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BiPAP® ventilatory assistance can increase minute ventilation and reduce respiratory effort, but does not always reduce Pa₄CO₂. We studied the effects of BiPAP ventilatory assistance on Pa₄CO₂ and examined specific mechanisms whereby BiPAP ventilatory assistance may not lower Pa₄CO₂. BiPAP ventilatory assistance using a non-rebreather valve and volume cycled ventilation at similar settings produced significantly lower Pa₄CO₂ than BiPAP ventilatory assistance using a standard exhalation device. The failure of Pa₄CO₂ to fall with the standard exhalation device was due to exhalation past the exhalation device into the ventilator tubing, subsequent rebreathing of the exhaled gases, and an increase in dead space ventilation. Use of other fixed-resistance exhalation devices also resulted in exhalation back into the ventilator tubing. Use of a new plateaus exhalation device or a non-rebreather valve eliminated CO₂ rebreathing and its effect on dead space ventilation. Changing exhalation devices had no significant effect on BiPAP pressure generation or sensing capabilities. Our results indicate that the use of a standard exhalation device during BiPAP ventilatory assistance causes CO₂ rebreathing, which can blunt any effect of BiPAP on Pa₄CO₂. Use of an appropriate alternative exhalation device can eliminate this problem.


BACKGROUND: Controversy exists as to the risk for postoperative apnea in former preterm infants. The conclusions of published studies are limited by the small number of patients. METHODS:
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The original data from eight prospective studies were subject to a combined analysis. Only patients having inguinal herniotomy under general anesthesia were included; patients receiving caffeine, regional anesthesia, or undergoing other surgical procedures were excluded. A uniform definition for apnea was used for all patients. Eleven risk factors were examined: gestational age, postconceptional age, birth weight, history of respiratory distress syndrome, bronchopulmonary dysplasia, neonatal apnea, necrotizing enterocolitis, ongoing apnea, anemia, and use of opioids or nondepoloarizing muscle relaxants. RESULTS: Two hundred fifty-five of 384 patients from eight studies at four institutions fulfilled study criteria. There was significant variation in apnea rates and the location of apnea (recovery room and postrecovey room) between institutions (P < 0.001). There was considerable variation in the duration and type of monitoring, definitions of apnea, and availability of historical information. The incidence of detected apnea was greater when continuous recording devices were used compared to standard impedance pneumography with alarms or nursing observations. Despite these limitations, it was determined that: (1) apnea was strongly and inversely related to both gestational age (P = 0.0005) and postconceptual age (P < 0.0001); (2) an associated risk factor was continuing apnea at home; (3) small-for-gestational-age infants seemed to be somewhat protected from apnea compared to appropriate- and large-for-gestational-age infants; (4) anemia was a significant risk factor, particularly for patients > 43 weeks' postconceptual age; (5) a relationship to apnea with history of necrotizing enterocolitis, neonatal apnea, respiratory distress syndrome, bronchopulmonary dysplasia, or operative use of opioids and/or muscle relaxants could not be demonstrated. CONCLUSIONS: The analysis suggests that, if it is assumed that the statistical models used are equally valid over the full range of ages considered and that the average rate of apnea reported across the studies analyzed is accurate and representative of actual rates in all institutions, the probability of apnea in nonanemic infants free of recovery-room apnea is not less than 5%, with 95% statistical confidence until postconceptional age was 48 weeks with gestational age 35 weeks. This risk is not less than 1%, with 95% statistical confidence, for that same subset of infants, until postconceptional age was 56 weeks with gestational age 32 weeks or postconceptual age was 54 weeks and gestational age 35 weeks. Older infants with apnea in the recovery room or anemia also should be admitted and monitored. The data do not allow prediction with confidence up to what age this precaution should continue to be taken for infants with anemia. The data were insufficient to allow recommendations regarding how long infants should be observed.

**OBJECTIVE:** To analyze temporal trends in acute respiratory distress syndrome (ARDS) fatality rates since 1983 at one institution. DESIGN: Cohort. SETTING: Intensive care units of a large county hospital. PATIENTS: Consecutive adult patients (>18 years of age) meeting ARDS criteria were identified through daily surveillance of intensive care units (N = 918 from 1983 through 1993). The major causes were sepsis syndrome in 37% and major trauma in 25%; 37% had other risks. Fifty-five percent were male. The median age was 45 years (range, 18 to 92 years); 70% were younger than 60 years. MAIN OUTCOME MEASURE: Mortality. RESULTS: Overall mortality rates showed no trend from 1983 to 1987, declined slightly in 1988 and 1989, and decreased to a low of 36% in 1993 (95% confidence interval, 25% to 46%). The crude rates were largely unchanged after adjustment for age, ARDS risk, and gender distribution. While patients both younger than 60 years and 60 years or older experienced declines in fatality rate, the larger decrease occurred in the younger cohort. In sepsis patients, ARDS fatality rates declined steadily, from 67% in 1990 to 40% in 1993 (95% confidence interval, 23% to 57%). The decline in sepsis-related ARDS fatality was confined largely to patients less than 60 years of age. Trauma patients and all other patients also experienced declines in fatality rates after 1987, although these trends were not as strong and consistent as in the sepsis population. CONCLUSIONS: In this large series, we observed a significant decrease in fatality rates occurring largely in patients younger than 60 years and in those with sepsis syndrome as their risk for ARDS. We are unable to determine the extent to which experimental therapies or other changes in treatment have contributed to the observed decline in the ARDS fatality rate. Institution-specific rates and temporal trends in ARDS fatality rates should be considered in clinical trials designed to prevent ARDS and the high mortality associated with this syndrome.

**OBJECTIVE:** Ethical concerns exist over the performance of medical procedures, such as endotracheal intubation, on newly deceased patients without family consent. This study examined the process of obtaining consent for the purpose of performing an invasive procedure in newly deceased adults. DESIGN: A prospective case series. PARTICIPANTS: The families of patients who died during a 5-month period were requested to provide consent to perform wire-guided retrograde tracheal intubation. MAIN OUTCOME MEASURES: Differences between success and failure in obtaining consent including information on the deceased, family reasons for their decision, and the experience of those requesting consent. RESULTS: Consent was requested from 44 families and 26 (59%) agreed to the procedure. This success rate was achieved despite the lack of a prior relationship with the family by the persons requesting consent. Consent was obtained more frequently in unexpected than expected deaths (77% versus 41%, P = .03). There were no differences in success rates for consent for age, race, sex, or do-not-resuscitate status of the deceased. Spouses consented more frequently than children (77% versus 50%, P = .25). The two physicians reported greater comfort in requesting consent than the nurse anesthetist investigator. In one instance, the consent process may have increased the emotional distress of the family. CONCLUSION: Consent can frequently be obtained from families for an invasive procedure in newly deceased adults. Physicians should reconsider the practice of performing postmortem procedures without obtaining family consent.

**Cervical Spine Movement during Laryngoscopy with the Bullard, Macintosh, and Miller Laryngoscopes—RH Hastings, AC Vigil, R Hanna. BY Yang, DJ Sartoris. Anesthesiology 1995;82(4):859-869.**

**BACKGROUND:** Direct laryngoscopy requires movement of the head, neck, and cervical spine. Spine movement may be limited for anatomic reasons or because of cervical spine injury. The Bullard laryngoscope, a rigid fiberoptic laryngoscope, may cause less neck flexion and head extension than conventional laryngoscopes. The purpose of this study was to compare head extension (measured externally), cervical spine extension (measured radiographically), and laryngeal view obtained with the Bullard, Macintosh, and Miller laryngoscopes. METHODS: Anesthesia was induced in 35 ASA I-3 elective surgery patients. Patients lay on a rigid board with head in neutral position. Laryngoscopy was performed three times, changing between the Bullard, Macintosh, and Miller laryngoscopes. Head extension was measured with an angle finder attached to goggles worn by the patient. The best laryngeal view with each laryngoscope was assessed by the laryngoscopist. In eight patients, lateral cervical spine radiographs were taken before and during laryngoscopy with the Bullard and Macintosh blades. RESULTS: Median values for external head extension were 11°, 10°, and 2° with the Macintosh, Miller, and Bullard laryngoscopy (P < 0.01), respectively. Significant reductions in radiographic cervical spine extension were found for the Bullard compared to the Macintosh blade at the atlantooccipital joint, atlantoaxial joint, and C3-C4. Median atlanto-occipital extension angles were 6° and 12° for the Bullard and Macintosh laryngoscopes, respectively. The larynx could be exposed in all patients with the Bullard but only in 90% with conventional laryngoscopes (P < 0.01). CONCLUSIONS: The Bullard laryngoscope caused less head extension and cervical spine extension than conventional laryngoscopes and resulted in a better view. It may be useful in care of patients in whom cervical spine movement is limited or undesirable.
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OBJECTIVE: To determine whether situations involving multiple options can paradoxically influence people to choose an option that would have been declined if fewer options were available. DESIGN: Mailed survey containing medical scenarios formulated in one of two versions. PARTICIPANTS: Two groups of physicians: members of the Ontario College of Family Physicians (response rate = 77%; n = 287) and neurologists and neurosurgeons affiliated with the North American Symptomatic Carotid Endarterectomy Trial (response rate = 84%; n = 352). One group of legislators belonging to the Ontario Provincial Parliament (response rate = 32%; n = 41). INTERVENTION: The basic version of each scenario presented a choice between two options. The expanded version presented three options: the original two plus a third. The two versions otherwise contained identical information and were randomly assigned. OUTCOME MEASURES: Participants' treatment recommendations. RESULTS: In one scenario involving a patient with osteoarthritids, family physicians were less likely to prescribe a medication when deciding between two medications than when deciding about only one medication (53% versus 72%; P < .005). Apparently, the difficulty in deciding between the two medications led some physicians to recommend not starting either. Similar discrepancies were found in decisions made by neurologists and neurosurgeons concerning carotid artery surgery and by legislators concerning hospital closures. CONCLUSIONS: The introduction of additional options can increase decision difficulty and, hence, the tendency to choose a distinctive option or maintain the status quo. Awareness of this cognitive bias may lead to improved decision making in complex medical situations.


PURPOSE: The purpose of this investigation is to evaluate the utility of the alveolar-arterial (A-a) oxygen gradient in the diagnosis of acute pulmonary emolosion (APE) among patients who participated in the Prospective Investigation of Pulmonary Emolosion Diagnosis (PIO-PED). METHODS: Pulmonary emolosion was diagnosed (n = 280) or excluded (n = 499) by angiography in all patients. Patients were then categorized as (1) the entire cohort, (2) no prior cardiopulmonary disease and no prior PE, and (3) no prior PE or deep venous thrombosis. Normal values of the A-a gradient were defined in three ways: (1) values ≤ 20 mm Hg; (2) values ≤ age/44; and (3) values based on age from the literature. RESULTS: When a normal A-a gradient was defined as ≤ 20 mm Hg, 11 to 14% of patients with PE in the three categories of patients had a normal A-a gradient. When the equation age/44 was used, 8 to 10% of patients with PE in the three categories of patients had a normal A-a gradient. With age-related values from the literature, 20 to 23% of patients with PE in the three categories of patients had a normal A-a gradient. The A-a gradient was normal in comparable percentages of patients who did not have PE. CONCLUSION: Normal values of the A-a gradient did not exclude the diagnosis of acute PE.


BACKGROUND: Atelectasis, an important cause of impaired gas exchange during general anesthesia, may be eliminated by a vital capacity maneuver. However, it is not clear whether such a maneuver will have a sustained effect. The aim of this study was to determine the impact of gas composition on reappearance of atelectasis and impairment of gas exchange after a vital capacity maneuver. METHODS: A consecutive sample of 12 adults with healthy lungs who were scheduled for elective surgery were studied. Thirty minutes after induction of anesthesia with fentanyl and propofol, the lungs were hyperinflated manually up to an airway pressure of 40 cm H2O. FIO2 was either kept at 0.4 (group 1, n = 6) or changed to 1.0 (group 2, n = 6) during the reanimation maneuver. Atelectasis was assessed by computed tomography. The amount of dense areas was measured at end-expiration in a transverse plane at the base of the lungs. The ventilation-perfusion distributions (VA/Q) were estimated with the multiple inert gas elimination technique. The static compliance of the total respiratory system (Crs) was measured with the flow interruption technique. RESULTS: In group 1 (FIO2 = 0.4), the reanimation maneuver virtually eliminated atelectasis for at least 40 min, reduced shunt (VA/Q < 0.005), and increased at the same time the relative perfusion to poorly ventilated lung units (0.005 < VA/Q < 0.1; mean values are given). The arterial oxygen tension (PAO2) increased from 137 mm Hg (18.3 kPa) to 163 mm Hg (21.7 kPa) before and 40 min after recruitment, respectively; P = 0.028. In contrast to these findings, atelectasis recurred within 5 min after reanimation in group 2 (FIO2 = 1.0). Comparing the values before and 40 min after recruitment, all parameters of VA/Q were unchanged. In both groups, Crs increased from 57.1/55.0 mL · cm H2O−1 · m2 (group 1/group 2) before to 70.1/67.4 mL · cm H2O−1 after the reanimation maneuver. Crs showed a slow decrease thereafter (40 min after recruitment: 61.4/66.0 mL · cm H2O−1), with no difference between the two groups. CONCLUSIONS: The composition of inspiratory gas plays an important role in the recurrence of collapse of previously reexpanded atelectatic lung tissue during general anesthesia in patients with healthy lungs. The reason for the instability of these lung units remains to be established. The change in the amount of atelectasis and shunt appears to be independent of the change in the compliance of the respiratory system.


BACKGROUND: Pneumocystis carinii pneumonia (PCP) remains a common and often fatal opportunistic infection among children infected with the human immunodeficiency virus (HIV). HIV-infected infants between three and six months of age are particularly vulnerable. Current guidelines recommend prophylaxis in children from birth to 11 months old who have CD4+ counts below 1500 cells per cubic millimeter. METHODS: We used national surveillance data to estimate the annual incidence of PCP among children less than one year old. We reviewed the medical records of 300 children given a diagnosis of PCP between January 1991 and June 1993 to determine why treatment according to the 1991 guidelines for prophylaxis against PCP either was not given or failed to prevent the disease. RESULTS: In our study the incidence of PCP in the first year of life among infants born to HIV-infected mothers changed little between 1989 and 1992. Among 7080 children born to HIV-infected mothers in 1992, PCP developed in 2.4 percent. Of 300 children with PCP diagnosed from January 1991 through June 1993, 199 (66 percent) had never received prophylaxis, and for 118 of those children (59 percent) exposure to HIV was first identified 30 days or less before the diagnosis of PCP. Among 129 children less than one year old, the CD4+ count declined by an estimated 967 cells per cubic millimeter (95 percent confidence interval, 724 to 1210 cells per cubic millimeter) during the three months before the diagnosis of PCP. Among infants in whom CD4+ counts were determined within one month of the diagnosis of PCP, 18 percent (20 of 113) had at least 1500 cells per cubic millimeter, a level higher than the currently recommended threshold for prophylaxis.

CONCLUSIONS: In the United States the incidence of PCP among HIV-infected infants has not declined. If this infection is to be prevented, infants exposed to HIV must be identified earlier, and prophylaxis must be offered to more children than the guidelines currently recommend.


BACKGROUND: Mechanical ventilator circuits are commonly changed at 48-h intervals. This fre-
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March 28 (Videotape Available)—Nancy Telford, RRT with Moderator Sam P. Giordano, MBA, RRT

Part II • Prevention and Management of Ventilator-Induced Lung Injury
April 25 (Videotape Available)—David J. Pierson, MD with Moderator Richard D. Branson, RRT

Part III • The Multidisciplinary Team Approach and Respiratory Care
June 20, 12:30 p.m. to 2 p.m. Eastern Time—Kevin L. Shrace, MA, RRT, CHE with Sam P. Giordano, MBA, RRT

Part IV • Emergency Respiratory Care: The Respiratory Care Practitioner’s Role
July 19, 12:30 p.m. to 2 p.m. Eastern Time—Charles G. Durbin, Jr., MD, FCCM with Richard D. Branson, RRT

Part V • Noninvasive Mechanical Ventilation: Its Role in Acute and Chronic Ventilatory Failure
August 2, 12:30 p.m. to 2 p.m. Eastern Time—Nicholas S. Hill, MD with Richard D. Branson, RRT

Part VI • Organizing a Respiratory Care Department without Walls
September 19, 12:30 p.m. to 2 p.m. Eastern Time—John R. Walton, MBA, MHA, RRT, CHE with Sam P. Giordano, MBA, RRT

Part VII • Shortening Length of Stay on Ventilators and in Hospitals
November 14, 12:30 p.m. to 2 p.m. Eastern Time—Neil R. MacIntyre, MD with Richard D. Branson, RRT

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frequency may be unnecessary because ventilator-associated pneumonia often results from aspiration of pharyngeal secretions and not from the ventilator circuit. We compared the ventilator-associated pneumonia rates and costs associated with 48-h and 7-day circuit changes. METHODS: Ventilator circuits were changed at 48-h intervals during the control period (November 1992 to April 1993) and at 7-day intervals during the study period (June 1993 to November 1993). Nosocomial pneumonias were prospectively identified using the criteria of the Centers for Disease Control and Prevention. The annual cost difference of changing circuits at 48-h and 7-day intervals was calculated using the distribution of ventilator days for the control and study periods. RESULTS: There were 1,708 patients, 9,858 ventilator days, and a pneumonia rate of 9.64 per 1,000 ventilator days in the control group (48-h circuit changes). There were 1,715 patients, 9,160 ventilator days, and 6.22 pneumonias per 1,000 ventilator days when circuits were changed at 1-week intervals (study group). Using a logistic regression model, there were significantly greater odds of developing a ventilator-associated pneumonia in surgical patients (odds ratio 1.77, P = 0.02) and patients in critical care units (odds ratio 1.54, P = 0.05), but no significant risk of ventilator-associated pneumonia in patients in whom circuits were changed at 1-week intervals (odds ratio 0.82, P = 0.22). Changing circuits at 7-day intervals resulted in a 76.6% ($111,530) reduction in the annual cost for materials and salaries. CONCLUSIONS: We found no difference in pneumonia rates with ventilator circuit changes at 48-h and 7-day intervals. Ventilator circuits can be safely changed at weekly intervals, resulting in large cost savings.


A meta-analysis of clinical trials of allergen immunotherapy was undertaken to assess the efficacy of this controversial form of therapy in asthma. A computerized bibliographic search revealed 20 randomized placebo-controlled double-blind trials of allergen immunotherapy for asthma. The results extracted included asthmatic symptoms, medication requirements, lung function, and bronchial hyperreactivity (BHR). Categorical outcomes were expressed as odds ratios and continuous outcomes as effect sizes. The combined odds of symptomatic improvement from immunotherapy with any allergen were 3.2 (95% CI 2.2 to 4.9). The odds for reduction in medication after mite immunotherapy were 4.2 (95% CI 2.2 to 7.9). The combined odds for reduction in BHR were 6.8 (95% CI 3.8 to 12.0). The mean effect size for any allergen immunotherapy on all continuous outcomes was 0.71 (95% CI 0.43 to 1.00), which would correspond to a mean 7.1% predicted improvement in FEV₁ from immunotherapy. Although the benefits of allergen immunotherapy could be Overestimated because of unpublished negative studies, an additional 33 such studies would be necessary to overturn these results. Allergen immunotherapy is a treatment option in highly selected patients with extrinsic ("allergic") asthma.


