking lot and obtain the belt from a Volkswagen Beetle (the old one) and place it in the Emerson to restore it to working order. I've never looked to see whether the two belt sizes are compatible because it's such a good story. In any event, the Emerson Postop Volume Ventilator was reliable and would allow ventilation of patients when other devices failed. Emerson's device was not the first of the piston ventilators (Mörch and Engstrom preceded him), but it was the first device to allow independent control of I:E.

For the intermittent mechanical ventilation (IMV) Emerson used continuous flow IMV and a unique water column PEEP (positive end-expiratory pressure) valve to allow successful use of IMV and a very low work of breathing. During the introduction of microprocessor ventilators with demand valves, numerous investigators compared the work of breathing of the new devices to the "Emerson" gold standard. In fact, the IMV champions of the 1970s were all great supporters of Emerson ventilators, because the work of breathing was low and the possibility of successful application of the technique maximized.

In 1954 Emerson was intrigued with the idea of a dog's ability to ventilate normally during panting. He developed a device for 'vibrating' the patient's airway. His patent² for this device, No. 2,918,917: *Apparatus for Vibrating Portions of a Patient's Airway*, issued in 1959 made several unique observations.

This invention pertains to an apparatus for treating a patient by vibrating a column of gas which is in communication with his airway at a rate which is greater than the patient's normal rate—from 100 to 1,500 vibrations per minute—vibrating the col-

umn of gas doubtless causes the gas to diffuse more rapidly within the airway and therefore aiding in breathing function by circulating the gas more thoroughly to and from the walls of the lungs.

The initial device used a reciprocating diaphragm, similar to the high-frequency oscillation devices used today. A continuous flow of air was provided from a blower and directed into the inspiratory circuit. The diaphragm was connected to a shaft, which was attached to a pulley. A second electric motor turned the pulley by means of a belt. As the pulley turned, the shaft 'vibrated' the diaphragm. In the 1970s Emerson experimented with a series of high-frequency devices, settling on a device that did not incorporate a diaphragm. The final high frequency device was simple and functional. A high-pressure gas source was delivered to a rotating ball. The ball had a hole drilled through the center to allow the passage of gas into the patient circuit. The faster the ball would spin, the higher the frequency. As the spinning speed was reduced, volume through the hole and duration of flow (% inspiratory time) increased. This device is frequently referred to as a High-Frequency Flow Interrupter (HFFI), owing to the mechanical design (Fig. 2).

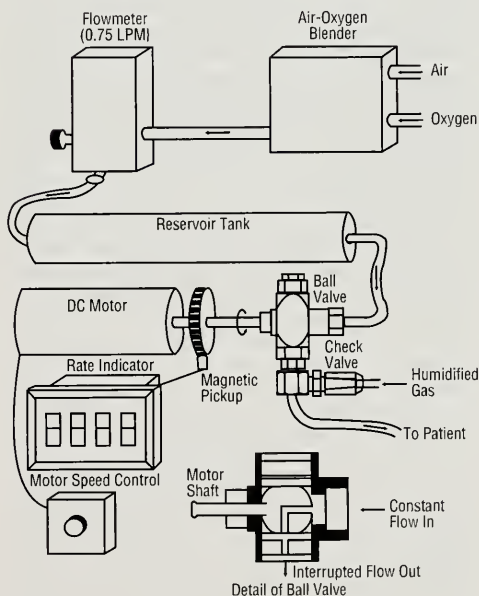


Fig. 2. Schematic of the Emerson High-Frequency Flow Interrupter used for high-frequency positive pressure ventilation.

Several authors reported successful use of this device in the 1980s.³⁻⁷

Throughout his lifetime, Emerson had long-standing relationships with the leaders in respiratory care, critical care, surgery, anesthesia, and pulmonary medicine. Alvin Barach was a close Emerson colleague and was instrumental in development of the "In-Exsufflator Cough Machine," a device gaining new acceptance now as a method of secretion removal in patients with neuromuscular disease. Many of you may recognize this device by its earlier name, the name given it upon its initial introduction, "The Cofflator." The Cofflator was Barach's invention and, despite his influence, never really caught on with clinicians. The In-Exsufflator improved on the idea and provided the flows required to aid in secretion removal. Emerson also developed Barach's walking cane, which contained 50 L of oxygen in an unobtrusive hollow cane.

I was introduced to Jack Emerson by Forrest Bird and enjoyed many conversations with Mr Emerson over the past decade. Despite his advancing age, Emerson was always patient and willing to discuss any matter of interest. He would always surprise me by reaching into his worn briefcase and pulling out an original manuscript or conference proceeding from the 1940s or 1950s. These would often have his handwritten notes and those of others (Barach, Mead, and others) in the margin. He would often produce this as evidence that all the new techniques we were talking about at the meeting had been done many times before. In the last years of his life, he would frequently lament over the state of relations between the government, clinicians, and manufacturers. He would produce the 1955 proceedings⁸ on the state-of-the-art conference on negative pressure ventilation and suggest that cooperation between the groups was a key to early success in mechanical ventilation. Emerson liked to reminisce about the days when a physician could call for a needed piece of equipment, and he could create the device in a couple of days and bring it in for a patient trial. In fact, the very first Emerson iron lung was never tested on anyone but Jack Emerson himself, before going on a patient. Perhaps this could be considered an Emerson innovation as well, true quality control.

This issue of RESPIRATORY CARE contains several pieces as a tribute to the life of a man who saved and changed the lives of others. I did not know Mr Emerson well, but he usually remembered my name and was always congenial and enlightening. The respect he garnered from Forrest Bird—who called him Jack, but always answered his questions with 'sir'—provided me some insight into the magnitude of his stature. A lot can be learned about a person by observing the respect shown them by leaders in their field. I was fortunate enough to have a photograph taken with Jack Emerson and Forrest Bird during the American Association for Respiratory Care meeting in Las Vegas in 1994 (Fig. 3); an enlarged version graces the wall of my office.

I do not know how Mr Emerson would have felt about this issue of the Journal. He was quiet and self-effacing. He didn't



Fig. 3. From left: John Haven Emerson, Richard D Branson RRT, and Forrest M Bird during the 1994 American Association for Respiratory Care annual meeting in Las Vegas.

seem to care much for attention or honors. He was like his devices—simple, functional, and reliable. The role Jack Emerson has played in the history of biomedical engineering has touched the lives of hundreds of thousands of people on both sides of his equipment. His entrepreneurial spirit and simple, innovative genius will be sorely missed in this microprocessor world.

ACKNOWLEDGMENT

This piece could not have been written without the kind cooperation and assistance of George Emerson. I am grateful for his honesty and hope that this issue of the Journal will invoke fond memories of his father.

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— Foreword —

Some Reflections on the Man Behind the Machines

From its origins in applying bellows to resuscitate drowning victims in the 1760s, the field of mechanical ventilation has advanced because clinical needs have been identified and then met by clever inventors and engineers, whose technical and engineering expertise engendered new and better machines. No exception to this pattern, the history of modern mechanical ventilation has been shaped by several creative innovators whose names are emblazoned on the equipment we commonly use today, for example, Bennett, Bird, Engstrom, and Emerson.

The field lost one of these innovators this past year—John Haven “Jack” Emerson—and it seems fitting for RESPIRATORY CARE to commemorate his career by publishing his own reflections on the contributions he made to the fields of mechanical ventilation and to respiratory care. The accompanying article is a transcript of a lecture that Emerson delivered to the Massachusetts General Hospital Department of Anesthesia Critical Care group in 1985. At that time, we invited Mr Emerson to address this group regarding these reflections.

A few words of explanation are needed for the reader. First, this lecture was invited as an informal seminar, accompanied by a few of Emerson’s selected slides (some of which are reproduced as figures). The article is a transcript of a lecture and therefore reflects the folksy spontaneity of conversation rather than the polish of a chapter-ready manuscript. Indeed, the editors have purposely avoided rigorous editing in order to preserve the folksy affability that was an endearing trait of Jack Emerson.

Also, Mr Emerson names specific persons in his lecture and we have done our best to transcribe the names accurately, while recognizing that the 13-year-old recording leaves room for errors in this regard. Most of the names of the questioners could not be deciphered.

Finally, I would like to add a personal introductory note. As a Fellow in Critical Care at the Massachusetts General Hospital then, I had the privilege of inviting and introducing Jack Emerson. I distinctly remember the self-effacing enthusiasm with which he agreed to deliver the lecture. My experience of inviting and hearing the lecture was framed by two main feelings: first, awe at meeting the man behind the machines that we used daily in our intensive care units then and second, reverence for the sense of craftsmanship that Jack Emerson exuded as he described a career of building sturdy, practical, life-saving machines. Overall, I believe that this article will pay tribute to Jack Emerson by putting his own words before the readership of RESPIRATORY CARE and, in so doing, celebrate thoughtful innovation and craftsmanship—values that will surely enhance our own practices of respiratory care.

