Effects of Bolus Normal-Saline Instillation

Transtracheal Gas Administration and the Perception of Dyspnea

Adult Disposable Resuscitators: Variables Affecting Volume

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Please see following page for brief summary of Prescribing Information.
Alupent
(metaproterenol sulfate)

Inhalation Aerosol 10 mL*

*10 mL; 15 mg per mL (each metered dose delivers 0.65 mg metaproterenol sulfate)

Bronchodilator

Brief Summary of Prescribing Information

CONTRAINDICATIONS Use in patients with cardiac arrhythmias associated with tachycardia is contraindicated.

Although rare, immediate hypersensitivity reactions can occur. Therefore, Alupent® (metaproterenol sulfate USP) Inhalation Aerosol is contraindicated in patients with a history of hypersensitivity to any of its components.

WARNINGS Paroxysms of asthma attacks may be precipitated by sudden discontinuation of a bronchodilator or by noncompliance to the prescribed regimen. The use of Alupent® (metaproterenol sulfate USP) Inhalation Aerosol should be limited to patients who have shown the necessity of such therapy, and who have had prior adequate trials of less potent bronchodilator agents. The use of Alupent® (metaproterenol sulfate USP) Inhalation Aerosol should not be extended by the physician beyond the specific circumstances for which it is indicated.

Adverse drug reactions associated with the use of Alupent® (metaproterenol sulfate USP) Inhalation Aerosol resulted in discontinuation of therapy in the few patients who have been treated. The majority of the patients have not experienced any significant side effects. The most frequent adverse reaction to Alupent® (metaproterenol sulfate USP) Inhalation Aerosol was nervousness. This reaction occurred in 6.8% of patients. Less frequent adverse experiences, occurring in 1% to 4% of patients were: headache, dizziness, palpitations, gastrointestinal distress, voice hoarseness, coughing, drowsiness and insomnia. Tachycardia occurred in less than 1% of patients.

Inhalation Aerosol contains 150 mcg of metaproterenol sulfate as a micronized powder in inert propellants. Each metered dose delivers through the mouthpiece 0.65 mg metaproterenol sulfate (each mL contains 15 mg). Alupent Inhalation Aerosol with Mouthpiece (NDC 0597-0070-17), net contents 14.9 g (10 mL), equipped with blue protective cap. Alupent Inhalation Aerosol Refill (NDC 0597-0071-16), net contents 14 g (10 mL).

Store between 59°F (15°C) and 77°F (25°C). Avoid excessive humidity.

Consult package insert before prescribing.

AL-BS-10-89

Reference:

Boehringer Ingelheim
Pharmaceuticals, Inc.
Ridgefield, CT 06877
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Reviews and Statements To Note


Toward Smoke-Free Medical Facilities (editorial)—RD Hurt. Chest 1990;97:1027. (Pertains to Hudzinski and Frohlich article abstracted below.)


Smoking and health are increasingly understood to be incompatible. To evaluate, prospectively and retrospectively, the attitudes of employees, staff physicians, and patients of a medical institution, a questionnaire was administered before and after implementation of a no-smoking policy. Of many questions, select ones reviewed here focused on the following concerns: (1) how tobacco smoke affects employees and patients, (2) employee acceptance of a no-smoking policy before and after its implementation, and (3) the consequences of the policy on employee smokers. Open-ended questions about smoking were constructed by a committee comprised of clinicians, investigators, and administrators. The questionnaire was given to 2,000 randomly selected patients and the institution's entire staff of 4,200 employees and 225 staff physicians. Data were obtained on three occasions: 6 months before, 5 months after, and 1 year after the implementation of the no-smoking policy. The majority of patients, employees, and physicians indicated that the smoke of others bothered them and ranked the following as most offensive: smell, eye irritation, provocation of sinus problems, coughing, and headache. Approximately 80% of employees and patients favored the policy before its inception; and employees increasingly favored it through the year after its implementation, with a favorable attitude increasing by nearly 10 percentage points. One year after implementation, 80% of patients were in favor of the policy. In the final survey, 74% of respondents indicated the policy had helped them. One year after policy implementation, employee smoking was reduced significantly from 22 to 14%; and of those employees who continued to smoke, 51% smoked less than 8 cigarettes per day. This study underscores the benefit of a work-site no-smoking policy in a health-care-provision setting. This overwhelming approval of smoke restriction supports the conclusion that employees, patients, and staff welcome a smoke-free environment.


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diagnostic tests on their ordering of such tests in an academic primary care medical practice. All tests were ordered at microcomputer workstations by 121 physicians. For half (the intervention group), the charge for the test being ordered and the total charge for tests for that patient on that day were displayed on the computer screen. The remaining physicians (control group) also used the computers but received no message about charges. The primary outcomes measured were the number of tests ordered and the charges for tests per patient visit. In the 14 weeks before the study, the number of tests ordered and the average charge for tests per patient visit were similar for the intervention and control groups. During the 26-wk intervention period, the physicians in the intervention group ordered 14% fewer tests per patient visit than did those in the control group (p < 0.005), and the charges for tests were 13% ($6.68 per visit) lower (p < 0.05). The differences were greater for scheduled visits (17% fewer tests and 15% lower charges for the intervention group; p < 0.01) than for unscheduled (urgent) visits (11% fewer tests and 10% lower charges; p > 0.3). During the 19 wk after the intervention ended, the number of tests ordered by the physicians in the intervention group was only 7.7% lower than the number ordered by the physicians in the control group, and the charges for tests were only 3.5% lower (p > 0.3). Three measures of possible adverse outcomes—number of hospitalizations, emergency room visits, and outpatient visits during the study period and the following 6 mo—were similar for the patients seen by the physicians in both groups. We conclude that displaying the charges for diagnostic tests significantly reduced the number and cost of tests ordered, especially for patients with scheduled visits. The effects of this intervention did not persist after it was discontinued.


Controversy exists on whether stimulation of the nasal mucosa results in reflex bronchoconstriction. To address shortcomings in previous experimental design, we performed double-blind randomized nasal challenges in asthmatic patients with allergic rhinitis and in controls. Using pledgets containing 10-μL aliquots of 0.9% saline or increasing concentrations of methacholine or histamine, we were able to increase nasal resistance significantly in both groups. Only methacholine caused an increase in lower-airway resistance, and this could be blunted by premedication of the nasal mucosa with phenylephrine. This suggests that the effect on lower-airway resistance was due to systemic absorption. Our study does not support the existence of a nasobronchial reflex from mechanical alteration of the nasal mucosa.


Spirometric data from 1586 healthy children, who did not smoke, were analysed to examine the effects of overweight as measured by the body mass index (weight/height²) on lung function. Overweight (72 boys, 88 girls) was defined as on or above the 90th percentile weight for height. After having controlled for the confounding variables of height and age, there were positive partial correlations between body mass index and lung function in girls whose weight was normal, in overweight girls, and in boys whose weight was normal, but not in overweight boys. In contrast to adults, body mass index has a positive effect on lung function in girls, whatever their weight. No such correlation between body mass index and lung function was seen in overweight boys. The observations may be accounted for by distinct sex-dependent patterns of fat distribution in children.


Asthmatic patients from western Canada and the United States have reported that after visits to an asthma clinic in Mexicali, Mexico, they return home substantially improved or cured having received ‘a bronchodilator medication unavailable in the United States or Canada because of the big drug companies.’ Analysis of these medications reveals that the most commonly prescribed combination is the glucocorticoid triamcinolone (unscored white tablets) and the
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antihistamine chlorpheniramine (coated biconvex orange or red tablets). Occasionally benzodiazepines are added to these medications. The patients are assured that the medications that they have been given are free of side effects and, specifically, that corticosteroids are not used. Such therapy is dangerous to the patient who not only is unaware of the medications that he or she is taking but is unlikely to mention this therapy to his or her physician. These patients risk drug interactions, medication side effects, and the possibility of adrenal failure either with a stress to their system or on withdrawal of drug treatment. Patients are also at risk of abandoning safer forms of asthma therapy for the miracle cure. We, too, are partially responsible for these unethical practices by avoiding the use of steroids and undertreating our patients at times, leaving them unnecessarily restricted and eager for any form of relief.


We studied the effects of an artificial surfactant, Exosurf, administered as an aerosol on respiratory system compliance (Crs), total respiratory resistance (RTh), and gas exchange (PO2) in anesthetized paralyz ed sheep with oleic acid (OA) induced lung injury. Paired experiments with OA were performed in 10 sheep, 5 of which received Exosurf in the first experiment and aerosolized 0.9% NaCl in the second, in the other 5 sheep, the order of Exosurf and NaCl was reversed. Paired experiments without OA were performed in 6 additional sheep that served as controls. In the first set of experiments, OA caused significant abnormalities, compared to control and baseline values (p < 0.02), in Crs, RTh, and PO2; there was no difference between animals that received Exosurf and those that received NaCl. Baseline values for PO2 and Crs during the second set of experiments with OA were lower than controls (p < 0.02), indicating that the animals had not fully recovered from their initial injury. After OA, the animals that received NaCl (ie, the ones that received Exosurf the first time) had higher PO2 and Crs values (p < 0.01) than those that received NaCl first and Exosurf second. There was no difference in postmortem lung water content between the animals that received Exosurf or NaCl first, both of which were higher than control (p < 0.01). Studies in 3 additional sheep showed peripheral deposition of aerosol. Thus, we failed to show an acutely beneficial effect of aerosolized Exosurf in OA-induced lung injury; Exosurf did, however, appear to provide protection against some of the consequences of repeated lung injury.


To assess the relationships among single-breath diffusing capacity for CO (DLCOsb), respiratory symptoms, and cigarette smoking in a general population sample, the data of 718 men and 894 women 20 y of age or older were analyzed, and comparisons were performed with flow-volume curve (MEVF) variables and the slope of the alveolar plateau (DNs%) as well. Percent of predicted DLCOsb and its correction for alveolar volume (DLvl/VA) were significantly lower in smokers than in nonsmokers. The relationship of presence/absence of respiratory symptoms and cigarette smoking with DlCOsb and Dl/VA was significant. DlCO indices were almost always selected as discriminant variables in multivariate analysis between asymptomatic and symptomatic subjects. Poor concordance among lung function tests was evident: in men, 30% with abnormal (ie, lower than the 97.5th percentile) and 21% with normal DlCO indices also had abnormal MEFV parameters and/or DN%/L. In women, the corresponding figures were 24 and 10%, respectively. In men, when considering only DlCO indices, the percentage of symptomatic subjects with abnormal lung function tests ranged from 33% in those with at least one symptom to 45% in those complaining of dyspnea. When the proportion of symptomatic subjects with DN%/L and MEFV abnormalities were added, it increased to 56 and 66%, respectively. However, in women the proportion of symptomatic subjects with abnormal lung function indices was very small. These results indicate the usefulness of including CO diffusing capacity in epidemiologic surveys in the detection of abnormalities.

Patient compliance with a standardized incremental regime of inhaled anti-asthma therapy has been assessed in a large, prospective study in general practice. Urine salbutamol estimations were made in 30 patients who had the largest improvement with therapy (mean increase in FEV1 0.45 L above baseline: Responsive) and in 30 patients whose airflow obstruction failed to improve (FEV1 -0.14 L: Nonresponsive). The urine salbutamol concentrations rose over the 9-mo period in the responsive patients as expected with the incremental doses prescribed, and were significantly higher than urine levels in nonresponsive patients at two dose levels. Poor compliance with prescribed inhaler therapy is an important cause of persistent morbidity from asthma.


Purpose: To determine the value of serum ferritin, mean cell volume, transferrin saturation, and free erythrocyte protoporphrin in the diagnosis of iron-deficiency anemia in the elderly. Methods and Patients: We prospectively studied consecutive eligible and consenting anemic patients over the age of 65 y, who underwent blood tests and bone marrow aspiration. The study consisted of 259 inpatients and outpatients at two community hospitals in whom a complete blood count processed by the hospital laboratory demonstrated previously undiagnosed anemia (men: hemoglobin level < 12 g/dL; women: hemoglobin level < 11.0 g/dL). Results: Thirty-six percent of our patients had no demonstrable marrow iron and were classified as being iron-deficient. The serum ferritin was the best test for distinguishing those with iron deficiency from those who were not iron deficient. No other test added clinically important information. The likelihood ratios associated with the serum ferritin level were as follows: >100 μg/L, 0.13; >45 μg/L but <100 μg/L, 0.46; >18 μg/L but <45 μg/L, 3.12; and <18 μg/L, 41.47. These results indicate that values up to 45 μg/L increase the likelihood of iron deficiency, whereas values over 45 μg/L decrease the likelihood of iron deficiency. Seventy per cent of those who were not iron-deficient had serum ferritin values >100 μg/L, and in populations with a prevalence of iron deficiency of <40%, values of >100 μg/L reduce the probability of iron deficiency to under 10%. Fifty-five percent of the iron-deficient patients had serum ferritin values of <18 μg/L, and in populations with a prevalence of iron deficiency of >20%, values of <18 μg/L increase the probability of iron deficiency to over 95%. Conclusion: In a general geriatric medical population such as ours, with a prevalence of iron deficiency of 36%, appropriate use of serum ferritin determination would establish or refute a diagnosis of iron deficiency without a bone marrow aspiration in 70% of the patients.


Thirty adult surgical patients admitted to the recovery room with an oral temperature ≤ 35.0°C were randomized into two groups. Group 1 patients were covered with cotton blankets warmed to 37.0°C, and group 2 patients were treated with a forced-air warming system. Mean oral temperature on admission to the recovery room was the same in both groups (34.3°C). Oral temperature and the presence or absence of shivering were recorded at 15-min intervals. After application of the selected warming method, patients in group 2 were warmer at all time intervals. Mean temperatures in the forced-air heating group and in group 1 were, respectively, 34.8°C and 34.3°C (p < 0.05) at 15 min; 35.0°C and 34.2°C (p < 0.01) at 30 min; 35.2°C and 34.5°C (p < 0.05) at 45 min; 35.8°C and 34.7°C (p < 0.001) at 60 min; 36.0°C and 35.0°C (p < 0.01) at 75 min; and 36.0°C and 35.0°C (p < 0.01) at 90 min. The incidence of shivering was significantly greater in group 1 at 15 and 45 min. In addition, time spent in the recovery room was significantly greater in group 1 than in group 2, 156.0 min vs 99.7 min (p < 0.003).


To our knowledge, the effects of corrective spinal surgery on total respiratory mechanics and its components in anesthetized patients with kyphoscoliosis have not been previously reported in detail. We studied 13 patients with kyphoscoliosis; their mean (± SD) age was 24.7 ± 2.1 y; eight underwent anterior and posterior spinal fusions (AF and PF, respectively) 2 wk apart (group A), four underwent PF alone (group B), and one had a three-stage procedure. Mean total respiratory elastance (Ers), static and dynamic lung elastance (Est, L and Edyn, L, respectively), chest wall elastance (Ew), and lung resistance (RL) were derived according to previously described methodology. In group A, Ers and Ew increased by 39%
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and 58%, respectively, following AF and by 20% and 129% following PF, while Est, L and Edyn, L did not change or declined following PF. Lung resistance increased 19% and 41% by the end of AF and PF, respectively, in group A. In group B, Ew more than doubled, resulting in a 39% increase in Ers. Increases in Ers, Ew, and respiratory flow resistance observed at the time of spinal corrective surgery for kyphoscoliosis may result from rib cage trauma and changes in airway caliber related to microatelectasis and uneven distribution of mechanical properties within the lungs. Spinal correction results in immediate and short-term deterioration of respiratory mechanics measured under anesthesia.


In lung sarcoidosis, the mutual relationships of three components of the extracellular matrix, fibronectin (FN), hyaluronan (HA), and type III procollagen peptide (PCP), were investigated in the bronchoalveolar lavage fluid. Furthermore, their relation to signs of alveolitis and of fibrosis was examined. Sarcoidosis patients (n = 74) had significantly (p < 0.001) increased bronchoalveolar lavage fluid concentrations of FN, HA, and PCP, as well as albumin and lymphocytes, compared to controls (n = 57). The increases were significantly higher in clinically active than in inactive sarcoidosis. FN, HA, and PCP were significantly correlated to markers of alveolitis, such as albumin (r = 0.6-0.7, p < 0.001) and lymphocytes (r = 0.4-0.5, p < 0.001 for FN and HA; p < 0.05 for PCP), indicating that an alveolar inflammatory process may be prerequisite for the increased production of the three components. Since correlations between FN and HA and functional parameters (VC, TLC, FEV1, and DLCO) were low (r = 0.2-0.3, p < 0.05-0.01 for FN and HA), the increased levels of the extracellular markers do not seem to reflect developed fibrosis. The three markers of extracellular matrix showed significant (p < 0.001) mutual correlations in the sarcoid patients (r ~ 0.7). FN and HA were correlated even in controls (r = 0.5; p < 0.01). The findings are in agreement with our hypothesis that these compounds may participate in the buildup of an extracellular network that supports the healing process but in excess may eventually lead to fibrosis.


High airway pressure may be injurious to lung parenchyma, but lowering airway inflation using conventional mechanical ventilation necessitates lowering tidal volume (VT). Intubated patients in the surgical intensive care unit (SICU) were randomly assigned to Group 1 (VT = 12 mL/kg, n = 56) or Group 2 (VT = 6 mL/kg, n = 47). Variables recorded included acute physiology and chronic health evaluation (APACHE II) score, mean peak airway pressure (MPAP), mean PA02/FIO2, incidence of pulmonary infectious complications (PIC), duration of intubation (DOI), and duration of SICU stay (DOS). Results in the table are means ± SE.

<table>
<thead>
<tr>
<th></th>
<th>Group 1</th>
<th>Group 2</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>APACHE-II score</td>
<td>13.3 ± 0.8</td>
<td>13.3 ± 0.8</td>
<td>0.776</td>
</tr>
<tr>
<td>MPAP, cm H2O</td>
<td>35.1 ± 0.2</td>
<td>26.0 ± 1.1</td>
<td>&lt; 0.001</td>
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<tr>
<td>PA02/FIO2</td>
<td>294 ± 11</td>
<td>260 ± 11</td>
<td>0.031</td>
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<tr>
<td>PIC, %</td>
<td>17.9</td>
<td>4.3</td>
<td>0.061</td>
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<td>DOI, days</td>
<td>3.9 ± 0.8</td>
<td>2.3 ± 0.5</td>
<td>0.066</td>
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<tr>
<td>DOS, days</td>
<td>4.6 ± 1.0</td>
<td>2.7 ± 0.5</td>
<td>0.064</td>
</tr>
</tbody>
</table>

The incidence of pulmonary infection tended to be lower and DOI and DOS tended to be shorter for non-neuroanatomical and noncardiac surgical patients randomized to low VT, suggesting that morbidity may be decreased. The use of low VT was associated with statistically significant but clinically irrelevant decrease in oxygenation. The routine use of low VT appeared to be
safe in a selected population of patients in the SICU.


The variability of peak flow measurements was studied in 24 patients with airway obstruction using 12 Assess peak flow meters and a Fleisch pneumotachograph Type 4 as a standard. The Assess peak flow meter gave systematic under-readings of 19.34% (low accuracy). The value scale of the Assess peak flow meter should be adjusted. As systematic under-readings do not influence the variability in peak flow measurements, variability was only caused by the random error of the instrument. The random error of the Assess peak flow meter was relatively low; about 4% of the measured peak flow value (high precision). The contribution of the Assess peak flow meter to the total variability of peak flow measurements showed a linear relationship \( r = 0.53 \) with the value of the peak flow rate itself and varied from 9% (at 3 L/s) to 86% (at 10.8 L/s). Reading errors of the peak flow meter were responsible for about 1% of the total variability. The remaining variability is probably caused by the correctness of the peak flow performance and the motivation of the patient.


Diaphragmatic function and biochemical changes were studied during respiratory failure induced by incremental inspiratory threshold loading in anesthetized rabbits (1) who were unbound and spontaneously breathing, (2) during lower thoracic and abdominal binding, and (3) while bound and undergoing transvenous phrenic nerve pacing of the diaphragm. There was no evidence of contractile fatigue or alterations in glycogen or lactate concentrations in the diaphragm of unbound spontaneously breathing animals. With thoracoabdominal binding, there was a fall in maximal transdiaphragmatic pressure \( \text{Pdi}_{\text{max}} \) and the ratio of diaphragmatic force divided by neural input \( \text{Pdi}/\text{Edi} \) ratio; there was no change in diaphragm glycogen, but there was a significant rise in lactate. In the bound and phrenic-paced animals \( \text{Pdi}_{\text{max}} \) and \( \text{Pdi}/\text{Edi} \) ratio fell, and there was significant glycogen depletion and lactate accumulation. There was a significant correlation between diaphragm function and the levels of diaphragm glycogen and lactate at the point of respiratory failure. We conclude that (1) respiratory failure induced by incremental inspiratory threshold loading was not associated with either contractile fatigue of the diaphragm or diaphragmatic biochemical changes in unbound spontaneously breathing animals, (2) when mechanisms that preserve diaphragmatic function are circumvented by phrenic pacing and/or thoracoabdominal binding, diaphragm fatigue and biochemical changes occur, and (3) there is a significant relationship between in vivo evidence of contractile fatigue of the diaphragm and diaphragmatic glycogen depletion and lactate accumulation.

Comparison of Scales Used To Quantitate the Sense of Effort To Breathe in Patients with Chronic Obstructive Pulmonary Disease—SR Muza, MT Silverman, GC Gilmore, HK Hellerstein, SG Kelsen. Am Rev Respir Dis 1990;141:909.

Several different scaling techniques, ie, Borg category (BC) and visual analogue (VA) scales have been used to quantitate the intensity of the respiratory sensations elicited during exercise, but their relationship is unclear. Six subjects with stable chronic obstructive lung disease \( \text{FEV}_{1} = 1.2 \pm 0.1 \) SE. L simultaneously rated the sense of effort to breathe with both BC and VA scales during progressive, maximal exercise tests performed three to five times on a cycle ergometer. The VA scores correlated linearly with minute ventilation in all subjects in all trials \( r = 0.98 \pm 0.01 \), and when converted to common units (ie, Z scores) correlated closely with simultaneous scores obtained using the Borg scale \( r = 0.99 \pm 0.01 \). Furthermore, VA scores varied minimally over several trials. Coefficient of variation for the maximal VA scores was 6 ± 1%, which was similar to the variation in maximal Borg score (ie, 3 ± 1%). We conclude that the visual analogue scale is reproducible and correlates closely with the Borg score when scaling the sense of effort to breathe during exercise in subjects with stable chronic obstructive pulmonary disease.

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The Effects of Bolus Normal-Saline Instillation in Conjunction with Endotracheal Suctioning

Joanne E Gray RN MSN, Neil R MacIntyre MD, and William G Kronenberger MA

Although the instillation of normal saline into the endotracheal tube prior to suctioning is common practice, the only available clinical data to support this practice were obtained in postoperative patients without apparent pulmonary disease. MATERIALS & METHODS: Fifteen critically ill, intubated patients with pulmonary disease were studied to determine the physiologic responses, the amount of material suctioned, and the degree of discomfort associated with two methods of endotracheal suctioning: suctioning with a 5-mL normal-saline instillation (NSI) and suctioning without NSI. Each subject was suctioned by each method, 90 minutes apart. Assessment of hemodynamics, gas exchange, and respiratory mechanics was carried out before, immediately after, and 15 minutes after each method was performed. The volume of material suctioned and subject discomfort were measured after each method was performed. RESULTS: While statistically significant changes in heart rate, blood pressure, respiratory rate, blood gas and pH values occurred immediately after suctioning in both methods, there were no significant differences in these variables between the two methods. The instillation of NSI resulted in coughing and a significant increase in the amount of material suctioned. There was no difference in subject discomfort between the two methods. CONCLUSIONS: We conclude that suctioning with NSI may enhance secretion clearance through cough stimulation, and that its effects on hemodynamics, respiratory mechanics, gas exchange, and patient comfort are not significantly different from the effects of suctioning without NSI. (Respir Care 1990;35:785-790.)