Recent prevalence data for childhood asthma in Switzerland suggest a substantial underdiagnosis that seems to be more pronounced in girls. We further analyzed our data trying to specify risk factors for underdiagnosis and undertreatment. Our special interest was focused on female sex as there is evidence for a sex-dependent diagnosis and treatment of chronic disease in adults, called the Yentl syndrome. The data are derived from a parent completed questionnaire survey of a stratified cluster sample of school children aged 7, 12, and 15 years. Besides the 12 months prevalence of asthma symptoms and bronchodilator treatment, the lifetime prevalence of an asthma diagnosis was noted. With a response rate of 97%, a total of 4353 completed questionnaires were analyzed. While age was not associated with undertreatment (except for exercise-induced symptoms in adolescents), the lack of a formal diagnosis of asthma and atypical asthma symptoms other than wheeze such as chronic...
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ic night cough were confirmed as significant risk factors for undertreatment. Of all boys reporting asthma symptoms 31% received bronchodilator treatment compared with only 15% of the symptom-reporting girls (P < 0.001). For all particular asthma-related symptoms (except wheeze), significantly more boys than girls (approximately double) received treatment. The physiological and psychological bases for these findings are discussed and suggest that gender is an important risk factor for underdiagnosis and undertreatment of asthma. Our research indicates that the Yentl syndrome may exist for childhood asthma.


BACKGROUND: Although epidemiologic studies have looked associated tobacco and alcohol use with the development of squamous-cell carcinoma of the head and neck, the molecular targets of these carcinogens have yet to be identified. We performed a molecular analysis to determine the pattern of mutations in the p53 gene in neoplasms from patients with squamous-cell carcinoma of the head and neck and a history of tobacco or alcohol use.

METHODS: Sequence analysis of the conserved regions of the p53 gene was performed in tumor samples from 129 patients with primary squamous-cell carcinoma of the head and neck. We then used statistical analysis to identify any patient characteristics associated with mutation of the p53 gene.

RESULTS: We found p53 mutations in 42% of the patients (54 of 129). Fifty-eight percent of the patients who smoked cigarettes and used alcohol (37 of 64; 95% confidence interval, 45 to 70%), 33% of the patients who smoked but abstained from alcohol (13 of 39, 95% confidence interval, 19 to 50%), and 17% of the patients who neither smoked nor drank alcohol (4 of 24, 95% confidence interval, 5 to 37%) had p53 mutations (P = 0.001). (Two patients used alcohol but did not smoke, and neither had a p53 mutation.) Furthermore, 100% of the mutations in the patients who neither drank nor smoked occurred at sites containing cytosine:guanine di- or tri-nucleotides (potentially representing endogenous mutations) within the p53 gene (5 of 5 mutations; 95% confidence interval, 48 to 100%), whereas only 23% of those in cigarette smokers consisted of such changes (12 of 53 mutations; 95% confidence interval, 12 to 36%; P = 0.001).

CONCLUSIONS: In our study, a history of tobacco and alcohol use was associated with a high frequency of p53 mutations in patients with squamous-cell carcinoma of the head and neck. Preliminary evidence linked cigarette smoking to p53 mutations at nonendogenous mutation sites. Our findings suggest a role for tobacco in the molecular progression of squamous-cell carcinoma of the head and neck and support the epidemiologic evidence that abstinence from smoking is important to prevent head and neck cancer.


OBJECTIVE: A meta-analysis of randomized trials was performed to estimate the effectiveness of antibiotics in treating exacerbations of chronic obstructive pulmonary disease (COPD). DATA SOURCES: English-language studies published from 1955 through 1994 were retrieved using MEDLINE, Index Medicus, bibliographies, and consultation with experts. MEDLINE search terms included “COPD,” “chronic bronchitis,” and “antibiotic(s).” STUDY SELECTION: Only randomized trials that enrolled patients having an exacerbation of COPD, used an antibiotic in the treatment group and placebo in the control group, and provided sufficient data to calculate an effect size were included in the meta-analysis. DATA EXTRACTION: Descriptive and outcome data from each study were independently abstracted by two authors. DATA SYNTHESIS: Overall summary effect size of the nine trials satisfying all inclusion criteria was 0.22 (95% confidence interval [CI], 0.10 to 0.34), indicating a small benefit in the antibiotic-treated group. Similar analysis of the six studies that provided data on peak expiratory flow changes revealed a summary effect size of 0.19 (95% CI, 0.03 to 0.35) and a summary change in peak expiratory flow rate of 10.75 L/min (95% CI, 4.96 to 16.54 L/min) in favor of the antibiotic-treated group. Sensitivity analyses did not significantly affect these results. CONCLUSIONS: These analyses suggest a small but statistically significant improvement due to antibiotic therapy in patients with exacerbations of COPD. This antibiotic-associated improvement may be clinically significant, especially in patients with low baseline flow rates.


The objective of this study was to determine the costs and effects of combined bronchodilator and anti-inflammatory therapy. In a 2.5-yr randomized controlled study, combined b2-agonist/ corticosteroid therapy (BA + CS) and combined b2-agonist/anticholinergic therapy (BA + AC) were compared with b2-agonist placebo therapy (BA + PL). Included in the study were 274 patients 18 to 60 yr of age with moderately severe obstructive airway disease. The main clinical endpoints were lung function, hyperresponsiveness, restricted activity days, and symptom-free days. The economic endpoints were the costs of health care utilization. Compared with BA + PL, BA + CS led to significant improvements in FEV1, PC20, and symptom-free days. BA + AC did not differ from BA + PL in this respect. The respective annual acquisition costs of BA + CS, BA + AC, and BA + PL were 532 US$, 277 US$, and 156 US$. Thus, BA + CS costs 376 US$ more than BA + PL. However, compared with BA + PL therapy, BA + CS led to statistically significant savings in other health care costs of about 175 US$ (95% CI from 46 to 303 US$). Thus, more than half of the additional costs of adding the inhaled corticosteroid are compensated for by a reduction in the costs of other health care services. Overall, inhaled corticosteroids lead to a small but net increase in health care costs of 201 US$ per patient per year. The incremental cost-effectiveness ratio of BA + CS compared with BA + PL ranges from 200 US$ per 10% increase in FEV1 to 5 US$ per symptom-free day gained. In order to reach net societal savings, the economic benefits of increased productivity due to inhaled corticosteroids have to be valued higher than 42 US$ per day. No significant differences in health care costs were found between the BA + AC and BA + PL groups. It can be concluded that the addition of an inhaled corticosteroid to a b-agonist leads to significant benefits in respiratory function and restricted activity days, which seem to be worth the relatively low additional health care costs, whereas addition of an anticholinergic agent appears expensive and of no long-term value.


BACKGROUND: Since the inflammatory response to chronic infection contributes to lung destruction in patients with cystic fibrosis we hypothesized that anti-inflammatory therapy might slow the progression of lung disease. METHODS: In a double-blind trial, 85 patients, 5 to 39 years of age, with mild lung disease (forced expiratory volume in one second [FEV1]; 360% of the predicted value) were randomly assigned to receive ibuprofen or placebo orally twice daily for 4 years. Doses were adjusted individually, to achieve peak plasma concentrations of 50 to 100 mg per milliliter. Changes in pulmonary function, the percentage of ideal body weight, the chest-radiograph score, and the frequency of hospitalization were assessed. RESULTS: Patients randomly assigned to ibuprofen had a slower annual rate of change in FEV1 than the patients assigned to placebo (mean ±SE slope, −2.14 ±0.57% vs. −3.60 ±0.55% in the placebo group; P = 0.02), and weight (as a percentage of ideal body weight) was better maintained in the former group (P = 0.02). Among the patients who took ibuprofen for four years and had at least a 70% rate of compliance, the annual rate of change in FEV1 was even slower (−1.48 ±0.69% vs. −3.57 ±0.65% in the placebo group; P = 0.03), and this group of patients also had a significantly slower rate of decline in forced vital capacity, the percentage of ideal body weight, and the chest-radiograph score. There was no significant difference between the ibuprofen and placebo groups in the frequency of hospitalization. One patient was withdrawn from the study.
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because of conjunctivitis, and one because of epis-taxis related to ibuprofen. CONCLUSIONS: In patients with cystic fibrosis and mild lung disease, high-dose ibuprofen, taken consistently for four years, significantly slows the progression of the lung disease without serious adverse effects.


Evidence that dying occurs as a natural, final event in the wholeness of human life is culturally, artistically and scientifically persuasive. Very elderly patients eventually undergo a process of functional declines, progressive apathy, and loss of willingness to eat and drink that culminates in death, even in the absence of acute illness or severe chronic disease. Despite clinical resemblances to depression and dementia, aging itself and a loss of will to live are the most probable explanations for natural dying. Acceptance of the naturalness of dying, however, directly conflicts with the medicalization and legalization of death that characterizes modern society’s treatment of dying elderly patients. We prefer instead to believe that dying results from disease and injury, which may yield to advances in medical technology. The progressive move of the dying out of the home and into acute and long-term care facilities suggests that medicalization may be an irreversible process. Viewing dying as an independent diagnosis in patients who are obviously undergoing terminal declines from aging and chronic diseases can facilitate communication about spiritual and palliative care needs, which tend to be neglected in the medically dying of patients. Physicians and nurses may need to assume the role of medical stewardship to help prevent the overtreatment and overtesting of modern medicine’s approach to the dying. The emotional burdens of caring for the dying elderly, however, must be addressed openly through collaborative work, institutional policies on limitation of treatment, and support building among physicians and other caregivers.


BACKGROUND: We describe a child who was identified shortly after birth as infected with the human immunodeficiency virus type 1 (HIV-1), but whose infection appears to have completely cleared. Asymptomatic HIV-1 infection was diagnosed in the mother during the fourth month of pregnancy. The infant was delivered vaginally at 36 weeks, received no blood products, and was not breast-fed. METHODS AND RESULTS: HIV-1 was detected by culture of the infant’s peripheral-blood mononuclear cells at 19 and 51 days of age. Plasma from the infant was also culture-positive for HIV-1 at 51 days of age by DNA polymerase chain reaction (PCR). Nucleotide-sequence analysis of HIV-1 DNA showed extremely close homology of the cultures obtained 32 days apart, and forensic markers of genetic identity for the two cultures were identical. Hence, inadvertent viral contamination or error in the collection of specimens was highly unlikely. At 12 months of age the infant was seronegative for HIV-1, and numerous subsequent cultures and tests by PCR have also been negative for HIV-1. The child is five years of age at this writing, is HIV-seronegative, and remains well, with normal growth and development and no laboratory or clinical evidence of HIV-1 infection. CONCLUSIONS: The infant we describe was infected perinatally with HIV-1, but the infection subsequently cleared and the infant remained without detectable HIV-1 infection five years later.


The contribution of ventilator circuit bacterial contamination to the occurrence of ventilator-associated pneumonia remains controversial. In a previous study, we found that the incidence of pneumonia was identical with ventilator circuit changes every 48 h and with no ventilator circuit changes. The present study prospectively assessed whether keeping ventilator circuits clean with a heat and moisture exchanger exhibiting antimicrobial barrier properties affects patient colonization and the incidence of nosocomial pneumonia in patients receiving mechanical ventilation for more than 48 h. Consecutive patients were randomly allocated to humidification with either a heat and moisture exchanger (Group 1, n = 61) or a heated humidifier (Group 2, n = 70). In both groups, no circuit changes were performed throughout ventilatory support. Duration of mechanical ventilation was identical in both groups (10 ± 8.6 d; range: 2 to 47) in Group 1 and 12.5 ± 14.2 d (range: 2 to 85) in Group 2. The incidence of pneumonia (positive quantitative culture of protected brush specimen) was similar in both groups (6/61 and 8/70 in Groups 1 and 2, respectively; p = 0.8), as was duration of ventilation prior to pneumonia (9 ± 5.9 versus 8.2 ± 5.7 d; p = 0.8). Ventilator tubing contamination was considerably reduced with the use of a heat and moisture exchanger. In contrast, bacterial colonization of the pharynx and trachea was identical in both groups. These results suggest that circuit colonization plays little or no role in the occurrence of ventilator-associated pneumonia, provided usual maintenance precautions are applied. It also indirectly confirms our previous finding that during mechanical ventilation with a heated humidifier, circuits need not be changed except between patients. Although it failed to reduce the incidence of pneumonia, the use of a heat and moisture exchanger may allow reduction of the number of septic procedures linked to circuit management during mechanical ventilation as well as substantial savings in cost provider time.

Early Pulmonary Inflammation in Infants with Cystic Fibrosis—T Z Khan, JS Wagen, T Bost, J Martinez, FJ Accurso. DWH Riches. Am J Respir Crit Care Med 1995;151:1075.

The mechanisms underlying the initiation of lung disease and early respiratory morbidity in cystic fibrosis (CF) are poorly understood. By identifying infants with CF through a statewide neonatal screening program, we investigated whether airway inflammation was present in these infants, with the goal of furthering our understanding of the early events in this lung disease. Bronchoalveolar lavage fluid (BALF) from 16 infants with CF (mean age, 6 mo) and 11 disease control infants (mean age, 12 mo) was examined for the following inflammatory parameters: (1) neutrophil count; (2) activity of free neutrophil elastase; (3) elastase/alpha antiprotease inhibitor complexes; and (4) the level of interleukin-8 (IL-8). We also quantified the spontaneous level of expression of IL-8 mRNA transcripts by airway macrophages. Each index of airway inflammation was increased in the BALF of infants with CF as compared with control infants. In addition, both the number of neutrophils and IL-8 levels were increased in infants with CF who had negative cultures (n = 7) for common bacterial CF-related pathogens, as well as for common respiratory viruses and fungi at the time of bronchoalveolar lavage (BAL). These findings suggest that airway inflammation is already present in infants with CF who are as young as 4 wk. Furthermore, although many different cells types (eg, epithelial cells) may express IL-8, airway macrophages appear to be a source of this chemokine, and may thus play a prominent role in early neutrophil influx into the lung.
Continuing Education: Motivated or Mandated?

Why do respiratory care practitioners participate in continuing education activities? This is a question that I have frequently asked myself. What is it that prompts them to spend a Saturday traveling to another part of the state to listen to lectures or symposia or to participate in a workshop? Are these practitioners imbued with the spirit of lifelong learning or are they meeting the letter of a licensure rule, requiring a certain number of credit hours per year?

Motivation is hard to define. It can’t be observed. It can’t be measured. But, behavior of practitioners can be observed. Inferences can be drawn when practitioners assess their own educational needs and then participate in educational activities designed to meet those needs. It is reasonable to say they are motivated.

On the other hand, many practitioners perceive continuing education as a chore—a hoop that one must jump through to obtain license renewal. Not a day goes by that a member of this latter group doesn’t call the Association’s executive office, frantic, because the state has sent out license renewals. This practitioner hasn’t even thought about continuing education during the biennium fast drawing to a close. “Help, I need 12 hours of CRCE credit.” The subject is immaterial. Timing is critical.

The leadership of the AARC, during a quality improvement session, described its vision of the roles and responsibilities of the respiratory care practitioner in the Year 2000. Among the 14 attributes identified for the practitioner of the future was “Remain professionally dynamic as manifested by a commitment to being a lifelong student.”

During the first Consensus Conference on Respiratory Care Education, each small working group reported that in the affective domain the therapist of the future should be committed to lifelong learning or professional improvement or a desire to learn.

The future is now. It is essential that practitioners today be motivated to expand their knowledge base. Whether the motivation is generated internally or externally is immaterial.

Content of the educational experience is paramount. Today more than ever before respiratory care practitioners should be searching for ways to expand their horizons. I know that weaning from mechanical ventilation is a solid-gold topic that never goes out of vogue. Is that the subject you need? Should you be looking beyond statutory requirements and consider skill building? How about performing ECGs? Completing an ACLS course? Participating in discharge planning? Learning I.V. line insertion?

You may be assured that unless you commit to lifelong learning your days as an RCP are numbered. You will not fit into the re-engineered hospital. You won’t be able to accept managed care. You won’t be able to function in other healthcare settings.

Further, if you are tasked with the planning of continuing education for your fellow workers, your responsibilities are important. Not only must you create a program that is congruent with the values of your staff, but you must also motivate the adult learners surrounding you. You must be aware of their educational and training needs—the needs generated by the changing clinical scene and financial climate. What is appealing and easy probably just won’t do. Last year’s program may not meet this year’s need!

Robert J Czachowski PhD
Director of Education
American Association for Respiratory Care
Dallas, Texas

REFERENCES

Bench Evaluation: Three Face-Shield CPR Barrier Devices

Mark Simmons MSEd RPFT RRT, Dan Deao BS RRT, Laura Moon BS RRT, Kristi Peters MS, and Sally Cavanaugh PhD

INTRODUCTION: Due to the fear of disease transmission, the practice of mouth-to-mouth (M-M) rescue breathing is rarely performed; to address this concern, many types of CPR barrier devices have been developed. These include bag-valve-mask devices, mouth-to-mask devices, and face shields (FS). The purpose of this study was to measure the volumes delivered during mouth-to-face shield (M-FS) breathing, to measure the back pressure and calculate the resistance to flow through their 1-way valves, and to test for backward leak of gas through the valves. METHODS: Three FS brands were evaluated: Kiss of Life (KOL), MicroSHIELD (Micro) and Res-Cue Key (RCK). Volume delivered during M-M and M-FS breathing was evaluated by 10 rescuers who used the devices while performing rescue breathing on a CPR mannequin. Back pressure was measured and resistance calculated by directing airflow through the 1-way valves. Backward leak was evaluated by measuring the O2 concentration at the rescuer side of the valve while 100% O2 was directed toward the patient side of the valve. Differences among the brands were evaluated using analysis of variance. RESULTS: The mean (SD) values for volumes in L were: M-M 1.00 (0.25), Micro 0.77 (0.20), RCK 0.64 (0.10), and KOL 0.24 (0.11). Mean values for back pressure in cm H2O at 50 L/min were Micro 16.7 (1.29), KOL 7.22 (0.13), and RCK 2.15 (0.16). Significant backward leak only occurred with RCK. CONCLUSION: Not one of the FSs tested met all of the requirements suggested by the American Heart Association and by the International Standards Organization. [Respir Care 1995:40(6):618-623]

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Mr Simmons is Program Director, School of Respiratory Therapy, York College and York Hospital, York, Pennsylvania. Mr Deao was a student at York College and York Hospital School of Respiratory Therapy when this study was conducted. He is now a Respiratory Care Practitioner at Prince George’s Hospital, Cheltenham, Maryland. Ms Moon was also a student at York College and York Hospital School of Respiratory Therapy when this study was conducted. She is now a Respiratory Care Practitioner at Johns Hopkins Hospital, Baltimore, Maryland. Ms Peters is a research assistant at York Hospital. Dr Cavanaugh is the director of research at York Hospital.

The authors have no financial interest in the products mentioned or in competing products.

This study was conducted at York Hospital, York, Pennsylvania. Findings were presented by Mr Simmons during the Respiratory Care Open Forum at the AARC Annual Meeting held in Las Vegas, Nevada, December 1994.

Reprints: Mark Simmons, School of Respiratory Therapy, York Hospital, 1001 South George Street, York PA 17405.