James K Stoller MD
Associate Editor
RESPIRATORY CARE

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Some Reflections on Iron Lungs and Other Inventions

John H Emerson

Transcribed from the videotaped lecture given by Mr Emerson during a meeting of the Department of Anesthesia Critical Care at Massachusetts General Hospital in 1985.

Thank you very much for asking me. I hope I can tell you some things you'll be interested in. Now, particularly, I gather you'd like to know how our ventilator came about. If I put these slides through, I might put them through right in the beginning, quickly—just so you can see how I got started.

I was from New York, and I didn't do very well in school at all. I never graduated from high school. My family sent me up here, because everybody went to Harvard or Radcliffe. That was traditional in my family. In fact, my father told me if I didn't go, I'd be the first Emerson since they landed on the shores of Ipswich—but that's me! My brother, who was in plant physiology at Harvard, got me a job in the physics lab. I swept floors and learned how to oil lathes and machine stuff.

Then next year I got a job at the medical school trying to make very, very fine fibers for a cousin of mine, Alexander Forbes, for a string galvanometer. He was trying to make one-centimeter-long fibers, gold plated, of quartz for a galvanometer, a special galvanometer he was having made, studying nerve action currents, which they were not able to study in those days, because the galvanometers wouldn't respond fast enough. So, if you had it short and small, and they wanted a half a μ —you know how big that is—you look at a spider web, and then you reduce it. You could hardly see these darned things! And I found a way to make them for him. Took me about a year, and they used to blow quartz in a room that was all covered with felt, and they'd pick them up off the wall, these little things. I had a bunch of things that spun around like that, and I wound them up.

Anyway, I decided then that, well, my brother being a plant physiologist, studied chlorophyll—what do they call it?—photosynthesis was his specialty, and he was trying to get pure cultures, and I developed a micromanipulator for him to get pure cultures, which is still on the market. In fact, there was a guy came to my shop very recently. He'd gone all over Europe, went to Zeiss and Leitz and came back here and some-

body said, why don't you come over to our place? And he bought the manipulator that I had made for my brother. It's different in that instead of having things that go that way and that way, to move that way and that way, I had a lever like an airplane control—no matter which way you moved the handle, it moved the needle that you were controlling, very little, in the opposite direction. It reversed it, so under a microscope, it was erected. This has been a useful instrument for people. In fact, talking about nerve action currents, it was used, a bunch of them together, at Boston University, way back then for tracing the fibers out of a cat's brain to find out what controlled what. See, that's the kind of thing they were doing in those days, and they used my manipulator.

Anyway, let's go quickly through this, and then I'll tell you. There's the oxygen tent I told you about, which I made for James Wilson. He was a resident at Children's Hospital. Let's see now, it was probably '29 or so that I made that. He had a milk pail that he bought at Sears Roebuck. There was a brand new Sears Roebuck store, that one that's over there. He used the milk pail and put a motor under it. The trouble was that—you put ice in that thing—and the motor was underneath to circulate the air to the tent. Only trouble was they were made of 'tinned' steel and in a very short time the salt and ice rusted the steel tank out that dripped on the motor, and it wasn't a good arrangement. So, we made one out of copper for him, and he wanted something that wasn't as cumbersome as Barach's tent for pediatrics.

OK, let's just go quickly. All right, here's the iron lung. Now, the same Jim Wilson, (I've just got to go a little into how I got into this) Jim Wilson then asked me if I had any ideas on how to improve the Drinker respirator (Fig. 1). That was a new thing, and they were selling them for around \$3,600. It was a rectangular tank affair with thick walls, because they were flat walls. They had valves, a motor to drive valves, and a blower to change the pressures. So that it made an awful lot of noise.

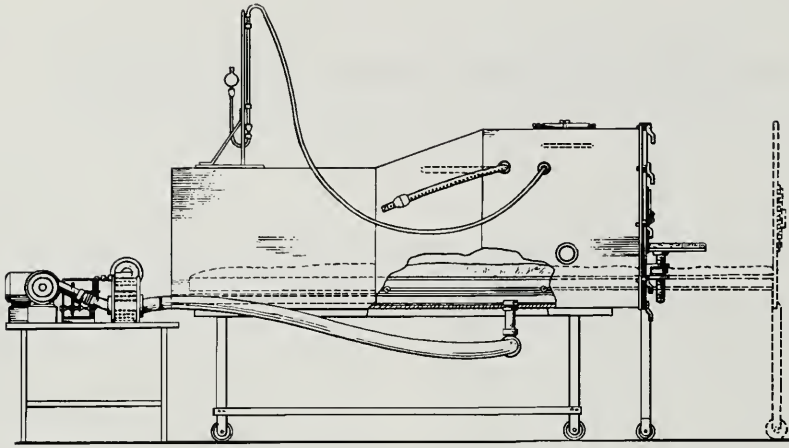


Fig. 1. The first "iron lung" to receive widespread use was developed in 1928 in Boston, and was patented by Philip Drinker and Dr Louis Agassiz Shaw. It was cumbersome and inconvenient but saved a number of lives. It had a sliding bed with head-wall attached, and a rubber collar. Pressure changes were created by a rotary blower and an alternating valve. During a polio epidemic the Consolidated Gas Company of New York paid for building large numbers of these respirators, and reports of their use spread quickly around the world. Reprinted, with permission of JH Emerson Co.

I told him I thought that a simple diaphragm would be better. I made a box that size, which I took to Children's Hospital recently to show them. I still have the box—a very simple thing with a big piece of leather [from] a car seat that I got from my junkyard. That's still in good condition, incidentally. You push the handle up and down and it would change the pressure in the box. Well, I took this thing over to Children's, and I gave it to Jim Wilson, and he said, "Could I keep it and take it to Drinker?" And Drinker said, "That won't work. We thought of that." And so they gave it back to me.

Well, I talked to my father about this ... (this was in '30-'31), and about maybe a month after, I got a short letter from my father. Now my father was the head of public health at Columbia. He simply said (he was an epidemiologist) and he said, "We're heading for a bad epidemic of polio this year. If you're ever going to try that idea of yours, now's the time to do it."

Well, the epidemic came pretty fast and so I went across the river here [to] Robertson Boiler Works, and I said (I had no drawings, absolutely nothing), I just went into that place and said, "Oh, make me a round tank about so big, well, 28 inches, maybe, in diameter, and put some legs on it. We'll take it from there." After I picked that tank up, in two weeks we had this thing completed (Fig. 2). That machine, I got into it and got a bad cold (I mean a sore throat) from one night. It worked, you see! I took [the tank] down and showed it for my father at Willard Parker and one other hospital, and he told me to stop at Rhode Island on the way back, at their conta-

gious hospital. I put it in there late in the afternoon and they told me, "Well, we have a patient. We have no machines." They had four Drinkers in use. You couldn't hear yourself think in the place it was so noisy. [The patient] was a young priest. They said, "He'll die at night. We have no way to ... we have no place to ..." We tried—so, we put the patient in—it worked perfectly. It ran for six months. The patient lived, and we decided we'd try and make iron lungs.

I asked the Boiler Works to make five more tanks. News got around here in Boston that I was doing this. And I decided that I would try to show it at the American Hospital show, which was coming up in Toronto. So, I had a call from Drinker. No! He came into my shop and told me if I tried to make this iron lung he'd put me out of business. He had patents coming up. Well, in those days, you know, doctors didn't patent things much (and especially life-saving things). It made us mad, anyway, to be threatened like that. But we went ahead and completed the machine and took the second machine to Toronto. We got it completed two days before the meeting, spent one day—no we started in the night, driving to Toronto with that iron lung across the back seat of a Dodge Touring car with the top down. As we went through Albany, it stuck out so far that my co-helper, Mr Garrison, was driving and the headrest knocked over some of these street signs.

We made it to Toronto. We set up this booth. The Drinker respirator was there. We were offering [ours] at \$1,000. All I can say is at the end of that meeting, everybody knew that I had the machine that was going to be used! This hospital



Fig. 2. Because of a severe poliomyelitis epidemic in 1931 John Haven Emerson of Cambridge, U.S.A., built a simplified respirator. It cost less than half as much as others, but contained many improvements. It operated quietly, using a bellows to create the changes of pressure (as Woillez's did, but with a motor added). A wide range of speeds was instantly available. Opening and closing were rapid and convenient. It could be pumped by hand if electricity failed. The first Emerson "iron lung" is now preserved in a glass case in the United States National Museum (the Smithsonian Institution) as the prototype of respirators constructed since 1931 in American and Europe. Reprinted, with permission of JH Emerson Co.

[Massachusetts General Hospital] ordered one on the spot. You had one of the first that I built. There was a hassle, you know. Harvard was mad. There was a lot of back and forth. Dean Edsel asked me not to advertise for six months, which I agreed to. And in six months, the new Drinker came out with all the five features that had made mine good, without my advertising. Anyway, we survived that, and that's just a smattering of what happened.