Introduction

Critically ill patients often require endotracheal intubation and mechanical ventilation. The presence of an endotracheal tube interferes with the mucociliary transport system—the normal mechanism by which respiratory tract secretions are removed. Thus, endotracheal suctioning is an important component of the mechanically ventilated patient’s care. In the presence of pulmonary disease, infection, or dehydration, respiratory secretions may become thick and tenacious, making removal by suctioning difficult.

It is a common practice in the critical care unit to instill a 5- to 10-mL bolus of sterile normal-saline solution into the endotracheal tube prior to suctioning. The use of normal-saline instillation (NSI) is purported to elicit a cough and to liquefy and mobilize secretions.1-6 In the only controlled clinical study7 that has compared suctioning with and without NSI, the amount of material suctioned was greater when a 5-mL NSI was used; however, no significant difference was observed in \( P_{\text{aO}_2} \) changes between the two techniques. Demers and Saklad8 have suggested that NSI might not be helpful because “while water in the form of an aerosol is of proven value in thinning secretions, mucus and water in bulk form are immiscible and occupy separate phases in vitro, even after vigorous shaking.” Finally, in a study that used a radiolabelled bolus of saline,9 only 10.7-18.7% of instilled material could be retrieved by suctioning.

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Ms Gray is a student anesthetist, Department of Anesthesia, North Carolina Baptist Hospital, Winston-Salem, North Carolina. Dr MacIntyre is Associate Professor of Medicine and Medical Director of Respiratory Care, Duke University Medical Center, Durham, North Carolina. Mr Kronenberger is a PhD candidate, Department of Psychology, Duke University, Durham, North Carolina.
Abbreviations Used in This Paper

FVC — Forced vital capacity
NSI — Normal-saline instillation
$P_{ACO_2}$ — Arterial carbon dioxide tension (pressure)
$P_{AO_2}$ — Arterial oxygen tension
$S_{PO_2}$ — Oxygen saturation determined by pulse oximetry
$T_1$ — Immediately prior to suctioning
$T_2$ — Immediately after suctioning
$T_3$ — Fifteen minutes after suctioning
$V_E$ — Minute ventilation

A Guide to the Use of SI in This Paper*

The SI unit for pressure is the kilopascal (kPa).

\[(torr)(0.1333) = \text{kPa}\]

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

Because the utility of NSI appears to be questionable, and because the only clinical data available were obtained in postoperative patients without apparent pulmonary disease, we designed a controlled study of NSI in patients with known pulmonary disease who required frequent suctioning. The specific purpose of this study was to assess the effect of suctioning with and without NSI on: (1) the amount of material suctioned, (2) gas exchange, (3) respiratory system mechanics, (4) cardiovascular function, and (5) patient comfort.

Materials and Methods

The study sample consisted of 15 critically ill adult patients who were orally or nasally intubated and being mechanically ventilated, and who met the following criteria: (1) were sufficiently alert to sign a consent form, (2) had a patent indwelling arterial catheter, (3) were hemodynamically stable (did not require titration of vasoactive medications), and (4) had previously been suctioned with and without NSI, at least once. Patients for whom an $F_{IO_2} > 0.70$ was required to maintain a $P_{AO_2} \geq 60$ torr [8.0 kPa] were excluded from the study, as were patients with tracheostomies or hematocrit < 25%. Characteristics of the study patients are shown in Table 1.

Heart rate; systolic and diastolic blood pressure; respiratory rate; arterial oxygen tension ($P_{AO_2}$), carbon-dioxide tension ($P_{ACO_2}$), $pH$; oxygen saturation; minute ventilation ($V_E$); peak inspiratory airway pressure; and forced vital capacity (FVC) were measured. These variables were measured at three points in time: immediately prior to suctioning ($T_1$), immediately after suctioning and returning the patient to the ventilator ($T_2$), and 15 minutes after suctioning and returning the patient to the ventilator ($T_3$). Heart rate was monitored and digitally displayed by a bedside cardiac monitor,* blood pressure was recorded by a bedside strip-chart recorder, respiratory rate was determined by manually counting the number of respirations in 15 seconds and multiplying by 4, blood gas and pH analysis was performed on blood drawn from the arterial catheter, and oxygen saturation was measured by a pulse oximeter ($S_{PO_2}$). Each patient was attached to one of three mechanical ventilators that digitally displayed $V_E$ (the BEAR 5, the Servo 900C, or the Puritan-Bennett 7200a). Airway pressure was measured at the proximal airway by the Novametrix airway pressure monitor and recorded by strip-chart recorder. FVC was measured with a hand-held ventilation monitor. After all data had been collected, the amount of material suctioned was determined by weighing the sputum trap with its contents and subtracting the weight of the empty trap. Lung sounds were assessed by auscultation. The degree of discomfort associated with each method was determined by asking the subject to rate his or her discomfort on a scale from 1 (least uncomfortable) to 6 (most uncomfortable).

Each subject was suctioned without NSI 90 minutes prior to beginning the study. Fifteen minutes prior to the study, the subject was placed in the supine position with the head of the bed elevated 30°; all transducers and recorders were zero-balanced. The two methods of suctioning (with and without NSI) were then carried out in each subject; the order of the methods was randomized, such that some subjects

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*Suppliers are identified in the Product Sources section at the end of the text.
Table 1. Characteristics of Patients Participating in Suctioning Study

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Primary Diagnosis</th>
<th>Secondary Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>51</td>
<td>M</td>
<td>bronchitis, COPD*</td>
<td>—</td>
</tr>
<tr>
<td>2</td>
<td>68</td>
<td>F</td>
<td>angina following infarction</td>
<td>COPD, sepsis</td>
</tr>
<tr>
<td>3</td>
<td>64</td>
<td>F</td>
<td>metastatic cancer</td>
<td>—</td>
</tr>
<tr>
<td>4</td>
<td>67</td>
<td>F</td>
<td>COPD</td>
<td>—</td>
</tr>
<tr>
<td>5</td>
<td>66</td>
<td>F</td>
<td>postsinus surgery</td>
<td>hypertension</td>
</tr>
<tr>
<td>6</td>
<td>78</td>
<td>M</td>
<td>postaneurysm repair</td>
<td>COPD</td>
</tr>
<tr>
<td>7</td>
<td>79</td>
<td>F</td>
<td>myocardial infarction</td>
<td>hypertension, CAD</td>
</tr>
<tr>
<td>8</td>
<td>43</td>
<td>M</td>
<td>pneumonia</td>
<td>—</td>
</tr>
<tr>
<td>9</td>
<td>73</td>
<td>M</td>
<td>COPD</td>
<td>anemia</td>
</tr>
<tr>
<td>10</td>
<td>74</td>
<td>M</td>
<td>post-CABG</td>
<td>reactive airway disease</td>
</tr>
<tr>
<td>11</td>
<td>20</td>
<td>F</td>
<td>leukemia, cardiomyopathy</td>
<td>sepsis</td>
</tr>
<tr>
<td>12</td>
<td>30</td>
<td>F</td>
<td>ARDS</td>
<td>sepsis</td>
</tr>
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<td>13</td>
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<td>M</td>
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<td>vasculitis</td>
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<tr>
<td>14</td>
<td>64</td>
<td>M</td>
<td>lung cancer</td>
<td>COPD</td>
</tr>
<tr>
<td>15</td>
<td>64</td>
<td>M</td>
<td>COPD</td>
<td>—</td>
</tr>
</tbody>
</table>

*COPD = chronic obstructive pulmonary disease.  
CAD = coronary artery disease.  
ARDS = adult respiratory distress syndrome.  
CABG = coronary artery bypass graft.

received suctioning with NSI first, and some received suctioning with NSI second. The two methods were separated by 90 minutes to allow for stabilization of the selected variables. During the 90 minutes between suctioning methods, all subjects remained supine with the head of the bed elevated 30°. No adjustments in vasoactive medications or ventilator settings were made.

The patients were preoxygenated prior to being suctioned by each method. A purge feature to administer a sigh breath with 100% oxygen was not available on every ventilator; therefore, to maintain consistency of technique, preoxygenation of patients prior to suctioning was accomplished by attaching the patient to a manual resuscitation bag that supplied 100% oxygen and by manually administering three sigh breaths. All preoxygenation was performed by the principal investigator. The suctioning procedure used in this study is described in Table 2. When suctioning with NSI was performed, a 5-mL bolus of normal saline at room temperature was instilled from a prefilled ampule into the orifice of the endotracheal tube prior to preoxygenation.

Statistical Analysis

The dependent variables measured at three points in time were subjected to a 2 (NSI versus no-NSI) × 3 (time) repeated measures analysis of variance (ANOVA), which revealed no differences or interaction-effects based on the presence of NSI. Therefore, the data were collapsed across the NSI/no-NSI condition and subjected to a one-way

Table 2. Suction Procedure Used in This Study

1. Three manual sigh breaths of 100% O₂ are delivered.
2. The suction catheter with collection trap is inserted and then withdrawn (using a twisting motion) over a period of 6 seconds while intermittent suction of ~120 torr (~16.0 kPa) is applied.
3. Repeat Step 1.
4. Repeat Step 2.
5. Repeat Step 1.
6. Reconnect the patient to the mechanical ventilator.
ANNOVA (time). Level of discomfort and weight of the material suctioned, which were measured at the end of each succioning procedure, were subjected to the t test and the Wilcoxon test (NSI versus no-NSI); p values of \( \leq 0.05 \) were considered significant.

**Results**

Figures 1-6 show the changes observed in the variables measured at the three points in time. At T2, a statistically significant change from T1 was found in heart rate, respiratory rate, systolic and diastolic blood pressure, \( P_{aO_2} \), \( P_{aCO_2} \), and pH (p < 0.05). These changes occurred regardless of succioning method. There were no significant differences in these variables between the two methods. The variables, in which changes were observed, returned to baseline levels 15 minutes after succioning with either method. Lung sounds did not change after succioning with either method. Subjects who had wheezing prior to succioning did not exhibit succioning-induced changes in wheezing with either method. No significant changes in peak inspiratory airway pressure, \( V_t \), \( S_pO_2 \), or FVC were observed with either succioning method. According to both the t test and the Wilcoxon test,
EFFECTS OF NORMAL SALINE ON SUCTIONING

Fig. 5. $P_{\text{aCO}_2}$ before ($T_1$), immediately after ($T_2$), and 15 min after ($T_3$) suctioning with and without NSI.

Fig. 6. Arterial pH before ($T_1$), immediately after ($T_2$), and 15 min after ($T_3$) suctioning with and without NSI.

Fig. 7. Level of discomfort expressed by subjects suctioned with and without NSI.

Fig. 8. Grams of material suctioned with and without NSI.

there was no significant difference—between suctioning methods—in subject-rated discomfort (Fig. 7). All subjects who received NSI coughed immediately after the instillation of saline as well as during stimulation by the suction catheter (cough was elicited more frequently during suctioning with NSI than during suctioning without NSI). Although the forcefulness of the cough was not subjected to statistical analysis, the cough observed after NSI did not appear to be stronger than the cough elicited by stimulation of the catheter during suctioning without NSI. A significant difference was found between the two methods in the weight of the material suctioned. Material suctioned following instillation of normal saline weighed more than that suctioned without NSI, according to both the $t$ test and the Wilcoxon test ($p < 0.05$) (Fig. 8).

Discussion

The results of this study suggest that suctioning with NSI results in a significantly greater amount of material being suctioned than suctioning without NSI. Unfortunately, more detailed analysis was not possible because it was not technically feasible in this study to determine the secretion content of the
material suctioned, nor was it possible to ensure that all subjects received the full amount of the NSI (ie, saline was often coughed out of the endotracheal tube prior to collection in the trap). Nevertheless, because all patients coughed immediately after the instillation of saline, these findings suggest that the major effect of NSI on secretion clearance is to stimulate a cough.

Changes in oxygenation and ventilation did not differ significantly between suctioning methods. These findings are in agreement with the findings of Bostick and Wendelgass.7 The increases in $P_{aO_2}$ that occurred immediately after suctioning in both methods may indicate that three manual breaths with 100% oxygen prior to and after suctioning were sufficient to prevent suction-induced hypoxemia in this patient population.

Bradycardia and hypotension due to vagal stimulation and prolonged coughing during the suctioning process have been reported in the literature.10 In this study, suctioning with and without NSI resulted in increases in heart rate and blood pressure. These changes, while statistically significant, do not appear to be clinically important in patients with stable cardiovascular systems. However, the occurrence of bradycardia and hypotension in one patient who was excluded from the study emphasizes that cardiovascular responses to suctioning can vary.

Most of the subjects in this study had underlying pulmonary disease and many had associated bronchospasm (an intravenous bronchodilator was being administered to 8 of the 15 subjects). If the instillation of normal saline had resulted in significant airway irritation, corresponding changes in peak inspiratory airway pressure, FVC, or wheezing (reflecting a change in airway resistance) would have been anticipated. However, no changes in these variables were observed.

We conclude that suctioning with NSI may enhance secretion clearance through cough stimulation and that its effects on hemodynamics, respiratory mechanics, gas exchange, and patient comfort are not significantly different from the effects of suctioning without NSI.

**PRODUCT SOURCES**

Blood-Gas Analyzers:
Radiometer ABL 4 Acid-Base Laboratory, Radiometer, Copenhagen, Denmark

Blood-Gas Syringes:
Monitor Medical Inc, Winston-Salem NC

Cardiac Monitor:
Mennen Horizon 2000, Mennen Medical Co, Clarence NY

Mechanical Ventilators:
BEAR 5, Bear Medical Systems Inc, Los Angeles CA
Puritan-Bennett 7200a, Puritan-Bennett Inc, Winston-Salem NC
Servo 900C, Siemens Elsa Inc, Los Angeles CA

Normal-Saline Ampules:
Monitor Medical Inc, Winston-Salem NC

Pulse Oximeter:
Nellcor N-200, Nellcor Inc, Hayward CA

Scale:
Ohaus Dial O Gram Balance, Ohaus Scale Corp, Florhan Park NJ

Strip-Chart Recorder:
Novametrix Pneumogard 1230A, Novametrix Medical Systems Inc, Wallingford CT

Suction Catheters:
Baxter Edwards Labs, Irvine CA

Sputum Traps:
Cheseborough Pond's Inc, St Louis MO

Ventilation Monitor:
BEAR LS 75, Bear Medical Systems Inc, Los Angeles CA

**REFERENCES**

Transtracheal Gas Administration and the Perception of Dyspnea

Marin H Kollef MAJ USA MC and Ray C Johnson MAJ USA MC

The effects of nasal cannula gas flow on dyspnea have been studied by Liss and Grant (Am Rev Respir Dis 1988; 137: 1285), who concluded that reduction of dyspnea was a placebo effect from the wearing of the nasal cannula. We conducted a similar study in subjects using transtracheal oxygen catheters (TTOCs). METHODS: Nine patients with chronic lung disease and hypoxemia were administered zero flow, 2 and 4 L/min of air, and 2 and 4 L/min of oxygen via their TTOCs. After each study segment, the subjects recorded their degree of dyspnea on a visual analog scale (VAS) calibrated from 0 (not short of breath) to 100 (extremely short of breath). After the first five tests, the subjects' tracheas were anesthetized with topical 1% lidocaine via their TTOCs, and the five tests were repeated. RESULTS: Prior to tracheal anesthesia, air and oxygen flows at 4 L/min produced significantly higher VAS scores (more dyspnea) than at baseline (air, p = 0.0233; oxygen, p = 0.0183). Air and oxygen at 2 L/min did not produce VAS scores significantly higher than baseline scores (air, p = 0.3196; oxygen, p = 0.0669). VAS scores associated with 4 L/min air and oxygen flows were significantly higher than scores associated with 2 L/min flows (p = 0.0179). VAS scores during zero flow prior to tracheal anesthesia were not significantly different from baseline VAS values (p = 0.5534). After tracheal anesthesia, no significant relationship was found between gas flowrate and VAS scores (p = 0.9618). The anesthesia appeared to eliminate the effect of transtracheal gas flow on dyspnea. CONCLUSIONS: Transtracheal gas administration can affect the perception of dyspnea in patients with chronic lung disease. This effect on flow-sensitive tracheal receptors appears to be abolished by tracheal anesthesia. (Respir Care 1990;35:791-799.)

Introduction

Oxygen therapy is known to prolong the lives of hypoxemic patients with chronic obstructive pulmonary disease (COPD), 1,2 and oxygen therapy will often improve such patients' dyspnea. 3 The conventional device for administering oxygen to these patients has been the nasal cannula, with oxygen-conserving cannulas receiving recent attention. 4 The transtracheal oxygen catheter (TTOC), a relatively new device, may also be desirable in the treatment of such patients. 5 Besides reducing the amount of supplemental oxygen needed and offering a cosmetic advantage, TTOC is also thought by some to decrease dyspnea, compared to nasal cannula oxygen therapy. 6,7 They believe this occurs because oxygen delivered directly into the tracheobronchial tree bypasses the anatomical dead space of the upper airway, and this decreases the work of breathing.

Because the direct effects of transtracheal gas administration on the perception of dyspnea have not been previously investigated, we studied the perception of dyspnea in hypoxemic patients with

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The opinions and assertions contained herein are the private views of the authors and do not necessarily reflect the views of the Department of the Army or the Department of Defense.

Reprints: Marin H Kollef MAJ USA MC, Director, Medical Intensive Care Unit, Pulmonary and Critical Division, Fitzsimons Army Medical Center, Aurora CO 80045-5001.
chronic lung disease who had TTOCs in place, subjecting them to various flowrates of gas, with and without tracheal anesthesia.

Methods

Subjects

Nine subjects—seven men and two women with chronic lung disease and resting hypoxemia when breathing room air—were recruited from the home oxygen therapy clinic at Fitzsimons Army Medical Center, Aurora, Colorado. Six subjects had a primary diagnosis of COPD based on clinical data and spirometry. Three subjects had mixed restrictive and obstructive lung disease.

All nine subjects met the following criteria: (1) resting \( P_{aO_2} \) below 55 torr when breathing room air (at an altitude of 5,280 feet) as a prerequisite to receiving long-term supplemental oxygen therapy, (2) use of conventional nasal cannula oxygen therapy for at least 6 months before use of TTOC therapy, (3) use of TTOC therapy for at least 6 months prior to the study, (4) voluntarily reported improvement in dyspnea at rest with TTOC therapy compared to dyspnea with previous nasal oxygen therapy, (5) clinical and spirometric evidence of chronic lung disease, (6) ability to sign informed consent, (7) no contraindications to placement of an indwelling radial artery catheter, (8) no allergy to lidocaine, and (9) no evidence of an acute exacerbation of their lung disease at the time of the study.

The subjects’ ages ranged from 54 to 73 years; their mean age was 65, with a standard deviation of 7, as shown in Table 1.

Table 1 also shows the subjects’ pulmonary function data, baseline resting TTOC oxygen flowrates, room-air blood gas values measured at least 1 week prior to the study period, and baseline dyspnea scores.

Study Design

A prospective experimental study design was used wherein all subjects were given, in a random, single-blinded manner, zero gas flow, air at 2 and 4 L/min, and oxygen at 2 and 4 L/min—via their TTOCs; after each period of flow, the subject indicated on a visual analog scale (VAS) the amount of dyspnea he or she had just experienced. Our study design followed that used by Liss and Grant,* who had examined the effects of nasally administered air and oxygen at different flowrates on the sensation of dyspnea in patients with COPD. We chose the same gas flowrates used by Liss and Grant in order to permit direct comparison of our findings with theirs. (Although a flowrate of 4 L/min is unusual in clinical TTOC therapy, it is not unknown.) In addition, by using increasing levels of gas flowrate, we could examine the data for any potential dose-response

Table 1. Baseline Data for Patients Receiving Oxygen by Transtracheal Catheter

<table>
<thead>
<tr>
<th>Patient Number</th>
<th>Age</th>
<th>FVC (L)</th>
<th>FEV1 (L)</th>
<th>( P_{aO_2} ) (torr)</th>
<th>( P_{aCO_2} ) (torr)</th>
<th>VAS* (baseline)</th>
<th>Flow (L/min)</th>
</tr>
</thead>
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<tr>
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<td>2.23 ± 0.69</td>
<td>1.42 ± 0.63</td>
<td>50 ± 4</td>
<td>45 ± 8</td>
<td>26 ± 13</td>
<td>1.14 ± 0.38</td>
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</table>

*VAS = Visual analog scale, 20-cm long, for recording dyspnea, from 0 (not short of breath) to 100 (extremely short of breath).
relationship between the gas flowrate and the perception of dyspnea.

**Study Protocol**

The protocol was approved by the Investigational Review Board and Human Studies Committee of the Fitzsimons Army Medical Center.

The subject sat in bed at a 45° angle, wearing a SCOOP-1 TTOC,* which had been placed prior to the study. Gas was supplied by wall sources located behind the head of the bed. Gas flow was regulated—without the patient's knowledge—by use of a system of stopcocks and tubing leading from the wall sources to the patient's TTOC. A separate source of air was kept flowing throughout the study so that its sound would keep the subject unaware when flow to the TTOC was zero.

A baseline dyspnea score was obtained just before the experiment, with the subject at rest and receiving his or her usual prescribed oxygen flow via the TTOC. The patient marked a point on a visual analog dyspnea scale to indicate his or her degree of dyspnea, and an arterial blood-gas sample was withdrawn from the radial-artery catheter and analyzed within 30 minutes. The blood sample was analyzed with a Corning 178 pH-blood gas analyzer. Analyzer quality control checks were performed every 4 hours as part of the institution's routine blood gas laboratory protocol.

The visual analog scale for dyspnea followed the design of the scale used by Liss and Grant; its 20-cm-long line was calibrated from 0 (not short of breath) to 100 (extremely short of breath). The use of visual analog scales to monitor and evaluate dyspnea has been examined and validated, with studies showing the results to be reproducible in a given patient and useful in comparing various measurements from a single patient.  

After the recording of the baseline dyspnea score on TTOC oxygen, the subject breathed room air for at least 15 minutes, after which another blood gas-pH determination was made to establish experimental baseline values.

Continuous electrocardiographic monitoring and continuous arterial oxygen saturation monitoring by a Lifestat 1600 finger pulse oximeter were conducted throughout the study. Gas flowrates were measured with a Collins water-seal spirometer.

Each gas flow was given for 3 to 5 minutes, with 3 minutes being the limit if the oxygen saturation dropped below 85%—to avoid complications associated with hypoxemia. At the end of each time interval, the subject again marked the VAS to indicate his or her dyspnea, and an arterial blood sample was drawn for blood gas-pH analysis. The five intervals of the study—zero flow, 2 and 4 L/min of oxygen flow, and 2 and 4 L/min of air—were administered in random, single-blinded order.

After the subject had undergone the first five study intervals, the same studies were repeated with the trachea anesthetized in order to mask the potential effect of gas flowrate on tracheal-receptor stimulation, thus allowing the examination of other potential variables, such as the arterial oxygen tension, and their effects on dyspnea in these patients.

The trachea was anesthetized with the instillation of 5 cc of 1% lidocaine solution into the TTOC. After allowing 5 minutes for the anesthetic to take effect, we again administered the five segments of the study—that is, zero flow, oxygen at 2 and 4 L/min, and air at 2 and 4 L/min—with the VAS and blood gas-pH data being collected as before.

**Statistical Analysis**

The data were analyzed by a nonparametric statistical method with the Kruskal-Wallis statistic.  

**Results**

Blood gas and VAS data are presented in Tables 2-5.

**Before Tracheal Anesthesia**

We found a significant relationship between gas flowrate and the degree of dyspnea before the trachea was anesthetized (Table 5). When air was administered at 4 L/min, VAS scores were significantly higher than the baseline VAS scores shown in Table 1 (p = 0.0233), indicating a higher level of dyspnea.