And the Lord God formed man of the dust of the ground, and breathed into his nostrils the breath of life; and man became a living soul.¹

Introduction

Restoration to life of those who are clinically dead has been an area of interest for many years. Of course, rescue breathing plays an important role in the process of trying to prevent biological death in the nonbreathing victim. The American Heart Association² (AHA) lists early CPR, of which rescue breathing is a vital part, as a link in the “chain of survival” along with early access to the emergency medical system, defibrillation, and advanced care. The story of the prophet Elisha³ is often referred to as the first documented case of resuscitation using mouth-to-mouth (M-M) rescue breathing. One of the first documented successes of the exhaled air method
of ventilation in more modern times was reported in 1771 by Tossach. In recent years authors have reminded us of the late 1950s and early 1960s work by Elam, Safar, and others who convincingly showed that M-M rescue breathing was effective, and could be taught to others for general use.

With increased concern about disease transmission during CPR, it is desirable to prevent rescuer-victim contact from respiratory secretions and other body fluids. The Centers for Disease Control (CDC) states in its guidelines that barrier devices should be used when performing rescue breathing, and this is routinely done by health-care personnel in hospital settings. Yet, today in many CPR courses for lay persons, the M-M technique without a barrier device for actual victims is still being taught. The AHA states that the perceived risk of disease transmission during CPR has reduced the willingness of lay persons to initiate M-M ventilation in victims with cardiac arrest. Although most CPR performed in the United States is done by health-care and public-safety personnel, the unwillingness of medically trained persons to initiate M-M ventilation due to the fear of disease transmission has also been demonstrated. To decrease the need for M-M breathing, many barrier devices have been developed. These include bag and mask devices, mouth-to-mask devices, and face shields (FS). (FSs are also referred to as face-shields, foils, and barrier masks). Although resuscitator bags and mouth-to-mask devices have been studied extensively, FSs have not. Consequently, FSs are not recommended as a primary means for delivery of ventilation by medical personnel in the hospital setting and should be considered AHA Class IIIb devices (acceptable, possibly helpful), reserved for use in basic life support (BLS) only.

Several characteristics of FSs should be considered. Some of these are cost, size, ease of use, portability, secretion-barrier function, durability, valve resistance, tidal volumes (VT), backward flow, impedance of exhalation, ability to allow administration of supplemental O2, application to patients of various ages, displacement and increased chance of vomitus aspiration, and use at extreme temperatures. However, the purpose of this study was threefold: to measure the volumes delivered during mouth-to-FS breathing (M-FS), to measure the back pressure and calculate the resistance to flow through the 1-way valves, and to test the integrity of 1-way valves in preventing backward leak of gas through the valve toward the rescuer.

Methods

To evaluate volumes delivered during rescue breathing, we performed rescue breathing on a mannequin. The mannequin's dynamic lung compliance, measured at 1 L with a slow inspiration, was 0.038 L/cm H2O. A ventilation monitor was placed in-line between the mannequin's 'trachea' and its 'lungs.' The monitor was tested for in-line accuracy by injecting 1 L of air in 1.5 to 2 seconds using a 1-L calibration syringe. Ten rescuers (3 men and 7 women), each certified in BLS, performed M-M and M-FS rescue breathing on the mannequin. Three different FS brands (10 devices for each brand) were evaluated: Kiss of Life (KOL) CPR Barrier Mask, CPR MicroSHIELD (Micro), and Res-Cue Key (RCK).

Description of Devices

The KOL comes in a flexible package with latex gloves and can be attached to a belt. The package is approximately 10 x 9 x 4 cm. This silicone FS is boat shaped. Once removed and unfolded from the package, it covers a large area of the victim's face (19 x 13 cm). Two straps placed around the victim's ears hold the FS in position (Fig. 1). The KOL also has ports to accept a nasal cannula for O2 administration.

The Micro is packaged in a variety of holders. One holder is a flexible plastic container 14 x 6 x 2 cm; and another, a belt holster that includes latex gloves. The Micro is also packaged as a key-chain arrangement. This clear plastic FS opens to cover a facial area of approximately 12 x 12 cm (Fig. 2).

Both the KOL and the Micro have a 1-way valve housed inside a hard plastic 'bite block' which is inserted into the victim's mouth between the teeth. The size of the bite blocks would likely prohibit their use in small children and infants.

The RCK is packaged in a rigid plastic container 3.5 x 3.5 x 1.5 cm and attached to a key ring. This FS is a thin flexible plastic covering that is also boat shaped and unfolds to cover a facial area 22 cm x 13 cm. It is held in place by two thin elastic straps that attach around the victim's ears (Fig. 3). The 1-way valve, encased in a small hard plastic housing, remains external to the victim's lips. According to the manufacturer, the RCK design makes mouth-to-nose ventilation possible.

* Manufacturers and suppliers are identified in the Product Source section at the end of the text.
Evaluation of Devices

Each rescuer was given a device of each FS brand. Each device was positioned on the mannequin and used in the manner suggested by the package insert from the manufacturer. Each participant was given time to become familiar with and practice with each device. All rescuers were then instructed to perform M-M and M-FS rescue breathing on the mannequin as they would on an actual patient. Each rescuer, following a randomization chart, performed M-M and M-FS breathing for 2 minutes with each device. A 5-minute rest period was given between each series to help reduce fatigue. Rescuers were not allowed to see the measured volumes until testing was completed. All VFs were measured, and mean and standard deviation values were calculated for each brand of FS and M-M ventilation.

To assess back pressure and resistance for the 1-way valves, we randomly selected two units from each brand for testing. The FSs were not placed in the mouth of the mannequin but held in a position that imitated actual use. A continuous flow of compressed air was directed through the valves at 10-60 L/min in increments of 10 L/min. Flow was verified by a calibration analyzer. Three measurements were made at each flow setting for each device. All back pressures were measured proximal to the 1-way valve using the calibration analyzer (Fig. 4). Resistance was calculated by dividing back pressure by the flowrate. The methodology was similar to that of others performing back-pressure studies to evaluate resistive pressure through valves.14-17 Mean values and standard deviations were calculated for the resistance of each of the FS brands at the various flows. We did not use flowrates higher than 60 L/min when testing for back pressure for two reasons: (1) Following the AHA1 guidelines of 0.8-1.2 L delivered in 1.5-2.0 seconds, the average flowrates would fall between 24 and 48 L/min. Because of this, we thought the range of 10 to 60 L/min was clinically appropriate. (2) We did not use flowrates above 60 L/min because once the flowrates were above 50 L/min with the KOL FS, the constant flow of gas made the valve flutter rapidly. The fluttering increased the back pressure reading to a much higher value and resulted in a loud whistle. For consistency, we stopped all measurements at 60 L/min.

To test the 1-way valve integrity for preventing backward leak, three randomly selected devices from each manufacturer were used. Each FS was placed on the mannequin in the proper position as suggested by the manufacturer. A constant flow of 100% O2 was directed through the trachea of the mannequin up to its oral cavity for 1 minute, simulating exhalation. Two different flowrates were used in the study, 30 L/min and 60 L/min. A flowrate of 30 L/min was intended to simulate normal expiratory flowrates due to normal lung recoil. A flow of 60 L/min was intended to simulate expiratory flowrates occurring when chest compressions are combined with rescue breathing, forcing air out of the lungs. A large-bore tube, 12 in. long, was connected to the rescuer’s side of the FS valves.
Face-Shield CPR Barrier Devices

The sensor of a calibrated polarographic O₂ analyzer with a digital readout was placed into the tube to within 1 inch of the 1-way valve (Fig. 5). This prevented the O₂ sensor from measuring any ‘stray’ O₂ not coming through the valve. This procedure for measuring O₂ was based on the premise that if the O₂ analyzer reading increased above 21% O₂, a valve leak or backward flow of O₂ had occurred. Three measurements were made for each device at each flow setting. The tube was thoroughly flushed with compressed air between each measurement and the O₂ analyzer recalibrated to 21%.

Differences between the devices for each test performed were evaluated using 1-way analysis of variance (ANOVA). Scheffé post-hoc analysis was conducted to determine differences between specific pairs of FSs, with p < 0.05 considered significant. All statistical analyses were performed using commercially available software and standard methodology.¹⁸

Results

Volume

The mean (SD) age of the rescuers was 24.8 (6.9) years and their mean height was 65.7 (3.8) inches.

The mean (SD) $V_T$ for each method of ventilation was: M-M = 1.00 (0.25) L, Micro = 0.77 (0.20) L, RCK = 0.64 (0.10) L, and KOL = 0.24 (0.11) L. Table 1 lists the mean values for each rescuer using each method of ventilation, and overall means for each device. Overall, there is a statistically significant difference among the mean values for the four methods of ventilation (ANOVA F test, p < 0.001). Significant differences were also found for comparisons between KOL and all other methods of ventilation as well as between M-M and RCK (Scheffé, p < 0.05).

Back Pressure and Resistance

Tables 2 and 3 list the means and standard deviation for back pressure measurements and resistance calculations for each brand of device at the different flows. There was a statistically significant difference among the mean values for each brand tested at each flow rate (ANOVA, p < 0.001). There was also a statistically significant difference for each comparison (Scheffé, p < 0.05) except between the KOL and Micro at 60 L/min.

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Table 1. Mean (SD) Tidal Volumes in Liters for Each Method of Ventilation.

<table>
<thead>
<tr>
<th>Rescuer</th>
<th>M-M*</th>
<th>Micro</th>
<th>RCK</th>
<th>KOL</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.88 (0.11)</td>
<td>0.60 (0.08)</td>
<td>0.62 (0.11)</td>
<td>0.05 (0.08)</td>
</tr>
<tr>
<td>2</td>
<td>1.33 (0.05)</td>
<td>1.12 (0.06)</td>
<td>0.78 (0.09)</td>
<td>0.20 (0.02)</td>
</tr>
<tr>
<td>3</td>
<td>0.87 (0.08)</td>
<td>0.86 (0.10)</td>
<td>0.60 (0.11)</td>
<td>0.30 (0.02)</td>
</tr>
<tr>
<td>4</td>
<td>1.41 (0.12)</td>
<td>0.74 (0.11)</td>
<td>0.71 (0.16)</td>
<td>0.16 (0.03)</td>
</tr>
<tr>
<td>5</td>
<td>1.07 (0.12)</td>
<td>0.83 (0.17)</td>
<td>0.75 (0.20)</td>
<td>0.41 (0.06)</td>
</tr>
<tr>
<td>6</td>
<td>1.04 (0.13)</td>
<td>0.53 (0.05)</td>
<td>0.61 (0.06)</td>
<td>0.19 (0.04)</td>
</tr>
<tr>
<td>7</td>
<td>1.19 (0.17)</td>
<td>1.02 (0.21)</td>
<td>0.62 (0.13)</td>
<td>0.38 (0.03)</td>
</tr>
<tr>
<td>8</td>
<td>0.81 (0.18)</td>
<td>0.57 (0.12)</td>
<td>0.55 (0.12)</td>
<td>0.19 (0.03)</td>
</tr>
<tr>
<td>9</td>
<td>0.77 (0.10)</td>
<td>0.72 (0.05)</td>
<td>0.44 (0.13)</td>
<td>0.27 (0.02)</td>
</tr>
<tr>
<td>10</td>
<td>0.64 (0.09)</td>
<td>0.65 (0.09)</td>
<td>0.71 (0.09)</td>
<td>0.27 (0.02)</td>
</tr>
<tr>
<td>Overall</td>
<td>1.00 (0.25)</td>
<td>0.77 (0.20)</td>
<td>0.64 (0.10)</td>
<td>0.24 (0.11)</td>
</tr>
</tbody>
</table>

* M-M, mouth to mouth rescue breathing; Micro, MicroSHIELD; RCK, Res-Cue Key; KOL, Kiss of Life
† analysis of variance (p < 0.001); a, b, c, matching letters denote significant comparisons as given by Scheffé’s post-hoc test (p < 0.05).

Table 2. Mean (SD) Values for Back Pressure in cm H₂O as a Function of Flow for the Face Shields Evaluated.

<table>
<thead>
<tr>
<th>Flow L/min</th>
<th>Micro*</th>
<th>KOL</th>
<th>RCK</th>
<th>p value (ANOVA)†</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>6.93 (1.16)</td>
<td>1.87 (0.36)</td>
<td>0.72 (0.10)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>20</td>
<td>10.08 (1.09)</td>
<td>3.08 (0.29)</td>
<td>1.15 (0.10)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>30</td>
<td>12.78 (1.37)</td>
<td>4.40 (0.27)</td>
<td>1.60 (0.15)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>40</td>
<td>14.73 (1.25)</td>
<td>5.80 (0.18)</td>
<td>1.90 (0.11)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>50</td>
<td>16.70 (1.29)</td>
<td>7.22 (0.13)</td>
<td>2.15 (0.16)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>60</td>
<td>18.47 (1.09)</td>
<td>15.25 (6.42)</td>
<td>2.42 (0.12)</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

* Micro, MicroSHIELD; RCK, Res-Cue Key; KOL, Kiss of Life.
† analysis of variance; a & b, matching letters denote significant comparisons as given by Scheffé’s post-hoc test (p < 0.05).

Table 3. Mean (SD) Values for Resistance in cm H₂O · s · L⁻¹ as a Function of Flow for the Face Shields Evaluated.

<table>
<thead>
<tr>
<th>Flow L/min</th>
<th>Micro*</th>
<th>KOL</th>
<th>RCK</th>
<th>p value (ANOVA)†</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>41.60 (6.93)</td>
<td>11.20 (2.17)</td>
<td>4.30 (0.59)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>20</td>
<td>30.25 (3.35)</td>
<td>9.25 (0.89)</td>
<td>3.45 (0.31)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>30</td>
<td>25.57 (2.74)</td>
<td>8.80 (0.54)</td>
<td>3.20 (0.22)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>40</td>
<td>22.10 (1.38)</td>
<td>8.70 (0.27)</td>
<td>2.85 (0.16)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>50</td>
<td>20.04 (1.54)</td>
<td>8.66 (0.16)</td>
<td>2.58 (0.20)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>60</td>
<td>18.47 (1.10)</td>
<td>15.25 (7.04)</td>
<td>2.42 (0.12)</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

* Micro, MicroSHIELD; RCK, Res-Cue Key; KOL, Kiss of Life.
† analysis of variance; a & b, matching letters denote significant comparisons as given by Scheffé’s post-hoc test (p < 0.05).
Backward Leak through the 1-Way Valve

After one minute, with 100% O₂ flowing continuously at 30 L/min, the O₂ analyzer recorded a mean (SD) of 21% (0.0) O₂ for the KOL, 21% (0.33) O₂ for the Micro, and 28% (1.2) O₂ for the RCK. After 1 minute with O₂ flow at 60 L/min, the O₂ analyzer measured a mean of 21% (0.0) O₂ for the KOL, 21% (0.0) O₂ for the Micro, and 30% (2.5) O₂ for the RCK. Overall, there was a statistically significant difference between the FS at both 30 L/min and 60 L/min (ANOVA, p < 0.001). Individual comparisons revealed that there were significant differences between the RCK and the other two FSs (Scheffé, p < 0.05), but there were no significant differences between the MICRO and the KOL.

Discussion

The three questions addressed by this study were volume delivery, resistance to breathing, and backward flow through the 1-way valve for three FS brands—KOL, Micro, and RCK. The results of our study show that only M-M breathing (mean value, 1.00 L) resulted in the delivery of the AHA’s suggested tidal volume of 0.8-1.2 L during rescue breathing on adults. However, we did demonstrate delivery of a mean volume of at least 0.6 L as recommended by the American Society for Testing and Materials (ASTM) with the Micro (mean value, 0.77 L) and RCK (mean value, 0.64 L). It must be noted that these volumes were collected for only 2 minutes, because our goal was to document volume delivery without the effect of fatigue. This time frame may have been too short for the rescuer to ‘settle in’ and find his own intrinsic rhythm and might have affected the volume results. Further study could be done in this area.

Volume delivery with both the Micro and RCK requires that the rescuer seals his mouth to that of the victim, much like M-M ventilation except that the thin material of the FS is preventing actual victim-rescuer contact. The instructions from the manufacturer of the KOL do not require the creation of an M-M seal. Instead, a seal occurs by design between the FS membrane and the patient’s face as the rescuer ventilates the patient. This occurs when the rescuer blows into the adapter protruding from the FS. This adapter is also designed to accept a resuscitator-bag valve (22-mm adapter) so that ventilation can occur with the use of a bag-valve device. However, this seal did not occur and a large leak resulted when rescuers attempted to ventilate a lung under positive pressure produced by M-FS breathing.

Lightsey et al. reported the following mean values for two rescuers: M-M = 1.042 L, KOL = 0.705 L, and Micro = 0.617 L. The values for M-M and the Micro are similar to ours, whereas the volume reported by Lightsey for the KOL is much higher. These volumes were measured with a pneumotachometer attached to the right bronchus of a mannequin, with the left bronchus sealed. The report does not say whether the pneumotachometer was attached to a test lung or left open to the atmosphere. If a test lung was not present, the results may not be valid because the rescuers would have been exhaling into an open system not into one under pressure. According to our results, only the back pressure of the RCK is below the recommended level of < 5 cm H₂O at a flow of 50 L/min suggested by the International Standards Organization (ISO). The RCK is also the only device that met the Emergency Care Research Institute (ECRI) recommendation of resistance < 10 cm H₂O · s · L⁻¹ at a flow of 60 L/min. For the RCK, Rossi et al. reported a mean value of 1.8 cm H₂O for back pressure over a flow range of 24-72 L/min. This reported value is similar to ours.

Lightsey and colleagues reported that the KOL had a resistance to flow of < 10 cm H₂O · s · L⁻¹ at a constant flow of 60 L/min thus meeting the ECRI guidelines. In our study, the back pressure for the Micro is > 5 cm H₂O at all flow rates.

A concern might be raised regarding the RCK FS even though the resistance is very low. The rescuer, in an attempt to seal his lips, must not encroach on the valve housing. If this happens, the vents where the rescuer’s air is exiting the valve will become obstructed and resistance will increase. The patient’s lips could possibly cause the same obstruction, thus making it difficult to ventilate the patient. The other two devices may also exhibit increased resistance if the victim’s tongue obstructs part of the valve housing. Increased resistance may lead to early rescuer fatigue and decrease tidal volume delivery. Vomitus and moisture may affect the function of all these devices. This is a topic deserving of further study.

Our study of backward leak was not quantitative (how much flow came through) but only qualitative (did any gas come through). We did not use intermittent flow of gas as occurs with exhalation nor did we alternate inspiration with exhalation, allowing opening and closing of the valve. We did however show that O₂ and presumably other gases, can make their way through some valves back to the rescuer, even within 1 minute. Of those studied, the KOL and Micro seemed to provide the best barrier. Lightsey et al. concluded from their study that the KOL valve prevented backward flow of gas. Cydulka and others, testing for aerobic bacterial contamination on the rescuer’s side of the Micro valve, found none after having a researcher exhale into the victim’s side of the valve for 60 seconds (12 breaths). Rossi et al. concluded that the backward flow of the RCK valve was very low; thus, airflow from the patient to the rescuer was essentially excluded. We found that the RCK did allow some backward leak of gas. If gases and/or liquids leak through the valve, the rescuer’s side of the FS may become contaminated. This may increase the chance of disease transmission. We recommend that further study be carried out on the backward leak of these devices.