So, let's go faster. That's a little one. You see how simple it could be. Just change the pressure inside by moving that handle. OK.

Now, in '36, there was an epidemic of flu, and Ralph Trabell was at Hartford. He wanted something better than the bubbling bottles. So, you remember for nasal oxygen they had these bottles just to—they were a tube under water, they would bubble. I wanted to split the bubbles up smaller. I got two bearings—from a Ford generator—that are porous bronze, compressed. I pushed the air through that, and I got little bubbles, like that. That was the first of a kind of humidifier where you broke the bubbles up real small. That was for Ralph Trabell, OK.

We did make one of the early IPPV [intermittent positive pressure ventilation] machines, too, and particularly for the Brigham, what was his name, [the] anesthesiologist there? What? Derek! Bill Derek. Bill Derek. We made an anesthesia machine ... a patient started to breathe, and it would assist them, and they tell me that it was the first with an anesthesia bag—you could encase or open the front of this thing and squeeze

the bag by hand. That was in—well—later. Let's go on.

This is the chest respirator with a cage, which I made for bronchoscopy, for one thing, for the Long Island College Hospital. Anyway, I guess you know pretty much about that. Next.

This machine, you'd think maybe was an iron lung, but this is for Al Barach. This is one of the most sophisticated things we ever made. That was for treating tuberculosis, and the patient was completely enclosed. The Swedes developed this. A fellow named Thunberg, but he just put the patients completely in the tank and changed the pressures up and down quite rapidly and got ventilation by compression and rarefaction. If you press here on your chest, and bring equal pressure inside, the lungs won't move. But you can ventilate if you get enough difference in pressure. This thing worked, but for closing (what do you call them?) the holes in the lungs, but we must have made 60 of those, and just at that time the sulfa drugs were coming, so that was out the window. Next.

Oh, there's Derek's thing, see? '53 again. That's Derek himself and the bag, and you could open that and squeeze the bag if you wanted to. It was an assisting anesthesia machine. Next.

Incidentally, we made the first hyperbaric tank in the U.S. That was for Presbyterian Hospital in New York. They used that for a good many years. I went over to England and saw what they were doing over there, and then made this tank. Next.

You all know about that. One of the chest surgeons came to my shop a long time ago and, well, it was way before that, Glover and O'Neil, that was one of the teams in Philadelphia.

He said, "We're trying to expand the chest after our surgery with something they called a Stedman pump. I [used] 15 pumps and I still can't get the lungs to come up." I said, "You're doing it the wrong way." In two weeks or so I fixed him a little vacuum cleaner affair with variable speed. We were in business making these things and everyone uses them. Next.

This was for Jere Mead at the School of Public Health. His volume rather than the pressure plethysmograph, and a lot of people bought these for research. Next.

Al Barach—he wanted oxygen for his patients. When they went out walking, they wanted the security of having oxygen. And he wanted a walking cane for them to ... that's a walking cane with oxygen in it. It had 50 L in it. The FDA [Food and Drug Administration] didn't want it sold, because they'd set a rule that nothing less than 70 liters—you know, it all depends on—he found it useful for somebody trying to get up a few steps at their home. Anyway, let's go on. Next.

There's the belt we were talking of, see, that Barach was using. You could cycle it, or the patient could cycle it. Next.

This old ... would be related to the stories I'm telling you—(that's my wife)—and it is a good way for manual ventilation—it was a lot better than what was being used. I proposed the use of lifting the hips because you stretch the rib cage that way. You make it like a bellows, you see, instead of just pushing on the back the way the Schäfer prone pressure worked, which was the standard way of rescue in those days. Next.

That's the resuscitator we made. That again. Next. And that's the house that I traveled and sold iron lungs out of. I bought that on Commonwealth Avenue from [the] General Electric Company. They'd used it to sell kitchens. See the gen-

erators up in front? As I drove, I'd charge batteries, and then I'd come into a town. I could run the iron lung that I had in the back, and the town would go through and see the iron lung they were going to spend their money for. Next.

That's the lung now. Next.

Now, two more. I tell you, I'm going to skip these. No, just go one after the other. Yeah. The last one was a French iron lung (Fig. 3), which saved the day in the lawsuit for us. We found the one at the bottom there, and here I have the actual picture. But the thing that saved the day was this hospital [Massachusetts General Hospital] forming, because you had a library, we came here. We found references to negative pressure cabinets from the world over! Everyone there had thought that Drinker was the only thing, see. And we found people that had this idea in many countries, a good many people in this country. So, this Frenchman had a real cute idea. Instead of a pressure gauge to tell how well he was ventilating, [he used] a glass tube with a rod in it, and if the chest went up and down, he could see how much he was ventilating. Very direct. Anyway. Let's go. Next.

There's a French one (Fig. 4)—worked with a steam boiler. You shift the valve and it would ... and the Venturi. Next.

Just run them through. This was in Vienna (Fig. 5). Vienna again. This is England (Fig. 6). All these things, now, this is 1905 this thing was made in Tennessee (Fig. 7). Nashville. OK. This one and the next one were made in Massachusetts, or designed. OK. This was in South Africa, and the fellow had a patient and built on the spot an iron lung for himself and saved his case. Right on the spot! OK.

This was a plethysmograph that recorded. You could breathe

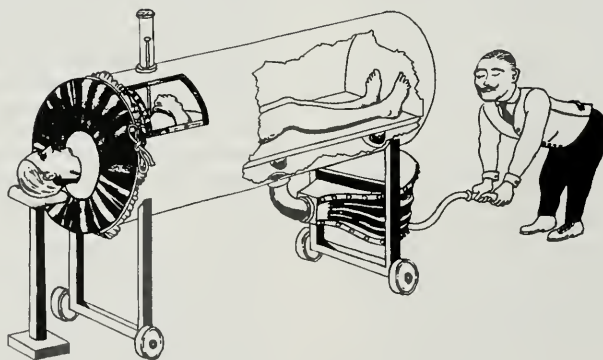


Fig. 3. In 1876 Dr Woillez of Paris built the first workable iron lung, which he called a "spirophore." It had the basic elements of modern respirators, including an adjustable rubber collar and a sliding bed. A unique feature was a rod which rested lightly on the patient's chest, to give visual proof of actual lung expansion. In a brilliant lecture presented before the Academy of Medicine on June 20, Woillez showed a thorough understanding of the physiology and mechanics of artificial respiration. He refused to patent his invention. A colleague suggested placing spirophores all along the Seine, for drowning rescues, but finances for the public service were lacking. (This illustration was reconstructed by Maxfield Parrish Jr for a legal battle ...) Reprinted, with permission of JH Emerson Co.

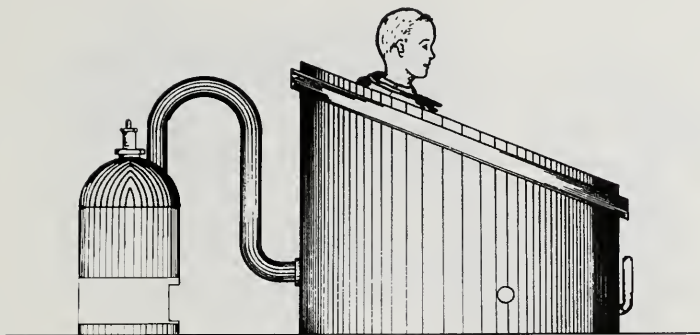


Fig. 4. Dr Charles Breuillard of Paris patented a "bath cabinet" type of respirator in 1887. For a source of vacuum he recommended "a steam ejector fed by a steam boiler ... heated by a spirit lamp." The patient himself was supposed to operate a valve, alternately connecting the cabinet with the vacuum, for inhalation, and with the atmosphere, for exhalation. Breuillard also described a chest respiratory "cuirass" to be operated in the same, and a face mask. Reprinted, with permission of JH Emerson Co.

dogs with it. All these were negative pressure cabinets. Go ahead.

This is do-it-yourself in Germany. OK.

And that's the Drinker, you see, with the pump and stuff and the rectangular tank. That was what was on the market. Next.

That's a room they had in Children's Hospital (Fig. 8). Five patients were taken at the same time. Negative pressure. OK.

Now, we found that Alexander Graham Bell had actually made one that was in the museum up in Canada. I believe we found reports that he came down to this hospital and tested it. I think he used to come to this hospital. OK. I have some infor-

mation about that. [Responding to an unintelligible comment from the floor.] There it is. The thing to the left is what he called the jacket. See, it's a pot tank concealed up here and down here. And that's the pump. See the bellows? OK. That's it.

I'm out of breath from trying to rush. I probably shouldn't have tried the pictures. [Question from the floor about development of positive-pressure ventilators.] Well, let's see what I can give you on how the ventilator came about.