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*Suppliers are identified in the Product Sources section at the end of the text.*
Table 2. $P_aCO_2$ (torr) before and after Tracheal Anesthesia in Patients Receiving Transtracheal Gas at Various Flows

<table>
<thead>
<tr>
<th>Subject Number</th>
<th>Zero Flow</th>
<th>Air at 2 L/min</th>
<th>Air at 4 L/min</th>
<th>O$_2$ at 2 L/min</th>
<th>O$_2$ at 4 L/min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before Tracheal Anesthesia</td>
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<tr>
<td>Mean ± SD</td>
<td>39 ± 7</td>
<td>39 ± 7</td>
<td>38 ± 6</td>
<td>39 ± 7</td>
<td>40 ± 7</td>
</tr>
</tbody>
</table>

| After Tracheal Anesthesia |           |                |                |                  |                  |
| 1              | 44        | 42             | 45             | 48               | 47               |
| 2              | 44        | 45             | 44             | 44               | 46               |
| 3              | 37        | 37             | 35             | 36               | 38               |
| 4              | 31        | 33             | 32             | 33               | 34               |
| 5              | 52        | 50             | 47             | 52               | 51               |
| 6              | 40        | 40             | 38             | 42               | 42               |
| 7              | 38        | 37             | 38             | 39               | 39               |
| 8              | 28        | 31             | 29             | 29               | 30               |
| 9              | 31        | 32             | 32             | 33               | 29               |
| Mean ± SD     | 38 ± 8    | 39 ± 6         | 38 ± 6         | 40 ± 8           | 40 ± 8           |

Oxygen flowing at 4 L/min produced a similar result compared to baseline ($p = 0.0183$). When air and oxygen were administered at 2 L/min, VAS scores did not differ significantly from baseline values (air, $p = 0.3196$; oxygen, $p = 0.0669$).

When the grouped pre-anesthesia VAS scores for air and oxygen at 2 L/min were compared to the grouped pre-anesthesia VAS scores for air and oxygen at 4 L/min, a significant difference was found ($p = 0.0179$), with the higher flowrates being associated with a greater degree of dyspnea (Table 6).

The VAS scores during zero flow prior to tracheal anesthesia (Table 5) were not significantly different from the baseline values in Table 1 ($p = 0.5534$).

After Tracheal Anesthesia

With the trachea anesthetized, no significant relationship was found between the degree of dyspnea and flowrate ($p = 0.9618$), $P_aO_2$ ($p = 0.1332$), $P_aCO_2$ ($p = 0.5459$), or arterial pH ($p = 0.4709$).

When oxygen was administered at 2 and 4 L/min during tracheal anesthesia, $P_aCO_2$ was higher and associated VAS scores were lower than when air was administered at 2 and 4 L/min (Tables 4 and 5); however, this was not statistically significant ($p = 0.1332$).

Other Data

The mean $P_aCO_2$ for each flow was similar before and during tracheal anesthesia (Table 2). Although ventilation was not directly measured, the subjects were clinically stable during the protocol period and the flows were administered for short time periods. The lack of any significant differences in the mean $P_aCO_2$ and pH values for each flow indicates that alveolar ventilation was unchanged during the study (Tables 2 and 3).
Table 3. pH Values before and after Tracheal Anesthesia in Patients Receiving Transtracheal Gas at Various Flows

<table>
<thead>
<tr>
<th>Subject Number</th>
<th>Zero Flow</th>
<th>Air at 2 L/min</th>
<th>Air at 4 L/min</th>
<th>O₂ at 2 L/min</th>
<th>O₂ at 4 L/min</th>
</tr>
</thead>
<tbody>
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<tr>
<td><strong>Median</strong></td>
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</table>

The $P_{aO_2}$ values during zero flow or flow of air (Table 4) were not significantly different from the baseline $P_{aO_2}$ values in Table I (zero flow, $p = 0.2239$; air flow at 2 L, $p = 0.4964$; air flow at 4 L, $p = 0.1448$).

The mean ± SD value for all VAS scores prior to tracheal anesthesia was 37 ± 17, which was not significantly different from the mean value for all VAS scores during tracheal anesthesia (36 ± 14), as collected from the Table 5 data ($p = 0.9732$). In addition, because of the relatively small sample size and large standard deviations, we analyzed the chance of a Type II error: failing to detect a difference between the VAS scores before tracheal anesthesia and those during anesthesia (Tables 5 and 6) when a difference may have actually existed. Using an alpha value of 0.05, we calculated the Type II error for the difference between the groups, which was a value of 1.0, and arrived at a beta value of 0.0027, or 0.27%. This suggests that the likelihood of a Type II error is small and that no significant difference exists between these groups.

**Discussion**

Our study was modeled on the experimental design of Liss and Grant, who investigated the effect of nasal gas flow on dyspnea in subjects with COPD who required oxygen therapy. Liss and Grant prospectively studied eight subjects, who experienced a reduction in dyspnea during nasal cannula gas flow; they found no significant effect of inspired oxygen concentration, gas flowrate, $P_{aO_2}$, or $P_{aCO_2}$ on the degree of dyspnea. However, they did find a significant increase in their subjects' dyspnea after the induction of nasal anesthesia with lidocaine spray and jelly. Liss and Grant concluded that the reduction in their subjects' dyspnea during nasal cannula gas flow was a placebo phenomenon caused by the wearing of the nasal cannula, and that it was unrelated to gas...
Table 4. \( \text{P}_{\text{aO}_2} \) before and after Tracheal Anesthesia in Patients Receiving Transtracheal Gas at Various Flows

<table>
<thead>
<tr>
<th>Subject Number</th>
<th>Zero Flow</th>
<th>Air at 2 L/min</th>
<th>Air at 4 L/min</th>
<th>( \text{O}_2 ) at 2 L/min</th>
<th>( \text{O}_2 ) at 4 L/min</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Before Tracheal Anesthesia</strong></td>
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<td>Mean ± SD</td>
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<td>52 ± 6</td>
<td>67 ± 8</td>
<td>78 ± 14</td>
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<td>Mean ± SD</td>
<td>52 ± 5</td>
<td>49 ± 4</td>
<td>51 ± 5</td>
<td>64 ± 8</td>
<td>69 ± 12</td>
</tr>
</tbody>
</table>

flowrate or to increased \( \text{P}_{\text{aO}_2} \). They hypothesized that the low flowrates of gas through the nasal canulas and the warm temperature of the gas were below the stimulation thresholds needed to sufficiently activate nasal receptors to produce effects on alveolar ventilation and dyspnea.

Before we discuss our findings, we will note several limitations of our study. The number of subjects was small, and they were studied at rest, which did not allow the assessment of TTOC use during exercise and its effect on dyspnea with exercise. In addition, we did not use a control group of measurements made with the transtracheal application of saline. This was omitted in an attempt to keep the protocol short and to minimize the number of arterial blood gas samples taken.

In our investigation, we were able to demonstrate that the perception of dyspnea was significantly influenced by tracheal gas flow via the TTOC. This effect was abolished after the trachea had been anesthetized with topical 1% lidocaine. The lack of difference between the overall VAS scores before tracheal anesthesia and those during anesthesia (Tables 5 and 6) suggests that the improvement in dyspnea reported by these patients with their TTOCs may not be a placebo effect of having their TTOCs in place. If the sensation of dyspnea were improved by the wearing of the TTOC alone, then we would expect significantly higher VAS scores (indicating greater dyspnea) after the induction of anesthesia, which was the finding of Liss and Grant in their study of nasal cannula usage.

The tracheobronchial tree contains irritant, stretch, and J-receptors, with the irritant receptors also termed rapidly adapting stretch or cough receptors. These irritant receptors appear to be most densely located in the large airways and differ from the nasal receptors involved in the study by Liss and Grant. Irritant-receptor stimulation has been implicated in the bronchoconstriction associated with asthma and in
Table 5. Dyspnea Scores from Visual Analog Scale before and after Tracheal Anesthesia in Patients Receiving Transtracheal Gas at Various Flows*  

<table>
<thead>
<tr>
<th>Subject Number</th>
<th>Zero Flow</th>
<th>Air at 2 L/min</th>
<th>Air at 4 L/min</th>
<th>O₂ at 2 L/min</th>
<th>O₂ at 4 L/min</th>
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<td>30 ± 13</td>
<td>44 ± 19</td>
<td>35 ± 13</td>
<td>48 ± 19</td>
</tr>
</tbody>
</table>

| After Tracheal Anesthesia |           |                |                |               |               |
| 1              | 52        | 47             | 35             | 25            | 27            |
| 2              | 70        | 50             | 60             | 40            | 50            |
| 3              | 35        | 30             | 30             | 35            | 30            |
| 4              | 50        | 35             | 35             | 25            | 20            |
| 5              | 35        | 40             | 25             | 25            | 30            |
| 6              | 40        | 35             | 80             | 40            | 50            |
| 7              | 30        | 35             | 35             | 30            | 30            |
| 8              | 12        | 12             | 20             | 12            | 12            |
| 9              | 40        | 50             | 40             | 40            | 40            |
| Mean ± SD      | 40 ± 16   | 37 ± 12        | 40 ± 19        | 30 ± 10       | 32 ± 13       |

*Score numbers reflect the subjects' rating of their dyspnea on a 20-cm-long visual analog scale, from 0 to 100, with 0 = not short of breath and 100 = extremely short of breath.

alterations of breathing patterns in patients with COPD.¹⁴ One explanation for the direct effect of tracheal gas flow on the degree of dyspnea in our patients may be that the threshold for tracheal irritant-receptor stimulation is different than that for the nasal receptors. This difference in threshold stimulation may explain why similar flows of gas in the nose and trachea produce different results in the sensation of dyspnea.

Nasal receptors have been shown to be flow-sensitive. McBride and Whitelaw demonstrated that the regular involuntary inspiratory-muscle contractions that occur in normal, conscious men during breath-holding could be reproducibly inhibited by a stream of cool air circulated through the nose.¹⁵ In another study, Burgess and Whitelaw demonstrated that the ventilatory response to carbon dioxide could be reduced by the nasal breathing of cold air.¹⁶ They concluded that the inhibition of respiratory output occurred by nasal-receptor stimulation by the cold air and was not due to changes in nasal or bronchial resistance. These studies suggest that nasal flow-sensitive and temperature-sensitive receptors exist that can modify ventilatory patterns. Similar studies of tracheobronchial receptors have not been done.

In our investigation, we used airway anesthesia to mask the effect of gas flowrate on tracheal-receptor stimulation. In normal subjects, airway anesthesia has been shown to produce no significant effects on the breathing pattern during resting air breathing.¹⁷ In patients with stable COPD, bilateral vagal block by lidocaine also did not appear to change the breathing
TRANSTRACHEAL GAS ADMINISTRATION AND DYSPNEA

Table 6. Grouped Dyspnea Scores (Mean ± SD) from Visual Analog Scales*

<table>
<thead>
<tr>
<th>All Scores before Anesthesia</th>
<th>All Scores after Anesthesia</th>
<th>p Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>37 ± 17</td>
<td>36 ± 14</td>
<td>0.9732</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>All Scores before Anesthesia (Flowrate 2 L/min)</th>
<th>All Scores after Anesthesia (Flowrate 4 L/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>32 ± 13</td>
<td>49 ± 19</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>All Scores after Anesthesia (Air Gas Source)</th>
<th>All Scores after Anesthesia (Oxygen Gas Source)</th>
</tr>
</thead>
<tbody>
<tr>
<td>39 ± 15</td>
<td>31 ± 11</td>
</tr>
</tbody>
</table>

*VAS = Visual analog scale, 20-cm long, for recording dyspnea, from 0 (not short of breath) to 100 (extremely short of breath).

pattern significantly. However, in patients with COPD and acute respiratory failure, lower-airway anesthesia with lidocaine has been shown to lead to rapid and shallow breathing, with a greater degree of dyspnea and deterioration in blood-gas values.

The effect of airway anesthesia in our subjects was two-fold. In two of our nine subjects, dyspnea seemed to acutely worsen after the induction of tracheal anesthesia; this was transient, lasting less than 5 minutes. In addition, tracheal anesthesia appeared to mask the effect of airflow stimulation on the tracheal-irritant receptors. This effect of local anesthesia in blocking the input to the flow-sensitive tracheal receptors is similar to that demonstrated for the nasal flow-sensitive receptors by McBride and Whitelaw.

We found no significant effect of inspired oxygen concentration, P\textsubscript{aO\textsubscript{2}}, or P\textsubscript{aCO\textsubscript{2}} on dyspnea in our subjects. However, during tracheal anesthesia there was a trend for the dyspnea scores to be higher when there was no flow or when air was flowing than when oxygen was flowing; however, this was not statistically significant. Table 5 indicates that following tracheal instillation of lidocaine, the zero-flow dyspnea index (40 ± 16) was substantially higher than the zero-flow dyspnea index before administration of lidocaine (28 ± 11). The higher zero-flow VAS scores associated with anesthesia may be an important reason for the fact that there was no flow-related rise in the dyspnea index during anesthesia. However, the VAS scores for air and oxygen at 4 L/min and air and oxygen at 2 L/min (Table 5) are not significantly different (p = 0.9618). This suggests that the tracheal administration of lidocaine did actually abolish the effects of gas flow rate on dyspnea.

The reason for the rise in the zero-flow dyspnea index after tracheal anesthesia is unclear. One possible explanation may be the residual numbness in the trachea or mouth if lidocaine was coughed up. Such a sensation of numbness may have upset the patients, leading to the greater sensation of dyspnea at zero flow.

The perception of dyspnea may be affected by many variables in patients receiving transtracheal oxygen cannula therapy. Their overall self-image may be improved with TTTOC therapy, producing psychological effects on dyspnea. Recently, two studies have demonstrated that TTTOC will decrease minute ventilation in patients with chronic lung disease.

In one of these studies, improved exercise tolerance was also described with TTTOC therapy compared to mouth-breathing. This decrease in minute ventilation may be associated with a decrease in the work of breathing and be one mechanism for the relief of dyspnea that has been described by other investigators in patients receiving TTTOC therapy.

In conclusion, our results differ from those found by analyzing the effects of nasal gas flow on dyspnea. In our subjects, tracheal gas flow rate did affect the perception of breathlessness. The clinical importance of this finding is unknown, as the flow rates used in this study were higher than those often used clinically with TTTOC therapy. Further studies are indicated to directly compare nasal oxygen therapy with TTTOC therapy prospectively to assess their effects on dyspnea in hypoxemic patients with chronic lung disease—and to examine the effects of other properties of tracheal gas administration on dyspnea.

PRODUCT SOURCES

Transtracheal Oxygen Catheters:
SCOOP-1, Transtracheal Systems, Denver CO

Blood Gas Analyzer:
Model 178, Ciba-Corning Diagnostics Corp, Medfield MA

Spirometer:
Collins Waterseal, Warren F. Collins Inc, Braintree MA
Lidocaine:
Xylocaine, Astra Pharmaceutica Products Inc,
Westborough MA

Pulse Oximeter:
Lifestat 1600, Physio-Control Corp, Redmond WA

ACKNOWLEDGMENTS
We thank Joyce Jones for her aid in the preparation of the manuscript, Robert Browning for his statistical support, and the nursing staff of the medical intensive care unit for their assistance.

REFERENCES
An Evaluation of Volumes Delivered by Selected Adult Disposable Resuscitators: The Effects of Hand Size, Number of Hands Used, and Use of Disposable Medical Gloves

Dean Hess MEd RRT and Christopher Spahr BS CRTT

Due to increasing concern over potential cross-infection during cardiopulmonary resuscitation (CPR), a number of disposable resuscitators have become commercially available. The wearing of disposable medical gloves by persons performing CPR has also become commonplace. In this study, we evaluated the effects of hand size, use of disposable medical gloves, and number of hands used (one versus two) on the volumes delivered by five adult disposable resuscitators. METHOD: Persons familiar with bag-valve ventilation were recruited to participate in the study—eight with small hands, eight with medium hands, and eight with large hands. Ventilation was delivered to one side of a Vent-Aid training test lung (TTL), and volumes were measured with a BEAR VM-90. In random order, each participant ventilated the TTL with all combinations of one hand/two hands, gloves/no gloves, and each of the following resuscitators: Code Blue, Hospitak, Pulmanex, Mercury, and Ambu SPUR. The participants were instructed to ventilate the TTL as they would ventilate a patient. RESULTS: The mean ± SD volumes (in liters) were small hands = 0.68 ± 0.15, medium hands = 0.71 ± 0.18, large hands = 0.81 ± 0.19 (p = 0.006); gloves = 0.73 ± 0.19, no gloves = 0.73 ± 0.18 (p = 0.80); one hand = 0.62 ± 0.12, two hands = 0.84 ± 0.17 (p < 0.0001; Code Blue = 0.79 ± 0.14, Hospitak = 0.56 ± 0.11, Pulmanex = 0.71 ± 0.15, Mercury = 0.77 ± 0.18, SPUR = 0.83 ± 0.2 (p < 0.0001). CONCLUSIONS: The use of gloves did not significantly affect volume delivery. Delivered volumes did increase significantly as hand size increased and as number of hands used to squeeze the bag increased, and observed differences in volume delivery between brands of resuscitators may be clinically important in some cases. This study emphasizes the importance of squeezing the resuscitator with two hands during bag-valve ventilation. (Respir Care 1990;35:800-805.)

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A version of this paper was presented by Mr Hess during the Respiratory Care Open Forum at the 35th Annual Convention of the American Association for Respiratory Care, December 2-5, 1989, in Anaheim, California.

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VARIABLES AFFECTING RESUSCITATOR VOLUMES

Introduction

Bag-valve resuscitators are used with adult patients during resuscitation, for hyperinflation during tracheal succioning, and to ventilate during patient transport. A recent study from our laboratory evaluated the effects of hand size, resuscitator brand, and the efficacy of two hands on the volumes delivered by adult bag-valve resuscitators.¹ In that study we evaluated nine adult resuscitators, including several disposable resuscitators. Due to the increasing concern over potential cross-infection during bag-valve ventilation, use of disposable resuscitators has become popular in the hospital and for emergency care in the field. It has also become common practice for caregivers to wear disposable medical gloves during bag-valve resuscitation, but the effect of wearing gloves on bag-valve resuscitator volume delivery has not been reported. This study evaluated the effects of hand size, use of medical gloves, and number of hands used to squeeze the bag.

Methods

We recruited 24 persons to participate in the study who were either respiratory care practitioners or nurses trained in adult critical care. All participants had been certified in Basic Cardiac Life Support by the American Heart Association (AHA), and were familiar with the use of bag-valve resuscitators. We specifically recruited eight persons with small hands, eight persons with medium hands, and eight persons with large hands. Hand size was determined by best right-hand fit into a medical glove (small: 6.5–7, medium: 7–8, large: 8–9).* The study design was modeled after a previously reported study of ours.¹ Ventilation was provided to one side of a Vent-Aid Training Test Lung (TTL). A compliance of 0.05 L/cm H₂O [0.51 L/kPa] was set on the test lung, and a 7-mm-ID endotracheal tube was fitted to the test lung. Volumes were measured with a BEAR VM-90 vortex pneumotachometer, the accuracy of which was confirmed at 1.0 L by use of a calibration syringe. The same volume monitor was used for all measurements. The TTL volume indicator was removed, and the VM-90 was positioned so that the participants could not see what volumes they were delivering.

Each participant used one hand and two hands, with and without wearing medical gloves, with each of five bags evaluated (Code Blue, Hospitak, Pulmanex, Mercury, and SPUR). The order of testing was randomized, and each subject participated in all levels of each aspect of the study. Because many of the participants were unfamiliar with some of the resuscitators, they were encouraged to practice using them before testing. The only instruction given to the participants was to ventilate the TTL as they would ventilate a patient. Volume and rate were measured for one minute, and tidal volume was calculated by dividing minute volume by rate. The participants were encouraged to rest as necessary between measurements to avoid fatigue.

The effects of hand size, use of gloves, number of hands used, and resuscitator brand were evaluated by a multifactorial (4-way) analysis of variance. Hand size was used as a between-groups factor. Number of hands used, use of gloves, and resuscitator brand were used as within-groups (repeated measures) factors. Post-hoc analyses were performed using the Scheffe test. A p value ≤ 0.05 was considered statistically significant. For p > 0.05, beta (power) analysis was performed to evaluate the power of the

Abbreviations Used in this Paper

AHA — American Heart Association
ASTM — American Society for Testing and Materials
CPR — Cardiopulmonary resuscitation
TTL — Training test lung

A Guide to the Use of SI in This Paper*

The SI unit for compliance is liters per kilopascal (L/kPa).

(L/cm H₂O)(10.20) = L/kPa.

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

*Suppliers are identified in the Product Sources Section at the end of the text.
VARIABLES AFFECTING RESUSCITATOR VOLUMES

analysis. Using pooled data, the mean and standard deviation (SD) were calculated for the tidal volumes delivered by the levels of each major factor (hand size, resuscitator brand, use of gloves, and number of hands used). Mean and standard deviation were also calculated for the cells of all interactions. Statistical analyses were performed using commercially available statistical analysis software.

Results

The results are summarized in Table 1 and Figures 1-5. Volume delivery was significantly affected by hand size (small hands = 0.68 ± 0.15 L, medium hands = 0.71 ± 0.18 L, large hands = 0.81 ± 0.19 L, p = 0.006); resuscitator brand (Code Blue = 0.79 ± 0.14 L, Hospitak = 0.56 ± 0.11 L, Pulmanex = 0.71 ± 0.15 L, Mercury = 0.77 ± 0.18 L, SPUR = 0.83 ± 0.2 L, p < 0.0001); and number of hands used to squeeze the bag (one hand = 0.62 ± 0.12, two hands = 0.84 ± 0.17, p < 0.0001). The only significant interaction effects were between hand size and resuscitator brand (p = 0.021) and resuscitator brand and number of hands used (p < 0.0001).

There were significant differences between the volumes delivered by small hands and large hands, and between medium hands and large hands (p < 0.05 by Scheffe analysis). There was no significant difference between the volumes delivered by small hands and medium hands. The volumes

Table 1. The Effects of Hand Size, Number of Hands Used, and Use of Gloves on Volumes Delivered by Selected Adult Disposable Resuscitators*

<table>
<thead>
<tr>
<th>Hand Size</th>
<th>Number of Hands Used</th>
<th>Use of Gloves</th>
<th>Resuscitator Brand</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Code Blue</td>
</tr>
<tr>
<td>small</td>
<td>one</td>
<td>yes</td>
<td>0.65 ± 0.02</td>
</tr>
<tr>
<td>small</td>
<td>one</td>
<td>no</td>
<td>0.67 ± 0.02</td>
</tr>
<tr>
<td>small</td>
<td>two</td>
<td>yes</td>
<td>0.85 ± 0.05</td>
</tr>
<tr>
<td>small</td>
<td>two</td>
<td>no</td>
<td>0.84 ± 0.06</td>
</tr>
<tr>
<td>medium</td>
<td>one</td>
<td>yes</td>
<td>0.64 ± 0.10</td>
</tr>
<tr>
<td>medium</td>
<td>one</td>
<td>no</td>
<td>0.67 ± 0.10</td>
</tr>
<tr>
<td>medium</td>
<td>two</td>
<td>yes</td>
<td>0.91 ± 0.13</td>
</tr>
<tr>
<td>medium</td>
<td>two</td>
<td>no</td>
<td>0.88 ± 0.11</td>
</tr>
<tr>
<td>large</td>
<td>one</td>
<td>yes</td>
<td>0.74 ± 0.06</td>
</tr>
<tr>
<td>large</td>
<td>one</td>
<td>no</td>
<td>0.77 ± 0.06</td>
</tr>
<tr>
<td>large</td>
<td>two</td>
<td>yes</td>
<td>0.95 ± 0.10</td>
</tr>
<tr>
<td>large</td>
<td>two</td>
<td>no</td>
<td>0.95 ± 0.09</td>
</tr>
</tbody>
</table>

*All volumes are in liters (L) and represent mean ± SD values.
delivered by the following pairs of resuscitators were significantly different (p < 0.05 by Scheffe analysis): Hospitak vs Code Blue, Hospitak vs Pulmanex, Hospitak vs Mercury, Hospitak vs SPUR, Pulmanex vs SPUR, and Pulmanex vs Code Blue.

There were no significant differences between the volumes delivered by gloved and ungloved hands (gloves = 0.73 ± 0.19, no gloves = 0.73 ± 0.18, p = 0.80). For this nonsignificant difference in volume delivery, beta was < 0.05 for a difference of 0.1 L between volumes delivered by gloved and ungloved hands.