Conclusions

Use of the FS in our study resulted in volume delivery less than the AHA recommended value of 800 mL. Use of the
Micro and RCK did result in volume delivery of at least 600 mL as recommended by the ASTM. Only the RCK met the back pressure and resistance recommendations as set forth by the ISO and ECRI. Neither the Micro nor the KOL valves had significant backward leak of gas. We recognize that some manufacturers are attempting to address the needs of the medical and lay community by producing CPR barrier devices. We encourage them to continue to improve their products. Few studies have addressed the safety and effectiveness of FS. We recommend that until FS have been shown to be safe and effective that they remain classified as Class IIb devices.

**PRODUCT SOURCES**

**CPR Mannequin:**
- Resusci Anne, Laerdal Medical Corporation, Armonk NY

**Ventilation Monitor:**
- Model VM-90, Bear Medical Systems Inc, Riverside CA

**Calibration Syringe:**
- Jones Medical Instrument Co, Oak Brook IL

**Face Shields:**
- Kiss of Life CPR Barrier Mask, Brunswick Biomedical Technologies Inc, Wareham MA
- CPR MicroSHIELD, Medical Devices International, Gurnee IL
- Res-Cue Key, Ambu Inc, Linthicum MD

**Flowmeter:**
- Timeter Instrument Corporation, Lancaster PA

**Calibration Analyzer:**
- RT-200, Timeter Instrument Corporation, Lancaster PA

**O2 Analyzer:**
- Ventronics, a Division of Hudson RCI, Temecula CA

**Statistical Analysis Software:**
- SPSS/PC+, SPSS Inc, Chicago IL

**REFERENCES**

Circadian Patterns of Emergency Asthma Presentations: Implications for Staffing and Treatment

Daniel D Buff MD, Samuel Z Bavli MD, Michael H Bloch MD, Boris M Serebryansky MD, and Chaudhry S Aman MD

STUDY QUESTION: Are there circadian patterns for time of presentation and clinical status in asthmatic patients admitted to an emergency department for acute exacerbations? DESIGN: Prospective observational study. SETTING: Urban community teaching hospital emergency department. PARTICIPANTS: 279 consecutive patients who presented a total of 310 times with asthma exacerbations between October 19 and December 31, 1993. We grouped patients aged 16 years and above as adults and patients younger than 16 years of age as children. INFORMATION COLLECTED: Time of emergency department presentation, time attack began (for adult patients), peak expiratory flow rate prior to emergency department treatment (for adult patients), need for hospital admission, ventilatory failure during an acute attack, and death during an acute attack. RESULTS: Circadian patterns were demonstrated for time of presentation. For the total study group, the peak time of presentation was 8:00 PM to 11:59 PM (p < 0.05) and the trough time of presentation was 4:00 AM to 7:59 AM (p < 0.01). There were differences in peak time of presentation for patients grouped by age. For adult patients only, the peak time of presentation was 8:00 AM to 11:59 AM (p < 0.01), whereas for children only, the peak time of presentation was 8:00 PM to 11:59 PM (p < 0.001). No statistically significant patterns in time of attack onset, hospital admission rates, or peak flow measurements were observed. CONCLUSIONS: There are circadian patterns for the time at which patients with acute asthmatic exacerbations present to our emergency department. Adult patients have a peak time of presentation between 8:00 AM and 11:59 AM, whereas children have an apparent peak time of presentation between 8:00 PM and 11:59 PM. For all age groups, there is a trough in presentation between 4:00 AM and 7:59 AM. [Respir Care 1995;40(6):624-630.]

Introduction

Recent decades have seen worldwide increases in asthma-related morbidity and mortality. In the United States, both hospitalizations and deaths due to asthma have consistently increased. These upward trends have generated considerable concern, and proposed contributing factors have included environmental allergens, lack of access to care, and the overuse of β-agonist bronchodilators.

DR. Buff is Assistant Chairman, Department of Medicine, St John’s Episcopal Hospital, Far Rockaway, New York and Assistant Professor of Medicine, State University of New York Health Science Center at Brooklyn. Dr. Bavli is Chief of Internal Medicine, Department of Medicine, St. John’s Episcopal Hospital, Far Rockaway, New York and Clinical Assistant Professor of Medicine, State University of New York Health Science Center at Brooklyn. Dr. Bloch is Chairman, Department of Emergency Medicine, and Drs. Serebryansky and Aman are associated with Department of Medicine, St. John’s Episcopal Hospital, Far Rockaway, New York. Beach 99th Street, Far Rockaway NY 11691.
Data have accumulated demonstrating circadian patterns of disease presentation for many disorders, including acute myocardial infarction, sudden cardiac death, and ischemic stroke. In a similar way, asthma has been shown to present in specific circadian patterns that peak in the early morning hours. Peak expiratory flowrates (PEFR) reach a nadir at 4:00 AM or when the patient awakens in the morning, with airway resistance progressively increasing between 12:00 AM and 6:00 AM. Studies in both asthmatic inpatients and outpatients have demonstrated that almost three fourths of those patients experience weekly nocturnal asthma attacks. Peaks in asthmatic ventilatory arrests between 12:00 AM and 6:00 AM and in asthma deaths between 8:00 PM and 8:00 AM have also been described.

Because the emergency department (ED) is the site where many patients with severe asthmatic episodes present for treatment, we examined the patterns with which asthmatic patients use the emergency services of our community-based teaching hospital. We sought to determine whether circadian patterns previously documented for asthma pathophysiology and symptoms would result in circadian patterns of ED asthma presentations. Such patterns in ED resource utilization could be important for scheduling and other responses formulated to address increases in asthma related morbidity and mortality.

Methods

Patients

We reviewed charts for all patients presenting to the ED of the St John’s Episcopal Hospital between October 19, 1993, and December 31, 1993. The time of emergency presentation for all patients was recorded, and those patients with a primary diagnosis of acute asthma exacerbation were studied further. Clinicians in the ED made the diagnosis of an asthma exacerbation and determined the treatment and need for admission, based on the patient’s history and clinical condition.

The triage nurse who initially interviewed and examined the patients and the treating ED physician identified asthma patients for study. Each day, a member of the study team reviewed the previous day’s log book (which lists all patients treated and their primary diagnoses on discharge from the ED) to ensure that all eligible asthma patients were included. If review of the log book identified an asthma patient who had not been surveyed in the ED, a member of the study team contacted the patient by telephone within 24 hours of discharge from the ED. All asthma patients who were admitted to the hospital were followed until discharge.

The ED staff or a member of the asthma study team completed an asthma study patient sheet (ASPS) on all asthma patients identified. One of the authors reviewed all ASPS for accuracy and completeness. One of the authors also reviewed the ED medical record for each patient entered into the study to confirm the accuracy of information entered on the ASPS.

Asthma patients were excluded from study for one or more of these reasons:

1. They had been treated at an emergency facility (either that of St John’s Episcopal Hospital or another hospital) for an asthma exacerbation in the 7 days preceding the study ED presentation.

2. They had been discharged from an acute care hospital within 7 days of a study ED presentation, and the previous hospitalization had been for treatment of an asthma exacerbation.

3. The ASPS had not been completed while the patient was in the ED, and the patient could not be contacted by a member of the study team within 24 hours of discharge from the ED.

4. Review of the ASPS or the ED medical record revealed that the principal diagnosis for ED presentation was not an acute exacerbation of asthma.

We classified asthma patients entered into the study into one of two groups, based on age. Adults were defined as patients 16 years or older, and children were defined as patients younger than 16 years of age. These definitions were chosen because at St John’s Episcopal Hospital, asthma patients ≥ 16 years who require admission are admitted to the adult medicine service whereas asthmatics < 16 years who require admission are admitted to the pediatric service.

Patient Characteristics

The time of presentation to the ED was recorded from triage sheets. For those with acute asthma exacerbation, the following information was recorded on the ASPS: time at which the acute asthma attack began, time at which the patient first arrived at the ED, medications taken at home prior to ED presentation, history of medical conditions other than asthma, initial peak expiratory flow rate performed by ED staff prior to treatment in the ED, treatment provided in the ED (eg, medications, endotracheal intubation), and final patient disposition (discharge home, hospital admission, or death).

Not all of the requested information was obtainable for every patient. Because we were concerned about the accuracy of information, we decided to collect information about the time of onset of the acute attack and medications taken at home only for adult patients. Additionally, because many children could not participate in PEFR measurements, we decided to collect peak flow data only for adult patients.

The hospital’s Institutional Research Review Board reviewed and approved the study protocol.

Statistical Analysis

The times when all patients and all asthmatic patients first presented to the ED were computed at 4-hour intervals during a 24-hour cycle: 12:00 AM-3:59 AM, 4:00 AM-7:59 AM, 8:00 AM-11:59 AM, 12:00 PM-3:59 PM, 4:00 PM-7:59 PM,
and 8:00 PM-11:59 PM. The time intervals were determined prospectively. The times at which acute asthmatic episodes began as reported by adult patients were also computed in the same 4-hour intervals. We used χ² tests for goodness of fit to test for differences in the frequency of events among these 6 time intervals. When such analysis showed a statistically significant difference to be present among the 6 time intervals, the peak and trough times were then determined using either χ² analysis or an exact binomial probability test for each of the 6 time intervals individually. We performed separate analyses for all patients presenting to the ED, the total study population of asthmatic patients, the two asthmatic patient groups based on age (adults or children), and for admission status of asthmatic patients (admitted or not admitted).

We analyzed prior medication usage by adult asthmatic patients by comparing medications used by those adult patients who required hospital admission to medications used by those who did not require hospital admission. Statistical significance was tested using χ² tests with Yates' continuity correction for fourfold tables. Alternatively, Fisher's exact test was used when appropriate. Differences in event occurrence between male and female patients were also tested using χ² tests with continuity correction. Mean ages were compared using the Student's t-test for unpaired data.

For analysis of PEFR, we grouped adult asthmatic patients into six 4-hour periods based on the time of presentation to the ED as described. We computed the mean PEFR for each of the 6 time intervals and used one-way analysis of variance to determine the statistical significance among the mean PEFRs. In all cases, the level of significance was chosen as p < 0.05.

Results

From October 19 through December 31, 1993, 6,137 patients presented to the ED. Of the 6,137 patient visits, 310 were for acute exacerbations of asthma meeting criteria for study (Table 1). We excluded 41 additional patient visits from the study: 10 patients had been treated either in an emergency facility or hospital for asthma within 7 days of the study presentation, 15 patients did not have the ASPS completed while they were in the ED and could not be contacted within 24 hours of ED discharge, and 16 patients on further review of the medical record did not have asthma as the primary reason for ED presentation.

The 310 visits that met study entry criteria were made by 279 different patients; 2 patients made 5 visits each, 1 patient made 4 visits, 2 patients made 3 visits, and 16 patients made 2 visits each.

The population studied—all patients meeting the study criteria—is described in Table 1.

The medications taken by adult asthma patients prior to presentation to the ED are listed in Table 2. Although a higher percentage of admitted patients (versus those not admitted) were taking each of the medications examined, none of the differences were statistically significant. Additionally, although a higher percentage of patients who were not admitted took no medications prior to ED presentation, the difference was not significant. Of note, fewer than 15% of adult study patients were taking a corticosteroid preparation, whereas 36% were taking a theophylline preparation.

<table>
<thead>
<tr>
<th>Medication*</th>
<th>All Adults (%)</th>
<th>Admitted (%)</th>
<th>Not Admitted (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Albuterol MDI</td>
<td>107 (70)</td>
<td>17 (77)</td>
<td>90 (69)</td>
</tr>
<tr>
<td>Albuterol nebulizer</td>
<td>10 (7)</td>
<td>2 (9)</td>
<td>8 (6)</td>
</tr>
<tr>
<td>Ipratropium MDI</td>
<td>9 (6)</td>
<td>2 (9)</td>
<td>7 (5)</td>
</tr>
<tr>
<td>Theophylline</td>
<td>55 (36)</td>
<td>11 (5)</td>
<td>44 (34)</td>
</tr>
<tr>
<td>Inhaled corticosteroid</td>
<td>7 (5)</td>
<td>2 (9)</td>
<td>5 (4)</td>
</tr>
<tr>
<td>Oral corticosteroid</td>
<td>16 (10)</td>
<td>4 (18)</td>
<td>12 (9)</td>
</tr>
<tr>
<td>Cromolyn sodium</td>
<td>3 (2)</td>
<td>1 (5)</td>
<td>2 (2)</td>
</tr>
<tr>
<td>Other bronchodilator</td>
<td>6 (4)</td>
<td>0 (0)</td>
<td>6 (5)</td>
</tr>
<tr>
<td>Oral antibiotic</td>
<td>4 (3)</td>
<td>0 (0)</td>
<td>4 (3)</td>
</tr>
<tr>
<td>No medication</td>
<td>31 (20)</td>
<td>1 (5)</td>
<td>30 (23)</td>
</tr>
</tbody>
</table>

MDI = metered dose inhaler.
* for none of the medications listed was the difference in use between patients admitted and not admitted statistically significant.

Figures 1-3 illustrate the times of ED presentation for all asthmatic patients, for adult asthma patients, and for the children with asthma. For the total study asthmatic group, peak time of presentation was from 8:00 PM through 11:59 PM (p < 0.05 compared to all time intervals) and trough time from 4:00 AM through 7:59 AM (p < 0.01 compared to all time intervals). This contrasts with the data for all patients presenting to the ED, in that no significant circadian patterns were found when patient presentations were analyzed regardless of the diagnosis (data not presented).

When asthmatic patients were separated by age group, different patterns emerged. For adult patients, the peak time for presentation was from 8:00 AM through 11:59 AM (p < 0.01 compared to all time intervals). For children, the peak time for

<table>
<thead>
<tr>
<th>Table 1. Characteristics of Asthmatic Patients Studied</th>
</tr>
</thead>
<tbody>
<tr>
<td>Variable</td>
</tr>
<tr>
<td>----------</td>
</tr>
<tr>
<td>Total visits (%)</td>
</tr>
<tr>
<td>Total patients (%)</td>
</tr>
<tr>
<td>Mean (SD) age in years</td>
</tr>
<tr>
<td>Male patients (%)</td>
</tr>
<tr>
<td>Female patients (%)</td>
</tr>
<tr>
<td>Number admitted (% of visits)</td>
</tr>
</tbody>
</table>

* p < 0.01 for comparison of number of adult male vs adult female patients.
† p < 0.001 for comparison of number of male children vs female children.
Circadian Patterns of Asthma

Fig. 1. Time of presentation to the Emergency Department for all asthma patients. A circadian pattern exists, with the peak time for presentation being the interval 8:00-11:59 PM (p < 0.05) and the trough time for presentation the interval 4:00-7:59 AM (p < 0.01).

Fig. 2. Time of presentation to the Emergency Department for adult asthma patients. A circadian pattern exists, with the peak time for presentation being the interval 8:00-11:59 AM (p < 0.01). The trough time for presentation, the interval 4:00-7:59 AM, does not reach statistical significance.

Fig. 3. Time of presentation to the Emergency Department for children with asthma. A circadian pattern exists, with the peak time for presentation being the interval 8:00-11:59 PM (p < 0.001) and the trough time for presentation the interval 4:00-7:59 AM (p < 0.01). An increase in presentations of lesser magnitude is seen at 4:00-7:59 PM (p < 0.05) and a decrease in presentations of lesser magnitude at 12:00-3:59 AM (p = 0.01).

Fig. 4. Time for onset of acute asthmatic attack in adult patients. Although the data appear to show a trend favoring onset of attack in the morning, the pattern is not statistically significant.

presentation appeared to be from 8:00 PM through 11:59 PM (p < 0.001 compared to all time intervals). In children, a significant increase in presentations of apparently lesser magnitude also occurred from 4:00 PM through 7:59 PM (p < 0.05 compared to all time intervals).

In both adults and children with asthma, the trough time for presentation appeared to be from 4:00 AM through 7:59 AM, although only in children did the trough achieve statistical significance (p < 0.01 for children, p = 0.10 for adults compared to all time intervals). For children, a significant trough time for presentation of apparently lesser significance was seen from midnight through 3:59 AM (p = 0.01 compared to all time intervals).

Analysis of our data showed no statistically significant patterns in the proportion of patients admitted versus those not admitted when the data were grouped by time of presentation.

Figure 4 illustrates the time at which asthma attacks began for adult patients. Of 173 adult visits, 118 patients (68%) were able to give an approximate time for when they became symptomatic. This included 99 (66%) of the patients who were not admitted and 19 (86%) of the admitted patients. Although the largest number of patients reported onset of symptoms in the early morning, no statistically significant pattern in time of onset of attack was found. When the same analysis was performed for admitted patients only and for patients who were
not admitted only, no statistically significant pattern was demonstrated (data not presented).

The peak flow data for adult asthma patients are presented in Table 3. Peak flow measurements were obtained in 142 of 173 (82%) adults presenting. The pattern obtained was not statistically significant. Data are not presented for admissions versus non-admissions because the number of peak flow measurements in the admitted group was too small to allow for meaningful comparison.

During the study period, there were no deaths among the 310 patient visits. One adult and one child required endotracheal intubation in the ED. Both presented in the 12:00 AM through 3:59 AM time interval.

Table 3. Peak Expiratory Flow Measurements in Adult Asthma Patients by Time Interval

<table>
<thead>
<tr>
<th>Time Interval</th>
<th>Mean (SD) Peak Flow Rate (L/min)</th>
<th>Number of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>12:00 AM-3:59 AM</td>
<td>188 [95]</td>
<td>20</td>
</tr>
<tr>
<td>4:00 AM-7:59 AM</td>
<td>205 [85]</td>
<td>16</td>
</tr>
<tr>
<td>8:00 AM-11:59 AM</td>
<td>213 [98]</td>
<td>41</td>
</tr>
<tr>
<td>12:00 PM-3:59 PM</td>
<td>214 [100]</td>
<td>21</td>
</tr>
<tr>
<td>4:00 PM-7:59 PM</td>
<td>202 [89]</td>
<td>23</td>
</tr>
<tr>
<td>8:00 PM-11:59 PM</td>
<td>220 [116]</td>
<td>23</td>
</tr>
</tbody>
</table>

No statistically significant circadian pattern was demonstrated.

Discussion

Previous studies have demonstrated that, from both a pathophysiologic and a clinical standpoint, asthma is a disorder of circadian nature. Measurements of lung function in normal patients and, to a greater extent, in asthmatic patients, show that pulmonary function is best in the late afternoon and worst in the early morning hours.13-16 It has been shown that most asthmatic patients have nocturnal symptoms17 and that there are peaks in ventilatory arrests18 and deaths in asthmatics patients19-21 during the nighttime and early morning hours. The factors proposed to contribute to the observed circadian patterns of asthma have been reviewed extensively elsewhere22-25 and include variations in mucociliary clearance of pulmonary secretions, pulmonary vagal neurologic function, and lower esophageal sphincter tone with reflux. Variations in airway temperature, cortisol secretion, and levels of several immunologic factors have also been implicated.

In the current study, we have attempted to determine whether there are circadian patterns in the utilization of emergency services by asthmatic patients, and whether the clinical status of those patients can be correlated with time of day. For all asthmatic patients, there was indeed a circadian distribution for presentations with a peak at night (8:00 PM through 11:59 PM) and a nadir in the morning (4:00 AM through 7:59 AM). Further analysis of patients based on age showed that the peak time of presentation for adults was in the late morning (8:00 AM through 11:59 AM), whereas the peak time of presentation for children was at night (8:00 PM through 11:59 PM). Both groups had an apparent nadir in time of presentation from 4:00 AM through 7:59 AM, although only in children did the nadir reach statistical significance. These patterns in asthma presentations contrast with the data for all patients (regardless of the diagnosis) presenting to the ED. There were no significant circadian patterns in the all-patient analysis.