I guess it's related to polio. Actually, I think around '52 or something like that, they had an epidemic in Denmark and there weren't enough iron lungs. They couldn't possibly get

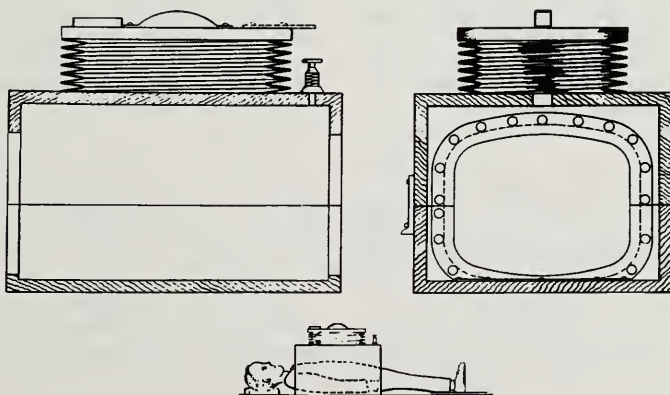


Fig. 5. In 1901 Rudolf Eisenmenger of Piski, Hungary, patented a portable respirator which consisted of a "simple, two-part box" enclosing only the patient's chest and abdomen. Later he became medical professor in Vienna, and there continued to improve his invention. He stressed the importance of access to the patient's throat and limbs, of portability, and of hand-operation. (Motors were also mentioned.) There are reports of "extraordinary success" with Eisenmenger's respirators. Reprinted, with permission of JH Emerson Co.

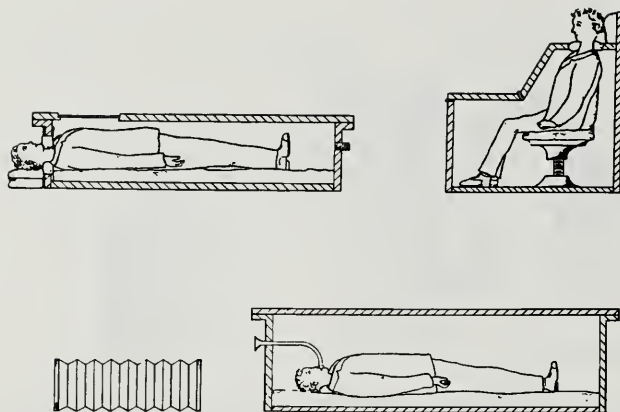


Fig. 6. William Davenport of London understood the mechanics of artificial respiration clearly. His patent in 1905 mentions a box, a rubber collar, and a simple bellows or piston pump. He lacked the sliding bed (of Woillez and modern iron lungs) but made several good suggestions, including the supplementary use of oxygen. He proposed several types, including a "collapsible form ... to facilitate transit." Reprinted, with permission of JH Emerson Co.

enough iron lungs. Any of you heard this story? You know about it? They got all the anesthesia machines they could find in the countryside and set up students squeezing anesthesia bags and kept those patients going, and they survived! A lot of them. Their survival was as good, I guess, as the iron lung cases' survival, so (I guess I'm not very polite this morning), but that brought a flood of anesthesiologists from Denmark over here. I think one of them is your Dr Pontoppidan. Dr Rotenberg is another one. Mörch is another. I guess there were maybe a dozen [who] got over here to show us how they'd done things.

Mörch was in Chicago at the University of Chicago and Cook County Hospital. He made a piston machine. As a matter of fact, just before Mörch, of course, it was the Engstrom. Maybe they were both around the same time, but they were

both piston machines. They were equal cycle, in and out, and Don Benson worked with Mörch on his machine. His, incidentally, went under the bed. Mörch's was a low machine, about that size and so high. And the drive was horizontal, so the whole thing could shove under the bed where you couldn't see it. Then he had a tube up to the patient, and an exhalation valve, which was a big steel ball that would pop up and let the patient exhale.

Anyway, Benson moved to Hopkins and he made himself a piston machine that he hoped would have better humidity, because that was one of the problems with the machine that Mörch had made. They still weren't trying to use very high pressures, because everyone thought you shouldn't use pressures above, oh, 20-25 cm H₂O. He had a water kettle and heated it. Then the gas that came out of the water kettle [passed

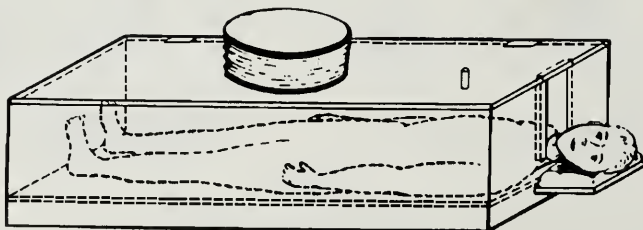


Fig. 7. Dr Charles Morgan Hammond of Memphis, U.S.A., built his first "artificial lung" in 1905 and a series of improved models throughout the next 20 years. He performed experiments and treated patients successfully, but his respirators were not produced commercially and remained unknown to the public for many years. Reprinted, with permission of JH Emerson Co.

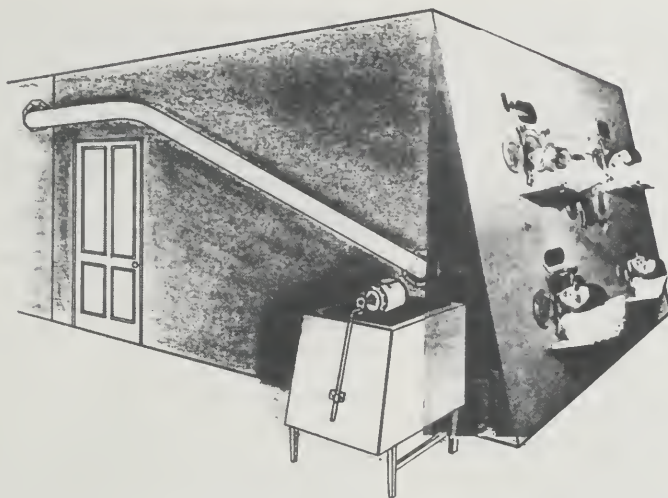


Fig. 8. In the early respirators it was difficult to give complete nursing care and to change the patients' position frequently. This care was found to be of life-saving importance, so Dr James L Wilson asked for a room in which several patients could be made to breathe simultaneously. A nurse could enter by the door and perform all procedures efficiently. The room pictured was built at the Children's Hospital in Boston, U.S.A., and was used successfully during several epidemics. Reprinted, with permission of JH Emerson Co.

through] this copper wool which you've seen in our machines. First, the main reason he put the wool in was because it was conductive. It would conduct the heat farther up the line, closer to the patient, and keep the humidity high and warm. He couldn't get any of the big manufacturers interested in making this thing for him, so one day I got a call from him. He wanted to know if I was interested, and I said "Surely." I went down to see him, and he told me why he built it and said he thought the people would want this thing.

So, we went back and worked on it for a year. I was involved in making underwater swimming stuff for the Navy, and I'd done that because the iron lungs had dropped out of the picture, pretty much, because of the vaccine. So it took us about a year to get a machine that we thought was about right. Benson wanted equal inhalation/exhalation time. He said he didn't want to worry about trying to have that variable. It was just a crank and a piston. Just as I was almost ready to take it down and show it to Benson, I get a call from Pontoppidan. Well, he'd been working here on a piston. I think he had Harvard Apparatus [a company at Harvard] get some electronic circuits made so that he could have I-E ratio variable. We knew from the polio days that with some of their cases, they wished they could have it not equal. So, I realized it was an important thing. We decided we'd hold up and try to add Pontoppidan's on top of what we made for Benson. That was the way the 3-PV came about.

You see, all the things I've done have been because doctors have come to me with something they were trying to do.

We didn't set out and say, "Now we're going to get in this field and make this." It's because of what doctors asked me for. That's the way my business is run.

Now, the next step I want to explain to you is why you're doing a lot of things you're doing now for rescue—the cardiac resuscitation you're doing as opposed to what was being done back then. In those days, they used Schäfer prone pressure for breathing, and a few people were questioning that. Researchers were saying, "Well, it doesn't even move the dead space. How can you ventilate a patient that way?" So, at any rate, the Army had a problem with chemical casualties. A friend of mine, Jim Elam, an anesthesiologist, also, I think he was in Chicago, for his tour with the army, he went down to Edgewood Chemical Center with a Dr Clements (you've probably heard of Clements—he's now at the University of California at San Francisco) to try to find out how to save these chemical casualties, and the way they started, he asked if I'd let them have a resuscitator.

Now, our resuscitator was pressure-limited. Went up to 18-cm positive pressure, and it went down to a negative phase of 9 cm, and went back and forth. Six months later he called me up and said, "We aren't getting anywhere. Those dogs all die. We give them the gas and we can't resuscitate them. I wonder whether you could make that thing go to any higher pressures." I said, "Sure." He said, "Well, make one that goes to 50 for me." And the dogs all lived! This was supposed to be a pressure that was dangerous. Of course everyone understands it now, but they didn't then. That if the patient's stiff

enough, it's perfectly safe to put the high pressure on.