**Discussion**

A number of studies published in the past 15 years have evaluated bag-valve resuscitators.\(^1\)\(^-\)\(^9\) Although the primary emphasis of some of these has been the oxygen concentration delivered, some have evaluated volume delivery, and others have evaluated the compliance of resuscitators to standards published by the American Society for Testing and Materials (ASTM). The ASTM recommends that adult resuscitators deliver a minimum volume of 0.6 L.\(^10\)

The AHA, however, recommends a tidal volume of 0.8-1.2 L be delivered during adult resuscitation.\(^11\)

The results of this study are consistent with previous studies that have shown that the volumes delivered by a resuscitator can be increased by using two hands (instead of one) to squeeze the bag.\(^1\)\(^,\)\(^2\)\(^,\)\(^12\) Our finding that hand size affects resuscitator volume delivery is also consistent with previously published work.\(^1\)\(^,\)\(^13\)\(^,\)\(^14\)

With small and medium hands, none of the resuscitators delivered a tidal volume over 0.8 L with...
a one-hand squeeze. With large hands, only the SPUR delivered tidal volumes greater than 0.8 L with a one-hand squeeze. With small and medium hands, only the Hospitak and Pulmanex failed to deliver greater than 0.8 L with a two-hand squeeze. Even with large hands, the Hospitak failed to deliver greater than 0.8 L with a two-hand squeeze.

In this study we found considerable variation in the volumes delivered by various brands of resuscitators; this finding was previously reported in the comparison of another group of resuscitators.1 There was nearly a 300-mL difference between the average volume delivered by the SPUR (0.83 L) and the Hospitak (0.56 L). As seen in Table 1, the volumes delivered by the Hospitak were consistently less than those delivered by the other resuscitators.

We recommend that the volumes reported in a previous study1 not be strictly compared to the volumes reported in this study. Although similar methodology was used in both studies, different subjects (therapists and nurses) were used. The data from both studies may be useful when comparing the performance differences of the various brands.

Because of infection control concerns, gloves are commonly worn when bag-valve resuscitators are used. The results of this study indicate that use of gloves does not grossly affect the volume delivery of bag-valve resuscitators. The subjects in this study wore gloves of the proper size (comfortable fit). It is possible that ill-fitted gloves might affect resuscitator volume delivery, but we did not study this.

The only significant interaction effects were between hand size and resuscitator brand, and between resuscitator brand and number of hands used to squeeze the bag. This means that there were differences among resuscitators and the volumes delivered by the three hand sizes, and that there were differences among resuscitators regarding the increase in delivered volumes between use of one hand and two hands to squeeze the resuscitator. However, a clinically important increase in volume delivery was observed with each of the devices as hand size increased, and when two hands (instead of one) were used to squeeze the resuscitator.

This paper re-emphasizes the importance of verifying volumes delivered during bag-valve resuscitation. On the average, persons with small and medium hands did not deliver tidal volumes that met AHA standards. Further study is required to determine whether bag-valve resuscitation produces hyperinflation during adult tracheal-suctioning procedures. When bag-valve resuscitators are used during patient transport, it might be useful to use a portable respirometer to ensure adequate tidal volume delivery. The results of this study might also help to explain why the volumes delivered by a one-person adult bag-valve-mask technique are often less than ideal.15-20

Airway resistance and lung compliance are also factors that can affect resuscitator volume delivery. A previous study7 demonstrated that volumes delivered during bag-valve ventilation decreased as airway resistance increased and as lung compliance decreased.

The ASTM has recommended a method to determine hand-size when evaluating the volumes delivered by bag-valve resuscitators.10 The ASTM method is used to assure that the person evaluating a resuscitator has a 'standard-size' hand. Medium hand-size by our method is roughly the ASTM standard dimensions. In this study, although we tried to use the ASTM method of measuring hand size, we found it to be very cumbersome, and in our opinion it added no advantage to the glove size method that we used.

Conclusions

The use of gloves when squeezing the resuscitators did not significantly affect volume delivery. There was a significant increase in delivered volume as hand-size increased and when two hands were used (instead of one) to squeeze the bag. There were also significant differences among the volumes delivered by resuscitators of different brands, which might be clinically important in some cases. This study re-emphasizes the importance of using two hands to squeeze the resuscitator during bag-valve ventilation.

PRODUCT SOURCES

Bag-Valve Resuscitators:
Code Blue, Vital Signs Inc, Totowa NJ
Hospitak, Hospitak Inc, Lindenhurst NY
Pulmanex, Life Design Systems, Carrollton TX
VARIABLES AFFECTING RESUSCITATOR VOLUMES

Mercury, Mercury Medical Inc, St Petersburg FL
Ambu SPUR, Ambu Inc, Hanover MD

Medical Gloves:
Medium and large sizes
Travenol Laboratories Inc, Deerfield IL
Small size
Becton Dickinson, Rutherford NJ

Test Lung:
Vent-Aid, Michigan Instruments, Grand Rapids MI

Spirometer:
VM-90, Bear Medical Systems, Riverside CA

Calibration Syringe:
Jones Medical Instrument Co, Oak Brook IL

Statistical Software:
SPSS/PC+, SPSS Inc, Chicago IL

REFERENCES

Extended Use of Prefilled Humidifier Reservoirs and the Likelihood of Contamination

David Seigel BS RRT and Bernice Romo MS

We sought to determine the potential for disposable prefilled humidifiers to become contaminated during extended patient use. METHODS & MATERIALS: We sampled the water in 55 humidifier reservoirs, which were being used by patients or which had been used by patients and then been placed on ‘standby,’ and had the samples cultured by a commercial clinical laboratory. RESULTS: None of the humidifiers showed contamination after 72 hours of continuous patient use, and 33 of the 55 were used by patients for an additional 3-9 days with no contamination. Fifteen humidifiers that had been in use on patients were put in a standby mode for 30-32 days, with 5 continuing for 60-62 days, with no contamination seen. CONCLUSIONS: An automatic 72-hour change-out for prefilled humidifiers used with low-flow oxygen (\( \leq 4 \text{ L/min} \)) is wasteful. Our study demonstrates that the use-time of humidifiers can be safely extended to as long as 6-12 days. (Respir Care 1990;35:806-810.)

Introduction

The prevention of nosocomial pneumonia is of major concern to the respiratory care practitioner in light of incidence rates for lower respiratory tract infection of 5.5/1000 hospital discharges. Water and the associated equipment used to deliver therapeutic gases are an excellent potential source of bacterial contamination especially from gram-negative aerobic bacilli, including *Pseudomonas* and *Klebsiella* species, that are known to cause nosocomial pneumonia.

The mechanisms by which prefilled water reservoirs for nebulizers and humidifiers can become contaminated are speculated to include (1) the oxygen or compressed air that powers the device, (2) the unwashed hands of hospital personnel handling the device, (3) contaminated water used to fill the reservoir, (4) reflux of contaminated condensate into the reservoir, and (5) inadequate sterilization or disinfection of associated equipment. The concern over contaminated aerosols generated by nebulizers has raised questions about the potential for the contamination of effluent gas by bubble-humidifier-generated microaerosols capable of carrying bacteria.

The use of prefilled reservoirs of sterile water for humidifying gases to be inhaled has eliminated the risk of contamination from organisms originating from improperly functioning distillation units or nonsterile reusable systems. The procedure used to assemble a system that incorporates a prefilled sterile reservoir could contribute contamination, but the possibility of this occurring is low because the hands do not touch the water. Once a prefilled humidifier has been set up for use, the potential sources of contamination of the unit become the patient and the gas being bubbled through the humidifier. Hospital policies have developed around infection-control measures with use-times and change-out periods of nebulizers set at 24 hours based on growth of contaminants in these systems. We have observed that a humidifier change-

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This study, which was conducted at Humana Hospital-West Hills, West Hills, California, was funded by Baxter Healthcare Corp.

Reprints: Bernice Romo, Baxter Healthcare Corp, Pharmaseal Div, 27200 N Tourney Rd, Valencia CA 91355.
out time of 72 hours appears to be a common practice. This practice is supported by studies conducted by Koss et al. and Stoler in which they concluded that the potential for contamination of humidifiers is low up to 72 hours.

When bubble humidifiers are used for low-flow oxygen delivery ($\leq 4 \text{ L/min}$), often a considerable portion of the water remains in the reservoir at the end of the 72-hour period of use. This has been perceived as wasteful, and the cost-effectiveness of and the need for humidification have been challenged based on subjective evaluations of benefit including patient comfort.9-11

Guidelines from the Centers for Disease Control suggest that disposable reservoirs for use with wall oxygen outlets may be safe for long periods. However, it is not known whether the reservoirs need to be changed routinely before they are empty.2

We conducted this study to evaluate, at 72-hour intervals, a disposable prefilled bubble humidifier's potential for becoming contaminated after assembly for patient use. The assembly procedure, during which the water bottle is opened and the sterile cap is screwed in place, has been characterized as an 'open' system as contrasted with so-called closed systems that require that a sterile connector puncture the clean top of a sterile bottle. Further, we sought to determine whether the open-system humidifier for continuous or intermittent low-flow use is safe up to 144 hours (6 days) or until the water supply is exhausted.

### Materials and Methods

Of 115 patients admitted to Humana Hospital-West Hills between June 12 and September 12, 1989, for whom low-flow humidified oxygen was prescribed, 55 met the minimum requirement of having used their oxygen systems continuously for at least 72 hours and were therefore enrolled in this study. The patient population included both medical and postsurgical patients (Table 1). Prescribed oxygen flow rates ranged from 0.5-5 L/min, with 36 of the 55 units (65.5%) run at 2-3 L/min, 8 units (14.5%) at $< 2 \text{ L/min}$, and 11 (20%) at $\geq 4 \text{ L/min}$. Of the 55 patients, 33 completed an additional 3-9 days of intermittent use with 72-hour contamination-level checks (Table 2).

Each humidifier unit tested consisted of a 500-mL bottle of sterile water for inhalation and a sterile humidifier cap.* A nasal oxygen cannula was attached to the humidifier (Fig. 1). As each humidifier was assembled for patient use, the flowmeter and quick-connect adapter were kept as a unit on the humidifier bottle. These humidifiers were not disassembled until empty or until the patient's oxygen was discontinued and the patient consequently left the study. A unit was considered 'empty' when the water level in the

| Pulmonary | 22 |
| COPD | 6 |
| Pneumonia | 5 |
| Lung carcinoma, advanced | 5 |
| Pulmonary infiltrate | 2 |
| Pleural effusion | 1 |
| Bronchospasm | 1 |
| Asthmatic bronchitis | 1 |
| Status asthmaticus | 1 |

| Kidney-Urinary Tract | 7 |
| Urosepsis | 3 |
| Renal failure | 3 |
| Hematuria | 1 |

| Skeletal | 2 |
| Hip replacement | 1 |
| Fractured hip | 1 |

| Cardiovascular | 15 |
| Congestive heart failure | 6 |
| Atrial fibrillation | 2 |
| Myocardial infarction | 2 |
| Angina | 2 |
| Cardiovascular accident | 2 |
| Coronary artery disease | 1 |

| Blood & Circulation-Related | 4 |
| Sepsis or septic shock | 2 |
| Anemia | 1 |
| Gastrointestinal bleeding | 1 |

| Other | 5 |
| Dehydration | 1 |
| Hypothermia | 1 |
| Fever, unknown origin | 1 |
| Obstructive jaundice | 1 |
| Hypoxemia, severe | 1 |

*Suppliers are identified in the Product Sources section at the end of the text.
bottle reached the bottom ribs of the container (ie, about 1 inch of water remained).

The first 22 humidifiers used in the study were cultured by swabbing the inlet port at the cannula connection prior to their being placed in use. After the results for these 22 units had been returned with a no-growth report, we decided that the remaining units need not be cultured prior to being put into patient use. This sample verified that the assembly procedure did not contribute to contamination. (Sterility of the contents of the unopened reservoirs is to be assumed from the manufacturer's quality control measures.) Each humidifier unit was sampled as follows: (1) After the patient's nasal oxygen cannula had been disconnected from the humidifier, 3-5 mL of humidifier water was poured from the cap connector into a sterile specimen cup; (2) the sample was immediately aspirated into a urine culture-and-sensitivity transport kit (containing preservative); and (3) the sample was labeled and sent to a commercial clinical laboratory for an environmental culture evaluation. A clean nasal cannula was placed on the unit, and the unit was held in 'quarantine.' After 48 hours of incubation, the sample for culture was read by laboratory personnel and the report was sent to us. When a no-growth report was received, the unit that had been held in quarantine was placed back in service on the patient. This sampling procedure was repeated at 72-hour intervals until the water in a unit was exhausted or the patient's oxygen therapy was discontinued.

Fifteen humidifiers that had been in use for 3 days were evaluated for level of contamination in a standby, or assembled, ready-to-use mode. These units were sampled for level of contamination as described, at 72-hour intervals for at least 30 days. An additional 5 units were evaluated for level of contamination for at least 60 days.

The 95% one-sided confidence limits for the 72- and 144-hour contamination rates were calculated using the cumulative probability curves for a Poisson variable with zero failures. For the standby units, a conservative method of estimating the one-sided confidence limit when there are no failures is to assume one failure (R = 1). The exponential model was used to estimate the probability of survival (ie, the unit remains uncontaminated) of the standby units because it predicts the survival of the product as a function of time.

Results

After continuous patient use of at least 72 hours, all 55 study humidifiers demonstrated no growth. Thirty three of these units remained in the study for extended-use evaluation, with all samples demonstrating no growth at 72-hour sampling intervals for a minimum of 6 days (144 hours) and a maximum of 12 days. One can state with 95% confidence that at least 94% of the units will not be contaminated after 72 hours of continuous use and that at least

### Table 2. Use-Times for the 55 Humidifiers Studied

<table>
<thead>
<tr>
<th>Days</th>
<th>No. of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>16</td>
</tr>
<tr>
<td>4</td>
<td>4</td>
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<tr>
<td>5</td>
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<td>8</td>
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<td>9</td>
<td>6</td>
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<td>10</td>
<td>0</td>
</tr>
<tr>
<td>11</td>
<td>2</td>
</tr>
<tr>
<td>12</td>
<td>1</td>
</tr>
</tbody>
</table>

Fig. 1. Humidifier setup as prepared for patient application.
Table 3. 95% Confidence-Interval Estimate for True Contamination Rate for Humidifiers

<table>
<thead>
<tr>
<th>Test Period (h)</th>
<th>No. of Units Tested</th>
<th>No. of Units Growth</th>
<th>Percentage not Contaminated</th>
</tr>
</thead>
<tbody>
<tr>
<td>72</td>
<td>55</td>
<td>0</td>
<td>≥ 94.5%</td>
</tr>
<tr>
<td>144</td>
<td>33</td>
<td>0</td>
<td>≥ 91.0%</td>
</tr>
</tbody>
</table>

The 95% confidence limits for the failure rate was calculated from cumulative probability curves.12,13

91% will remain uncontaminated with extended use on the same patient (Table 3).

A total of 15 units in the standby mode demonstrated no growth for an average of 27 days with 72-hour sampling intervals. An additional 5 units were evaluated for an extended period of time, 3 units demonstrated no growth after 60 days and 2 units after 62 days (Fig. 2). Although no failures were observed, the lower confidence limit of the mean time to failure was calculated (Table 4) by assuming one failure (as if the next unit evaluated would be positive). By assuming one failure, the estimate of the 95% lower confidence limit of the average time to contamination for standby units is at least 195 days. The probability of a unit’s surviving without contamination in standby use for 3 days is at least 98.4%. For a unit in use for 10 days, the probability of survival without contamination is at least 95%.

Table 4. Probability of Standby Humidifiers Remaining Uncontaminated

<table>
<thead>
<tr>
<th>Days in Standby Use</th>
<th>Probability Remaining Uncontaminated*</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>≥ 98.4%</td>
</tr>
<tr>
<td>10</td>
<td>≥ 95.0%</td>
</tr>
<tr>
<td>15</td>
<td>≥ 92.5%</td>
</tr>
<tr>
<td>30</td>
<td>≥ 85.7%</td>
</tr>
<tr>
<td>60</td>
<td>≥ 73.5%</td>
</tr>
</tbody>
</table>

*Assumes one failure to estimate probability.11 Based on exponential failure model with \( m = 195 \) days.

\[ p = e^{-t/m} \]

\( e = \) natural logarithm. \( t = \) total time of survival in days.

\( m = \) mean time to failure. \( y = \) test time for a unit.

\( \alpha = \) significance level of 2 degrees of freedom.

\[ m = \frac{2(y_1 + \ldots + y_n)}{X^2(\alpha;2)} \]

Discussion

In order to achieve a greater confidence limit or to assure that the defect rate is below 1% (ie, that 99% of the units will not be contaminated) with 95% assurance, the sample size would need to be increased to 300 units, with no incidence of failure.13 A < 0.1% defect rate (ie, 99.9% of the units will not be contaminated) with a 95% assurance, would require a sample size of at least 1000 units without a failure.14

The CDC Guidelines suggest that oxygen humidifiers can be used for long periods (beyond 72 hours). This study confirms that premise and suggests that water reservoirs on low-flow oxygen units remain uncontaminated for at least 6 and up to 12 days. In our study, humidifiers on standby did not become contaminated for up to 60 days, even when they had previously been used by a patient. Further, our study suggests that no contamination results from use of the open assembly procedure from setup and that this potential source of contamination is probably negligible.

Conclusions

Investigators have questioned the use of humidifiers for low-flow nasal oxygen delivery by nasal cannula
because of cost and questionable benefit based on subjective evaluation for patient comfort. Our study demonstrated that it is a safe practice to use humidifiers intermittently until they are empty and that a 72-hour change-out period can be safely extended. This extended use will eliminate the waste associated with replacing partially used units for patients on long-term therapy and will make the humidification of low-flow oxygen delivery more economical. We believe that the decision to humidify low-flow oxygen in an effort to avoid drying of nasal mucosa and secretions and the associated epistaxis should rest with the individual institution based on their own experience and their perception of individual patient needs.

ACKNOWLEDGMENTS

We thank Hernando Gonzalez for his assistance with the statistical evaluation of the data, the Respiratory Therapy Department staff at Humana Hospital-West Hills for their participation in the study, and Smith Kline Beecham Clinical Laboratories, Van Nuys, California, for environmental culture evaluation.

PRODUCT SOURCES

Prefilled Disposable Humidifiers:
Humidifier, Catalog # 2D0802, Baxter Healthcare Corp, Valencia CA

Nasal Oxygen Cannulas:
Cannula, Catalog # 001312, Baxter Healthcare Corp, Valencia CA

Culture and Sensitivity Kits:
Vacutainer Urine Collection and Sensitivity Kits, Becton Dickinson, Rutherford NJ

REFERENCES

Case Reports

Sarcoidosis in the Elderly:
Expect the Unexpected—A Case Report

Robert J Albin MD

Sarcoidosis is a common disease with radiographic findings so characteristic that the diagnosis is often initially suggested by the radiologist. However, atypical clinical or radiographic features such as hoarseness, asymmetric hilar adenopathy, atelectasis, parenchymal masses, or isolated mediastinal adenopathy may be present when the disease has its onset after the age of 50 years. The patient described in this report was initially suspected of having bronchogenic carcinoma, on the basis of her age, radiographic findings, and history of hoarseness. Despite the clinical features for carcinoma at the time of presentation, lymph-node biopsy confirmed a diagnosis of sarcoidosis. Recognition of these atypical radiographic features, coupled with supportive biopsy material, assists in arriving at the correct diagnosis in older patients and helps to avoid confusion with neoplastic or infectious etiologies. (Respir Care 1990;35:811-814.)

Introduction

Sarcoidosis is a multi-system granulomatous disease of undetermined etiology usually affecting young adults, with a mean age of onset in the third decade. The most frequent presenting findings are bilateral hilar adenopathy, pulmonary infiltration, and eye or skin lesions. Recent reports have suggested that patients over the age of 50 years presenting with sarcoidosis may have very atypical radiographic and clinical findings.¹,² This report describes a 58-year-old patient with several atypical features of sarcoidosis that initially suggested a diagnosis of neoplasm. Knowledge of the atypical clinical and radiographic features associated with older patients with sarcoidosis will help to avoid diagnostic confusion and rapidly lead to the correct diagnosis.

Case Summary

A 58-year-old white woman was referred for evaluation of an abnormal chest radiograph. Her past medical history was significant only for hoarseness three years previously. Following a sinus operation and vocal cord “stripping,” her voice returned to normal. Three months prior to presentation, she again became hoarse. She denied symptoms of an upper-respiratory tract infection, sinus drainage, fever, or dysphagia. She had no history of tobacco use and was taking no medications.

The physical examination was unremarkable. There was no palpable peripheral adenopathy or splenomegaly. Lung auscultation revealed only scattered crackles at the lung bases. There was no clubbing or cyanosis. The laboratory evaluation revealed a white blood cell count of 5800/mm³, hematocrit 39.5%, globulin 2.9 g/dL, calcium 9.4 mg/dL, and alkaline phosphatase 125 U/L. These values were all within normal limits. Chest radiographs revealed a mass in the region of the left hilum, with a more peripheral infiltrate in the left upper lobe (Fig. 1). There was lobulation along the right hilar border, which, by lateral chest radiograph, suggested hilar adenopathy (Fig. 2). An unenhanced chest CT

When this report was prepared, Dr Albin was Assistant Professor of Medicine, Division of Pulmonary and Critical Care Medicine, Department of Medicine, University of Maryland School of Medicine and Hospital, Baltimore, Maryland.

SARCOIDOSIS IN THE ELDERLY

scan revealed extensive adenopathy within the mediastinum, subcarinal fullness, a mass-like density in the region of the left hilum, and bilateral infiltrates. In addition, two pleural-based soft-tissue densities were noted in the left lung (Fig. 3).

The working diagnosis was bronchogenic carcinoma with vocal cord paralysis from recurrent laryngeal nerve involvement. At bronchoscopy, there was bilateral supraglottic fullness, but both vocal cords moved normally. The trachea was erythematous and the main carina was full, but the overlying mucosa was normal. The main-stem, lobar, and segmental bronchi were erythematous, with a "cobblestone" mucosal appearance noted in the right lower lobe. There were no endobronchial lesions. Endobronchial biopsies from the area of cobblestone mucosa revealed only severe chronic bronchitis. The bronchial washings showed cellular atypia, and cultures were negative. The patient was subsequently referred for mediastinoscopy and lymph-node biopsy.

Mediastinal lymph-node biopsies revealed noncaseating granulomata completely replacing the normal nodal architecture. Special stains and cultures for acid-fast bacilli and fungi were negative. Pulmonary

Fig. 1. PA chest radiograph demonstrates a left hilar mass, bilateral parenchymal infiltrates, and an irregularly enlarged right hilum.

Fig. 2. Lateral chest radiograph reveals fullness of both hila consistent with adenopathy.

Fig. 3. CT image just below the level of the carina demonstrates a pleural-based nodule on the left along with a mass in the aortopulmonary window.
function testing revealed a mild restrictive defect with a reduced diffusing capacity. On the basis of all these findings, the patient was diagnosed as having sarcoidosis. She was begun on systemic prednisone (0.6 mg/kg/d), with marked improvement in her dyspnea and hoarseness. A follow-up CT scan 6 months later demonstrated no changes in the sizes of the lymph nodes or nodules, and she has continued to experience improvement of her symptoms.

**Discussion**

This patient manifested several unusual features of sarcoidosis, including laryngeal involvement, parenchymal nodules, asymmetric hilar enlargement, and advanced age at the time of diagnosis. Sarcoidosis is classically a disease of young adults, with the mean age at the time of onset being in the third decade. Only 3-15% of all cases will present after the age of 50 years. However, within this group of patients, atypical radiographic patterns are frequently noted. Conant et al recently reported the radiographic findings in 29 patients 50-78 years of age who were newly diagnosed as having sarcoidosis. Only 41% had "typical" radiographs for sarcoidosis, meaning symmetric hilar adenopathy with or without right paratracheal adenopathy. In the 59% with "atypical" chest radiographs, the most common findings were atypical adenopathy (unilateral or isolated mediastinal), atelectasis, parenchymal masses, or asymmetric alveolar disease. These findings were suggestive of a diagnosis of bronchogenic carcinoma, metastatic carcinoma, lymphoma, or tuberculosis, and commonly led to a delay in establishing the correct diagnosis.