The only previously published report we could locate that examined time of emergency department presentation for asthma exacerbations was by Rossi et al, who found that the peak time of presentation for adults in a Finnish population was 10:00 to 11:00 AM.26 This correlates well with our peak of 8:00 AM to 11:59 AM, and would appear to confirm the significance of this result.

The finding that adult patients have a peak for presentation in the late morning would at first appear surprising. After all, this peak is about 6 hours later than what would be expected in view of known circadian data for pulmonary function in asthmatics. However, this 6-hour discrepancy may reflect attempts by patients to treat their asthma at home. It may be that most attacks did start in the early morning but only the minority of patients with severe symptoms came to the ED at the onset of an attack. The majority of patients whose symptoms were not as severe may have spent several hours attempting to treat their asthma at home before coming to the ED.

An alternative explanation is that the morning peak in ED presentations of adult asthma patients may be explained by social rather than medical factors. Patients may simply have preferred to wait until daylight or came to the hospital when they realized they were too sick to go to work or school.

Most data on circadian patterns of respiratory function are from studies on adults, but there is good evidence that the same patterns hold true for children.26,27 Although we could not find a previous study on pediatric asthmatic emergency department presentations, we assumed that the utilization patterns for children and adults would be the same. Our findings of significant differences in presentations based on age were therefore unexpected, especially because the children in our study presented more often between 8:00 PM and 11:59 PM, a period when pulmonary function would be expected to be at its best.

A possible explanation for this finding may lie in differing sleep patterns between adults and children. The pediatric patients in our study were quite young and, therefore, would be expected to go to sleep earlier than the adult patients. Although time of day is important in determining the diurnal variations in pulmonary function, sleep has been shown to be a major contributor to nocturnal bronchoconstriction.22 Ballard et al16 examined lower airway resistance in asthmatic patients at night while they were asleep and while they were awake. Results showed that although airway resistance in-
creased in asthmatic patients at night independent of sleep, the increase in resistance doubled when the study subjects were asleep.\textsuperscript{16} The earlier onset of sleep in young children may result in an earlier peak in asthma presentations (nighttime versus early morning).

As in our adult patients, social factors may have contributed to the nighttime peak we observed in the presentation of pediatric asthma patients. Most of the children in our study were brought to the hospital by their parents, and it is possible that children waited at home or were kept at home until a parent returned from work in the evening. It is also possible that the children were brought to the ED when they or their parents were unable to fall asleep because of the child’s shortness of breath. An alternative explanation may have to do with children’s play activities. It may be that when children finished school in the afternoon and went to play, the exercise itself or returning to a warmer indoor environment when the exercise was completed triggered asthmatic exacerbations.\textsuperscript{28}

No statistically significant circadian patterns could be demonstrated for the clinical indices we studied or for the estimated time for the beginning of exacerbations. It is possible, however, that a larger study would allow some of the trends we observed to reach statistical significance.

The data on medications taken prior to presentation deserve some discussion. A relatively large number of patients were taking no asthma medications prior to ED presentation, and rate of corticosteroid use was low. The latter finding is particularly alarming given the inflammatory nature of asthma. The fact that the hospital at which this study was performed is located in an urban setting and that many of our patients did not receive regular medical care may account for these findings. It may also be that, at the time of this study, many physicians practicing in the urban community were unaware of or did not follow the most recent recommendations regarding the treatment of chronic asthma.\textsuperscript{29} Further education of both patients and their primary physicians about the optimum treatment of asthma appears warranted.

An incidental finding of the study was that 18\% of our adult patients did not have peak expiratory flow measurements taken prior to therapy, and an even larger percentage of pediatric patients did not have objective measures of lung function performed. There may be a need to further educate ED staff about the importance of monitoring measures of pulmonary function during an acute exacerbation of asthma.

We chose the months October through December in part because records at the study hospital indicated that these were the months for peak utilization of the ED by asthma patients. This is consistent with published data showing peaks in hospitalizations and deaths due to asthma during this time of the year.\textsuperscript{40} It is possible that our findings are limited to the months examined and that different patterns exist at other times of the year. However, previous data have demonstrated that circadian patterns in the pulmonary function of asthma patients remain constant throughout the year. Moreover, in the emer-

gency department study done by Rossi et al\textsuperscript{15} monthly asthma presentation rates were relatively stable.

Conclusions

In summary, we have identified peaks in the ED presentation of asthmatic adults (8:00 AM through 11:59 AM) and children (8:00 PM through 11:59 PM) and a nadir in both groups from 4:00 AM through 7:59 AM. We were not able to demonstrate statistically significant circadian patterns in our analysis of admission rates, time of attack onset, or in clinical data. Further research is needed to determine whether the patterns we demonstrated can be reproduced in other populations. Emergency Department staffing targeted to accommodate peaks in asthma presentations and treatment strategies targeted at circadian peaks in asthma activity should be considered as physicians and health-care providers address the rising worldwide rates of asthma-related morbidity and mortality.

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16. Ballard RD, Saathoff MC, Patel DK, Kelly PL, Martin RJ. Effect of
Effects of Aerosolized Synthetic Surfactant in a Canine Model of Wood Smoke Inhalation

Robert R Fluck Jr MS RRT, Andrew M Paskanik
William R Clark Jr MD, and Gary F Nieman BS

BACKGROUND: Pulmonary surfactant replacement therapy is beneficial for premature infants with surfactant deficiency, and preliminary studies indicate that it may also be beneficial for the treatment of the surfactant deficiency associated with the acute respiratory distress syndrome (ARDS). Surfactant deactivation is an important factor in the pathophysiology of wood smoke inhalation. In this study, we examined the effects of surfactant replacement with aerosolized EXOSURF on smoke-inhalation injury in dogs.

METHODS: All dogs were anesthetized and placed on a ventilator with provision for airway pressure measurement. Vascular access was established for sampling, pressure measurements, and cardiac-output determinations. Smoke was delivered to the animals at 26 breaths/min for 5 minutes. The treatment consisted of either aerosolized surfactant (experimental group, n = 6) or aerosolized saline (control group, n = 6). We measured P_{aO_2}, carboxyhemoglobin, lung compliance, and pulmonary shunt at 30-minute intervals for 4 hours. Following sacrifice of the animal 4 hours after smoke exposure, we determined the wet-dry lung-weight ratio and dynamic function of pulmonary surfactant with a Wilhelmy balance.

RESULTS: There was no statistically significant difference for any of the measured variables between the control and experimental groups. CONCLUSION: EXOSURF does not ameliorate the acute pulmonary pathophysiology of severe smoke inhalation within 4 hours of injury. [Respir Care 1995;40(6):631-637]

Introduction

Smoke inhalation increases the mortality rate associated with cutaneous burn injury.\(^1\)\(^2\) Venus et al\(^1\) demonstrated a mortality rate of 7.1% in 914 burn patients without associated smoke inhalation and 54.7% in those with inhalation injury. The pathophysiology of smoke inhalation varies greatly as a result of the multitude of combustion byproducts generated by many different kinds of fuel, the temperature of combustion, the concentration of the smoke, and the length of time the victim is exposed. As a result, the clinical picture varies from mild (upper airway irritation without respiratory compromise) to severe (respiratory and cardiac arrest).\(^3\)

Smoke inhalation in animal models results in hypoxemia, carboxyhemoglobinemia, increased intrapulmonary shunt, decreased lung compliance, atelectasis, and high-permeability pulmonary edema.\(^3\)\(^12\)

Because wood smoke inhalation decreases pulmonary compliance directly by inactivation of surfactant and indirectly as a consequence of alveolar flooding with proteinaceous liquid, we postulated that replacement of the surfactant with exogenous surfactant would ameliorate the acute effects of wood-smoke inhalation.
Methods and Materials

Model Preparation

The experiments described in this article were performed in adherence with National Institutes of Health guidelines for the use of experimental animals in research. The protocol was approved by the university’s Committee for the Humane Use of Animals (CHUA).

Twelve mongrel dogs weighing between 15 and 22 kg were anesthetized with 30 mg/kg nembutal, placed in the supine position, and ventilated with 15 breaths/min, 40% oxygen and 5 cm H₂O positive end-expiratory pressure, using a ventilator* equipped with a special nebulizer initially operating without any liquid. (See the Protocol section for a description of this nebulizer.) Adequate anesthesia was maintained with boluses of intravenous nembutal (65 mg) as needed based on eye reflexes, and paralysis was maintained with boluses of intravenous succinylcholine chloride (20 mg) as needed based on ventilation asynchrony after deep anesthesia had been confirmed. Tidal volume was adjusted initially to maintain arterial PCO₂ between 35 and 45 torr. If the base excess (BE) was below -2 mEq/L, it was corrected by the intravenous administration of sodium bicarbonate; the dose was calculated by

\[
\text{HCO}_3^- (\text{mEq}) = \text{BE (mEq/L)} \cdot \text{BW (kg)} \cdot \text{EF (0.3 L/kg)},
\]

where BE = base excess, BW = body weight, and EF = extracellular fluid.

Administration of the total estimated dose (as opposed to the half dose routinely given to humans) usually resulted in correction of BE to within normal limits (0 ± 2 mEq/L).

Femoral arterial and venous cutdowns were performed and lengths of 2-mm-ID polyethylene tubing were placed for drug administration, blood-gas sampling, and arterial blood-pressure measurements. A 7-French flow-directed thermodilution pulmonary artery catheter was inserted into the pulmonary artery through the external jugular vein for pulmonary artery (Ppa) and pulmonary-artery wedge (Ppw) pressure measurements, mixed-venous blood sampling, and cardiac output determination. All pressures were measured using disposable transducers that had been leveled at the heart; values were traced continuously on a chart recorder. Peak airway pressure (Paw) and PEEP were measured from a side port in the ventilator circuit connector 10 cm from the endotracheal tube. Static lung compliance (Cstat) was measured by disconnecting the ventilator and injecting twice the tidal volume (VT) with a 1-L calibrating syringe. The plateau pressure (Pplat) was recorded and compliance was calculated as 2 VT/Pplat.

Arterial, pulmonary-artery, and airway pressures; arterial and mixed-venous blood gas values; cardiac output; and carboxyhemoglobin levels were recorded at baseline, at 5 minutes after smoke exposure, and then every 30 minutes for 4 hours.

When blood gas values were stable and baseline values had been recorded, the animal was removed from the ventilator and placed on an animal ventilator modified so as to deliver smoke. The breathing gas was passed through a chamber containing burning fir-wood sawdust that had been moistened with kerosene. The technique has been described in detail elsewhere. The total exposure time was 5 minutes. VT was the same as that used on the original ventilator, and the respiratory rate was 26 breaths/min. Following smoke inhalation, the animals were returned to the original ventilator with the original settings. No further changes in ventilator settings were made for the remainder of the experiment.

Protocol

Animals were randomly separated into an experimental (surfactant aerosol treatment) or control (saline aerosol treatment) group. The surfactant preparation used for this experiment was EXOSURF, a totally synthetic preparation that is supplied as a dry lyophilized powder and must be reconstituted before use.

Surfactant or saline was aerosolized using a nebulizer designed especially to deliver surfactant (VISAN) driven by an inspiratory flow signal from the ventilator. This nebulizer has 3 jets; 1, 2, or all 3 can be operating at any given time. The number of jets operating and the driving pressure were adjusted to optimize the concentration and volume of aerosol delivered; in all cases, the nebulizer delivered at least two thirds of the VT. The mass median aerosol diameter generated by the VISAN is 1-3 μ, and the concentration of aerosol it generates varies inversely with the driving pressure. At a pressure of 50 psi, the aerosol concentration is 18.3 μL/L, and at 40 psi, 30.8 μL/L.15 (Although the aerosol concentration peaks at 36.6 μL/L at a driving pressure of 30 psi, this provides insufficient gas flow and thus fraction of the delivered VT even with all three jets operating.) Source gas for the nebulizer and the ventilator was provided by a blender so that the FIO₂ was the same for both sources of VT. Each vial of surfactant contained 2,025 grams of dipalmitoylphosphatidylcholine, 225 mg cetyl alcohol, 150 mg tyloxapol, and 146.1 mg NaCl.

The VT was set at approximately 15 mL/kg, inspiratory time was approximately 0.45 seconds, and the nebulizer was operated at 40 psi with 3 jets running, delivering approximately 75% of the tidal volume. (It is important to note that the flow from the nebulizer can be affected both by the number of operating jets and also the driving pressure; the driving pressure also affects the concentration of aerosol. Thus the actual volume of aerosol particles delivered during a breath is determined both by the volume the nebulizer delivers and also the concentration of aerosol particles in that volume.) Each vial of surfactant was reconstituted with 25 mL of sterile water and added

* Products and suppliers are identified in the Product Sources section at the end of the text.
to the nebulizer immediately after the 5-minute post-smoke data collection was complete. An additional 5 mL of sterile water was used to collect the surfactant remaining in the vial; this was added to the nebulizer 2 hours after smoke exposure. Each animal received the contents of one vial of surfactant during the 4 hours after smoke exposure. All animals survived the 4-hour time period. At the conclusion of the experiment, a median sternotomy was performed on each dog.

Dynamic surfactant function of lung lavage fluid was measured on a modified Wilhelmy balance (WHB). The right cardiac lobe (RCL) of the lung was excised with the airway intact and inflated with a 60-mL syringe to eliminate any atelectasis. Saline (60 mL) was injected and withdrawn 5 times from the RCL; this process was repeated 3 times for a total lavage volume of 180 mL. This lavage fluid was placed into the trough (351 cm$^2$) of the WHB, which had been primed with 750 mL of saline. The samples were allowed to ‘cure’ for 30 minutes before surface compression. The trough surface was compressed with a movable barrier from 100% (maximum) to 20% (minimum) of total trough area and decompressed back to 100%. This compression-decompression cycle was repeated 5 times. The surface tension (ST) was continually measured with a platinum paddle connected to a pressure transducer with microscale accessory that was coupled with an amplifier and recorder. Each time cycle was 2 minutes; all samples were run at room temperature. The ST at minimum area (ST$_{\text{min}}$) and maximum area (ST$_{\text{max}}$) from the fifth compression-decompression cycle were used. Surfactant was considered functional if ST$_{\text{min}}$ was $\leq$ 10 dyn/cm.

Random samples of tissue from the remaining lung were cut, weighed, and placed in an oven at 65°C until all the water was removed. Lung water was expressed as wet-to-dry lung weight ratio (W/D).

Statistical significance between groups (normal saline versus surfactant) and within groups (post-smoke exposure versus other times) was determined using a 2-way analysis of variance (ANOVA) with repeated measures. For statistically significant ANOVA, Newman-Keul’s test was used to identify the individual comparisons that were significant.$^{16}$

**Results**

$P_{\text{aO}_2}$ and COHb data are shown in Figures 1 and 2. The $P_{\text{aO}_2}$ dropped significantly immediately after smoke inhalation. Surfactant treatment did not significantly change $P_{\text{aO}_2}$ compared to saline aerosol at any time point. COHb increased to the same initial level in both groups and demonstrated a similar ‘washout’ with time. The presence of similar post-smoke COHb levels between the groups suggests that their exposure to smoke was similar.

The hemodynamic values that were measured (cardiac output, systemic arterial blood pressure, and pulmonary artery pressure) were not statistically significantly different between groups at any measurement period (p $> 0.05$).

Static compliance fell immediately following smoke exposure and continued to fall until 150 minutes following smoke exposure (Fig. 3). However, these decreases were not significantly different from the pre-smoke baseline values, and neither treatment had any effect on compliance. Postmortem examination of the lungs indicated that both surfactant- and saline-treated lungs were approximately equally atelectatic.

Intrapulmonary shunt increased equally in the groups at 5 minutes following smoke exposure (Fig. 4) but was not significantly different from the pre-smoke baseline. Shunt continued to rise with time in both groups. The surfactant treated group actually had a higher shunt fraction than the saline-aerosol group from 150 to 240 minutes post smoke exposure, although not significantly so. These findings are consistent with the atelectasis seen postmortem.
Pulmonary surfactant function, expressed as the surface tension at (20%) compression, was inhibited in both groups (Fig. 5). Surface tension minimum was significantly elevated in both groups as compared to that of historic controls but was not significantly different between the two treatment groups.

Lung water (expressed as wet-dry ratio) following smoke inhalation was significantly increased over historic controls in both the surfactant and saline treatment groups (Fig. 6). Lung water was less in the surfactant group compared to the normal saline group, but the difference was not statistically significant.

**Discussion**

In this study, neither surfactant nor saline aerosol effected an improvement in any parameter, except for a slight and insignificant decrease in lung water. Wood smoke is known to deactivate surfactant and, presumably, the decreased compliance and increased intrapulmonary shunt following inhalation injury is secondary to altered surfactant function. An in vitro study, utilizing a Wilhelmy balance, demonstrated that wood smoke deactivates surfactant and that the addition of surfactant re-establishes normal surface tension. A preliminary study in a smoke-inhalation model identical to that used in the present study demonstrated that administration of a bovine surfactant markedly improved lung function. These studies suggest that surfactant replacement is a viable treatment for smoke inhalation and suggest that the synthetic surfactant preparation used in our study may be less effective than other surfactant preparations in the treatment of acute smoke inhalation.
Smoke inhalation injury has been studied clinically in humans and experimentally in animals using a variety of sources of smoke and has been shown to result in a wide array of pulmonary pathology. In humans, smoke inhalation causes a deactivation of surfactant, increased work of breathing, increased airway resistance, increased physiologic dead space, hypoxemia, and an edematous and inflamed tracheobronchial tree. Electron microscopy of rabbit lungs exposed to pine wood smoke shows the development of epithelial inflammatory changes and destruction of the cilia and secretory cells. Sheep exposed to cotton smoke develop diffuse pulmonary mucosal sloughing, pulmonary edema, and a decreased Pao2 from 6 to 15 hours after exposure. Wood smoke inhalation in dogs produces an injury similar to that seen in sheep, with hypoxemia, an increased intrapulmonary shunt, and pulmonary edema, but does so much more rapidly (within 5 minutes of exposure) than does exposure to cotton smoke. Earlier work from our laboratory showed that surfactant deactivation is one of the primary pathologic changes seen following wood smoke inhalation.

Surfactant deactivation is found in patients with acute respiratory distress syndrome (ARDS). Gregory and colleagues showed that surface tension at minimum compression of surfactant harvested from patients with ARDS is 4 times higher than normal. Lewis and Jobe postulated that inactivation or abnormal production of surfactant may play a role in the pulmonary edema, decreased compliance, and increased intrapulmonary shunt that are the hallmarks of ARDS. In a recent multicenter trial in sepsis-induced ARDS, EXOSURF aerosolized continuously for 5 days seemed to improve survival compared to saline placebo. However, only a dose-response relationship is reported on a small number of patients, without statistical analysis. A similar trial wherein the drug was continuously aerosolized showed a decreased intrapulmonary shunt and mortality in the surfactant group compared to saline controls. As with the previous study, there was no statistical analysis; determination of efficacy was made by rank order.