So a big meeting was called down there, and for one thing they decided that they ought to throw out the Schäfer prone pressure, and Elam and Clements were advocating mouth-to-mouth, because that's about all you'd have out on the battlefield, and you could generate enough pressure to save these cases, they felt. So, the meeting, I think NIH [National Institutes of Health] held it in Washington, and Dr Whittenberger was chairman. Now, Whittenberger was at the School of Public Health here. He used to visit our place up in Essex with his family. One day, just before the meeting, he said to me, "You know, a dog when he pants, he doesn't move more than a seventh (I think he said a seventh) of his dead air space. But the dog lives. Maybe it isn't as serious as they're trying to say about the Schäfer."

That triggered something with me. It got me thinking about high frequency, about rapidly vibrating. I got so excited about that that I got a patent back then. I guess you know I got a patent around 1955. I made a machine to do this, but I couldn't really get anyone too much interested in it. But that was the reason, and people ask me why I did this, you see.

The other thing that was going on down there in Baltimore, of course, you know about Kouwenhoven, an electrical engineer at Hopkins. He was having trouble with the doctors, too. I mean, I don't know why I say "too," but ... he would go to meetings, showing how he could push on the sternum of a dog and circulate blood. Up to that time, the doctors had been—if you had to try to start the heart up—they'd been chopping you open and grabbing your heart and squeezing it. Now, these two things were put together, and that's what makes your present cardiopulmonary resuscitation.

The next addition to our machine, we went down to Gainesville in Florida, and Dr Downs and Dr Kirby ... I'm going to back up a little bit, because I believe that before that, again in this hospital, they were trying to get heat, and we made some things for them to retard inhalation, but the people in Gainesville were using what they called 'constant positive pressure.' Now, I go to my father's thing, which Barach said to him was the classic paper on CPAP [continuous positive airway pressure]. Alvin Barach had been trying to get people to see the value of positive pressure, constant positive pressure. In 1940, well, he wrote a lot about it, but I think my father's paper,* well, it was the earliest thing he could find where the suggestion of raising the pressure somewhat could be beneficial.

Anyway, Downs and Kirby had our ventilators down there, but they wanted, they were trying to get manufacturers to put constant positive pressure, be able to raise the pressure. CPAP they called it. Puritan Bennett wasn't interested. They said,

"We're selling our machines. We don't care about it ... " Forrest Bird was working on it for Downs and Kirby, and so, of course, he had it. He made the one for Kirby for babies, but was unable to come up at that time with one for adults. And so, that's when they came and said "Can't you make this machine to be positive?" So, the next step was adding that to our machine. Of course, our machine was far better suited for it than any other machines there were on the market, because they all had pumps that they can shove gas through so they're small piping systems, and our machine was already made so you could breathe very, very freely, and I think that's one of the great benefits—I guess you all know that—of our simple machine that we have, ventilator. So we added that on to the machine, and now I don't know what else there is on it. Kirk, you tell me.

Kirk: Well, there's updated alarm systems.

Emerson: Oh, alarms, I haven't talked about. I had the first patent on the alarm for a ventilator, I believe, on the iron lung, and I had a lot of misgivings about it, and I still have a lot of misgivings. To tell the truth, I think you've got your ventilators so complex that there's a lot of trouble ahead. Do many of you people know about the present ... problems [of a ventilator manufacturer]? You do? Well, they are the little piston machines. They've jammed [the components] in so small and tried to add so much into them, that they can't keep them running. Now, the big machines, of course, I think we still have the most reliable breathing machine or ventilator on the market. We had troubles, but they've got so much electronic stuff that it's prone to breaking down, and then they try to make up for it by putting a whole bunch of alarms on, and before you get through, it's a bunch of alarms and machines that just isn't reliable. I don't know how far you go. I've been on the standards committees and they're going to demand more and more sophistication. I think they're going to go on getting into more and more trouble. For instance, [for] home care, what [their ventilator] is for, they say ours is definitely the most reliable by far, our big machine, but they can't drag that into a home. It's too big, you see. We haven't really tried to make it much smaller.

Anyway, I thought I might see if anyone had any questions, anything anybody would like to ask me about the history that I might know about.

Unidentified Member of the Audience: We took your machine into a home in Somerville to help a child, who—when this child would go to sleep at night—would stop breathing. This child's had this machine in the home for the past 13 years, and I remember going out there 13 years ago, to this old rickety house, dark night over there, and going into this home and the machine was there next to a wardrobe next to the bed, and this child was being ventilated. The mother called me because apparently the child was getting a little blue at night,

* Emerson HA. Artificial respiration in the treatment of edema of the lungs: a suggestion based on animal experimentation. *Arch Intern Med* 1909;3:368-371. Reprinted on Pages 583-584 in this issue, with permission.

and she thought the machine may need some adjustment. While I was watching the child being ventilated, lying there very quietly, this child was about a year of age, I saw the tubes from the child's tracheostomy to the machine wobbling in the breeze. I had no idea why, because I wasn't touching the tubes. I looked down and I saw the family cat there. He was clawing at the tubes. And I was trying to picture this scenario in the home with my intensive care unit ... in Burnam Six. It is really doing a remarkable job, and this child is 13 years of age and has lived at home on this respirator and is a thriving child. He goes to a normal school.

Emerson: Well, Children's in Philadelphia sent many children home on our machine, and also Children's in Chicago, and maybe you have, I don't know. This Children's has here. One of the real problems now with this [manufacturer's] machine, though, is that, for instance, Dr Goldberg at Children's in Chicago has all these people at home. He's pushed to get standards on these things, because there have been so many problems. Now the FDA has closed the door to this company—won't even let them sell parts. I can't understand that, because I don't know how they'll resolve it, but here they have all these patients with machines that are breaking down, what do you do? Anybody else got any questions on the history?

Stoller: Well, what's on the horizon? I mean, given the fact that things are getting more and more complicated and probably should get less and less complicated, how's it going to go? What do you think?

Emerson: Well, I think they are going to find uses for high frequency. Is anything happening with that here?

Unidentified: Not a lot, no.

Emerson: Not even with the pediatrics?

Unidentified: They want to use it down in lithotripsy ... to minimize the movement of the patient who's being treated with the lithotripsy.

Emerson: That's really sort of a problem, that is high frequency, because the FDA says you can't sell a machine that goes faster than 150 [breaths per minute]. And that isn't really high frequency, you see. And so everybody's confused. They don't know what they're talking about. High frequency really should be substantially higher than breathing rates, and Kirk and you people have used our standard ventilator at 150 right here in the hospital, and people aren't calling that high frequency.

I've made two types of very simple high frequency machines. One of them is an interrupting affair, which they use at Children's successfully. And quite a few other places—down at Hershey Medical Center and Orlando General Hos-

pital they've been using it. They've been doing it, in fact, the Critical Care show—this last one—had an exhibit on what they're doing down at the hospital in Orlando.

Now, the other one is an oscillator, which is what I described originally, the motion down, just a diaphragm or a piston. They used that successfully at San Diego Children's ... Mannio and ... Kopotic ... Now the NIH has finally come around—Mary Ellen Avery, because of the success they had at Children's, was pushing to have a test, an NIH-run test. NIH has set up to run the test and they asked all the manufacturers to bring machines down to Miami a year ago, and they were going to rush through getting this machine, 100 machines, putting them in 10 hospitals, and finding out what happened if they put the machine on every other baby that needed to be ventilated in the first 24 hours. Well, there were about six manufacturers, and they chose a machine that had not been made yet. I think it's absolutely outrageous what they did. Getting a machine called the Hummingbird that [a company in Japan] agreed to make. They hadn't made it yet for babies.

On Tuesday I'm going out to a meeting in Salt Lake. Dr Harris, a neonatologist at Temple, runs these meetings. They're going to have a discussion of this. I guess the NIH is going to be there and try to explain what they're doing—why they've done this. Again, Kopotic set up with our simple oscillator, it couldn't be simpler. And incidentally, right after the decision to get this Japanese machine—the cost is going to be \$30,000 per machine—you know, they really don't care. Anyway, I'm going to go out there and see what happens. I'm going to show my machine. You know, they want to have it there, and Kopotic and Mannio are probably going to describe the thing. But right after their decision, I got orders from three good California hospitals, Stanford, Sunshine—Dr Sunshine and Loma Linda—and the University of California at Irvine where Dr Whittenberger is, and Kopotic was going to show them how to use them. I don't know how they made out with them. I'll find out, probably, at this meeting.

Unidentified: Two questions. The first one: in a very limited sense, we could use on occasion in the operating room a high-frequency ventilator with which you can also give anesthesia. I don't know if there are any on the market, but people have tried to put them together, but that's not currently available, as I understand it, for us to use. We have a high frequency ventilator in the O.R.—a small box.

Emerson: Well, it isn't really high frequency. It doesn't go over 150.