The incidence of laryngeal involvement in sarcoidosis is low, being only 1.3% in one large series. Bower and colleagues described four cases of biopsy-proven laryngeal sarcoid in 1980. As in our case, their four patients complained of hoarseness, as did 63% of the 40 other cases they reviewed. Interestingly, 28% of these 44 patients had no evidence of disease beyond the larynx. The differential diagnosis for granulomatous laryngitis includes blastomycosis, histoplasmosis, tuberculosis, syphilis, leprosy, and actinomycosis. This diagnostic differential stresses the importance of submitting biopsy materials for culture. These authors cautioned that high-grade upper-airway obstruction and asphyxiation may occur in these patients and warrants close observation for this complication. Systemic corticosteroids have been the mainstay of therapy, although the use of intralesional steroids, radiation therapy, and surgical excision of localized, obstructing lesions has been reported. Spontaneous remission has also been noted.

Nodular pulmonary sarcoidosis occurs in only 2-4% of all cases. The nodules vary in size, ranging from 1 to 4 cm. They tend to be well circumscribed, though coalescence of nodules with blurring of margins has been suggested as a clue to the diagnosis of sarcoidosis. In a review of nodular sarcoidosis, Onal and colleagues reported radiographic improvement in all their cases either spontaneously or following steroid therapy. This resolution led these authors to propose that nodular infiltration represented an early stage of sarcoidosis. Despite the radiographic improvement, these patients had either no improvement or their pulmonary function worsened. In contrast, previous reports had suggested the nodules failed to improve in over one half of cases despite therapy.

Classically, sarcoidosis is suspected when the chest radiograph reveals bilateral hilar adenopathy in an otherwise asymptomatic individual. How does one decide which patients need further work-up (including biopsy) and which patients can be followed without the need for tissue diagnosis? Winterbauer and his colleagues addressed this question by reviewing a series of 100 patients with bilateral hilar adenopathy on their chest radiographs. They found that all asymptomatic patients with normal physical examinations proved to have sarcoidosis. Also, all patients with bilateral hilar adenopathy and either uveitis and/or erythema nodosum had sarcoidosis. The patients with sarcoid were younger than those with neoplastic adenopathy, by an average of 10 years (34.8 vs 44 years). In symptomatic patients with abnormal physical examinations, however, granulomatous disease could not be reliably separated from neoplastic hilar adenopathy. These authors concluded that asymptomatic patients demonstrating bilateral hilar adenopathy, who had normal physical examinations or evidence of either uveitis and/or erythema nodosum, could be presumed to have sarcoidosis and no further diagnostic evaluation was necessary.

This case demonstrates several atypical features of sarcoidosis, which are more commonly associated with advanced age at the time of presentation.
Recognition of these atypical findings will facilitate making the correct diagnosis and direct the diagnostic workup toward procuring appropriate biopsy material.

REFERENCES

High Frequency Ventilation of Infants: An Analysis of the Literature

Kim Cavanagh MEd RRT

Introduction

Historical Background, Applications, and Scope

Ulf Sjöstrand, a Swedish anesthesiologist, pioneered the use of high frequency ventilation (HFV) in the late 1970s.¹ He and his colleagues² observed that high frequency positive pressure ventilation (HFPPV) depressed or abolished spontaneous ventilation and postulated that the technique would be useful in cases in which the subject’s chest must remain motionless (eg, during surgeries or certain diagnostic procedures).

Since that time, investigator-clinicians worldwide have employed a variety of devices to subject lungs to supraphysiologic rates, and HFV has been addressed in hundreds of journal articles. Today, HFV encompasses a variety of techniques and devices whose only apparent commonality is operation at rates higher than the normal physiologic range.³

Although the suppression of ventilation observed by Sjöstrand and colleagues has not been seen by all investigators, HFV deviates from long-held notions of pulmonary mechanics sufficiently to pique the interest of physiologists and clinicians alike. The intriguing feature of HFV is its capacity to ventilate subjects at tidal volumes (VT) close to or less than anatomical dead space.⁴

Sjöstrand began the high-rate trend with a relatively modest rate of 60 breaths/minute,¹ but HFV rates were soon numbered in hundreds of breaths/min and, eventually, the term hertz (Hz), or cycles per second (c/s), was applied.⁵

Tested first on animal models⁶ and healthy adult volunteers,⁷ HFV was quickly hailed as a panacea for a gamut of problems from the hypoventilation and hypoxemia of patients undergoing bronchoscopy⁸ to hyaline membrane disease⁹ to bronchopleural fistula.¹⁰ The use of HFV for the support of high-risk infants has brought disappointing results in some cases and promise with others.

A review of the English-language literature from 1977 to 1989 produced 25 reports of high-frequency-ventilation trials on infants (Table 1). During this period, 732 subjects were reported with diagnoses of respiratory distress syndrome (RDS), air leak (pneumothorax and/or pulmonary interstitial emphysema [PIE]), congenital diaphragmatic hernia (CDH), persistent fetal circulation (or persistent pulmonary hypertension) (PFC), pneumonia, meconium aspiration syndrome (MAS), and nonpulmonary surgeries.²,⁹,¹¹⁻⁻¹³

Only 4 of the papers reported randomized clinical trials.¹¹,¹⁵,¹⁸,²⁴ Of the remaining trials, 18 were ‘rescue’ in nature,⁹,¹²⁻⁻¹⁴,¹⁶,¹⁷,¹⁹⁻⁻²¹,²₃,²₅⁻⁻²⁹,³₁⁻⁻³₃ attempting to salvage infants who were moribund or otherwise failing conventional mechanical ventilation (CMV). The remaining 3 trials utilized HFV for a minimal time period for measurements only.²,²²,³₀

Infants were ventilated with HFV for periods ranging from less than an hour²⁶ to 1,680 hours.¹² Frequencies varied from 50¹⁰ to 2,400¹² cycles/min. Survival rates of 0%²³ to 100%¹² were reported in the rescue trials.

The diseases in which HFV has appeared promising in anecdotal reports are those that have typically been refractory to CMV—air leaks (particularly PIE)¹⁷,¹⁹,²¹,²₅,²₈,³⁴ and CDH.¹₂,¹₆,²₃,³₃
### HFV OF INFANTS

#### Table 1. Clinical Trials and Case Series Involving High Frequency Ventilation, Published from 1977-1989

<table>
<thead>
<tr>
<th>Year</th>
<th>No. of Subjects</th>
<th>Principal Pathology</th>
<th>Mode of Ventilation</th>
<th>Device</th>
<th>Subject Selection for Entry</th>
<th>Ventilator Frequency</th>
<th>Hours of HFV Ventilation</th>
<th>Reference</th>
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</thead>
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<tr>
<td>89</td>
<td>327</td>
<td>RDS</td>
<td>HFOV</td>
<td>Hummingbird</td>
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<td>HFOV</td>
<td>Hummingbird</td>
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<td>20 Hz</td>
<td>1680</td>
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<td>45</td>
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<td>HFO-IMV</td>
<td>Emerson &amp; Babybird</td>
<td>rescue</td>
<td>20 Hz</td>
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<td>13</td>
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<td>88</td>
<td>41</td>
<td>PFC</td>
<td>HFOV</td>
<td>Metrex</td>
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<td>Percussionaire</td>
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<td>87</td>
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<td>CDH, PFC MAS, etc.</td>
<td>HFOV</td>
<td>Texas Research</td>
<td>rescue</td>
<td>15-20 Hz</td>
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<td>16</td>
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<td>PIE</td>
<td>HFFI</td>
<td>Percussionaire</td>
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<td>2-4 Hz</td>
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<td>Emerson &amp; Babybird</td>
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<td>HFFI</td>
<td>Emerson</td>
<td>RDS-stable PIE-rescue</td>
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<td>VS 600 Bannell</td>
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<td>PIE</td>
<td>HFJV</td>
<td>VS 600</td>
<td>rescue</td>
<td>200-300/min</td>
<td>246</td>
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<td>HFPPV</td>
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<td>Bourns LS 104-150 &amp; Babybird</td>
<td>rescue</td>
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<td>PFPPV</td>
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<td>60/min</td>
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<td>60-180/min</td>
<td>?</td>
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</table>

RDS = respiratory distress syndrome.
CDH = congenital diaphragmatic hernia.
MAS = meconium aspiration syndrome.
PFC = persistent fetal circulation, or persistent pulmonary hypertension.
PIE = pulmonary interstitial emphysema.
Complications of HFV have included increased air leak, bleeding diatheses, necrotizing tracheobronchitis (NTB), hypotension, bronchopulmonary dysplasia (BPD), increased tracheal secretions, and intraventricular hemorrhage.

The benefits cited have been improved oxygenation and ventilation at reduced \( P_{aw} \) or reduced mean airway pressure (\( P_{aw} \)), and decreased air leak. Obvious overlaps of benefits and complications exist. For example, pulmonary air leaks, i.e., pneumothorax and PIE have been reported to improve dramatically when treated with HFV. On the other hand, application of HFV has been reported to produce (or worsen pre-existing) air leaks.

In spite of the obvious interest demonstrated in the medical community by the number of patient series and laboratory testing reported, confusion continues to surround HFV, and evaluation is difficult.

I believe that factors contributing to the confusion are (1) inadequate descriptions of ventilator-operating characteristics, (2) lack of agreement on measurements of ventilatory variables and indications of efficacy, (3) use of different and possibly inappropriate animal models, (4) differing study designs and patient series, and (5) diversity in patient management. I will discuss aspects of each of these factors, but first I will describe and define the submodes of ventilation encompassed by HFV and the mechanisms of gas transport operative in HFV. (Terms are defined in the Glossary at the end of the text.)

Definitions and Descriptions of Modes and Devices

The first issue related to devices is the lack of clear delineations among the different modes of HFV. Classification on the basis of frequency alone is inadequate because the operating ranges of different modes overlap. A more comprehensive classification takes ventilator design as well as frequency into account. The major types of HFV are: (1) HFPPV, (2) high frequency jet ventilation (HFJV), (3) high frequency flow interruption (HFFI), and (4) high frequency oscillatory ventilation (HFOV).

Six papers have reported the use of HFPPV, five HFJV, and 14 HFOV or HFFI. In several instances, HFOV has been combined with intermittent mandatory ventilation (IMV) delivered by a conventional ventilator. Reports of the use of 17 different devices have been published. The device cited most frequently is the Bourns LS 104-150 (used in three trials). Eight other ventilators were used in two different studies, and eight more were mentioned in only one study.

HFPPV employs ventilators similar or in some instances identical to those used to deliver CMV but at rates of 60-150 breaths/min. Not all conventional ventilators perform well at these high rates because of flow limitations or the inertia of valving mechanisms. Difficulty with these machines can also occur if the expiratory time becomes too short for exhalation to be complete and gas is trapped in the patient’s lungs. The magnitude of gas-trapping depends on the rate of ventilation, the mechanics of the lung, and the dynamic behavior of the ventilator.

HFJV employs ventilators that operate at rates of 100-600 breaths/min. Short bursts of gas under high pressure are delivered at the airway through a jet injector (Fig. 1). HFJV has been used extensively in human subjects. Its use is advocated during bronchoscopy and laryngoscopy. Some investigators suggest that it is an ideal form of support for patients with major airway disruptions such as...
bronchopleural fistula. Evidence of necrotizing tracheobronchitis discovered during pathologic examinations of subjects ventilated by HFJV has raised serious concerns about the safety of the technique. Controversy continues regarding the mechanism of tracheal damage and whether the damage is associated only with jet ventilation. Inadequate humidification systems, proximity of the jet injector to the airway mucosa, or some other unrecognized factor may be responsible. HFFI represents yet another type of HFV. The device may employ a so-called set-back jet with the jet located proximal to the patient's airway, the exhalation valve positioned between the jet and the airway. Another design consists of a ball-valve rotated by a variable speed motor. The turning of the ball interrupts the stream of gas directed toward the airway in a pulsatile manner (Fig. 2). One HFFI device (Infrasonics, San Diego CA), which has recently been licensed by the Food and Drug Administration, uses a bank of microprocessor-controlled solenoid valves to produce high-frequency pulsations. A venturi creates subambient pressures during expiration; so, the device performs like an oscillator.

HFV achieves the highest frequency range (900-3000 cycles/min, or 10.5-50 Hz). HFV is generated by a piston or diaphragm that oscillates a column of gas in the airway, generating a sinusoidal waveform (Fig. 3). (The magnitude of pressure fluctuation in a cycle is the amplitude. Frequency denotes the number of cycles per unit of time.) HFOV is unique in that, in addition to the positive stroke during inspiration, an active negative 'pull' stroke aids expiration. No bulk flow or convective motion of the column of gas in the airway occurs. Therefore, a stream of fresh gas, known as the bias flow, intersects the oscillatory pathway to deliver oxygen and flush out carbon dioxide. The expiratory portion of the bias-flow circuit is designed to impede high frequency oscillations while letting a steady flow of gas escape, ie, it functions as a low-pass filter.

The distinction between HFFI and HFOV is not always clear-cut. Terms such as set-back jet and quasi-oscillator, as coined by one manufacturer to describe its machine, further cloud the issue. A true oscillator is differentiated from other HFV devices by an active expiratory phase resulting when the backstroke of the piston or membrane produces a fall in airway pressure. This negative pressure during expiration accelerates the expiratory flowrate and reduces gas trapping.

**Mechanisms of Gas Transport during HFV**

Gas transport in HFV is not yet fully understood, although observations of two different cardiorespi-
Inhalatory phenomena shed some light on what is believed to occur. According to the 1985 review by Smith and Sjostrand,\textsuperscript{43} apneic diffusion oxygenation (ADO) was first studied in animals and humans in the early 1950s. The term describes what occurs in paralyzed subjects in an environment of high-flow oxygen. After minutes of apnea, although the $P_aCO_2$ increases, $P_aO_2$ does not decrease as one might expect. Oxygenation occurs as a result of diffusion, rather than convection. In terms of ventilatory frequency, ADO is the opposite of HFOV, yet there are functional similarities.\textsuperscript{45}

The second phenomenon, cardiogenic mixing, occurs when the motion of the beating heart is transmitted to the surrounding lung, resulting in a five-fold increase in diffusion.\textsuperscript{44} This observation inspired investigators to provide a “more energetic mixer” by directing the output of a loudspeaker into tubing attached to a mouthpiece to fashion one of the original HFOV generators.\textsuperscript{44}

Speculation has persisted about the mechanisms by which HFOV accomplishes gas exchange. Chang\textsuperscript{45} has identified five modes of transport that may interact within the lung during HFOV. The first mechanism is direct alveolar ventilation by bulk convection. In other words, if the $V_T$ delivered is equal to at least one half to three quarters of the anatomical dead space, some fresh gas may reach some alveoli situated relatively close to the mouth.

The second mechanism identified by Chang\textsuperscript{45} is gas mixing by pendulluf—-the movement of air between neighboring lung units. The rate of filling and emptying of any lung unit is determined by its time constant (the product of resistance and compliance). Pendulluf is greatly enhanced during HFOV and facilitates interregional gas mixing between adjacent lung units with disparate time constants.\textsuperscript{37,45} This recirculation of gas between lung regions is thought to produce a greater tidal volume in the parenchyma than that delivered to the trachea.\textsuperscript{45}

The study of axial velocity profiles within a bifurcating network suggests another mechanism of gas transport. Laminar flow through a tube produces a parabolic or asymmetric velocity profile in which the lamina, or layer, of molecules next to the wall is essentially motionless, with the velocity of each layer increasing toward the center of the tube.\textsuperscript{46} It has been shown experimentally that with asymmetric velocity profiles, a full cycle of oscillatory flow produces convective forward movement of the molecules in the center of the tube (Fig. 4\textsuperscript{47}). The molecules near the wall are displaced backward. The final position of the molecules represents a net forward movement of gas past a reference point.\textsuperscript{46}

![Fig. 4. Cross-sectional illustration of asymmetric velocity profile of gas molecules within a tube. Reprinted from Reference 47, with permission.](image)

The mechanism of augmented diffusion is one that is frequently cited as playing a major role in HFOV.\textsuperscript{4,37,45,48,49} The concept derives from Taylor-type dispersion. According to Chang,\textsuperscript{45} Taylor determined mathematically that laminar dispersion in a long straight tube is the result of interaction between axial convection and radial, or lateral, diffusion. When flow becomes turbulent, random convective eddies provide transport between lamina—rather than lateral movement's occurring as a result of molecular diffusion. Therefore, mixing between the central core of fluid and the periphery is much faster during turbulent flow. Turbulent flow is likely to be present in the airways during HFOV as suggested by measurement of Reynold’s numbers in the trachea as high as 20,000.\textsuperscript{50} (The Reynold’s number is a value that indicates the presence of
laminar or turbulent flow through a tube. The value is derived from velocity, density, viscosity, and tube diameter, with Reynold's numbers $> 2000$ denoting turbulent flow.\textsuperscript{41} Oscillatory dispersion depends not only on the speed of axial convection but also on the unsteadiness of the flow. Inertia, which influences the process of acceleration and deceleration, becomes important during oscillation because the pressure gradient that drives the fluid must reverse direction twice in every cycle. In the central core of a tube, particles possess greater momentum than do those in the periphery and cannot accelerate or decelerate in phase with the pressure gradient. (This unsteadiness of oscillatory flow is quantified as the Wormersley number.\textsuperscript{45})

Because these theories of gas transport are based on observations of fluid flow through rigid, uniform cylinders, it is not likely that they can be rigorously applied to the human airway, which consists of a highly complex branching network of compliant structures. The difficulty encountered in mathematically analyzing branching networks has hampered development of a theory that fully explains augmented diffusion.\textsuperscript{45}

The final mode of gas transport proposed by Chang\textsuperscript{45} is simple molecular diffusion, which is dominant near the alveolocapillary membrane during HFOV as it is during any other type of ventilation.

These five modes of gas transport probably interact during HFOV, and one or another may dominate in a particular area of the lung. However, because of the limitations of mathematical predictions of gas dispersion and the limitations of the experimental method, I believe that much remains to be learned about the mechanisms operative in the lung.

Factors Contributing to Confusion
Surrounding Applications and Efficacy

Inadequate Descriptions of
Ventilator-Operating Characteristics

The variety of HFV devices in use compounds the confusion encountered in evaluating HFV efficacy and outcomes. Operating characteristics of ventilators, particularly their limitations, have often been inadequately described.\textsuperscript{52} Results derived from one HFV system do not necessarily apply to other systems.\textsuperscript{41} Assessment of mechanical performance using an in-vitro model of the neonatal respiratory system can be a useful means of comparison between HFV devices.\textsuperscript{53} Lack of standardization in ventilator design coupled with inadequate understanding of a ventilator's operating characteristics can introduce unwanted variables into clinical investigation.

Some devices, for example, sacrifice tidal volume as frequency is increased.\textsuperscript{21} Another problem occurs if a machine's expiratory time is shortened beyond the time constant or critical emptying time of that particular device and circuit. (The time constant of the ventilator and delivery system must not be confused with the time constant of the respiratory system, which in the healthy infant is about 0.12 seconds.\textsuperscript{54}) During mechanical ventilation, the actual emptying time of the lung depends on the time constants of the respiratory system and those of the ventilator, circuit, and endotracheal tube.\textsuperscript{36,54,55} Incomplete emptying results in gas trapping and progressive increase in distal airway pressures.\textsuperscript{36}

Lack of Agreement on Measurements of
Ventilatory Variables and Indicators of Efficacy

Monitoring of ventilatory variables is another dilemma that adds to the confusion surrounding the efficacy and application of HFV. Delivered $V_T$ is difficult to measure, particularly in neonates who typically have uncuffed endotracheal tubes. Although some investigators have used body plethysmography to assess $V_T$, intrathoracic gas compression may result in a discrepancy between $V_T$ at the mouth and body surface displacement.\textsuperscript{56} Electrical impedance plethysmography, which utilizes transthoracic electrical impedance measurements, has been proposed as a noninvasive means of determining $V_T$ during HFV.\textsuperscript{57} Another approach uses a pneumotachograph on the expiratory limb of an HFJV device to measure $V_T$.\textsuperscript{58}

During HFV, pressures measured at the airway opening provide poor estimates of pressures in the trachea or alveoli.\textsuperscript{37} Alveolar pressure swings can be substantially greater than those measured at the airway opening at certain frequencies. At higher frequencies, however, alveolar pressure swings may be much smaller than those at the airway opening.\textsuperscript{59} We know that a big drop in pressure occurs from the proximal to the distal end of the endotracheal tube. The
important question is what occurs in the rest of the lung.  

Direct measurement of alveolar pressure presents problems. Although an alveolar capsule has been used to measure regional alveolar pressures in animals, its use requires puncturing the pleura, and the technique has not been perfected for application to human subjects. Airway occlusion has been proposed as a means to extrapolate alveolar pressure measurements in infants.

Pressure monitoring systems used on conventional ventilators may give erroneous results when used with an HFV system because of inadequate frequency response and phase lag. (Frequency response indicates the degree of fidelity with which the original phenomenon is reproduced. Phase lag is the time delay between the original event and its reproduction.) These problems are magnified in the small-lumen pressure-measurement catheters that must be used for neonates. Volume of gas delivered by an HFV system depends on a number of factors including oscillatory frequency, configuration of the ventilator circuit, and diameter of the endotracheal tube. Because delivery circuits are not standardized, no consistent relationship between driving pressure and actual amount of delivered ventilation exists that is applicable to different systems.

Impedance of the respiratory system, an index of the impedance a body presents to oscillatory flow, affects the delivered gas volume. The point of minimal impedance corresponds to the natural or resonant frequency of the lung, at which point air movement is opposed only by airway resistance. The value of impedance is derived from the lung's compliance, resistance, and inertance. Therefore, pathologic processes that change resistance and compliance can alter impedance of the respiratory system. The presence of an endotracheal tube adds inertance. Impedance can even change in a particular subject over time as clinical condition changes.

In theory, impedance should determine the mechanical efficiency of HFV. At certain frequencies, resonant amplification of delivered gas volume exceeds the stroke volume of the oscillator. It is theorized that the best gas transport at the least pressure will occur at or above resonance. Data on the resonant frequency of the lungs of human infants are scarce. If the available data (suggesting that resonance occurs in intubated infants with RDS at 13-23 Hz) are correct, then the frequency used in many of the clinical trials (10 Hz) has been too low. At resonance, pressure excursions are at a minimum in the trachea and a maximum in the terminal airways. As frequency increases, pressure in the terminal airways falls and tracheal pressure rises. Thus, the selection of an optimal frequency involves a choice between pressure exposure in central versus terminal airways.

In clinical application, a critical frequency exists that varies from patient to patient and among systems. Below that frequency, carbon dioxide removal depends on the product of frequency and VT. Above that frequency, VT is relatively more important for effective ventilation than is frequency. A method for rapid determination of the critical frequency in each patient would facilitate rational application of HFV.

Different and Possibly Inappropriate Models

Although some interesting physiologic data have been gathered using excised animal lungs, this approach eliminates the effect of the chest wall and so may not have true clinical relevance. Even measurements made on lungs of infants at autopsy do not necessarily relate well to living children. Animal experimentation provides a valuable approach to studying how HFV works in living lungs. For this approach, disease states in the animal lung need not be identical to those in humans. However, it is important to recognize the limitations and advantages of different animal models.

Subject size is important in animal research. Many HFOV devices are limited clinically by the size of the subject they can effectively ventilate. Some can ventilate large animals with normal lungs but not large animals with poorly compliant lungs. Rabbits are commonly used for study of neonatal ventilation because they are about the same size as newborn infants. Differences among species can be important in observations of lung mechanics. In mammals, lung volume and compliance are generally proportional to body weight, whereas ventilation and conductance usually vary with mass. Smaller animals breathe more rapidly and have more compliant chest walls. Alveolar diameter increases in larger animals.
However, certain species of animals deviate from these generalizations.