The effects of EXOSURF treatment have been extensively investigated for treating respiratory distress syndrome in the newborn. Early work with this drug by Durand and colleagues showed that preterm lambs in the treatment group had improved survival at 11 hours compared to controls. Bose et al reported that very-low-birthweight infants given a single dose of EXOSURF had both lower rates of pulmonary interstitial emphysema and lower combined outcome of death rate or survival with bronchopulmonary dysplasia compared to controls. In a similar study, Corbet and colleagues found a single dose of EXOSURF resulted in a decrease in the number of deaths due to respiratory distress syndrome (RDS), a decrease in pulmonary air leaks, and a decrease in oxygen and mean airway pressure requirements. They concluded that although there was no reduction in the incidence of RDS, there was a decreased severity of lung disease in treated infants.

In spite of the reported success of the previously cited investigators, others have not found EXOSURF to be effective. Cummings et al found no differences in any measured variable in preterm lambs between controls and those treated with instillation of EXOSURF before the first breath. Hall and colleagues found that EXOSURF had negligible effect on lowering surface tension in vitro; it had similar minimal effect on restoring normal mechanics to excised lavaged rat lungs. The failure of EXOSURF to improve lung function in the cited studies may be related to the absence of the apoproteins that are present in natural surfactant because the addition of these apoproteins has been shown to significantly improve the ability of the drug to reduce surface tension in vitro.

Smoke inhalation causes a significant increase in lung water. It has been shown that high alveolar surface tension can cause pulmonary edema in and of itself. Our lab also previously demonstrated that wood smoke inhalation resulted in increased endothelial permeability. This combination of increased surface tension and increased vascular permeability may act together to result in alveolar edema. Treatment with Exosurf resulted in no significant change in lung water compared to saline.

One of this study’s limitations was that it only evaluated the acute effects of surfactant treatment. Armsby and colleagues found no increase in compliance at 4 hours following EXOSURF treatment of preterm infants yet there was a significant improvement at 1 week of age (following a second dose). Pfenninger et al reported no change in lung mechanics or gas exchange 20 minutes following surfactant treatment, yet significant improvement in Pao2 and compliance occurred over the next 12-36 hours. Bhutani and colleagues found no change in pulmonary mechanics 2 hours following EXOSURF treatment, yet at 24 hours, there was significant improvement. However, preliminary results from our laboratory, utilizing a smoke-inhalation model identical to the one used in this study, found that instillation of a bovine surfactant markedly improved shunt, Pao2, and compliance within a 4-hour period.

Conclusion

There was no significant improvement in any measured variable following a single aerosol dose of EXOSURF compared to saline placebo in this canine model of smoke inhalation, except for a slight decrease in lung water. Although treatment with EXOSURF might show some benefit over an extended period of time, it was acutely ineffective. These results cannot necessarily be extrapolated to other types of surfactant, to surfactant administered by means other than aerosol, or to larger or multiple doses. Further research using other surfactant preparations, different doses, multiple doses, and other administration methods is needed to determine the effectiveness of surfactant replacement for treating smoke inhalation.
PRODUCT SOURCES

Ventilator:
Bear 5, Bear Medical Systems, Riverside, CA

Cardiac Output Computer:
Model 9520, Edwards Labs, Irvine, CA

Transducers:
Sorensen, Transpac MK4-04DTNVF, North Chicago IL

Chart Recorder:
Model 7754A, Hewlett Packard, Waltham, MA

Amplifier:
Model 8805B, Hewlett Packard, Waltham, MA

Calibration Syringe:
1-mL, Warren E. Collins Inc., Braintree, MA

Blood-Gas Analyzer:
Radiometer ABL2, Copenhagen, Denmark

CO-oximeter:
Radiometer OSM-2, Copenhagen, Denmark

Surfactant:
EXOSURF, Burroughs-Wellcome Co, Research Triangle Park, NC

Surfactant Nebulizer:
VISAN model VN8, VN4, Vortran Medical Technology Inc., Sacramento CA

Wilhelmy Balance Transducer:
Gould Inc., Ohio, CA

Statistics Package:

Air-Oxygen Blender:
Bird 3M, Palm Springs CA

REFERENCES


FACE TO FACE WITH CHANGE

AARC 41st Annual Convention and Exhibition
December 2-5 • Orlando, Florida
Evaluation of the Ciba Corning 840 Blood Gas Analyzer

Ronald J Wong BA, John J Mahoney BA RCP, and Antonius L Van Kessel BS RCP

BACKGROUND: Because our laboratory had used the Ciba Corning 200 series blood-gas analyzers for a number of years, we were asked to participate in the evaluation of a premarket unit of the Model 840 analyzer (C840).

DESCRIPTION OF DEVICE: The C840 is a bench-top instrument that combines a menu-driven user interface with an automated sampler and integrates software for data management and system diagnostics. EVALUATION METHODS: We compared the performance of the C840 to a laboratory-based Ciba Corning 278 (C278), analyzing a total of 325 blood samples. We also evaluated the software for routine laboratory applications. EVALUATION RESULTS: The bias and imprecision (± 2 SD) between the C840 and C278 was calculated for pH (+0.004 ± 0.014 pH units), $P_{CO_2}$ (+1.8 ± 3.3 torr), and $P_{O_2}$ (+0.01 ± 9.0 torr for all $P_{O_2}$ ranges; -0.17 ± 4.8 torr for $P_{O_2} < 150$ torr). CONCLUSIONS: We conclude that the analytical performance of the C840 is comparable to the C278, and its data storage and interface capabilities should help laboratories meet CLIA-88 requirements.[Respir Care 1995;40(6):638-643]

Introduction

A number of current blood gas instruments incorporate computers to help laboratories conform with the Clinical Laboratory Improvement Amendments of 1988 (CLIA-88). An example of this type of analyzer is the Ciba Corning 840 (C840).* We performed a clinical study according to the American Association for Clinical Chemistry (AACC) guidelines on a "pre-release" C840 in order to assess its performance in comparison to our established blood-gas analyzer. We also evaluated the integrated computer system with respect to its use in routine laboratory practice and its ability to comply with governmental rules regulating storage and retrievability of patient results, quality-control data, and instrument maintenance records.

Description of Device

The C840 is used to determine pH, $P_{CO_2}$, and $P_{O_2}$. It also calculates standard and actual bicarbonate ($HCO_3^-$), total carbon dioxide (ct$CO_2$), blood and extracellular base excess [BE(B) and BE (ect)], oxygen saturation ($S_O_2$), oxygen content ($C_O_2$), alveolar-arterial oxygen tension difference ($P(A-a)O_2$), and arterial-alveolar oxygen tension ratio ($P(A-a)O_2$).

The approximate dimensions of the analyzer are width 56 cm, depth 48 cm, height 51 cm, and weight 29.5 kg. Data entry and instrument commands are accessed by a soft-key touch pad situated below the adjustable LCD screen or by an alphanumeric keyboard. The analyzer can be externally interfaced via 3 RS232 ports with data management systems (DMS), laboratory information systems (LIS), hospital information systems (HIS), CO-oximeters, and/or ticket-end 8.5 in. x 11 in. printers.

In the hydraulic system of the C840, three peristaltic pumps have replaced the vacuum pump of earlier analyzers. Each peristaltic pump is dedicated to its own function (reagent delivery, sample aspiration, or waste elimination). The use of peristaltic pumps has eliminated the need for waste filters and

* Suppliers are identified in the Product Sources section at the end of the text.
water traps. The sample pathway has been redesigned to eliminate the segmentation valve assembly used in the 200 series, and the sample tubing is short and connects directly to the measuring block. An automated aspiration assembly holds the sample syringe in place on the instrument, and a metal probe enters the syringe to aspirate the sample. The device automatically identifies the sampling device and draws the precise volume required for analysis. This not only ensures consistent sample delivery but also ensures accurate sample volume and should minimize personnel exposure to biohazardous sample aerosols and spills. The C840 incorporates the same electrodes as those found in the 200 series analyzers. A spring-loaded latch locks open for electrode removal and also keeps the modules aligned in the measurement block. Also, the throughput now averages 35 samples per hour. Fiberoptic cables continuously illuminate the sample chamber and, with the help of a magnified viewfinder, allow the user to view the sample during analysis. Sample size requirements are: 90 μL for syringe and capillary samples; 45 μL for microsyringe and split-capillary samples; 35 μL for pH only samples; and 10 mL for expired gas samples.2

A menu with prompts is displayed on a high resolution LCD screen. A status/event log (which stores diagnostic codes and system messages from the last 72 hours) and a step-by-step diagnostic display (which lists possible solutions associated with a specific error code) can be used to assist in unscheduled instrument maintenance and may facilitate troubleshooting. The software for quality control ranges, patient reference ranges, automatic calibration times, and printing options can be customized. The C840 has the capacity to store up to 1,500 patient sample reports on its internal hard drive. In addition to patient reports, it can also display on-screen, quality control data with range checking and statistical summary reports, Lavey-Jennings charts for up to 12 control files (each file holds up to 100 data points), and all measured parameters for one month of quality control. All data can be printed on a built-in roll printer or on an externally interfaced printer. The user has the option of incorporating an alphanumeric keyboard and/or laser bar code scanner for patient identification and quality control range entry. All data are stored on the internal hard drive and can be downloaded onto diskettes for archiving and back-up.

Because the C840 shares the same single-base platform as the other 800 series analyzers, it may be adapted to measure additional analytes by inserting a new measurement block that contains the desired electrode modules (e.g., Na⁺, K⁺, Cl⁻, Ca²⁺, glucose, and lactate). The software associated with the additional modules is readily accessible.

Evaluation Methods

Ciba Corning 278 Operation

A single Ciba Corning 278 (C278) was arbitrarily chosen from several available instruments in the Blood Gas Laboratory at Stanford University Hospital and used throughout the study. This instrument was operated in accordance with the manufacturer’s instructions. Its performance was checked with a calibration-gas mixture (3.0% CO₂, 12.0% O₂, balance N₂) and buffer (pH 7.382 at 37 °C) every 30 minutes, and with this same gas and buffer and a second (slope) gas (10.0% CO₂ and 90.0% N₂) and buffer (pH 6.838 at 37 °C) every 2 hours. Limits for the deviations from the theoretical target values for the calibrations were ± 2.0 torr for both PCO₂ and PO₂, and ± 0.010 pH units for pH. (These were limits recommended by the manufacturer.)

For quality control of pH, PCO₂, and PO₂, a reduced bovine hemoglobin preparation was tonometered as described by Mahoney et al.3 using a three-channel bubble-type syringe tonometer attached to a precision gas mixer. Every 24 hours, three different gas-tonometered samples listed in Table 1 were tested. Because the pH for each level is lot-dependent, the pH data can only be used to indicate instrument imprecision. The same lot of each level of control material was used throughout the study.

Corning 840 Operation

Prior to the start of the study, we received hands-on training from the manufacturer’s representatives. The training included operation, maintenance, and troubleshooting of the instrument, and the C840 was operated in accordance with the manufacturer’s instructions. Its performance was checked with

Table 1. Tonometry Results [Mean ± SD (CV) [% Inaccuracy]]. Using a Bovine-Hemoglobin-Based Tonometry Fluid to Assess the Inaccuracy of pH, PO₂, and PCO₂, with a Single Ciba Corning 278 Analyzer. The pH Values Are Lot-Dependent and Only Indicate Instrument Imprecision.

<table>
<thead>
<tr>
<th>Level</th>
<th>pH</th>
<th>PCO₂</th>
<th>PO₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>pH = 7.200</td>
<td>P&lt;sub&gt;CO₂&lt;/sub&gt; = 70</td>
<td>P&lt;sub&gt;O₂&lt;/sub&gt; = 100</td>
</tr>
<tr>
<td>(n = 23)</td>
<td>7.210 ± 0.003 (0.043)</td>
<td>68.7 ± 1.2 (1.7) [98.1%]</td>
<td>99.1 ± 0.7 (0.7) [99.1%]</td>
</tr>
<tr>
<td>2</td>
<td>pH = 7.400</td>
<td>P&lt;sub&gt;CO₂&lt;/sub&gt; = 40</td>
<td>P&lt;sub&gt;O₂&lt;/sub&gt; = 70</td>
</tr>
<tr>
<td>(n = 20)</td>
<td>7.415 ± 0.002 (0.029)</td>
<td>39.5 ± 0.7 (1.7) [98.8%]</td>
<td>69.1 ± 0.5 (0.8) [98.7%]</td>
</tr>
<tr>
<td>3</td>
<td>pH = 7.600</td>
<td>P&lt;sub&gt;CO₂&lt;/sub&gt; = 20</td>
<td>P&lt;sub&gt;O₂&lt;/sub&gt; = 40</td>
</tr>
<tr>
<td>(n = 20)</td>
<td>7.607 ± 0.003 (0.042)</td>
<td>20.0 ± 0.3 (1.5) [100%]</td>
<td>39.3 ± 0.3 (0.7) [98.3%]</td>
</tr>
</tbody>
</table>
Evaluation of Blood-Gas Analyzer

A calibration gas mixture (5.0% CO₂, 12.0% O₂, and balance N₂) and buffer (pH 7.382 at 37°C) every 30 minutes and with a second calibration (slope) gas (10.0% CO₂ and 90.0% O₂) and buffer (pH 6.838 at 37°C) every 2 hours. Limits for the deviations from the calibration values were set by the manufacturer prior to this study and were 5.6% of the target value for PₐCO₂, 4.1% of the target value for PₐO₂, and 0.015 pH units for pH. (These limits, however, can be modified by the user in the market version of the C840.) When these calibration drift limits were exceeded, the instrument would automatically calibrate again. No patient samples were analyzed until the calibrations were again within these tolerance limits.

For quality control of pH, PₐCO₂, and PₐO₂, an aqueous quality control material was used. Every 24 hours, 3 different levels of this material were tested in duplicate. The same lot of each level of control was used throughout the study.

Blood Specimens

We used 325 random patient samples, both arterial and mixed-venous blood drawn for routine blood-gas analysis, in an attempt to obtain a wide range of values. Most of these blood samples originated from patients in the hospital’s intensive care units, cardiac catheterization laboratory, and pulmonary physiology laboratory. The study was performed in accordance with the revised Helsinki Declaration of 1983 and our institution’s Human Subjects Research Committee.

Clinical Trial Protocol

All blood samples were collected in 3-mL plastic syringes preheparinized with 100 to 200 USP units of lyophilized lithium heparin. Each sample contained at least 2 mL blood, and care was taken not to place any of the samples on ice during the preanalytical period. All patient samples were first analyzed with the C278 by regular blood-gas laboratory personnel and then with the C840 within 5 minutes by one of two designated staff members. If the difference between the two analyses was in excess of the ranges listed in Table 2, the sample analysis was repeated with the C840 and with the C278, provided there was adequate sample volume. The study was performed during a 2-week period and in accordance with AACC guidelines.

Statistical Methods

Measured values of all quality control and tonometered samples are expressed as mean ± SD and coefficient of variation expressed as a percent (CV%).

Measured values of quality control samples by the C278 for PₐCO₂ and PₐO₂ are also expressed by the following formula: (mean of the measured value/target value) x 100 = % inaccuracy (ie, % of target value).

Because the "true" blood pH, PₐCO₂, and PₐO₂ values for the patient samples are not known, we use the method of Bland and Altman to calculate the bias and imprecision for the C278 and C840. The bias is defined as the mean difference between measurements made on the C840 and the C278, and imprecision is defined as the value for 2 SD from this mean.

Evaluation Results

Corning 278 Performance

Table 1 summarizes the mean (SD) values, CV%, and % inaccuracies for the routine quality control of the C278.

Corning 840 Performance

Table 3 summarizes the mean (SD) values and CV% for the routine quality control of the C840. For Level 1, 2 additional samples (on Day 5 and Day 6) were analyzed because values were outside of the expected ranges and, therefore, n = 30. All other results were within the expected ranges listed by the manufacturer.

Clinical Trial

Figure 1 illustrates the imprecisions and biases for the C840 when compared to the C278 for pH, PₐCO₂, and PₐO₂.

Some data pairs were discarded due to clots in the blood sample (n = 1) and excessive calibration drifts in either instrument (n = 2).

Repeat analyses (for samples with differences exceeding the limits defined in Table 2) were performed on 18 of the 325 patient samples analyzed (18/325 = 6%). Analysis was repeated for 1 sample at PₐCO₂ < 70 torr, 6 samples at PₐCO₂ > 70 torr, 8 samples at PₐO₂ < 150 torr, 2 samples at PₐO₂ < 250 torr but > 150 torr, and 1 sample at PₐO₂ > 250 torr.

Experience

We found the C840 easy to operate, maintain, and troubleshoot, mostly because of the redesigned sample pathway.
and simplified hydraulics. The optional alphanumeric keyboard was useful for patient identification entry. The laser bar code scanner facilitated quality control range and identification entries. The throughput was fast in comparison to the C278.

The automated sampler left air bubbles in the syringe during aspiration. Although these bubbles were expelled quickly, some contamination of the remaining blood sample probably occurred. The new waste system, because of its location and mechanics, was accidentally triggered twice to falsely indicate "full," but our testing of later commercially available models suggests that this problem has been resolved. Also, on two separate occasions, we could not manually exit a wash cycle or initiate a calibration sequence. Consequently, we were forced to shutdown and then restart the system, which takes approximately 10 minutes. None of the previously stored data were lost during this procedure. Unfortunately, we did not have the opportunity to measure any expired air samples in this evaluation.

**Discussion**

We were one of five laboratories nationwide selected to participate in the clinical evaluation of a prerelease C840 analyzer. We found the analytical performance of the C840 comparable to that of the C278—not surprising because they use the same type of electrodes. For the PO₂ data (n = 325), the bias between the C840 and the C278 was essentially zero with an imprecision of ±9.0 torr for PO₂, ranging from 30 to 500 torr. When the PO₂ was < 150 torr (ie, in physiologic range), the bias was −0.17, with a smaller imprecision of only ±4.8 torr (n = 294). When the PO₂ was ≥ 150 torr, the bias was +2.0 with a larger imprecision of ±24.7 torr (n = 31). For the entire data set (ie, with the inclusion of the 18 repeat analyses), the bias was +0.26 with an imprecision of ±10.0 torr (n = 347) for the total range of PO₂ tested.

For the PCO₂ data, the bias between the C840 and the C278 was +1.8 torr with an imprecision of ±3.3 torr for the entire range tested. At PCO₂ levels > 70 torr (n = 17), the C840 showed a large positive bias (+6.5 torr with an imprecision of ±4.8 torr) when compared to the C278. However, at the more physiologic PCO₂ levels of ≤ 70 torr (n = 318), the bias was only +1.7 with an imprecision of ±3.0 torr. For the entire data set (ie, with the inclusion of the 18 repeat analyses) of PCO₂, the

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**Table 3. Daily Quality Control Results [Mean ± SD (CV %)] Using an Aqueous Solution.** To Assess pH, PCO₂, and PO₂ Measurements (n = 28) with the Ciba Corning 840 Analyzer.