Unidentified: I don't think so ... it's a jet ventilator. It works for the short term, but we have no ability to give anesthesia.

Emerson: In fact, they've been doing that this way, the same darned thing. I may come to do that. Well, if Kirk can tell us

what you want, I made one which they wanted, the same thing in Florida over at Clearwater, and I made it for them, and I got it back here. It's hard to work with somebody in Clearwater. It's too far away. If you want to try one of those slow kinds of things, now I've made—see, with FDA, you can't do these things anymore.

Unidentified: That was my second question, actually. Could you give us, as you've gone through the history of what you've done—you went through it very quickly and it sounded a lot easier than it probably was—but in today's times versus in the 30s and 40s, the bureaucracy is so deep.

Emerson: It's terrible. Look, I built my iron lung essentially in two weeks. I slept in it one night, and put a patient in it. And it worked, see! I don't think that the FDA should have been given the authority by Congress or whatever it is to put the identical rules that they have for drugs. I think there should be some control, but I don't think that they are equivalent, you see. I think the mechanical thing, you know better what it's going to do, but it's very difficult to do; in fact, I don't know, I often think of going out of business. Of course, I have the same trouble all the doctors have of lawsuits and stuff. Life is different. We're going to go on, till we get put out of business.

Unidentified: It seems that there are a lot of things that are written that say that the nondemand valve ventilators are very good, but one thing I've noticed is that some of the other ones are much prettier than yours. How much is appearance important in your sales or in sales of ventilators?

Emerson: Oh, I hear a lot of that.

Unidentified: I think the Emerson is pretty.

Emerson: I don't know what to say about that. I know the old green machine looked like a household appliance. But it worked.

Unidentified: Do you find that the hospitals would get rid of those, even if they were still working, to get a new machine?

Emerson: All right, that brings up another story. Who's going to ... He isn't here anymore? Who's going to Cleveland Clinic?

Stoller: Oh, I am.

Emerson: Oh, there you are! I was looking for you.

Stoller: I had to sit down. I was going to ...

Emerson: Who came from this hospital and took over the anesthesiology department at Barnes?

Stoller: Bill Owens.

Emerson: Bill Owens! I was trying to think of his name. Well it seems to me that maybe the same thing's going to happen at Cleveland Clinic that happened at Barnes. Bill Owens trained here, right? And he went down there and he threw all their MA-1's out for his intensive care unit. He went down to the cellar and found about 18 of our machines that they had discarded. He brought them up, and they did a show themselves. They painted them; they looked like Rolls Royces. They have padded covers that go over them. They're beautiful! And that's what they use in their intensive care. Now, I understand that in Cleveland Clinic, they had an anesthesiologist who used our machine for all their anesthesia for their heart surgery. They simply fed anesthetic gas into it. And then he left. They threw them all out. It's an awfully inefficient system, I must say. Maybe you've got someone here who's going to ...

Stoller: I'll give you an update.

Emerson: So, anymore questions?

Unidentified: Was James Wilson the same James Wilson who went to Michigan as chairman of pediatrics?

Emerson: Absolutely wonderful guy! Yeah. He got me started on the iron lung. I think I've gone over time.

Artificial Respiration in the Treatment of Edema of the Lungs: A Suggestion Based on Animal Experimentation

Haven Emerson AM MD
New York

On three separate occasions, in 1906, 1907 and 1908, while demonstrating the effects of extreme peripheral resistance on the heart and pulmonary circulation, I have noticed a definite result of artificial respiration when administered to an animal apparently dying from acute pulmonary edema.

The physical causes of the benefit apparently derived from this procedure seem to agree so well with facts already accepted in physiology, and the possibility of application of the method in certain kinds of clinical cases seems so reasonable, that I offer this communication in the hope that practical tests may, before long, be sufficiently conclusive to establish its value therapeutically, or to relegate it to the mass of theories that have failed.

It will save time if I call attention to a few points regarding the effect of respiration on the circulation. The respiratory fluctuations in blood pressure which anyone can appreciate in the radial pulse are due to the variation in the ease of passage of blood between the right and left side of the heart and to the inherent elasticity of the lungs. The expansion of the lungs allows a wider path for the blood and an increase in the blood in the pulmonary vessels, and at the same moment a diminished resistance to the passage of the blood through the lungs, a lessened burden for the right ventricle. When the lungs collapse in expiration, the elastic recoil empties the pulmonary vessels, and at the same time narrows the path through which the right ventricle must now pump the blood. So we find in the last two thirds of inspiration and the first third of expiration a rising pressure, the remainder of the respiratory cycle showing a falling pressure.

If we watch the results of positive pressure respiration properly applied, we notice an entire reversal of the blood pressure changes above described. During the inspiratory phase, which is due to the forcing of air into the lungs under posi-

tive pressure, the normal conditions in the chest and in the pulmonary spaces are altered. The positive pressure exerted on the vessels in the lungs tends to empty them, or at least to obstruct their lumen, by just the amount of pressure exerted. The small vessels are squeezed, as it were, against the resistant pulmonary tissue, by air forced into the terminal vesicles through the trachea. During the expiratory phase the release from positive pressure permits a filling of the vessels again and a diminished resistance to the passage of blood from the right to the left heart. So it will be found that during positive pressure respiration, the so-called artificial respiration of laboratory procedure, the blood pressure falls during inspiration and rises during expiration.

For our present purposes the important thing to bear in mind is that rhythmical variation of pressure, applied at any point of the circulation, will serve to assist in the onward movement of the blood, and will in proportion to its extent assist the action of the heart. It has been found possible to continue a circulation of the blood simply by artificial respiration in an animal in which the heart is no longer capable of contracting, the valves allowing an onward movement with each inspiratory phase and preventing any regurgitation to fill the vessels during expiration.

If we modify the procedure of Professor Leo Loeb, who first called my attention to the use of adrenalin to cause edema of the lungs, we can develop gradually an acute cardiac insufficiency. Massive and repeated doses of adrenalin given intravenously in a cat will produce acute dilatation of the left ventricle, due to sudden and extreme constriction of all the systemic arteries. The dilatation of the left ventricle allows of a mitral regurgitation, an acute congestion of the lungs and a dilatation and failure of the right heart. The inability of the right ventricle to force the blood received from the auricle against the back pressure of blood regurgitating from the left auricle allows of increase in the stagnation of the pulmonary circulation. Edema—that is, a collection of blood serum in the air spaces of the lungs—occurs, increasing until pink or clear serous frothy fluid appears in the trachea. Respiratory movements become exaggerated and later feeble and spasmodic, and the animal will presently die of asphyxia due to a flood-

From the Department of Physiology of the College of Physicians and Surgeons of Columbia University in the City of New York. Read at the meeting of the Section on Medicine of the New York Academy of Medicine, March 16, 1909.

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ing of the air spaces of the lungs by blood serum.

If, when we find respiration showing definite signs of beginning asphyxia, when the veins are becoming distended and deepened in color, cardiac insufficiency is established and the incompetency is increasing, and when we can hear moist râles over the lungs, and when we know that cardiac insufficiency is established and the incompetency is increasing, we then apply artificial respiration through the tracheotomy tube, gently distending the lungs and allowing them to collapse with or without suction, we shall find presently an amelioration in the animal's condition. The full expansion of the lungs, due to distention from within, forces a considerable amount of blood onward to the left auricle, and as the respiratory phase extends over two or three heart beats, an increased amount of blood will have passed the mitral valve and there will be more room in the pulmonary vessels when expiration occurs for the blood held in the distended right ventricle, and a diminished resistance in the lungs against which the right ventricle can now successfully empty itself.

This at least seems the probable explanation for the improvement in the circulation which presently occurs. The lungs appear free from moist râles, the heart beats more vigorously, the distention of right and left side diminishes and when the artificial respiration is discontinued after about half an hour the animal is able to breathe normally and shows none of the signs of insufficient circulation or respiration. The effect of the adrenalin has worn off, the heart muscle has recovered from its acute overloading, the pulmonary circuit is no longer engorged with regurgitated blood, and to all intents and purposes the heart and lungs are again performing their functions normally.

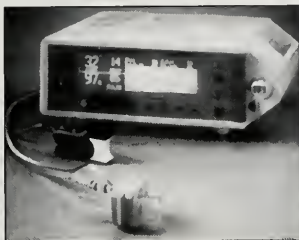
The bearing of this purely experimental procedure on the individual case of edema of the lungs in the human subject may not appear quite clear, and I shall try to point out the conditions in which I believe this lesson can be applied with advantage.