The lungs of dogs demonstrate some important differences from the lungs of other mammals. For one thing, dog lungs have extremely short time constants. The short time constant appears to be advantageous when dogs pant to dissipate body heat.\textsuperscript{75} More important in regard to HFV studies is the presence of multiple collateral channels in the dog lung. These allow for extensive horizontal gas-mixing among airways at equivalent levels (alveolus to alveolus). Also, the trachea and bronchi of the dog are disproportionately large compared to those of man.\textsuperscript{76} These differences suggest that some benefits ascribed to HFV based on the early dog studies reflect not efficacy of the method but rather difference in the lung structure of that particular species. Because of these structural differences, the dog is now considered to be a poor model in which to study HFV.\textsuperscript{44,56}

It is interesting to note that the bird is frequently referred to in discussions of HFV. The avian respiratory tract is a unidirectional high velocity system in which there is continuous gas exchange during inhalation and exhalation.\textsuperscript{43} Hummingbirds (after which an HFOV device has been named) synchronize wing-beat with respiration and probably facilitate diffusion in a manner similar to HFOV.\textsuperscript{77}

A variety of animals (including lambs, rabbits, and cats) have been studied using HFV. These investigations, which utilize mature animals, may not take one critical factor into account: The immature lung is probably particularly and uniquely vulnerable to pressure-induced injury. Animal models, if they are to be truly useful, should simulate the pathologies seen in the lungs of premature infants.\textsuperscript{41}

A model of RDS has been produced by washing surfactant out of the lungs of rabbits. Lung lavage is performed using isotonic saline at body temperature. The severity of lung injury is determined by the number of lavages performed on the animal.\textsuperscript{78}

Some investigations of HFOV are also being done using primates.\textsuperscript{79-81} Baboons born prematurely develop RDS and bronchopulmonary dysplasia (BPD) analogous to those diseases in human infants.\textsuperscript{56} Investigations done using baboons support the efficacy of HFOV in the treatment of RDS in premature subjects.\textsuperscript{81}

Models of other neonatal diseases are more difficult to simulate in animals. Not all animal lungs are capable of developing PIE as do the lungs of human infants subjected to barotrauma.\textsuperscript{32} Furthermore, PIE cannot be documented in living animals unless x-ray facilities are available.

Although some investigators have attempted to produce meconium aspiration syndrome by instillation of slurries of meconium into animal lungs,\textsuperscript{83} the results are not truly representative of neonatal meconium aspiration syndrome. The passage of meconium in utero signals an initial episode of fetal asphyxia, which may have negative repercussions of its own.\textsuperscript{84} A true model of the disease should mimic that mechanism.

Study Designs and Patient Series

A large body of information based on the use of HFV in adults is available, but the findings are not applicable to neonates.\textsuperscript{23} The filtering effect of the much smaller endotracheal tubes used in infants is profound—accounting for more than one third of the total impedance to CO\textsubscript{2} removal.\textsuperscript{85}

The information available about infants treated with HFV, as mentioned earlier, is daunting in its lack of consistency. Many investigators called for a large randomized clinical trial of HFV in infants to answer some questions about its efficacy.\textsuperscript{9,15,17,24,25,31,56} In response, The Division of Lung Diseases of the National Heart, Lung and Blood Institute conducted a multicenter randomized clinical trial to compare the efficacy and safety of HFV with conventional ventilation in the treatment of respiratory failure in preterm infants. The principal end-point of the study was the incidence of BPD.\textsuperscript{11}

Between March 1, 1986 and March 31, 1987, 673 infants were enrolled, 327 of whom received HFOV. Oscillatory ventilation was the type of HFV selected for the trial, based on its association with minimal air trapping and the relative absence of major complications associated with HEV.\textsuperscript{11} Investigators concluded that the incidence of BPD was comparable in the HFOV and CMV groups. HFOV was not found to reduce mortality or the need for ventilatory support. In fact, HFOV was associated with an increased incidence of pneumoperitoneum of pulmonary origin and Grades 3 and 4 intracranial hemorrhage.\textsuperscript{11}
Although the trial has been concluded, a number of questions remain. Could the disappointing outcome reflect a problem with the device, how it was managed, or the patient population—rather than with the mode of ventilation itself?

HFOV was provided in the study by a Japanese piston oscillator called the Hummingbird. It was selected after bench-testing of several available machines. Reports of its clinical application in neonates are scarce in the English-language literature.

Infants were managed briefly on CMV before being ventilated by HFOV. $P_{aw}$s were kept the same in both HFOV and CMV groups at all times, and ventilatory frequency for HFOV was maintained at 15 Hz. The use of sigh breaths and muscle relaxants was optional, and little attention was paid to recruitment of effective lung volume. Any of these factors could have affected outcome.

Although a variety of diseases were represented in the study population, hypoplastic lungs, and diaphragmatic hernia, which are thought to respond well to HFOV, were excluded.

The study was not designed to test the significance of differences in incidence of air leaks in the two groups; so, that issue remains speculative. It did confirm, though not consistently, that desperately ill infants may improve when their treatment is changed from CMV to HFOV. The HIFI trial has provoked controversy among investigators, some of whom suggest re-evaluation of HFOV using other ventilatory strategies. Variability in the incidence of BPD among the 10 centers involved in the trial suggests that differences in ventilatory management could have played a significant role in outcome. Obviously, the chapter on HFV is not yet closed.

The existing literature suggests that some neonatal pathologies appear responsive to HFV. In theory and application, air leaks seem to respond well to HFV. It is not fully understood how HFV is able to sustain gas exchange despite major airway or parenchymal disruption. The relatively shorter time at peak airway pressure during HFV may prevent large volume losses because lung tissue defects expand at peak pressure. It is also possible that if the time constant of the leak is greater than the inspiratory time, less volume escapes during HFV than during CMV.

Treatment of CDH with HFOV appears promising. PFC can be reversed using HFV. Infants with pneumonia have been reported to respond well to HFV. Patients with meconium aspiration syndrome, on the other hand, have shown a poor response to HFV.

The HFV literature is inconsistent in defining the outcome that indicates efficacy of treatment. The NIH in a past trial considered the incidence of BPD a fairly long-range outcome. Some investigators in rescue trials have noted an increased incidence of BPD, but concede that the initial lung injury was so severe that normal healing was very unlikely. Investigators have used survival rates of historical control populations for comparison. They have also considered survival based on the assumption that the investigative subjects were nearly moribund when HFV was instituted. Many investigators cite improved arterial blood gas values (ABGs), or comparable ABGs at lower $P_{aw}$s, as evidence of improved outcome. Improvement in air leak as seen on radiograph is another determinant of positive outcome.

Many complications have been observed in infants treated with HFV. Some appear to be linked predominantly with one mode of HFV, such as NTB with HFJV. Other problems, such as that of increased tracheal secretions, are found in various modes of HFV.

Increased air leak has been seen in a number of studies and may include pneumoperitoneum and pneumopericardium. Hypotension has been reported and may be due in part to the relatively constant intrathoracic pressure that may impede venous return to the heart. Bleeding diatheses have complicated the course of some subjects treated with HFV. Hemorrhages in the lung and brain have been observed. Some of these problems may result from the initial insult rather than as a consequence of HFV therapy. Patent ductus arteriosus may also be more prevalent in infants managed with HFV.

**Diversity in Patient Management**

Considerable diversity exists in the manner in which HFOV is managed in infants by different investigators.
The results of manipulations of some HFV parameters are documented. $P_{aO_2}$ correlates directly with $F_{O_2}$ and $P_{aw}$, $P_{aCO_2}$ depends on both oscillatory amplitude and frequency. However, at higher frequencies, $CO_2$ elimination is influenced more strongly by tidal volume (amplitude) than by frequency. If IMV or sigh breaths are interspersed, they will contribute to gas exchange also.

How then, are the initial frequency and amplitude settings for HFOV chosen? Amplitude is often selected by incrementally increasing the stroke volume of the generator until the subject’s chest begins to visibly vibrate. Changes in amplitude then are based on degree of ventilation as determined by $P_{aCO_2}$, or expansion of lung on chest radiograph, or both. The piston displacement volume required to maintain normocarbia has been observed to be strongly influenced by endotracheal tube size.

The design of the ventilator may restrict setting selection if frequency or amplitude are fixed or if changing one setting affects another. (For example, increasing frequency results in decreased amplitude.) Amplitude may be considered as the pressure differential (peak inspiratory pressure – positive end-expiratory pressure) measured at the airway. That value does not represent a true estimation of the amplitude of pressure swings in the lung, however, and may be misleading. In one patient, peak inspiratory pressure (PIP) measured at the proximal end of the endotracheal tube was 240 cm H$_2$O and dropped to 10 cm H$_2$O at the distal end. Multilumen tubes are useful in the determination of distal airway pressure.

Investigators concede that there is probably an optimum frequency that will maximize gas exchange. That frequency depends on a number of factors and varies between patients. In many clinical trials, one frequency, usually between 10 and 20 Hz, is selected and used exclusively.

In an HFOV system, $P_{aw}$ is adjusted by varying the amount of egress of the bias flow through the low-pass filter. Increasing resistance on the distal side of the circuit decreases gas escape and increases $P_{aw}$.

Management philosophies differ regarding the level of $P_{aw}$ most effective for HFOV. Some investigators keep the $P_{aw}$ the same as it was when the infant was on CMV. One of the potential advantages of HFOV, however, is the reduction of barotrauma by lessening distension of the lung parenchyma. The question is whether it is advantageous to use a lower distending pressure at the cost of a higher $P_{aw}$ during HFOV. If distension rather than pressure is the crucial factor in the development of barotrauma, then HFOV theoretically would be preferable to CMV. High $P_{aw}$s, however, have been associated with protein leaks into the alveoli and decreased surfactant production.

Numerous studies defend the efficacy of HFOV based on its ability to achieve carbon dioxide elimination at lower $P_{aw}$ than those used in CMV. Other investigators hypothesize that vibration of the chest wall during HFOV subtly alters compliance so that a higher $P_{aw}$ is necessary to achieve the same lung volume. In that case, increased $P_{aw}$ need not result in greater lung distension.

It appears that at equivalent $P_{aw}$s, mean lung volumes and oxygenation are markedly higher during HFOV than CMV, provided that the lung is inflated to total lung capacity prior to institution of HFOV.

Recruitment and maintenance of open alveoli during HFOV can be a problem. HFOV is characterized by near-constant airway pressure. Pressure fluctuations in the distal airways are fairly static compared to the large phasic pressure swings that occur with CMV. It appears that when subjects are ventilated for more than a brief period with HFOV alone, they may develop diffuse atelectasis accompanied by hypoxemia and hypercarbia.

In order to maintain adequate alveolar volume, various methods of delivering sigh breaths have been attempted. Manual sighs may be provided with an anesthesia bag. The subject may be disconnected from the oscillator for a period of time and be given mechanical breaths with a conventional neonatal ventilator. Early lung expansion is now being touted as a factor in improved outcome.

Mixed-mode ventilation or combined HFV-CMV represents another approach to the problem of volume recruitment. In some devices, conventional breaths are interposed between periods of oscillation to function as mechanical sighs (Fig. 5). Other protocols use an HFOV device coupled with a conventional ventilator to superimpose oscillations on conventional breaths. Periodic sighs can result in clinical stabilization, but may also produce interstitial air leak. If an infant already has PIE, clinical deterioration may occur during or following sighing.
HFV OF INFANTS

Such patients have been observed to remain stable with sufficient alveolar patency when adequate $P_{aw}$ is used.\(^{19}\)

Whether subjects do or should spontaneously breathe during HFOV is even a subject for dispute. Some investigators observed cessation of breathing in conscious patients with normal gas exchange during HFV.\(^ {1,2,3,6,8,7}\) Infants have been reported to appear relaxed while being oscillated and to take spontaneous breaths only when stimulated by pain or handling.\(^ {9}\) Patients treated with HFV who could communicate have reported that the sensation of HFOV is very different from CMV but not unpleasant.\(^ {71}\)

In some clinical trials, infants have been kept paralyzed during HFV.\(^ {15,17,20}\) In others, the use of muscle relaxants and sedation has been arbitrary.\(^ {11,19}\)

Inadequate humidification and warming of delivered gases had serious repercussions in the early days of HFV. Inadequate humidification was cited as the cause of devastating tracheal damage.\(^ {3,4,1}\) HFJV in particular has been associated with necrotizing tracheobronchitis.\(^ {41}\) One reason was that the original jet ventilators provided humidification of entrained gases but not of the gases delivered through the jet injector.\(^ {28}\) The problem is apparently not simply one of humidification. HFJV appears in some studies to produce more tracheal damage than CMV even when adequate humidity systems are employed.\(^ {25,89}\) Shear stress, mechanical trauma from the high flow of gas produced in the airway at the site of the jet injector, has been proposed as one culprit.\(^ {37,41}\) Some investigators maintain that HFJV is no more likely to produce tracheobronchial damage than CMV\(^ {30,31}\) or other forms of HFV.\(^ {32}\) HFOV has not been clearly linked with similar tracheal lesions.\(^ {14,41}\) Humidification of gases delivered by HFOV is technically simple compared to methods used in HFJV.\(^ {37}\)

Variations on HFV are now being investigated. Mixed-mode ventilation, as previously mentioned, has shown promising clinical results.\(^ {20}\) Combination of HFOV with extracorporeal membrane oxygenation (ECMO) has potential benefits. It is proposed that the use of HFOV during ECMO could shorten the duration of ECMO and provide safer ventilator back-up by maintaining critical lung inflation in minimally traumatic fashion.\(^ {16}\)

HFOV is being provided experimentally to animals by external means. Some devices use negative-pressure oscillation around the chest with a modified respirator-incubator\(^ {93}\) or a thoracoabdominal chamber.\(^ {94}\) Another approach employs pneumatic compression of the subject in a body plethysmograph.\(^ {95}\) These techniques have the advantage of avoiding endotracheal intubation.\(^ {94}\) The potential for clinical application is limited by the necessity for strict enclosure of most of the body during ventilation.\(^ {96}\)

High-frequency chest-wall compression (HFCWC) is produced when pressure oscillations are delivered by an air-filled cuff wrapped around the lower thorax. Like HFOV by other means, HFCWC provides normal gas exchange at $V_{T} = \text{dead-space volume}$.\(^ {90}\)

In Conclusion

HFV continues to tantalize investigators and clinicians with its potential for successfully treating various types of pulmonary pathology that remain intractable to conventional ventilation. Nowhere is that need more obvious than in the care of critically ill infants, in whom the immature lung appears to be uniquely vulnerable to ventilator-induced trauma.

Inadequate understanding of gas transport mechanisms coupled with a plethora of different HFV devices and management techniques cloud what is already a complex issue. Lack of adequate experimental models and non-applicability of data gathered from adult subjects compound the dilemma.

Many questions must be answered before we will know the role—if one exists—that HFV can play in salvaging neonates. The cost of conventional mechanical ventilation of premature lungs, in terms of morbidity and mortality, remains sufficiently high to justify the search for a better alternative.
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High frequency ventilation (HFV)—ventilation at rates higher than the normal physiologic range.

High frequency positive pressure ventilation (HFPPV)—high frequency ventilation provided by devices similar or identical to conventional mechanical ventilators but at rates of 60-120 breaths per minute.

High frequency jet ventilation (HFJV)—high frequency ventilation provided by a jet injector that delivers short bursts of gas under high pressure at the airway; typically operates at rates of 100-600/min.

High frequency flow interruption (HFFI)—high frequency ventilation that is similar to HFJV but the location of the jet is not directly at the patient airway; may also employ a turning-ball valve that interrupts a stream of gas directed toward the airway.

High frequency oscillatory ventilation (HFOV)—high frequency ventilation generated by a piston or diaphragm that oscillates a column of gases to produce a sinusoidal waveform; no bulk flow of gas is produced; HFOV is unique in that it produces an active negative pull stroke during expiration; operates at rates of 900-3000 cycles/min.

High frequency chest wall compression (HFCWC)—high frequency ventilation that results from external oscillation delivered to the chest wall.

Adiabatic—expansion or compression without loss or gain of heat.

Amplification—the process of making larger or stronger.

Amplitude—magnitude of pressure fluctuation in the oscillatory waveform; the peak inspiratory pressure minus positive end-expiratory pressure measured at the airway; similar to tidal volume.

Apneic diffusion oxygenation (ADO)—the phenomenon that occurs in apneic subjects in an oxygen-rich environment; maintains adequate alveolar oxygenation by simple diffusion.

Attenuation—dilution or reduction of intensity.

Augmented diffusion—the enhancement of simple gas diffusion that occurs in the presence of turbulent flow.

Axial velocity—the speed of a fluid that flows in a direction parallel to the walls of a tube.

Bias flow—a stream of fresh gas that intersects the oscillatory pathway in order to provide oxygen and flush out carbon dioxide.

Boundary layer—layer of molecules adjacent to tube wall where fluid velocity is zero.

Cardiogenic mixing—increase in diffusion that occurs in neighboring lung units as a result of transmission of motion from the beating heart.

Conductance—the reciprocal of resistance; equal to flow divided by change in pressure.

Convection—forward fluid flow; mass movement of fluid molecules.

Critical frequency—that ventilatory frequency above which CO₂ removal is dependent solely on tidal volume, independent of frequency; value varies among subjects.

Frequency—cycles/unit of time.

Frequency response—degree of fidelity with which a monitoring system reproduces the original phenomenon.

Hertz (Hz)—cycles per second (c/s).

Impedance—index of impedance a body presents to oscillatory flow; derived in the lung from compliance, resistance, and inertance; the ratio of pressure to flow at a given frequency.

Inertance—opposition to acceleration, negligible in the lung during normal ventilation.

Inertia—the tendency of matter at rest to remain at rest and of matter in motion to remain in motion.

Lamina—a thin layer.

Laminar flow—smooth, nonstumbling flow, characterized by a concentric series of thin, cylindrical layers of molecules; the velocity in the center of the tube is greatest; denoted by a Reynolds number less than 2000.

Low-pass filter—a term borrowed from electronics; in high frequency ventilation, the term refers to a portion of the expiratory circuit that is designed to impede high frequency oscillations while letting a steady flow of gas escape.

Parabolic or asymmetric velocity profile—cone-shaped front of fluid molecules during laminar flow; results from molecules in the center of the tube moving at greatest velocity while those next to the wall are essentially motionless.

Pendelluft—movement of air between neighboring lung units during cyclic ventilation.

Phase lag—time delay between the original event and its reproduction on a monitoring system.

Pneumotachometer—an electronic flow-sensing device.

Radial or lateral diffusion—diffusion of molecules from the center of a tube out toward the walls.
Resonant frequency—natural vibration frequency of a body; the point of minimal impedance, the frequency at which air movement is opposed only by airway resistance.

Reynold's number—value that indicates the presence of laminar or turbulent flow through a tube; derived from velocity, density, viscosity, and tube diameter; values > 2000 denote turbulent flow.

Shear stress—force causing two contacting layers to slide against each other in opposing directions; a type of mechanical trauma produced at the airway by a high flow of gas.

Solenoid valve—electrical on-off valve.

Taylor-type dispersion—molecular mixing process that results from the interaction between axial convection and radial diffusion.

Time constant—product of resistance and compliance; critical filling or emptying time needed for pressure equilibration.

Time constant of the respiratory system—measure of the time necessary for alveolar pressure to reach 63% of the change in airway pressure; for practical purposes, volume delivery is complete after interval equal to five time constants has elapsed. The time constant of a healthy infant is about 0.12 seconds; so, pressure equilibration will be complete in 0.6 seconds.

Turbulent flow—uneven, tumbling molecular flow pattern with eddy currents.

Wormersley number—value that denotes the degree of unsteadiness of oscillatory flow; derived from the ratio of the radius of the tube to the thickness of the molecular boundary layer adjacent to the tube wall.
Mouth-to-Mouth Resuscitation by Lay Rescuers—Should They or Shouldn’t They?

From the first publication of Standards and Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiac Care, the emphasis has been on the ABCs—Airway, Breathing, and Circulation. In fact, if someone has suffered a respiratory arrest, opening the airway and instituting artificial ventilation can be literally vital maneuvers. In 1980, the (then) Emergency Cardiac Care (ECC) Subcommittee of the American Heart Association (AHA) emphasized that lack of spontaneous ventilation could be due to complete upper airway obstruction. In 1986, the ECC Subcommittee recognized the public concern generated by the potential for the transmission of Hepatitis B virus (HBV) and human immunodeficiency virus (HIV)—both during practice on manikins and during actual performance of CPR on human subjects. In fact, in the 1986 Standards, the subcommittee points out that mouth-to-mouth ventilation requires close physical contact with the victim and the potential rescuer should overcome any hesitancy about such contact. The report goes on to say that “students may practice on manikins contaminated by the hands and oral fluids of previous students.” In those 1986 Standards, it was estimated that 40 million people had had contact with manikins during CPR training; yet, at that time the statement was made “The use of CPR training manikins has never been documented as being responsible for an outbreak or even an isolated case of bacterial, fungal, or viral disease.” (Italics ours.)

However, in November 1989, the Supplemental Guidelines were published to address the risk of infection during CPR training and rescue. Those guidelines emphasize that two groups of investigators (Fox et al and Friedland et al) have concluded that saliva has not been shown to be a causative agent for the transmission of HIV. In spite of the purely theoretical possibility of HIV transmission via saliva (due to the presence of lesions invisible to the unaided eye), Sande makes a strong case against casual contagion in the spread of AIDS—defining casual in this instance as any mode of transmission other than sexual, blood-borne, or intrauterine. It seems, therefore, that it can be concluded from the current literature that the likelihood of contracting AIDS from performing mouth-to-mouth ventilation on a person or a manikin is essentially nonexistent.

In spite of the preponderance of the literature against the casual spread of HIV and the continuing recognition of the importance of ventilation, the latest Guidelines go on to state “As a minimum action, in situations perceived as high-risk for disease transmission, the lay rescuer should assess the victim’s responsiveness, call for help, position the victim, open the airway, and, in the absence of a pulse, perform chest compressions.” Ventilation is conspicuously absent. The Guidelines continue by pointing out that lack of ventilation can mean death or disablement of an otherwise healthy person, although “risk to the rescuer, even with a known HBV-HIV-positive victim, is considered very low.”

Upon reviewing all of the documents, we conclude that it seems the AHA is violating what has been an unwritten rule—but nonetheless good science—that recommendations from the ECC be based on conclusions from well-designed studies and literature reviews. Thus, in spite of the fact that the literature supports the virtual nonexistence of risk of transmis-
sion of Hepatitis B or HIV via mouth-to-mouth ventilation, the ECC does not recommend performing artificial ventilation by that method when the perceived risk is high (a position that, at the same time, appears to minimize the fact that apparently healthy persons can transmit HIV contagion). The recommendation does no service to communities (such as our county) that are attempting to increase the numbers of lay rescuers; it could potentially reduce both the number of people who take CPR courses and the number of qualified rescuers who will choose to perform CPR on a stranger. The AHA emphasizes the role of airway maintenance and ventilation; yet, under the ECC Supplemental Guidelines, a person who has suffered a complete upper airway obstruction may not receive proper emergency care because the airway obstruction will go undetected unless the rescuer attempts to ventilate the victim.

In light of this scenario—and as food for thought and discussion—we propose the following addition to the ECC Supplemental Guidelines: In resuscitation efforts, a patent airway must be established and adequate ventilation must be provided. We suggest that all courses in Basic Life Support (BLS) include instruction in the use of pocket mask and one-way valve and that such a pocket mask be supplied to participants with further emphasis on storing the device in a readily accessible place for emergency use. The mask is an additional expense that some may find unacceptable, but an additional $10 to $12 (with some minimal additional cost for replacement valves and disinfection when necessary) seems a small price to pay to help ensure that maximal efforts will be made to save one’s fellowman.

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REFERENCES


Mouth-to-Mask Resuscitation by Lay Rescuers—Will They or Won't They?

Concern over disease transmission to rescuers providing mouth-to-mouth ventilation is not new. In the late 50s, 60s, and early 70s when mouth-to-mouth techniques were being developed, tuberculosis was still endemic to many Northeastern cities. Concern about disease transmission was aired by trainers of lay rescuers. To overcome what was believed to be a public concern about such intimate contact, two oropharyngeal airways were glued together to form the notorious S-tube. The rescue
breather positioned the tube in the victim's mouth, sealed the lips, and blew into the tube rather than providing mouth-to-mouth ventilation. These devices were demonstrated in lay courses and were offered free or for a nominal fee to those rescuers who desired them.