<table>
<thead>
<tr>
<th>Level</th>
<th>pH (pH units)</th>
<th>PCO₂ (torr)</th>
<th>PO₂ (torr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1†</td>
<td>7.174 ± 0.002 (0.03%)</td>
<td>75.0 ± 1.10 (1.4%)</td>
<td>65.8 ± 3.6 (5.5%)</td>
</tr>
<tr>
<td>2</td>
<td>7.420 ± 0.004 (0.05%)</td>
<td>46.4 ± 0.64 (1.4%)</td>
<td>105.5 ± 3.4 (3.2%)</td>
</tr>
<tr>
<td>3</td>
<td>7.621 ± 0.003 (0.04%)</td>
<td>22.9 ± 0.40 (1.8%)</td>
<td>148.2 ± 4.4 (3.0%)</td>
</tr>
</tbody>
</table>

* Ciba Coming Certain® Elite  
† n = 30.
bias was slightly different, +1.7 with an imprecision of ±3.9 torr (n = 347) for the total range of \( P_{CO_2} \) tested. The pH bias was small (0.004 units with an imprecision of 0.014 pH units) for the patient data set and approximately the same (0.003 with an imprecision of 0.012 pH units) with the inclusion of the repeat analyses. (Adjustment of the C840 algorithms based on our findings, reduced the inaccuracies in subsequently manufactured units of the instrument. (Michael Layman, CIBA Corning Diagnostics, July 27, 1994: personal communication)

We observed minimal calibration drifts after the performance of maintenance and troubleshooting procedures such as clot removal, power shutdowns, and scheduled weekly deproteinizing and conditioning. We also detected minimal calibration drifts between each day of the study.

During the course of the 2-week evaluation, an entire calibration-gas cylinder was depleted, but we were unable to locate any external leaks in the system tubing or cylinder valves to explain why this had happened. The other four testing laboratories, however, did not experience this problem. (Robert Evans, CIBA Corning Diagnostics, July 28, 1994: personal communication) The manufacturer claims that the normal calibration gas consumption is one cylinder every 12-18 months. (Robert Evans, CIBA Corning Diagnostics, July 28, 1994: personal communication)

Under CLIA-88 guidelines, blood gas analyzers are classified as moderately complex instruments only if they do not require extensive operator intervention during the analytical process, including sample introduction. As a result, manufacturers have been designing instruments with automated processes for sample intake, calibration, and flushing of sample lines. However, a shortcoming of automatic aspiration sampling devices is the formation of air bubbles during sample aspiration, which can lead to sample contamination. Immediate evacuation of the air is essential to the maintenance of sample integrity to allow reproducible duplicate measurements of the same sample when indicated. This may increase the risk of biohazard exposure when the air is evacuated and the syringe re-capped. Therefore, we question the perceived benefit of automated samplers.

The integrated data management system for patient- and calibration-data storage should fulfill all of the CLIA-88 requirements for data management. However, a more sophisticated networking data management system would be more useful in a large volume laboratory with several blood gas analyzers. We would like to have used the Levey-Jennings charts option now available in the quality control software, but this feature was not available at the time of this evaluation. The ability to generate fully annotated Levey-Jennings charts is important because it allows laboratories to fulfill virtually all of the CLIA-88 regulations concerning instrument quality control.6

With the adoption of CLIA-88, laboratory patient records and quality control data, including maintenance and calibrations, must be stored for 2 years and be readily retrievable in a timely fashion. CLIA-88 also states that patient reports forms must include specific information, such as patient ID numbers, the date and time of test reports, person(s) performing the test, and an explanation if the sample is rejected. Although we only observed the capabilities of the integrated computer for storage and interface, the C840 should provide the means to comply with the CLIA-88 rules and regulations regarding data management.

We believe that the incorporation of computers into blood gas analyzers has improved laboratory efficiency. Less technologist time is now spent performing instrument maintenance or patient or quality-control data entries. Presumably, more time is now available for patient sample analysis and, therefore, faster reporting of results. The one disadvantage of the increased dependency on computers is that any system failure may lead to complete analyzer shutdown and prolonged analyzer start-up. To overcome this, we believe that each laboratory should have a minimum of two blood gas analyzers at all times, although our position remains controversial.9

Conclusions

The analytical performance of the C840 is comparable to the C278 analyzer, and its data storage and interface capabilities should help laboratories meet CLIA-88 requirements.

PRODUCT SOURCES

Blood gas analyzers:
278, Ciba Corning Diagnostics Corp, Medfield MA
840, Ciba Corning Diagnostics Corp, Medfield MA

Precision Gas Mixer:
192, Ciba Corning Diagnostics Corp, Medfield MA

Tonometer:
Equilibrator, RNA Medical, Acton MA

Tonometry Solution:
Equil, RNA Medical, Acton MA

Aqueous Quality Control Material:
Certain Elite, Ciba Corning Diagnostics Corp, Medfield MA

Syringes:
Arterial Blood Aspirator #478487, Ciba Corning Diagnostics Corp, Medfield MA
Quick A.B.G. #P4023, Marquest Medical Products Inc, Englewood CO

ACKNOWLEDGMENTS

We thank the Blood-Gas Laboratory staff at Stanford University Hospital for their technical assistance in this evaluation.

REFERENCES

Nonsurgical Airway Management: General Considerations and Specific Considerations in Patients with Coexisting Disease

Burkhard F Spiekermann MD, David L Bogdonoff MD, 
George S Leisure MD, and David J Stone MD

I. Introduction

In this review, we consider the patient with coexisting medical disease and the special considerations necessary for urgent or emergency airway intervention. We have limited our discussion to nonsurgical interventions and the more common clinical problems encountered in the emergency room and the intensive care unit. We do not review airway management in the traumatized patient because this has been extensively reviewed.1,2

II. General Considerations

A. Local Anesthesia
B. Sedation
C. Muscle Relaxants
D. Other Pharmacologic Adjuvants
E. Confirmation of Intubation

III. Specific Considerations in Coexisting Disease

A. Coronary Artery Disease
B. Cardiopulmonary Arrest
C. Drug Overdose
D. Bronchospasm
E. Chronic Obstructive Airway Disease
F. Neurological Diseases
G. Upper Gastrointestinal Bleeding
H. Hemoptysis
I. End-Stage Liver Disease

IV. Infectious Risk to the Respiratory Care Practitioner

V. In Summary

Introduction

The practitioner is often asked to provide airway management for patients with coexisting medical problems. The method chosen for management of the airway depends heavily on the urgency of the situation, the practitioner’s expertise, and the special concerns regarding the patient’s underlying medical condition. The critically ill patient should be cared for by the most skilled person available, which in many cases is the respiratory care practitioner.

Dr Spiekermann is Assistant Professor of Anesthesiology, Dr Bogdonoff is Associate Professor of Anesthesiology and Surgery, Dr Leisure is Assistant Professor of Anesthesiology, and Dr Stone is Associate Professor of Anesthesiology and Neurological Surgery—Department of Anesthesiology, University of Virginia Health Sciences Center, Charlottesville, Virginia.

The authors have no financial interest in any of the commercial products mentioned in this paper.
Elective and risk associated intubated have been observed. Proper airway management depends on the technical skills of the practitioner and on his or her ability to anticipate the potential effects of specific interventions on various organ systems, especially the cardiovascular system, the central nervous system, and the respiratory system. This is crucial in the patient with coexisting medical problems, to avoid undesirable adverse physiologic sequelae. Flexibility of technical and pharmacologic approaches must be maintained in the treatment of each patient.

Emergency airway management differs from controlled elective intubation in the operating room. We believe that safety mandates a conservative approach to the use of sedatives and muscle relaxants, and, in our practice, we try to maintain the patient’s ability to breathe spontaneously whenever feasible. It must always be remembered that, after relaxants have been given, one may be left with a patient who cannot be ventilated by mask and whose trachea cannot be intubated. When associated with hypoxemia, regurgitation, aspiration and esophageal intubation, airway manipulation carries a significant risk of cardiopulmonary arrest. Topical airway anesthesia and a knowledgeable assistant may be all that is needed to facilitate intubation. A higher complication rate during emergency airway management involving drug-facilitated intubations as compared to awake intubations has been reported (23 versus 13%).

Local Anesthesia

In unresponsive patients, airway anesthesia is not usually required. In awake, cooperative patients the ability to block sensation from the oropharynx is often the only intervention needed to facilitate intubation. This may be achieved with topical anesthesia, eg, 10% lidocaine spray, viscous lidocaine, or nebulized lidocaine. However, topical anesthesia does not anesthetize the mechanoreceptors at the base of the tongue, which are responsible for much of the deep-pressure discomfort during laryngoscopy. This discomfort can be reduced or abolished with bilateral glossopharyngeal nerve blocks. The block is easily performed by injecting 5 mL of 1% lidocaine 5 mm lateral to the edge of the tongue where it forms the palatoglossal arch. (Fig. 1) This blockade of noxious sensory stimuli is effective in providing lasting, complete analgesia for awake intubation and can effectively blunt the hypertensive response and the adverse intracranial pressure dynamics that can be associated with laryngoscopy and intubation. Prior to intubation, the larynx, vocal cords, and upper part of the trachea can be anesthetized during laryngoscopy with additional lidocaine using an LTA (laryngotracheal applicator) kit. In our experience, transtracheal injection of 2-3 mL of 1% lidocaine through the cricothyroid membrane with a 23-gauge needle provides excellent anesthesia of the larynx below the vocal cords. However, anesthesia of the glottis and trachea may increase the patient’s risk of aspiration.

Sedation

If local anesthesia is not feasible, is contraindicated, or is inadequate, intravenous sedation is often used as an adjunct for airway management. No single drug is appropriate for all settings. As with the technical aspects of intubation, drug selection needs to be tailored to the specific needs of the patient and should only be given by a person knowledgeable of drug actions and potentially serious side effects. A full discussion of anesthetic pharmacology is beyond the scope of this review but can be found in textbooks of anesthesia. All the doses mentioned are intended for intravenous use.

Narcotics are effective in reducing the pain associated with airway instrumentation. They also suppress cough reflexes and provide some degree of sedation. Their ability to blunt the laryngeal closure reflex, however, may predispose the patient to the risk of pulmonary aspiration. Fentanyl is often used in small increments of 25-50 mg, and the more rapidly and shorter-acting agent alfentanil has also proven to be useful. The availability of an antagonist such as naloxone provides an added safety feature when narcotics are used. Narcotics are powerful respiratory depressants, especially when used in combination with other sedatives, and should therefore be titrated carefully, particularly if maintenance of spontaneous ventilation is desired.

Benzodiazepines are effective in producing amnesia and reducing anxiety but their sedative action is not as predictable.

![Fig. 1. Glossopharyngeal nerve block. Injection of 5 mL of 1% lidocaine lateral to the edge of the tongue into the palatoglossal arch. (Original drawing courtesy of AM Woods.)](image)
as that of other drugs. In combination with narcotics, they may create dangerous degrees of sedation, with a higher incidence of apnea and hypoxia than either drug alone. Midazolam has replaced diazepam as the most useful benzodiazepine for acute parenteral use and should be carefully titrated in small doses (0.5-1 mg).

The hypnotic agents are a diverse group of drugs; Those employed in the acute care situation are chosen for their rapid onset and relatively short duration of action. In high doses, they can be used to rapidly produce an unconscious patient, and with incremental small doses, optimal sedation can be achieved. When used in small, sedative doses, individual differences among these drugs are not clinically detectable. (Dosages are further discussed in the section Coronary Artery Disease.) The ultrashort-acting barbiturates (eg, thiopental) and propofol must be used cautiously in hypovolemic patients and in patients with decreased cardiac function. Venodilation and myocardial depression by these drugs can cause profound hypotension leading to myocardial ischemia and arrest. Barbiturates do not provide analgesia, and patients often respond to painful stimuli with tachycardia and hypertension. Propofol, a lipid soluble isopropyl phenol, has depressant effects on the cardiovascular system similar to the barbiturates. It can be titrated to achieve adequate sedation and has an even shorter duration of action than thiopental. Etomidate, an imidazole derivative, provides more cardiovascular stability and therefore is often the agent of choice in patients with hypovolemia, poor myocardial function, or ischemic heart disease. It should not be used for long-term sedation (unless corticosteroid replacement is ensured) because it blocks the function of the adrenocortical enzyme 11-beta hydroxylase, thereby interfering with cortisol synthesis. Ketamine, a phencyclidine derivative, is the only hypnotic with a profound analgesic effect. In contrast to other hypnotic drugs mentioned, respiratory drive is maintained and protective airway reflexes are left intact when ketamine is used in small doses. The cardiovascular system is stimulated due to sympathetic activation, unless the reserve of the patient’s adrenergic system is already exhausted. Ketamine may be chosen for patients with symptoms of congestive heart failure, hypovolemia, or symptomatic hypothyroid disease. Caution should be exercised in using ketamine in patients with intracranial pathology because it can raise intracranial pressure because of its direct cerebral vasodilator action.

The use of any sedative demands attention to the possibilities of vomiting and aspiration, complete upper airway obstruction, apnea and cardiovascular collapse.

Muscle Relaxants

Muscle relaxants are used to facilitate intubation by producing skeletal muscle relaxation even to the point of complete respiratory muscle paralysis. They should be used only with extreme caution and only by health-care providers knowledgeable in their pharmacology and with much experience in airway management. The choice of a specific muscle relaxant depends on the pharmacologic profile of the agent and the clinical situation.

Succinylcholine, the only available depolarizing agent, has the most rapid onset and shortest duration of action of all the muscle relaxants and does not require pharmacologic reversal. These qualities may make it the most useful muscle relaxant in an emergency situation. Metabolism of succinylcholine is relatively unaffected by hepatic and renal disease. Late administration (≥24 hours after the insult) to patients with derangement injuries, such as spinal cord injuries or serious strokes, or to patients with skeletal muscle injuries, as associated with burns or massive trauma, is contraindicated due to the risk of hyperkalemia and the potential for subsequent cardiac arrhythmias and arrest.

Non-depolarizing muscle relaxants all have a longer duration of action and a slower speed of onset than succinylcholine. Selection is dependent on the differences in duration, metabolism, and side effects primarily on the cardiovascular system. Because of their neutral cardiovascular profile and short duration of action (30-60 min), rocuronium, vecuronium, and atracurium are used frequently. A full discussion of these drugs can be found in any current textbook of anesthesia.

Other Pharmacologic Adjuvants

Intravenous glycopyrrolate, an anticholinergic agent, is useful in decreasing airway secretions and thereby improving visualization during conventional and fiberoptic intubation. Its antiallalogue effect may also improve the anesthetic effect of nebulized or topical lidocaine. Other beneficial effects include the attenuation of bronchospasm and improvement of pulmonary function in obstructive airway disease. The main side effect of glycopyrrolate is occasional mild tachycardia. Unlike atropine and scopolamine, it has no effects on the central nervous system.

Intravenous lidocaine is effective in decreasing airway reactivity and airway reflexes. It has been shown to decrease the hemodynamic response to intubation similarly (but less effectively) than a sedative dose of narcotics, but may not be quite as reliable as aerosolized lidocaine in this regard. Lidocaine also reduces elevated intracranial pressure, without adverse effects on hemodynamics.

In our opinion, droperidol, which is commonly used in the operating room for its sedative effects, is rarely helpful in an emergency situation due to its lack of reversibility, alpha-receptor blockade, long half life, and high incidence of dysphoric reactions.

Confirmation of Intubation

Tracheal placement of the endotracheal tube must be verified without delay. Furthermore, the classic physical signs of
successful endotracheal intubation may be unreliable at times.\textsuperscript{21}

Capnography, the detection of CO\textsubscript{2} in expired gas, is commonly accepted as the gold standard for confirmation of proper tube placement. Battery-powered, portable capnometers are available for use outside the operating room.\textsuperscript{22} These employ infrared detectors and are able to detect CO\textsubscript{2} concentrations of 2\% or greater (Fig. 2). A colorimetric device that attaches to the end of an endotracheal tube is also on the market. (Easy Cap, Nellcor Inc, Hayward CA; Fig. 3) This device changes color when CO\textsubscript{2} is present in the expiratory gases.\textsuperscript{23-25} However, the initial detection of CO\textsubscript{2} is not 100\% reliable. There are several circumstances in which CO\textsubscript{2} is transiently detectable with esophageal intubation. Prolonged mask ventilation or recent ingestion of carbonated drinks or antacid may produce sufficient CO\textsubscript{2} accumulation in the stomach so that CO\textsubscript{2} measurement during the first few breaths might lead one to assume correct tube placement.\textsuperscript{26} In some situations, CO\textsubscript{2} is not detectable even with proper tube placement. These include absent or low cardiac output, severe airway obstruction or bronchospasm, and malfunction of the detection system.

Physical examination alone to assure proper tube placement is most commonly used in emergency situations. Direct visualization of the tube in the glottic opening during intubation is one of the most reliable physical signs. This can be repeated once the tube is secured. The view can be facilitated by posterior displacement of the tube towards the palate.\textsuperscript{27,28} Symmetric bilateral chest movement with manual ventilation may be helpful, but can be misleading especially in obese patients, patients with large breasts, or patients with a ‘barrel’ chest from chronic obstructive pulmonary disease.\textsuperscript{29} The presence of bilateral breath sounds and absence of sounds over the epigastrium does not guarantee proper tube placement. The ‘feel’ for compliance of the reservoir bag, condensation around the endotracheal tube during exhalation, and palpation of the inflated endotracheal tube cuff at the sternal notch can be helpful contributory physical findings, especially when used collectively and in conjunction with a high index of suspicion and continued observation.\textsuperscript{30}

A simple device that reliably differentiates esophageal from tracheal intubation has been described.\textsuperscript{31,32} A 60-mL syringe is connected to a 15-mm right-angle connector that attaches to the end of the endotracheal tube. (Fig. 4) Rapid aspiration through the syringe immediately after intubation results in resistance due to collapse of unsupported esophageal mucosa against the end of the tube in the case of esophageal intubation. This is not the case with proper endotracheal tube placement because the trachea is held open by C-shaped cartilages. A modification of this esophageal detector device, consisting of an inflatable bulb connected via a 15-mm plastic fitting to the endotracheal tube was recently studied in 500 patients.\textsuperscript{33} The results showed 100\% sensitivity and specificity (using capnography as a reference) with the conclusion that the esophageal detector device is a useful diagnostic tool if capnography is not available. There are reports, however, that this device may fail to confirm proper tracheal intubation in patients with airway or tube obstruction, tracheomalacia, severe asthma, and morbid obesity.\textsuperscript{34-36}

If a fiberoptic bronchoscope is available, visualization of tracheal rings and carina is another reliable way of confirming intubation. However, a bronchoscope is often unavailable in emergency situations outside the operating room.

The pulse oximeter can be a useful monitor of proper oxygenation after successful intubation but should not be relied upon to confirm tube placement. Desaturation, especially when...
the patient has been adequately mask ventilated with 100% oxygen prior to intubation, may be a relatively late sign of improper intubation. A chest radiograph can also confirm appropriate tube placement but may not be immediately available.

**Specific Considerations in Coexisting Disease**

**Coronary Artery Disease**

Patients with ischemic heart disease often require intubation for hypoxemia from pulmonary edema or for institution of controlled ventilation when the decompensated heart is unable to meet the demands associated with the increased work of breathing. The goal in managing the airway is to avoid further disturbance of the balance between myocardial oxygen supply and demand and to avoid any drugs that would further depress the failing myocardium. As stated previously, the urgency of the situation and the status of the patient dictates the approach to the airway.

Heart rate and blood pressure are major determinants of myocardial oxygen requirements. The faster the heart rate and the higher the pressure the heart must work against, the higher the myocardial oxygen consumption. Airway manipulation is stimulating and can significantly increase both systemic and myocardial oxygen consumption, the latter primarily by eliciting tachycardia and hypertension.37 Blunting undesirable hemodynamic responses mediated through the sympathetic nervous system is, therefore, a goal during laryngoscopy, and adjunctive drugs and/or topical anesthesia are required in most patients.39 It is equally important to avoid hypotension, which may compromise O2 delivery to the myocardium by decreasing coronary perfusion pressure.