In many instances a hypertrophied and properly compensating heart, which has adjusted itself gradually to a valvular defect or to an increasing inelasticity of the arteries or per-

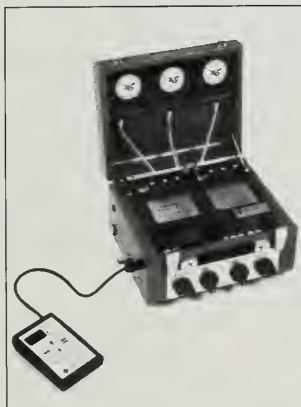
sistent increase of peripheral resistance from any one of a number of causes, will, if a sudden strain is put on it, develop an acute incompetence. Overexertion physically, overindulgence in food or wine, excess of psychical excitement or an unfortunate combination of all three, or an attack of contracted arteries or bronchi may be the determining factor. With a heart just able to maintain its competence under favorable conditions, even if it is not the seat of myocardial degeneration, insufficiency is easily precipitated and pulmonary edema is likely to be developed unless the failing heart action is of very brief duration. Under such conditions as I have above described, I believe it would be a valuable aid to the necessary medication if artificial respiratory movements were used. With the patient in the semirecumbent position, which is usually assumed when cardiac dyspnea is marked, raising the arms above the head and then pressing them against the sides of the thorax or, better, across the upper part of the abdomen, ought to establish the accessory pumping action which, under normal conditions, facilitates the flow of blood through the lungs, but which the patient, in his enfeebled condition, is unable to do for himself. This assistance, I believe, should prove more prompt and effective than any medication, and would at least be giving mechanical relief to the overloaded heart muscle, while arterial relaxation and cardiac stimulation are being accomplished by drugs. I think such treatment would be indicated whenever the edema and cardiac incompetence are of sudden development and are due to causes which are likely to prove of brief duration or can be removed by appropriate treatment. Edema, when due to cardiac failure in the course of pneumonia or appearing as the inevitable terminal feature of a chronic endocarditis, could not be expected to respond to such temporary relief as artificial respiration would offer. Moreover, I hope I shall not be misunderstood as advocating forced respiration by intubation or tracheotomy, for I certainly think such measures would be quite unjustifiable. My belief, based on experimental observations, is that artificial respiratory movements, directed to establishing a rhythmical expansion and contraction of the thorax, are worthy of clinical trial in cases of acute cardiac insufficiency accompanied by edema of the lungs.

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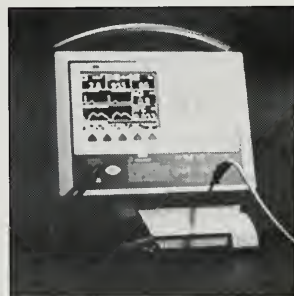


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A. Patient information

1. Patient identifier	2. Age at time of event: or Date of birth:	3. Sex <input type="checkbox"/> female <input type="checkbox"/> male	4. Weight ____ lbs or ____ kgs
In confidence			

B. Adverse event or product problem

1. <input type="checkbox"/> Adverse event and/or <input type="checkbox"/> Product problem (e.g., defects/malfunctions)	
2. Outcomes attributed to adverse event (check all that apply)	
<input type="checkbox"/> death (mo/day/yr)	<input type="checkbox"/> disability
<input type="checkbox"/> life-threatening	<input type="checkbox"/> congenital anomaly
<input type="checkbox"/> hospitalization - initial or prolonged	<input type="checkbox"/> required intervention to prevent permanent impairment/damage
	<input type="checkbox"/> other: _____
3. Date of event (mo/day/yr)	4. Date of this report (mo/day/yr)
5. Describe event or problem	

6. Relevant tests/laboratory data, including dates

7. Other relevant history, including preexisting medical conditions (e.g., allergies, race, pregnancy, smoking and alcohol use, hepatic/renal dysfunction, etc.)

C. Suspect medication(s)

1. Name (give labeled strength & mfr/labeler, if known)	
#1 _____	
#2 _____	
2. Dose, frequency & route used	3. Therapy dates (if unknown, give duration) from/to (or best estimate)
#1 _____	#1 _____
#2 _____	#2 _____
4. Diagnosis for use (indication)	5. Event abated after use stopped or dose reduced
#1 _____	#1 <input type="checkbox"/> yes <input type="checkbox"/> no <input type="checkbox"/> doesn't apply
#2 _____	#2 <input type="checkbox"/> yes <input type="checkbox"/> no <input type="checkbox"/> doesn't apply
6. Lot # (if known)	7. Exp. date (if known)
#1 _____	#1 _____
#2 _____	#2 _____
8. Event reappeared after reintroduction	
#1 <input type="checkbox"/> yes <input type="checkbox"/> no <input type="checkbox"/> doesn't apply	
#2 <input type="checkbox"/> yes <input type="checkbox"/> no <input type="checkbox"/> doesn't apply	
9. NDC # (for product problems only)	
- -	
10. Concomitant medical products and therapy dates (exclude treatment of event)	

D. Suspect medical device

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

E. Reporter (see confidentiality section on back)

1. Name & address		phone #
2. Health professional?	3. Occupation	4. Also reported to
<input type="checkbox"/> yes <input type="checkbox"/> no		<input type="checkbox"/> manufacturer <input type="checkbox"/> user facility <input type="checkbox"/> distributor
5. If you do NOT want your identity disclosed to the manufacturer, place an "X" in this box. <input type="checkbox"/>		

ADVICE ABOUT VOLUNTARY REPORTING

Report experiences with:

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

Report **SERIOUS** adverse events. An event is serious when the patient outcome is:

- death
- life-threatening (real risk of dying)
- hospitalization (initial or prolonged)
- disability (significant, persistent or permanent)
- congenital anomaly
- required intervention to prevent permanent impairment or damage

Report even if:

- you're not certain the product caused the event
- you don't have all the details

Report product problems – quality, performance or safety concerns such as:

- suspected contamination
- questionable stability
- defective components
- poor packaging or labeling
- therapeutic failures

How to report:

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

Important numbers:

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

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

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Case Report: A report of a clinical case that is uncommon, or was managed in a new way, or is exceptionally instructive. All authors must be associated with the case. A case-managing physician must either be an author or furnish a letter approving the manuscript. Its components are Title Page, Abstract, Introduction, Case Summary, Discussion, References, Tables, Figures, and Figure Captions.

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

Graphics Corner: A brief case report incorporating waveforms for monitoring or diagnosis—with Questions, Answers, and Discussion.

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

PFT Corner: Like Blood Gas Corner, but involving pulmonary function tests.

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Bunch D. Establishing a national database for home care. *AARC Times* 1991;15(Mar):61,62,64.

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American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. *Respir Care* 1988;33(11):1044-1046.

Article in journal supplement: (Journals differ in their methods of numbering and identifying supplements. Supply sufficient information to promote retrieval.)

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

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

Editorial in journal:

Enright P. Can we relax during spirometry? (editorial). *Am Rev Respir Dis* 1993;148(2):274.

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Letter in journal:

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Paper accepted but not yet published:

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Personal author book: (For any book, specific pages should be cited whenever possible.)

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

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☐ 2 years ☐ Other, specify _____

Expected Date of Graduation (REQUIRED INFORMATION)

Month _____ Year _____



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- ☐ High School
- ☐ RC Graduate Technician
- ☐ Associate Degree
- ☐ Bachelor's Degree
- ☐ Master's Degree
- ☐ Doctorate Degree

Number of Years in Respiratory Care

- ☐ 0-2 years
- ☐ 3-5 years
- ☐ 6-10 years
- ☐ 11-15 Years
- ☐ 16 years or more

Job Status

- ☐ Full Time
- ☐ Part Time

Credentials

- ☐ RRT
- ☐ CRIT
- ☐ Physician
- ☐ CRNA
- ☐ RN
- ☐ LVN/LPN
- ☐ CPFT
- ☐ RPFT
- ☐ Perinatal/Pediatric

Salary

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- ☐ \$20,001-\$30,000
- ☐ \$30,001-\$40,000
- ☐ \$40,000 or more

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Date _____

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Specialty Sections

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

- ☐ Adult Acute Care Section \$15.00
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AARC & AFFILIATES

July 17-19—American Association for Respiratory Care

1998 Summer Forum at the Registry Resort, Naples, Florida. Sessions will cover education and management, plus additional topics including "Technology and Credentialing," and "The Lewin Group Report: Implications for the Respiratory Care Community." Following the Summer Forum, a Spirometry Workshop will be conducted at the same facility July 19-20; and participants can earn seven hours of CRCE credit.

CRCE: Up to 20 hours of continuing respiratory care education (CRCE) credits.
Contact: The AARC at (972) 243-2272.

July 20-21—Nevada Society

Annual Conference and Business Meeting at the Atlantis Hotel Casino in Reno, Nevada.

CRCE: 10 CRCE credits have been requested.

Contact: John Steinmetz, 181 Brooks Circle, Sparks NV 89431, (702) 331-0721

August 5-7—TriState Respiratory Care Conference

27th annual meeting—Changing Times: A Continuation—at the Grand Casino Biloxi Bayview Hotel, Biloxi, Mississippi.

CRCE: 14 hours; nursing units have also been approved.

Contact: Rocco Tretola, (504) 482-3530, e-mail corr@ochsner.org, or visit the web site at <http://members.aol.com/akramer259/documents>.