The S-tube device, known as The Resuscitator, had a very brief history. The lay public, despite the concerns of CPR instructors, adopted mouth-to-mouth ventilation wholeheartedly. It turned out that the instructors' concerns over this most intimate contact were not shared by the public at large. The result was that mouth-to-mouth ventilation was adopted and performed apparently without squeamishness or reservation by the public.

In 1990, we face a similar problem—that is, CPR instructors are concerned that the lay public will respond hysterically and with inappropriate concern for personal safety and will decrease resuscitation efforts. As amply explained by the authors of the opposing view, the risk of contracting AIDS, Hepatitis B, or any other infectious disease is extremely remote, if not zero, during practice sessions on manikins and from public rescue attempts.

I interpret the Supplemental Guidelines as a disclaimer intended to hold the American Heart Association blameless should any rescuer believe that he has contracted an infectious disease from performing CPR. I believe the intent is to avoid possible legal action.

Certainly, persons who are required to perform artificial ventilation in high-risk populations—trauma victims, hospitalized patients, and groups in whom endemic infection rates for contagious diseases are high—are trained in the use of pocket masks and other devices that preclude mouth-to-mouth ventilation. The CDC Guidelines mandate availability of such devices in hospitals.

What's the problem with providing lay rescuers with the opportunity to learn to use and purchase protective devices? I believe that the major problem is that instead of reducing fear of infectious disease transmission such an overly cautious approach will lead to an increased fear of this remote possibility.

Second, the mask will be helpful only if it is physically present with the rescuer at the time he or she is called upon to perform resuscitation. This means carrying the mask on one's person practically every waking moment. This is a practical problem if one wishes to increase the likelihood of field resuscitation's occurring in the general population—a circumstance in which resuscitation has been shown to increase survival. If resuscitation is undertaken only when such masks are available, the responder pool will diminish dramatically simply because would-be responders forgot their masks that day.

Has the fear of becoming infected by performing resuscitation diminished the public's interest in learning these techniques? My own informal survey of the local AHA's courses showed no decline in the number of lay persons requesting and attending these training sessions. In fact, over the last several years in Virginia, both available training slots and the length of the waiting list to receive training has increased. Certainly the public is showing no decreased interest in learning to resuscitate—even though mouth-to-mouth ventilation remains the standard at this time. The concerns seem to rest with CPR instructors only.

Because there is no apparent medical reason to suspect that a problem exists with disease transmission during manikin practice or in field resuscitations in the general population, is a solution necessary? For high-risk populations in whom resuscitation is expected and is likely to be performed—by hospital staff members, emergency medical technicians, police and fire personnel—protective devices (including gloves, masks, and gowns) are required as part of the employment environment. Lay rescuers many years ago rejected the instructors' solution to avoid mouth-to-mouth contact and made it a nonissue. I believe that the same is true today. Infectious disease transmission arising from population-based lay-provided CPR has not been a problem. The mandatory provision of training in mouth-to-mask ventilation and the necessity for purchasing a device to provide it would only serve to inappropriately increase the concern of the lay public. If it ain't broke, don't fix it!

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Blood Gas Corner #26—Pulmonary Artery Catheterization: Where Did the Blood Come From?

Joseph E Ollivier BA RRT

A 65-year-old man who had undergone an exploratory laparotomy was being mechanically ventilated with the following settings: SIMV 8, tidal volume 0.80 L, PEEP 8 cm H2O, and FIO2 0.40. A Swan-Ganz pulmonary artery catheter (PAC) (American Edwards Laboratories, Santa Anna CA) was placed 24 hours after surgery to optimize fluid management because of associated cardiac and renal failure.

The initial pulmonary artery pressures were 38/22 torr [5.1/2.9 kPa], and the pulmonary capillary wedge pressure was 12 torr [1.6 kPa].

The following morning the PAC failed to wedge and, therefore, was repositioned. Following repositioning, a blood sample was drawn from the distal port of the catheter simultaneously with a sample drawn from the radial artery catheter. Blood gas and pH values from these two blood samples are shown in Table 1. The therapist performing the procedure noted that the blood obtained through the distal port of the PAC was bright red. Because of the incompatibility of the values from these two blood samples, a second set of blood samples was drawn simultaneously with a sample from the proximal port of the PAC. Blood gas and pH values from this second set of samples are shown in Table 2.

**Table 1. Blood Gas and pH Values of the First Set of Samples Drawn from the Radial (RAC) and Pulmonary (PAC) Artery Catheters of a Patient Ventilated with 40% Oxygen**

<table>
<thead>
<tr>
<th></th>
<th>RAC (distal port)</th>
<th>PAC (distal port)</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.41</td>
<td>7.52</td>
</tr>
<tr>
<td>P\textsubscript{CO2} (torr)*</td>
<td>42</td>
<td>30</td>
</tr>
<tr>
<td>P\textsubscript{O2} (torr)*</td>
<td>100</td>
<td>199</td>
</tr>
<tr>
<td>HCO\textsubscript{3} (mEq/L)*</td>
<td>27</td>
<td>25</td>
</tr>
<tr>
<td>S\textsubscript{O2} (%)</td>
<td>98</td>
<td>98</td>
</tr>
</tbody>
</table>

*See box for SI conversion.

**Table 2. Blood Gas and pH Values of the Second Set of Samples Drawn from the Radial (RAC) and Pulmonary (PAC) Artery Catheters of a Patient Ventilated with 40% Oxygen**

<table>
<thead>
<tr>
<th></th>
<th>RAC (distal port)</th>
<th>PAC (distal port)</th>
<th>PAC (proximal port)</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.40</td>
<td>7.52</td>
<td>7.37</td>
</tr>
<tr>
<td>P\textsubscript{CO2} (torr)*</td>
<td>42</td>
<td>29</td>
<td>48</td>
</tr>
<tr>
<td>P\textsubscript{O2} (torr)*</td>
<td>97</td>
<td>218</td>
<td>41</td>
</tr>
<tr>
<td>HCO\textsubscript{3} (mEq/L)*</td>
<td>26</td>
<td>23</td>
<td>28</td>
</tr>
<tr>
<td>S\textsubscript{O2} (%)</td>
<td>98</td>
<td>98</td>
<td>73</td>
</tr>
</tbody>
</table>

*See box for SI conversion.

**Study Questions**

1. What is your interpretation of the pH and blood gas values displayed in Tables 1 and 2?

2. Where do you think the blood obtained through the distal tip of the PAC came from?

Answers and Discussion on the next page
**Answers**

1. **Interpretation of pH and blood gas values.** The acid-base status of both radial-artery blood samples is within normal limits; the oxygenation is consistent with that of a patient with an increased alveolar-arterial oxygen pressure gradient. The acid-base and oxygenation status of the blood sample drawn from the proximal port of the PAC is consistent with that of venous blood. Venous pH, P$_{O_2}$, and S$_O_2$ values are characteristically lower than their arterial counterparts; venous P$_{CO_2}$ is characteristically higher than arterial P$_{CO_2}$. The acid-base and oxygenation status of the blood samples drawn from the distal port of the PAC is not consistent with that of the radial-artery blood samples, nor is it representative of mixed-venous (pulmonary artery) blood.

2. **Source of blood.** The pH and blood gas values of the samples drawn from the distal port of the PAC are consistent with those of ‘wedged’ blood—blood beyond the distal tip of the PAC obtained when the balloon is inflated and wedged in the pulmonary artery preventing mixed-venous blood from being aspirated. Brewster and Mcllroy have shown (during room air breathing) that the P$_{O_2}$ of wedged blood can be 15 to 55 torr [2.0 to 7.3 kPa] higher than systemic arterial P$_{O_2}$, the P$_{CO_2}$ of wedged blood can be 2 to 21 torr [0.3 to 2.8 kPa] less than systemic arterial P$_{CO_2}$, and the pH of wedged blood can be 0.02 to 0.21 units higher than systemic arterial pH.

**Discussion.** The pH and blood gas values of the samples drawn from the distal port of the PAC are consistent with blood flowing through a compartment of the lung with high ventilation-to-perfusion (V-Q) ratios—the P$_{O_2}$ is higher than arterial P$_{O_2}$ and P$_{CO_2}$ lower than arterial P$_{CO_2}$. In this case, it is likely that the high V-Q ratios were artificially induced by vascular occlusion caused by the improper positioning of the PAC. A chest radiograph taken subsequent to the blood drawing revealed that the tip of the PAC was positioned in the upper one third of the right lung. The most probable hypothesis to explain the blood gas values of wedged blood is that the PAC balloon or the catheter itself obstructs blood flow through the specific vessel in which it is situated; then, while ventilation continues, blood is drawn from adjacent, normally perfused areas of the lung. This causes oxygenated postcapillary blood to pass back through ventilated portions of the lung a second time. Because of increased exposure time to alveolar gas, the oxygen content of the blood increases and the CO$_2$ content decreases.

It has also been shown that the speed with which blood is drawn from the distal port of a PAC can affect the blood gas values of the sample. It is therefore possible that a wedged blood sample may also be obtained when blood is withdrawn too rapidly through the distal port of a properly positioned PAC.

When drawing blood from the distal port of a PAC, it is important to verify that the catheter tip is located proximally in the pulmonary artery. It is also advisable to withdraw the blood slowly; this will decrease the likelihood of contaminating the mixed-venous blood sample with wedged (oxygenated postcapillary) blood.

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Superior Mediastinal Mass with Bilateral Innominate Vein Obstruction

Michael F Tenholder MD and James K Smith MD

A 79-year-old black man was evaluated at our hospital for swelling of the left arm and pain in the left shoulder that had persisted for one week. The patient was 5-ft 9-in, 188-lb, and afebrile, with a pulse of 90 and blood pressure of 135/80 (equal in both arms). A palpable, firm, nonpulsatile cervical mass extending into the left supraclavicular fossa and moderate edema of the left arm were noted.

The patient denied experiencing heat or cold intolerance, fever, chills, cough, or weight loss. He was a nonsmoker, but had chewed tobacco for 50 years. Medical history included a partial hemicolecotomy (performed two years before the present admission, for diverticular disease) and pacemaker placement (performed one year before for coronary artery disease complicated by ventricular dysrhythmias).

A chest radiograph was obtained and revealed a large superior mediastinal mass, with deviation of the trachea to the right (Fig. 1). Bilateral upper-extremity venography was performed, and revealed obstruction of the left subclavian vein and both left and right innominate veins. A large collateral vein was visible on the right, with numerous small collateral veins present bilaterally (Fig. 2).

Questions

Diagnosis: What is the probable diagnosis?
Actions Indicated: What further diagnostic testing and therapeutic intervention is indicated?

Fig. 1. The chest roentgenogram demonstrates superior mediastinal widening, rightward deviation of the trachea, and permanent pacemaker with lead traversing the great veins terminating in the apex of the right ventricle.

Fig. 2. Bilateral upper-extremity venography shows obstruction of the superior vena cava and both innominate veins.

Answers and Discussion on next page
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Answers and Discussion

Diagnosis: On the basis of the physical examination, chest radiograph, and bilateral upper-extremity venography, the presence of a thyroid goiter may be suspected; however, other diagnostic possibilities must be entertained including bronchogenic carcinoma with cervical node metastasis, lymphoma, mesenchymal tumor, and vascular aneurysm.

Actions Taken: Computed tomography (CT) of the chest with contrast vascular enhancement outlined a homogenous mass, small calcification within the mass, and smooth rightward displacement of the trachea and esophagus (Fig. 3). Thyroid function tests were within the normal range. A technetium thyroid scan demonstrated poor uptake. Fiberoptic bronchoscopy showed extrinsic tracheal compression without lesions in the tracheobronchial tree. Transbronchial needle aspirate was nondiagnostic. A CT-directed needle biopsy of the left neck mass yielded follicular cells consistent with thyroid tissue. Anti-thyroid globulin antibodies and anti-microsomal antibodies were negative. Pulmonary function testing revealed FVC 2.81 L (71% predicted), FEV₁ 2.2 L (89% predicted), and FEV₁/FVC 0.80. The flow-volume loop demonstrated a normal pattern of inspiratory and expiratory airflow.

The diagnostic testing confirmed the suspicion of a thyroid goiter with a large substernal component. The patient refused surgery, and thyroid suppression therapy was initiated. Thyroxine dosage was increased (over the next 2 years) to 0.076 mg/day, and the patient became mildly thyrotoxic. Thyroid suppression was steadily reduced and discontinued 6 months later. The results of follow-up chest radiography and computed tomography (performed 1½ years after thyroid suppression was discontinued) were unchanged from those obtained 4½ years earlier when the initial diagnosis had been made.

Discussion: The presence of cervical and mediastinal masses in the older patient most often denotes a neoplastic process. This generalization is especially true when signs of vascular obstruction are also present. Fortunately in the case being discussed, a relatively benign diagnosis—thyroid goiter—accounted for the patient's signs and symptoms.

Goiters are a relatively common cause of substernal masses; intrathoracic goiters occur in some 4 to 10% of patients undergoing thyroidectomy.¹,² Intrathoracic goiters generally occupy the superior and anterior mediastinum, less frequently involve the posterior compartment, and rarely occur in the inferior mediastinum.³ Approximately 10% of surgically removed intrathoracic goiters lie entirely within the thorax and lack any associated cervical mass.¹,² Masses appear with equal frequency on right and left sides of the trachea.¹,² Although multinodular and follicular histology is characteristic of the majority of intrathoracic goiters, the incidence of thyroid cancer has been reported to be 1 to 3 percent.¹

Many intrathoracic goiters are asymptomatic,¹ however, tracheal and esophageal deviation is commonly observed and a definite risk exists for sudden and potentially life-threatening glandular enlargement resulting from hemorrhage, cystic degeneration, or malignant changes.²,³ Katlic and colleagues¹ described symptoms in 80 patients undergoing resection of substernal goiter: Dysphagia was experienced by 16 patients, dyspnea and stridor (often related to posture) by 22, hoarseness by 10, wheezing by 7, cough by 6, facial flushing (related to position) by 2; and a cervical mass was present in 55 of these patients. Ten patients were asymptomatic.

Edema and vascular engorgement of the face and/or upper extremities is an unusual but well-defined complication that results from the entrapment of veins by a large intrathoracic goiter. In a series of 733 cases of superior vena cava (SVC) syndrome, reported by Kamiya and colleagues,⁴ only four cases resulted from intrathoracic goiter. In a series of 70 cases of intrathoracic goiter, reported by Cho and colleagues,²
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Table 2. Results of Symptom-Limited Exercise Test

<table>
<thead>
<tr>
<th>Minutes into the Test</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>4</th>
<th>6</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td>Work load (mph/grade)</td>
<td>0.0</td>
<td>1.0</td>
<td>1.0</td>
<td>3.0</td>
<td>3.0</td>
<td>3.0</td>
</tr>
<tr>
<td>(F_{\text{O}_2})</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>82</td>
<td>85</td>
<td>87</td>
<td>89</td>
<td>94</td>
<td>98</td>
</tr>
<tr>
<td>Systolic pressure (torr)†</td>
<td>112</td>
<td>118</td>
<td>120</td>
<td></td>
<td></td>
<td>90</td>
</tr>
<tr>
<td>Diastolic pressure (torr)†</td>
<td>62</td>
<td>68</td>
<td>60</td>
<td></td>
<td></td>
<td>58</td>
</tr>
<tr>
<td>Arterial pH</td>
<td>7.40</td>
<td>7.41</td>
<td>7.41</td>
<td>7.41</td>
<td>7.40</td>
<td>7.40</td>
</tr>
<tr>
<td>(P_{\text{ACO}_2}) (torr)†</td>
<td>36</td>
<td>35</td>
<td>35</td>
<td>36</td>
<td>36</td>
<td>38</td>
</tr>
<tr>
<td>(P_{\text{AO}_2}) (torr)†</td>
<td>56</td>
<td>40</td>
<td>38</td>
<td>36</td>
<td>33</td>
<td>32</td>
</tr>
<tr>
<td>(S_{\text{PO}_2}) (%)</td>
<td>89</td>
<td>72</td>
<td>68</td>
<td>67</td>
<td>59</td>
<td>53</td>
</tr>
<tr>
<td>(S_{\text{SPO}_2}) (%) (measured)</td>
<td>88</td>
<td>71</td>
<td>67</td>
<td>66</td>
<td>57</td>
<td>54</td>
</tr>
<tr>
<td>(S_{\text{SPO}_2}) (%) (calculated)</td>
<td>89</td>
<td>75</td>
<td>72</td>
<td>71</td>
<td>63</td>
<td>61</td>
</tr>
</tbody>
</table>

*Predicted maximum heart rate: \(220 - \text{age} = 198\).
†See box for SI conversions.

(20 min after the resting patient began breathing 100% oxygen) and sequentially during the exercise test. Calculated \(S_{\text{SPO}_2}\) values (IL 1306 pH-Blood Gas Analyzer, Instrumentation Laboratory, Lexington MA), directly measured \(S_{\text{SPO}_2}\) values (Il 282 CO-Oximeter Instrumentation Laboratory, Lexington MA), \(S_{\text{PO}_2}\) values (Ohmeda 3700 Pulse Oximeter, Ohmeda, Louisville CO), and other exercise data are reported in Table 2. The patient reached only 6% of her predicted maximum work capacity before exhibiting profound hypoxemia with a concomitant fall in systolic blood pressure to 22 torr [2.9 kPa] below her resting level, at which point the test was terminated. No dysrythmias were noted.

Study Questions

1. Do results shown in Table 1 suggest that the patient’s pulmonary function contributes to her dyspnea and exercise intolerance?
2. What do you think is the source of this patient’s dyspnea and exercise intolerance?
3. Which of the oxyhemoglobin saturation values is most likely to be correct?

Answers and Discussion on the next page

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signs of vascular compression were noted in only 5 patients (3 of the 5 had overt SVC syndrome). In an analysis of 80 patients with substernal goiter from Massachusetts General Hospital, only one patient is reported to have exhibited facial plethora. Likewise, out of 872 patients undergoing thyroidectomy at the University of Michigan, only 5 manifested symptoms of vascular compression. Of these 5, 4 had benign disease; and in no patient were symptoms associated with SVC syndrome responsible for the patient seeking medical attention.

Bilateral vocal cord paralysis with acute stridor and “downhill” varicosities (a bleeding diathesis from esophageal varices) are unusual complications that have been reported in patients with intrathoracic goiter and SVC compression.

As the present case demonstrates, symptomatic but otherwise uncomplicated vascular compression may follow a rather benign course when associated with substernal goiter.

Diagnosis of a substernal goiterous mass may be difficult. A noninvasive approach based upon the demonstration of substernal extension of cervical thyroid tissue is preferred, and only rarely is mediastinal exploration necessary for diagnosis or therapy. Most patients are euthyroid, but hypothyroidism may be seen—depending on goiter type. An enlarged cervical gland is detected in at least half of the cases. Radionuclide scanning can be of great diagnostic utility, but poor iodine-uptake and/or prior administration of iodine (as a radiopaque dye) limits the usefulness of this test. Computed tomography has proved to be a valuable diagnostic adjunct by demonstrating clear continuity of the goiter with a cervical thyroid gland and delineating well-defined borders. Goiters characteristically appear on a CT scan as nonhomogenous masses with discrete nonenhancing low-density areas. Coarse ring-like calcification appears in a majority of cases (Fig. 3). Only rarely is an intrathoracic goiter assessable by fine-needle aspiration. Flow-volume loops are indicated in all patients with goiters involving the thoracic inlet because important tracheal obstruction can sometimes be detected prior to the onset of respiratory symptoms. Flattening of the inspiratory limb of the flow-volume loop (variable extrathoracic obstruction) is expected when the extrathoracic trachea is significantly narrowed by cervical goiter. Similarly, attenuation and plateau of expiratory flow (variable intrathoracic obstruction) results when the thoracic trachea is sufficiently compromised. A combined pattern may result if thoracic and cervical sections of the trachea are both involved. Modifying patient position, including extending the arms and neck, may demonstrate postural decrement in airflow.

The collective experience to date suggests that exogenous thyroid suppression therapy has little effect on gland size. Because of the potential for respiratory compromise, dysphagia, and, more rarely, thyrotoxicosis and cancer, the treatment of choice is surgical removal. (In the case being discussed, thyroid suppression therapy was performed because the patient refused to undergo surgery.) Most glands irrespective of size or degree of mediastinal extension are surgically accessible by means of a cervical approach; therefore, mediastinal exploration is often unnecessary. All cases not surgically corrected warrant close observation for the appearance of complications.

REFERENCES

A 22-year-old female medical technology student, with diagnoses of tetralogy of Fallot and Eisenmenger syndrome, was referred to the pulmonary function laboratory for evaluation because she had noticed increasing dyspnea (during fast walking on level ground and occasionally while resting at night). A Waterson-Cooley anastomosis had been performed on the patient when she was 3 years old, in an attempt to improve blood flow to the lungs. She had been able to participate in many social activities and to keep up with her friends during physical activities until she reached about 20 years of age, at which time she began experiencing dyspnea on exertion (eg, during moderate exercise or after stairclimbing). She also began experiencing severe headaches after playing volleyball and football, and occasional hemoptysis. Even though she was chronically hypoxemic and polycythemic, she had been unwilling to wear any type of supplemental oxygen device because of her perception of the 'social stigma' associated with these devices.

Physical examination revealed a well-nourished female (height 67 in and weight 128 lb [94.8% ideal body weight]) in no apparent distress despite digital clubbing with mild cyanosis. Chest auscultation revealed clear breath sounds throughout all lung fields.

The analysis of arterial blood drawn while the patient (at rest) breathed room air, revealed: pH 7.40, PaCO₂ 36 torr [4.8 kPa], PaO₂ 46 torr [6.1 kPa], Table 1. Pulmonary Function Values

Table 1. Pulmonary Function Values

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value (83)</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC (L)</td>
<td>3.57</td>
</tr>
<tr>
<td>FEV₁ (L)</td>
<td>2.93</td>
</tr>
<tr>
<td>FEV₁/FVC%</td>
<td>82</td>
</tr>
<tr>
<td>PEFR (L/s)</td>
<td>6.30</td>
</tr>
<tr>
<td>FEF₂⁵⁻⁷⁵ (L/s)</td>
<td>2.79</td>
</tr>
<tr>
<td>MVV (L/min)</td>
<td>101</td>
</tr>
<tr>
<td>Raw</td>
<td>2.20</td>
</tr>
<tr>
<td>TLC (L)</td>
<td>5.32</td>
</tr>
<tr>
<td>RV/TLC%</td>
<td>33</td>
</tr>
<tr>
<td>DlCO (mL·min⁻¹·torr⁻¹)</td>
<td>26.40</td>
</tr>
</tbody>
</table>

*Values in parentheses are % of predicted.
†See Box for SI conversions.

To assess the refractory nature of the hypoxemia and to evaluate the patient’s cardiopulmonary response to minimal exertion, a symptom-limited treadmill exercise test was carried out with the patient breathing 100% oxygen. Supplemental oxygen was administered via a large two-way non-rebreathing valve, with a 5-L reservoir bag attached with sufficient oxygen flow to prevent deflation of the bag during rapid, maximal patient inspiration (the patient wore nose clips and breathed through a mouthpiece). Three-lead electrocardiogram (ECG) monitoring and pulse oximetry were performed continuously, and blood pressure was measured sequentially during the exercise test. A right-radial artery catheter was placed for blood gas sampling; blood gas samples were obtained just prior to the exercise test.