August 14—Live Teleconference

"Care Plans: Developing, Implementing, and Measuring Outcomes," Part 6 of the AARC's 1998 "Professor's Rounds," from 12:30-2 pm Eastern Time.

Contact: The AARC at (972) 243-2272.

August 21—Ohio Society

Independence, Ohio
Critical Care Committee announces its annual seminar to be held at the Holiday Inn Rockside Road, Independence, Ohio.
CRCE: 6 hours.

Contact: Nancy Johnson, (330) 929-7166 or abbyru@aol.com.

September 11-13—AARC Patient Assessment Course

Chicago, Illinois

The course will be offered at the Clarion International Quality Inn at O'Hare, Chicago, Illinois.

CRCE: 16 hours are available.

Contact: Preregistration is required, call (972) 243-2272.

September 16-18—Maryland/District of Columbia Society

18th Annual Conference by the Sea at the Sheraton in Ocean City, Maryland. Two certification programs, "Advanced Respiratory Skills for Skilled and Subacute Care" and "Patient Assessment," will also be offered.

Contact: Jeanette Ledbetter at (202) 574-6348.

October 8—New York

Society—Southeastern Chapter

30th Annual Symposium at the Marriott Marquis in Manhattan, New York, New York. Featured speaker is AARC

Executive Director Sam Giordano, MBA, RRT, speaking on "The RCP: Practice in the 21st Century." Also, the NYSSRC and SUNY Stony Brook will be hosting a clinical assessment workshop Oct. 9-10 at the Cornell Club in Manhattan.

Contact: (516) 444-3181 or www.nyssrc.org.

October 9—Southern Chapter of the Colorado Society

Fifth Annual Southern Colorado Pulmonary Medicine Symposium at the Holiday Inn Garden of the Gods, Colorado Springs, Colorado.

Contact: Cathy Fletchall at (719) 776-5025 or Barry Beard at (719) 776-5212.

November 7-10—International Respiratory Congress

The American Association for Respiratory Care hosts its 44th annual International Respiratory Congress at the Georgia World Congress Center in Atlanta, Georgia. More than 7,000 people will experience programs appealing to all levels of health care providers—from clinicians to managers and administrators, to manufacturers and distributors of equipment and supplies. Program content will include neonatal, pediatric, and adult critical care; acute, continuing and rehabilitative care; diagnostics; management; and case and disease

management—truly a comprehensive program on respiratory care. Exhibits by all manufacturers of cardiopulmonary equipment in the world will be featured.
Contact: For program brochure and registration information, contact the AARC, 11030 Ables Lane, Dallas TX 75229-4593; (972) 243-2272; fax (972) 484-2720; e-mail: meetings@aacrc.org; or visit the web site at www.aarc.org.

OTHER MEETINGS

August-December—National Subacute Care Association

Seven, 2-day, regional seminars, covering Minimum Data Set (MDS) to classify patients in the Resource Utilization Group System (RUG). These will be held in Chicago, Illinois; Hartford, Connecticut; Philadelphia, Pennsylvania; Dallas, Texas; Seattle, Washington; Los Angeles, California; and Orlando, Florida.
Contact: (301) 961-8680.

December 9-11—Diagnosis and Treatment of Sleep Breathing Disorders

An international conference that will feature 50 speakers from all over the world with all sessions translated either into English or French, Grenoble, France. Abstracts for presentation are being sought and are due Oct. 1.

Contact: Congress Secretariat at ADTSAS, Hôpital de la Croix Rousse, Service de Réanimation et d' Assistance Respiratoire, Hôpital de la Croix Rousse, 93 Grande Rue de la Croix Rousse, 69317 Lyon Cedex 04, France. Phone 33 (0)4 76 76 55 16, fax 33 (0)4 76 76 56 17, e-mail Patrick.levy@imag.fr.

June 12-16, 1999—International Society for Aerosols in Medicine

12th International Congress at the Austria Center in Vienna, Austria.
Contact: Vienna Academy of Postgraduate Medical Education and Research, Alser Strasse 4, A-1090 Vienna, Austria. Phone (+43/1) 405 13 83-22, fax (+43/1) 405 13 83-23. E-mail medacad@via.at.



Helpful Web Sites

American Association for Respiratory Care

<http://www.aarc.org>

- Current job listings
- American Respiratory Care Foundation fellowships, grants, & awards
- Clinical Practice Guidelines

National Board for Respiratory Care

<http://www.nbrc.org>

RESPIRATORY CARE online

<http://www.rcjournal.com>

- 1997 Subject and Author Indexes
- Contact the editorial staff

The American College of Chest Physicians

<http://www.chestnet.org>

The National Board for Respiratory Care—1998 Examination Dates and Fees

Examination	Examination Date	Examination Fee
CRTT Examination	November 14, 1998	\$100 (new applicant)*
	Application Deadline: September 1, 1998	60 (reapplicant)*
RRT Examination	December 5, 1998	250 Both (new applicant)
	Application Deadline: August 1, 1998	210 Both (reapplicant)
RPFT Examination	December 5, 1998	180 (new applicant)
	Application Deadline: September 1, 1998	150 (reapplicant)

*In 1999, this examination fee will increase by \$20.

For information about other services or fees, write to the National Board for Respiratory Care, 8310 Nieman Road, Lenexa KS 66214, or call (913) 599-4200, FAX (913) 541-0156, or e-mail: nbrc-info@nbrc.org



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New Federal Register Notices Now Available

The Center for Devices and Radiological Health announces the publication of new Facts-on-Demand FOD notices in the *Federal Register*. The new publications: FOD#774—Medical Devices; Preemption of State Product Liability Claims; Proposed Rule; FOD#607—Rebuilders, Reconditioners, Services, and "As Is" Remarketers of Medical Devices; Review and Revision of Compliance Policy Guides and Regulatory Requirements; Request for Comments and Information; Proposed Rule; and FOD#513—Medical Devices; Reports of Corrections and Removals; Stay of Effective Date of Information Collection Requirements; Stay of Effective Date of Final Regulation. For more information about Facts-on-Demand call (800) 899-0381 or (301) 826-0111. The FOD system is also on the Internet at www.fda.gov/cdrh/fedregin.html.

NAMES 1998 Education, Conference Schedule Set

The National Association for Medical Equipment Services (NAMES) announces its 1998 national conferences and regional education seminars. For information about upcoming events, call the NAMES Education & Meeting Department at (703) 836-6263, or visit the web site: www.names.org.

Web Site Link to Fellowships, Scholarships, & Grants

The American Association for Respiratory Care's web site contains important information about fellowships, scholarships, and research grants. International fellowships, education scholarships, research fellowships, and other grand programs are described in detail. The site also contains information about the \$1,000,000 Research Fund, a restricted fund to sponsor research initiatives that document the clinical and economic impact of respiratory care professionals in the delivery of health care. To apply, a "Research Plan Abstract" must be submitted to the AARC by October 1, 1998. To find out more about these programs, log on at www.aarc.org.

Year 2000 Date Problem Addressed by FDA

On June 24, 1998, the Food and Drug Administration announced the availability of the document, "Guidance on FDA's Expectations of Medical Device Manufacturers Concerning the Year 2000 Date Problem." The document is available via telephone (800) 899-0381 or (301) 827-0111 or via the Internet at www.fda.gov/cdrh/yr2000/y2kguide.html.

Consumer Information Catalog Available

The United States General Services Administration provides a catalog of consumer information that may be helpful to health care providers. For a copy of the catalog, write to R Woods, CIC - 8A, P O Box 100, Pueblo CO 81002, or call (719) 948-4000, or visit the Internet web site at www.pueblo.gsa.gov.

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\$7; add \$9 for air mail postage to overseas countries. Checks should be made payable to *RESPIRATORY CARE* and sent to the subscription office at 11030 Ables Lane, Dallas TX 75229-4593.

SUBSCRIPTION RATES FOR ASSOCIATIONS. An association may offer individual subscriptions of *RESPIRATORY CARE* to its members at a reduced rate. The rates based on membership are: \$8.50 per year for 101-500 members; \$8 for 501-1,500 members; \$7.50 for 1,501-10,000 members; \$6.50 for more than 10,000 members. For information, contact Ray Masferrer at (972) 243-2272.

CHANGE OF ADDRESS. Notify the AARC at (972) 243-2272 as soon as possible of any change in address. Note the subscription number (from the mailing label) and your name, old address, and new address. Allow 6 weeks for the change. To avoid charges for replacement copies of missed issues, requests must be made within 60 days in the US and 90 days in other countries.

MANUSCRIPTS. The Journal publishes clinical studies, method/device evaluations, reviews, and other materials related to cardiopulmonary medicine and research. Manuscripts may be submitted to the Editorial Office, *RESPIRATORY CARE*, 600 Ninth Avenue, Suite 702, Seattle WA 98104. Instructions for authors are printed in every issue. An expanded version of the Instructions is available from the editorial office.

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