A Guide to the Use of SI in This Paper*

The SI unit for pressure is the kilopascal (kPa).

\[(\text{torr}) \cdot (0.1333) = \text{kPa}\]

The SI unit for amount of substance is millimoles per liter (mmol/L).

\[\text{mEq/L} = \text{mmol/L}\]

The SI unit for resistance is kPa·s·L⁻¹.

\[(\text{cm} \cdot \text{H}_2 \cdot \text{O} \cdot \text{s} \cdot \text{L}^{-1}) \cdot (0.09806) = \text{kPa} \cdot \text{s} \cdot \text{L}^{-1}\]

The SI unit for gas transfer (diffusion) is mL·min⁻¹·kPa⁻¹.

\[(\text{mL} \cdot \text{min}^{-1} \cdot \text{torr}^{-1}) \cdot (0.1333) = \text{mL} \cdot \text{min}^{-1} \cdot \text{kPa}^{-1}\]

*For further information on SI (le Systeme International d'Unites), see Respir Care 1988;33:661-673 (October 1988) and Respir Care 1989;34:145 (February 1989).
Answers and Discussion

1. Interpretation of Findings: With the exception of the slightly elevated \( R_{aw} \) (predicted normal is \(< 2.0 \text{ cm} H_2O \cdot s \cdot L^{-1} (0.18 \text{ kPa} \cdot s \cdot L^{-1}) \)),² which may be indicative of mild airflow limitation, the other pulmonary function data in Table I are essentially normal. Therefore, it is doubtful that pulmonary function contributes to this patient's dyspnea and exercise intolerance.

2. Source of Dyspnea: The source of this patient's dyspnea and exercise intolerance appears to be cardiovascular in nature.

3. Correct Oxyhemoglobin Saturation: Of the three methods used in this case to determine arterial oxyhemoglobin saturation, only direct measurement using a spectrophotometric technique can be depended upon to provide correct values.

Discussion: It is likely that the exercise-induced oxyhemoglobin desaturation and drop in systolic blood pressure were attributable to this patient's cardiovascular disease. During the Waterston-Cooley procedure performed on this patient when she was 3 years of age, a shunt was created between the ascending aorta and the right main pulmonary artery. The procedure was merely palliative, and therefore the four defects of tetralogy of Fallot¹ continued to exist: a large ventricular septal defect (VSD), pulmonic or right-ventricular infundibular stenosis, an aorta that overrides the VSD, and right-ventricular hypertrophy. In the presence of these cardiac defects, it is not surprising that blood pressure dropped as the subject attempted to exercise; her cardiac output simply could not be increased in proportion to the increased systemic demand and associated vasodilation.

Pulmonary hypertension is a consequence of the chronic hypoxemia associated with tetralogy of Fallot. Eisenmenger syndrome is also characterized by severe pulmonary hypertension (secondary to obstructive pulmonary vascular disease).³ It results from intracardiac shunting and V/Q mismatch, the lungs of our subject were unable to adequately oxygenate the blood; this inadequacy became more profound during exercise. This patient was eventually admitted to our hospital for heart-lung transplant evaluation, the only viable form of corrective surgery for her.

Pulse oximetry readings are often rendered inaccurate by motion and are dependent upon adequate perfusion of the monitored site. During every exercise procedure performed in our laboratory, three-lead (minimum) ECG monitoring is performed along with pulse oximetry at two sites—finger and ear. Because inflation of the blood-pressure cuff reduces perfusion to the fingers of the affected hand, a finger on the opposite hand should be used for \( \text{SpO}_2 \) monitoring. Whenever perfusion to the monitored site is reduced, the oximeter signal quality decays. This may be seen as a fluctuating or failing pulse-oximeter heart rate (HR) reading. By employing the two-site method, the oximeter probe can rapidly be changed to the alternate site if the pulse-oximeter HR begins to differ from the electrocardiogram HR by more than 5 beats/min. Assuring the match of pulse-oximeter HR with electrocardiogram HR helps to optimize and better validate pulse oximetry measurement.

\( \text{SpO}_2 \) measurements have traditionally been considered unreliable or suspect at values \(< 60\% \). Because we knew that this patient was chronically hypoxemic at rest and potentially could desaturate further during exercise (even while breathing 100% oxygen), we inserted an arterial catheter so that arterial blood-gas sampling and analysis could easily be performed in addition to pulse oximetry. Interestingly, there was a close correlation between \( \text{SpO}_2 \) and measured \( \text{SaO}_2 \) throughout this exercise test. It may be that insufficient datapoints have been available for comparison of \( \text{SpO}_2 \) and measured \( \text{SaO}_2 \) at these lower levels.

The calculated \( \text{SaO}_2 \) is essentially derived from measured pH, measured PO\(_2\), and the ideal oxyhemoglobin dissociation curve. As a result of chronic hypoxemia, the patient's 2-3DPG levels may have been elevated to facilitate oxygen release to the tissues. An elevated 2-3DPG level causes a rightward shift of the oxygen dissociation curve, which, in this case, could be the reason why the 'calculated' resting \( \text{SaO}_2 \) was higher than the actual 'measured' resting \( \text{SaO}_2 \). During exercise, it is also possible that the subject's body temperature may have increased sufficiently to cause the oxygen dissociation curve to shift even further to the right, which, in this case, could be the reason why the disparity between calculated and measured \( \text{SaO}_2 \) widened as exercise continued. In the presence of a rightward shift of the oxygen dissociation curve (which can result from elevated body temperature, 2-3DPG, \( P_{ACO_2} \), and hydrogen ion concentration), calculated \( \text{SaO}_2 \) will always be an overestimation of actual measured \( \text{SaO}_2 \).

While pulse oximetry may provide an easy means to noninvasively assess oxygenation, its accuracy depends upon meeting certain absolute criteria. The same holds true for calculation of oxyhemoglobin saturation from blood gas variables. At the present time direct measurement is the method of choice for determining oxyhemoglobin saturation; the other methods have too many factors that can affect their accuracy.

REFERENCES

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Fatalities have been reported in association with excessive use of inhaled sympathomimetic drugs. The exact cause of death is unknown, but cardiac arrest following the unexpected development of a severe acute asthmatic crisis and subsequent hypoxia is suspected.

Immediate hypersensitivity reactions may occur after administration of albuterol inhaler; as demonstrated by rare cases of urticaria, angioedema, rash, bronchospasm, anxiety, and oro-pharyngeal edema.

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Large doses of intravenous albuterol have been reported to aggravate pre-existing diabetes mellitus and ketoads. Adverse beta-agonists, including albuterol, given intravenously may cause a decrease in serum potassium, possibly through intracellular shifting. The decrease is usually transient, not requiring supplemental hydration. The relevance of these observations to the use of Ventolin® Inhalation Aerosol is unknown, since the aerosol dose is much lower than the doses given intravenously.

Although there have been no reports concerning the use of Ventolin Inhalation Aerosol during labor and delivery, it has been reported that high doses of albuterol administered intravenously inhibit uterine contractions. Although this effect is extremely unlikely as a consequence of aerosol use, it should be kept in mind.

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See illustrated Patient’s Instructions for Use section of the package insert.

**Drug Interactions:**
Other sympathomimetic aerosol bronchodilators should not be used concomitantly with albuterol. If additional adrenergic drugs are to be administered by any route, they should be used with caution to avoid deleterious cardiovascular effects.

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

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

Carcinogenesis, Mutagenesis, Impairment of Fertility: Albuterol sulfate, like other agents in its class, caused a significant dose-related increase in the incidence of benign neoplasms of the mesonephros in a two-year study in the rat, at doses corresponding to 111, 555, and 2,900 times the maximum human inhalational dose. In another study this effect was blocked by the coadministration of propranolol. The relevance of these findings to humans is not known. An 18-month study in mice and a limited study in hamsters revealed no evidence of tumorigenesis. Studies in rats with albuterol revealed no evidence of mutagenesis. Reproduction studies in rats revealed no evidence of impaired fertility.

Pregnancy: Teratogenic Effects: Pregnancy Category C: Albuterol has been shown to be teratogenic in mice when given in doses corresponding to 14 times the human dose. There are no adequate and well-controlled studies in pregnant women. Albuterol should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus. A reproduction study in CD-1 mice given albuterol subcutaneously (0.035, 0.2, and 2.5 mg/kg, corresponding to 1.7, 14, and 140 times the maximum inhalational dose, respectively) showed cleft palate formation in 5% of 111 (5%) fetuses at 0.25 mg/kg, and in 10 of 166 (6%) fetuses at 2.5 mg/kg. None was observed at 0.025 mg/kg. Cleft palate also occurred in 22 (3.5%) fetuses treated with 2.5 mg/kg spiroterenol (positive control). A reproduction study with oral albuterol in Sprague-Dawley rats revealed canoide scars in 7 of 19 (36%) fetuses at 50 mg/kg, corresponding to 2,800 times the maximum human inhalational dose of albuterol.

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

Pediatric Use: Safety and effectiveness in children below 12 years of age have not been established.

**ADVERSE REACTIONS:**
The adverse reactions to albuterol are similar in nature to reactions to other sympathomimetic agents, although the incidence of certain cardiovascular effects is less with albuterol. A 13-week double-blind study compared albuterol and isoproterenol aerosols in 57 asthmatic patients. The results of this study showed that the incidence of cardiovascular effects was palpitations, less than 10 per 100 with albuterol and less than 15 per 100 with isoproterenol; tachycardia, 10 per 100 with both albuterol and isoproterenol; and increased blood pressure, less than 5 per 100 with both albuterol and isoproterenol. In the same study, both drugs caused tremor or nausea in less than 15 patients per 100, and dizziness or heartbeat in less than 5 per 100 patients. Nervousness occurred in less than 10 per 100 patients receiving albuterol and in less than 15 per 100 patients receiving isoproterenol.

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

In addition, albuterol like other sympathomimetic agents, can cause adverse reactions such as hypertension, angina, vomiing, vertigo, central nervous system stimulation, insomnia, headache, unusual taste, and drying or irritation of the oropharynx.

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Serial Number(s)

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Date event occurred ________________

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To the manufacturer/distributor ☐

Other ___________________________

If requested, will the actual product involved in the event be available for evaluation by the manufacturer or FDA? YES ☐ NO ☐

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*In Maryland, call collect (301) 881-0256 between 9:00 AM and 4:30 PM

Obstructive Sleep Apnea Syndrome provides a broad-ranging synopsis of epidemiology, pathophysiology, and therapeutic regimens related to multiple obstructive apneas during sleep. The text provides a summary of research presented at a recent international symposium held in Siuntio, Finland. The editors (Dr Christian Guilleminault of the Sleep Research Center, Stanford University School of Medicine, and Dr Markku Partinen, Department of Neurology, University of Helsinki, Finland) are internationally recognized for their expertise in the diagnosis and treatment of sleep-related breathing disorders.

The reader will notice that material presented in one chapter may be disputed by the author of a different chapter. For instance, Chapter 4, Snoring and Sleep Apnea, offers a new, objective definition of gauging the severity of sleep apnea. The Italian group Lugaresi et al proposes that greater than 30 apneas/h of sleep be used as the criterion to distinguish between subclinical and clinical disease states. They also hypothesize that the somnolence associated with sleep apnea is caused by a lowered reactivity of the respiratory and vigilance centers of the brain rather than by frequently disrupted sleep patterns.

This definition of abnormality (greater than 30 apneas/h of sleep) is quite different than the currently accepted definition of greater than five apneas/h of sleep as discussed in Chapter 1, Epidemiology of Sleep Apnea.

Just as the diagnostic criteria vary from chapter to chapter, so do the suggested points of therapeutic intervention. Lugaresi’s group (Chapter 4) suggests that sleep apnea syndrome should not be treated until after a subject has reached the defined point of clinical relevance (> 30 desaturations/h). However, Dr Guilleminault suggests in Chapter 9 that therapy should be instituted in anyone with disrupted sleep and a complaint of tiredness or fatigue, even if desaturations do not occur. Hence, his clinical decision is based on symptoms, hypopneas, and sleep interruptions rather than some number of apneas during sleep.

Chapter 9, Treatments in Obstructive Sleep Apnea, highlights Stanford University’s experience with pharmaceutical trials, nasal continuous positive airway pressure (CPAP), and orthodontic and surgical approaches to sleep apnea. Their conclusion is that, when indicated, corrective surgeries such as uvulopalatopharyngoplasty, maxillofacial surgery, or linguoplasty should be used. Even though the reported cure rate for surgical intervention is approximately 50-60%, the Stanford group appears to suggest that aggressive surgical management is the treatment of choice, with nasal CPAP being reserved only for surgical failures.

While the book does focus a great deal on Stanford’s experience with surgical interventions, it unfortunately does not present a chapter written by a representative of an institution with a high success rate with noninvasive therapies for sleep apnea. This bias probably reflects the lack of information on uniform success or failure with different interventions at various institutions.

Chapter 13, Technical Issues Related to Obstructive Sleep Apnea Syndrome, describes two devices used in Europe for outpatient assessment of sleep-disordered breathing. While these devices are less sophisticated than those recently available in the United States, they have been used successfully by European physicians and sleep researchers since the late 1980s.

It appears that the Europeans are more comfortable with ambulatory assessment of sleep-disordered breathing than are their American counterparts. This may be due in part to the limited availability of fully equipped polysomnographic laboratories in Europe or to greater acceptance of outpatient diagnostic procedures by European physicians.

In summary, Obstructive Sleep Apnea Syndrome describes current diagnostic and therapeutic approaches to the sleep apnea syndrome from primarily Scandinavian and European countries. The text illustrates the need for establishing sensitive and specific criteria for the diagnosis of sleep apnea syndrome, for determination of severity, and for therapeutic approaches and outcome.

Although some chapters present data developed from established, reproducible protocols, others offer unique information and ideas that have yet to be rigorously validated. Therefore, rather than considering this book a standard reference, the reader should view this book as being a resource that provides insight into the variety of clinical practices and research related to sleep-disordered breathing.

Michael J Decker CRTT
Research Assistant
Clement Cahan MD
Research Fellow
Kingman P Strohl MD
Chief
Pulmonary and Critical Care Medicine
University Hospitals of Cleveland
Case Western Reserve University
Cleveland, Ohio
Notices of competitions, scholarships, fellowships, examination dates, new education programs, and the like will be listed here free of charge. Items for the Notices section must reach the Journal 60 days before the desired month of publication (January 1 for the March issue, February 1 for the April issue, etc). Include all pertinent information and mail notice to Respiratory Care Notices Dept, 11030 Ables Lane, Dallas TX 75229.

ARCF Literary Award

- The American Respiratory Care Foundation announces a $1000 Literary Award—funded by Radiometer America Inc—for the best case report published in Respiratory Care from October 1989-December 1990. The winner will be announced on December 8, 1990, at the AARC Annual Meeting, and in the January 1991 issue of Respiratory Care. All case reports will be considered for the award, and no application is necessary.

AARC ANNUAL CONVENTION SITES & DATES

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1990 Examination and Fee Schedule

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**Fee Schedule**

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8310 Nieman Road • Lenexa, Kansas 66215 • (913) 599-4200
August 22-24 in Atlantic Beach, North Carolina. The North Carolina Society for Respiratory Care presents its 12th Annual "Symposium by the Sea" at the Atlantic Beach Sheraton and Conference Center. Featured speakers include Paul Cheney MD, speaking on "Chronic Fatigue Syndrome." Contact Ms Barbara Chaney RRT, Mercy Hospital South, Charlotte NC 28210. (704) 543-2031.

August 23-25 in Savannah, Georgia. The Georgia Society for Respiratory Care presents its Annual Summer Meeting. Special activities include a reception for department managers and a golf tournament. Contact Bob Burnauh at (404) 944-5189.

September 5-6 in Pittsburgh, Pennsylvania. The Pennsylvania Society for Respiratory Care sponsors its 17th Annual Seminar on Pulmonary Medicine and Physiology at the Sheraton Hotel at Station Square. The conference topics include recent advances in pulmonary medicine. Small discussion groups will focus on specific problems and intervention. Contact Lynda Brynner RRT, Respiratory Care Dept, North Hills Passavant Hospital, 9100 Babcock Blvd, Pittsburgh PA 15237. (412) 367-5428.

September 13-14 in Glen Ellyn, Illinois. The Illinois Society for Respiratory Care's Home Care Committee and the College of DaPage/Business and Professional Institute present "Continuity of Pulmonary Care" at the College of DaPage Open Campus Center. This conference is designed for health care professionals involved in home care issues. The conference is valuable for home-health nurses, respiratory therapists, discharge planners, home medical equipment providers, social service professionals, physical therapists, occupational therapists, and others interested in respiratory care. Contact Carol Kozlowski at (708) 858-2800, ext 2904.

September 13-14 in Jackson Hole, Wyoming. The Wyoming Society for Respiratory Care presents its Roundup '90 Annual Educational Workshop at the Teton Village, just 12 miles west of Jackson Hole. Beneath the breathtaking Teton Mountain Range and 12 miles south of Yellowstone National Park, this seminar offers scenic beauty and a list of outstanding speakers. Golf and whitewater rafting round out the workshop. Contact Mike Arndt, Memorial Hospital, 300 E 23rd St, Cheyenne WY 82001. (307) 775-7701.

September 15-16 in Boise, Idaho. The Idaho Society for Respiratory Care presents its 1990 Annual Educational Seminar at the Anderson Center of St Luke's Regional Medical Center. Contact Jeanne O'Hara, 10 Highway 95, Payette ID 83661. (208) 642-9293.

September 18-19 in Honolulu, Hawaii. The Hawaii Society for Respiratory Care presents its 17th Annual Respiratory Care Conference at the Hilton Hawaiian Village Hotel. Contact Helen M Ono RRT, 1717 Palolo Ave, Honolulu HI 96816. (808) 547-9532.

September 19-21 in St Cloud, Minnesota. The Minnesota Society for Respiratory Care presents its 21st Annual Educational Meeting. Topics include respiratory care practice, current and future. Contact Carolyn Chaon, University of Minnesota Hospitals & Clinic, Cardio-Respiratory Services, Harvard Street at East River Rd, Box 247, Minneapolis MN 55455. Or phone (612) 625-3976 (8 AM-4:30 PM CST).

September 19-22 in Guatemala City, Guatemala. The Central American Association for Respiratory Care presents its 2nd Congress on Respiratory Care at the El Dorado Hotel in Guatemala. Contact Susan P Pilbeam, Respiratory Care, Greenville Tech, PO Box 5616, Greenville SC 29606-5616. (803) 250-8000, ext 2308.

September 20-21 in Napa, California. The California Society for Respiratory Care (Chapter 10), Napa Valley College, and the American Lung Association of the Redwood Empire cosponsor the 8th Annual Napa Valley Conference "Current Concepts in Cardiopulmonary Care." Topics include heart/lung transplantation, pressure support/control ventilation, care of the infant with BPD, pre- and post-op cardiac care, the intravascular oxygenator, and autogenous drainage. Nine CEUs offered. Contact Kate Benscoter at Napa Valley College. (707) 253-3141.

September 25-27 in Atlantic City, New Jersey. The New Jersey Society for Respiratory Care presents its annual Shore Conference at the Trump Castle Hotel and Casino. Contact Ed Mellon RRT at Shore Memorial Hospital, Somers Point NJ 08244. (609) 653-3729.

September 26-28 in Niagara Falls, New York. The Western New York Chapter of the New York State Society for Respiratory Care hosts the 11th Annual Statewide Respiratory Care Symposium. Neal Machtyre MD, Bruce Wilson MD, William Ferguson CRRT, and Donald Greenblatt MD are featured speakers. Contact Emilie Walker at Mount St Mary's Hospital, 5300 Military Rd, Lewiston NY, or call (716) 298-2142.

September 26-28 in Charleston, South Carolina. The South Carolina Society for Respiratory Care presents its 19th Annual Meeting, "Challenges of a New Decade," at the Marriott Hotel in Charleston. Contact Sandy Byrdie, SC SRC Annual Meeting, PO Box 8500, Florence SC 29501. (803) 661-3629.

September 26-28 in Rockland, Maine. The Maine Society for Respiratory Care presents its Annual Fall Seminar, "The Maine Event," at Sebasco Lodge. Topics include hemodynamics, BIPAP/CPAP, pediatric asthma, surfactant therapy, ethics, communication, and case-study workshops, as well as other topics of interest. Contact Janey Barthellette, Respiratory Care Department, Penobscot Bay Medical Center, Rockland ME 04841. (207) 596-8485.

September 26-28 in Frankenmuth, Michigan. The Michigan Society for Respiratory Care presents its Annual Fall Meeting at the Bavarian Inn Motor Lodge. Program topics include hemodynamic monitoring, capnography, metabolic assessment, ventilation techniques, pediatrics, sleep disorder concerns, and pulmonary rehabilitation. Special events include a golf outing and an outdoor steak fry. Contact Beth Hill RRT, Bay Medical Center, Respiratory Care Department, 1900 Columbus, Bay City MI 48708. (517) 894-3166.


October 7-13 in U.S.A. Respiratory care practitioners across the nation celebrate Respiratory Care Week. Turn to the special RC Week section in the July issue of AARC Times for more information.

November 2 in Jackson, Mississippi. The MSRC presents a mini-seminar on the ABCs of neonate/pediatric critical care. This meeting will be at the Holiday Inn-Medical Center. For more information, contact Donna Lindsey CPFT RRT, Northeast MS Community College, Cunningham Blvd, Booneville, MS 38929. (601) 728-7751, ext 387.

December 8-11 in New Orleans, Louisiana. The AARC presents its 36th Annual Convention and Exhibition at the New Orleans Convention Center. Contact the AARC, 11030 Ables Ln, Dallas TX 75229. (214) 243-2272.
Instructions for Authors and Typists

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

General Requirements

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

Publication Categories

Research Article (Study): A report of an original investigation.
Evaluation of a Device/Method/Technique: A description and evaluation of an old or new device, method, technique, or modification.
Case Report: A report of a clinical case that is uncommon or of exceptional teaching value. The author(s) must have been associated with the case. A case-managing physician must be one of the authors or, if not an author, must supply a letter approving the manuscript.
Case Series: Like a Case Report but including a number of cases.
Review Article: A comprehensive, critical review of the literature and state of the art of a pertinent topic that has been the subject of 40 or more published research papers.
Overview: A critical review of a pertinent topic about which not enough research has been published to merit a Review Article.
Update: A report of subsequent developments in a topic that has been critically reviewed (not necessarily in this journal).
Point of View: A paper expressing the author's personal opinions on a pertinent topic.
Special Article: If a paper does not fit one of the foregoing categories but is pertinent, the editors may consider it a Special Article.
Editorial: A paper that draws attention to a pertinent concern.
Letter: A signed communication about material published in this journal or on topics of interest or value to readers.

Blood Gas Corner: A brief, instructive case report (real or fictional) involving invasively or noninvasively obtained respiratory care blood data, followed by questions for readers— with answers and discussion.
PFT Corner: Like Blood Gas Corner but involving pulmonary function testing.
Test Your Radiologic Skill: Like Blood Gas Corner and PFT Corner but involving pulmonary-medicine radiography and including one or two 4 x 5 or 5 x 7 inch prints of radiographs. The case must be real.
Review of Book, Film, Tape, or Software: Anyone interested in writing a review can discuss it with an editor.

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

Preparing the Manuscript

General Concerns—Typist

• Double-space ALL lines, including those in references, figure legends, and tables. Do not justify right margins.
• Number pages in upper right corner and leave margins of 1¼ or more on all four sides of the page.
• For research articles, follow format of Model Manuscript, Respir Care 1984;29:182 (Feb 1984).
• Meticulously follow instructions for typing references.

General Concerns—Author:

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

Manuscript Structure

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


Evaluation of Device/Method/Technique: Title page, abstract page, continuous text (Introduction, Description of Device/Method/Technique, Methods of Evaluation, Results of Evaluation, Discussion), Product Sources page, Acknowledgments page, references, tables, figure legends.

Case Report or Case Series: Title page, abstract page, continuous text (Introduction, Case Summary, Discussion), Acknowledgments page, references, tables, figure legends. Also see “How To Write a Better Case Report,” Respir Care 1982;27:29 (Jan 1982).

Review Article: Title page, Table of Contents page, continuous text (Introduction, History, Review of Literature, State of the Art, Discussion, Summary), references. May include figures & tables. No abstract. Table of Contents optional. Other formats may be appropriate.

Overview, Update, Point of View, or Special Article: Title page, text (introduction, message), references, tables, figure legends. No abstract.

Letter: Title page (provide a title), text, writer’s name & affiliation, references. Tables & figures may be included. Double-space everything. Write “For Publication” on title page.

Structure: Important Details

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