If intubation can be approached without extreme urgency, intubating conditions and drug selection can be planned carefully to improve patient comfort and to meet the delineated goals. Ketamine is the only sedative-hypnotic that stimulates the sympathetic nervous system. This may be beneficial if severe congestive heart failure is present but can be detrimental to the ischemic heart because it can cause tachycardia in the high risk patient.40 At low doses, up to 1 mg/kg, it is not likely to cause hypotension. Etomidate in doses of 0.1 mg/kg is virtually devoid of hemodynamic side effects and has mild coronary vasodilating properties.41 Thiopental in small doses (25-75 mg) causes brief unconsciousness but, due to its venodilating and myocardial depressant effect, should be used with caution in the severely compromised patient.42 Propofol has similar properties to thiopental but may be even more likely to cause hypotension because it is dilates both arteries and veins.43

Narcotics do not depress the myocardium and, therefore, do allow for excellent hemodynamic stability.44-46 The short-acting agents fentanyl and alfentanil are effective in blunting heart rate and blood pressure responses to intubation. When these agents are used in combination with benzo diazepines, baroreceptor reflexes are attenuated and undesirable hypotension is possible.47-49

The safety of benzodiazepines, even in large doses, has been documented in patients with ischemic heart disease.50 They are useful to allay anxiety and to decrease the response to tracheal intubation. As already mentioned, care must be taken when these drugs are used with opioids.

Intravenous lidocaine in a dose of 1-2 mg/kg given 3 minutes prior to intubation is more effective than topical administration to the airway and in some studies it compares favorably to small doses of narcotics.51-53 An added benefit is that lidocaine decreases the incidence of ventricular arrhythmias associated with intubation.53

Other cardiovascular drugs may be useful in maintaining a favorable oxygen-supply-to-demand ratio in the myocardium. Nitroprusside in a dose of 1-2 µg/kg given 15 seconds prior to laryngoscopy can prevent increases in blood pressure.54 Nitroglycerin is effective when administered nasally in a dose of 0.75 mg given 60 seconds before intubation55 and is also a coronary vasodilator that should improve myocardial blood flow. Some evidence suggests that patients have a lower incidence of myocardial ischemia during intubation when nitroglycerin is infused at 1 µg/kg/min.56 Sublingual nifedipine (10 mg) given 10 minutes prior to intubation also blunts hypertensive responses to intubation, but excessive hypotension has been reported.58 The drugs mentioned do not block the increase in heart rate associated with airway manipulation, a major cause of myocardial ischemia in this patient population.59-61

Esmolol, an ultrashort-acting cardioselective beta blocker is very effective in blocking the tachycardia associated with intubation. The usual recommended dose is 0.5 mg/kg, but more recent studies have shown that higher doses of 100 to 200 mg given approximately 90 seconds prior to intubation are most effective and have only minimal effect on blood pressure.62-64 When compared to intravenous lidocaine and fentanyl, esmolol was the only drug in one study that consistently prevented tachycardia and hypertension during laryngoscopy.63 It is ideally suited for use in combination with drugs that block hypertensive responses but is relatively contraindicated in patients on inotropic support with severely compromised ventricular function and in patients with bradycardic rhythm disturbances. Bronchospasm is a potential problem with beta blockers but esmolol is the drug of choice in this regard.65

The optimal approach to the cardiac patient often includes the careful use of hypnotic and sedative drugs in conjunction with local anesthesia of the airway, as described. In the face of reduced cardiac output, caution with respect to intravenous and topical drug dosing is necessary. The practitioner must be especially patient before re-dosing because each dose takes effect more slowly than in patients with normal cardiac output.

The technical aspects of intubation are important in reducing the level of stimulation to the patient. Direct laryngoscopy of short duration (ideally less than 15 seconds) is beneficial in minimizing the magnitude of circulatory changes.66 It has
been suggested that fiberoptic techniques are less stimulating, but one study of oral fiberoptic intubation did not result in any major difference compared with direct laryngoscopy. The use of a lightwand device may cause less hypotension when compared to standard laryngoscopy. A relatively easy, blind oral or nasal intubation technique may be used to avoid the additional stress associated with laryngoscopy in dyspeptic and tachypneic patients.

We find it helpful to have an assistant watch closely for changes in vital signs and for signs of myocardial ischemia. Changes may occur rapidly and could be missed initially when the practitioner is distracted by the demands of intubation.

**Cardiopulmonary Arrest**

Although cardiac arrest usually mandates rapid intubation to provide adequate oxygenation and airway protection, some patients who can be resuscitated rapidly may not require an endotracheal tube and can be managed with basic airway maneuvers to alleviate upper airway obstruction.

Intubation usually does not require any pharmacologic aid in the unconscious patient. Airway management in a patient receiving chest compressions may be technically difficult, and often compressions must be briefly interrupted until the airway is secured. Confirmation of proper tube placement is also difficult in this situation. Auscultation of breath sounds and other physical signs indicating adequate ventilation are hard to discern during the ongoing resuscitation effort. In addition, poor cardiac output may make CO2 detection with capnometry impossible or inaccurate, although if CO2 is present in the expired gases during cardiac arrest, it is a good prognostic indicator for patient survival. The esophageal detector device may be useful in this situation to confirm endotracheal tube placement if capnometry is not available or no CO2 can be detected on the capnogram.

**Drug Overdose**

A patient with acute drug overdose may require tracheal intubation to protect the airway (especially during gastric lavage), to allow suctioning of secretions, and to provide mechanical ventilation. These patients are at risk for aspiration pneumonitis because airway reflexes are generally obtunded. The approach to the airway depends on the level of consciousness of the patient. In our experience, the unresponsive patient can be intubated with simple oral direct laryngoscopy with maintenance of cricoid pressure. The completely alert patient, on the other hand, may need only to be observed for signs of deterioration of mental and respiratory status. The greatest challenge is the agitated, uncooperative patient who often cannot be intubated without further sedation and or muscle relaxation. It is important to note that hypoxia may be the cause of agitation and may be alleviated by simply increasing the fraction of inspired O2. In our experience, blind nasal intubation is often successful in these situations. The endotracheal tube whistle as an adjunct to blind nasal tube placement has been described. This device attaches to the endotracheal tube adapter and produces whistle sounds with spontaneous respirations, enhancing the detection of air movement, which may allow for easier intubation. However, repeated attempts at blind nasal approaches can cause epistaxis, vomiting, and aspiration, and succinylcholine-assisted oral laryngoscopy may be safer if initial trials are unsuccessful. Airway anatomy and ease of intubation should be assessed—to the extent possible—before any muscle relaxant is given because relaxants are no guarantee of establishing a successful airway. Fiberoptic, lighted stylets and other intubating techniques may be safer than techniques requiring muscle relaxants if difficult intubation is anticipated.

**Bronchospasm**

Asthmatic patients with acute bronchospasm are generally treated medically but at times may need endotracheal intubation for acute respiratory failure. Indications for intubation include failed medical therapy with progressively deteriorating mental status, respiratory fatigue, and deterioration of arterial blood gas values. Intravenous glycopyrrolate, 0.4-1 mg, blocks vagal reflex pathways to the bronchial tree and is useful if given prior to intubation. Although endotracheal intubation allows for mechanical ventilation in order to decrease the work of breathing and increase FIO2, it is a powerful airway irritant and may aggravate bronchospasm. Other pharmacologic adjuvants to decrease airway irritability during intubation include intravenous lidocaine and narcotics. If sedation is not contraindicated, ketamine is a useful drug because it can produce bronchodilation and has no significant respiratory depressant effect when used in small doses.

Stimulation of the carina with the endotracheal tube and right main stem intubation should be avoided because these may trigger severe bronchospasm. If the clinical situation worsens after intubation, proper tube position should be carefully checked, if necessary with a chest radiograph. On occasion, positive end-expiratory pressure and muscle relaxants may be tried when the situation is life-threatening.

**Chronic Obstructive Airway Disease**

Exacerbations of COPD most commonly occur when the patient develops a pulmonary infection, as evidenced by increased sputum production and sputum purulence. The work of breathing required to maintain adequate oxygenation may become so high that more aggressive airway intervention is needed. Continuous positive airway pressure via a tight fitting mask may initially be tried, but often endotracheal intubation and positive pressure ventilation is required to maintain adequate oxygenation and prevent respiratory acidosis.
Intubation requires considerations similar to those in patients with asthma. Sedatives should be given carefully because some of these patients already have a compromised hypoxic ventilatory drive. Local airway anesthesia in the cooperative patient may be a safer alternative. Although an FIO2 of 1.0 may result in some degree of hypercarbia, actual respiratory arrest as a result of oxygen administration is uncommon. The barrel chest of COPD makes auscultation to confirm tube placement difficult, and other signs may have to be relied upon to assure proper intubation. Ventilation should be performed with large tidal volumes and a slow rate to minimize the likelihood of turbulent airflow through airways and to provide sufficient time for complete exhalation to occur so that auto-PEEP is avoided.

**Neurological Diseases**

Patients with nontraumatic neurologic diseases may require airway intervention for airway protection and provision of adequate oxygenation or heavy sedation or therapeutic hyperventilation to control intracranial pressure. Common clinical situations include massive strokes, seizures, intracranial hemorrhage from ruptured aneurysms, tumors, infection, or other intracranial pathology.

The effective approach to the patient with intracranial disease requires an understanding of the patient's risk factors, most importantly the issue of brain swelling and concomitant increase in intracranial pressure (ICP). ICP is directly related to total intracranial volume, which consists of brain tissue, cerebrospinal fluid, and cerebral blood volume. Cerebral blood flow is held constant in the uninjured brain with a mean arterial pressure between 50 and 150 mm Hg due to the phenomenon of cerebral autoregulation. Autoregulation may be compromised with cerebral pathology, and a rise in systemic blood pressure during laryngoscopy can translate into acute ICP elevations compromising cerebral oxygenation, worsening cerebral hemorrhage, or causing catastrophic cerebral herniation. Precise control of blood pressure during laryngoscopy is important and can be achieved with gentle and skillful manipulation during intubation in conjunction with pharmacologic intervention. The barbiturates, propofol, and etomidate are effective sedatives for ICP reduction. Their mechanism of action is mainly cerebral vasodilation secondary to a decrease in cerebral metabolic rate. Lidocaine as an intravenous adjunct is also effective in this regard. Ketamine, a cerebral vasodilator, should be avoided. Often complete muscle paralysis is necessary to avoid coughing and 'bucking' the endotracheal tube, which may lead to pronounced ICP elevations. Succinylcholine has been shown to mildly raise ICP in patients with reduced intracranial compliance, but in an urgent situation this is clearly less of a concern than other potential problems facing the patient if immediate airway control is not established. The possibility of succinylcholine-induced hyperkalemia in a patient with a subacute neurologic deficit needs to be carefully considered.

Other causes of increased ICP that can be controlled with appropriate airway management are hypercarbia, hypoxia, and patient position. The P<sub>aco2</sub> and cerebral blood flow are linearly related in the P<sub>aco2</sub> range of 20-80 torr. Hypocarbia causes cerebral vasoconstriction: Cerebral blood flow at a P<sub>aco2</sub> of 20 torr is roughly 50% of its normal value. Hyperventilation to maintain the P<sub>aco2</sub> between 25 and 30 torr has been a mainstay of ICP control in the acute setting. A P<sub>aco2</sub> of less than 50 torr is a powerful cerebral vasodilator, making it necessary to provide adequate oxygenation in conjunction with hyperventilation. Avoidance of the head-down position and any pressure on the vascular structures of the neck is important. Venous congestion or any impedance to cerebral venous outflow will increase ICP by increasing cerebral blood volume.

**Upper Gastrointestinal Bleeding**

Massive gastrointestinal bleeding with passive regurgitation or active vomiting of blood into the oropharynx can cause airway obstruction and impair the ability to ventilate. It may also make visualization of the airway structures impossible, thereby complicating intubation attempts. The definitive initial approach involves removal of blood from the airway and provision of adequate ventilation and oxygenation. Adequate suctioning capability is mandatory. In our experience, blind passage of an endotracheal tube into either the esophagus or trachea is a reasonable initial step if definitive visualization is impossible. If placed in the esophagus, the tube diverts the blood flow from the intestinal tract and may allow for better exposure of the laryngeal structures. Placement in the trachea protects the airway from further blood aspiration.

Pharmacologic intervention, including skeletal muscle paralysis, may sometimes be required to rapidly gain control of the airway. Paralysis stops active vomiting but does not prevent bleeding from the esophagus. It completely eliminates any protective airway reflexes and may convert a precarious situation into a life-threatening one, if intubation is unsuccessful. Cricoid pressure should be maintained until proper tube placement is confirmed.

**Hemoptysis**

Upper-gastrointestinal-tract bleeding and hemoptysis may be initially indistinguishable in a patient with respiratory distress, and the first approach is the same, namely, removing blood from the upper airway to provide adequate oxygenation and ventilation. Most commonly, hemoptysis is secondary to carcinoma of the lung but other causes include infection, bronchiectasis, and cystic fibrosis.

Uncontrollable hemorrhage from the respiratory tract carries a high incidence of mortality. Rapid identification of the affected lung is desirable so that the unaffected side can be isolated via endobronchial intubation. Bronchial blockers and double-lumen tubes may not be available in the emergency
infecting diseases from exposure to patients’ blood and body secretions and aerosolized respiratory pathogens. Often the patient’s history is unknown, and it is wise to approach each patient as a potential source of disease. The implementation of Universal Precautions for blood and body fluids is essential to prevent exposure to potentially infectious material. This includes protective barriers—gloves, gown, mask and goggles—and extreme caution when handling sharp items and needles. Needles should not be recapped because this is a major source of accidental needle sticks, and containers for the disposal of sharp devices should be readily available. Often this is forgotten in emergency situations and health-care workers involved in the resuscitation effort can be victims of such oversight. Personnel with exudative skin lesions should refrain from direct patient care, and equipment for cardiopulmonary resuscitation should be used that obviates the need for mouth-to-mouth resuscitation.

The most worrisome infectious agents are the HIV virus, hepatitis B, non-A, non-B hepatitis, and hepatitis C. Both HIV and hepatitis are bloodborne infections that may also spread by intimate personal contact. Hepatitis C is the prime agent involved in transfusion-associated hepatitis. The prevalence of drug resistant tuberculosis is also increasing in the immunocompromised HIV-infected patient, and the use of masks may serve to diminish the inhalation of this respiratory pathogen. So far there have been no known instances of transmission of bloodborne pathogens by aerosols (10–100 μ), and no virus particles have been found to be present in aerosols.

As of 1991, at least three health-care workers had developed AIDS following well-documented exposure to HIV-infected blood and at least 20 additional health-care workers had seroconverted to HIV after documented percutaneous injury or mucous membrane or skin exposure to blood. Through 1992, this number grew to 33, with 69 other cases strongly suspected of being related to occupational exposure. Oral mucosa contains a large number of Langerhans cells, the cells most readily infected by HIV. It is theoretically possible that exposure of the oral mucous membranes to HIV-infected material can lead to transmission of the HIV virus. Most documented seroconversions were secondary to hollow needle stick injuries with contaminated needles.

One study has shown that approximately 25% of the medical interns and residents studied had been exposed to HIV-infected blood during the 12 months preceding the study. The subjects were working in hospitals in which approximately 15% of the patients were infected with HIV. The risk of seroconversion after percutaneous injury is thought to be about 1 in 250. With the increasing spread of AIDS with no successful therapy in sight and the potentially serious sequelae of hepatitis infection, strict adherence to observance of Universal Precautions for blood and body fluids is mandatory and the wisdom of such adherence should be self-evident. Studies have consistently shown that most

End-Stage Liver Disease

Liver transplantation is becoming an increasingly frequent option for patients in hepatic failure, and more patients in hepatic coma require ventilatory support in intensive care units. End-stage liver disease is often due to cirrhosis resulting from alcohol abuse or viral infections. Other causes include cancer or autoimmune, metabolic, and biliary diseases. End-stage liver disease affects all other organ systems of the body, and many factors need to be taken into consideration when managing the airway.

Hepatic encephalopathy is often associated with increased intracranial pressure and ICP precautions should be followed when intubation is required, as discussed previously. The patient in hepatic coma is unable to protect the airway, which mandates intubation with a cuffed tube. Hyperventilation may be required to decrease ICP. Coagulopathy is almost always present in the cirrhotic patient, and, therefore, blind or fiberoptic nasal intubation may be contraindicated. A careful approach to instrumentation of the airway is necessary because rupture of esophageal varices from inadvertent esophageal intubation can cause dangerous bleeding. Ascites causes increased intra-abdominal pressure and predisposes the patient to a significant risk of aspiration, warranting cricoid pressure. Alcoholic cardiomyopathy is often present in the alcoholic patient with end-stage liver disease and care must be taken when administering drugs with myocardial depressant properties for facilitating intubation. These patients are also prone to develop hypoxia if an airway cannot be established rapidly. Atelectasis secondary to ascites and pleural effusions, a hyperdynamic circulation with pulmonary arteriovenous shunting, and portopulmonary venous anastomoses are all thought to be responsible for decreased arterial oxygen saturation. In the cirrhotic patient, cumulative drug effects are likely if the liver disease is so severe that drug metabolism is slowed. In the patient with hepatorenal syndrome, careful attention must be given to determining which drugs depend on renal excretion.

Infectious Risks to the Respiratory Care Provider

Health-care workers who provide airway management in the emergency situation may be at considerable risk of
skin and mucous membrane contact can be prevented with appropriate protection and meticulous technique.  

In Summary

Airway management in emergency situations in patients with significant coexisting medical diseases demands an understanding of the underlying physiologic problems and expertise and flexibility in the technical aspects of intubation. The overall goal is to secure the airway in an expedient fashion without further aggravating the ongoing disease process. This may involve the use of topical airway anesthesia alone, intravenous sedation, or complete anesthesia with muscle relaxation. The choice of drugs is influenced by their pharmacologic profiles and the patient's medical condition. With the wide variety of technical and pharmacologic options available today, it is possible to meet this goal in most clinical settings.

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New Avenues of Asthma Therapy

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Introduction

In view of the large number of asthma patients, which is apparently increasing each year, it is not surprising that many pharmaceutical companies are actively engaged in asthma research. The major focus is no longer on agents for control of acute bronchospasm but rather on compounds to prevent these episodes. The recognition of inflammatory changes that underlie the asthmatic syndrome has drawn attention to the mediators released that are bronchoactive. The extensive studies of histamine, occupying most of this century, fixed attention on human basophils and mast cells as mediator sources. However, the relative ineffectiveness of antihistamines in asthma therapy indicated strongly that other mediators were at least of equal importance.

Emphasis in recent years has shifted to nonpreformed mediators, that is compounds formed in response to inflammatory or allergic stimuli. These can be derived from basophils, mast cells, macrophages, eosinophils, and lymphocytes. What was originally described (1938) as slow-reacting substance of anaphylaxis (SRS-A) was found to be a mixture of bronchoconstrictor arachidonic acid metabolites called leukotrienes. These are lipid compounds, biosynthesized upon release of arachidonic acid from cell walls and other depot sources. Activation of an enzyme called phospholipase A₂ was shown to be pivotal in the liberation of arachidonic acid. Another bronchoactive lipid, termed platelet activating factor (PAF), was similarly shown to depend upon phospholipase A₂ for its release from cell-membrane lipid stores. The effectiveness of corticosteroids in asthma prevention can be attributed in part to their inhibiting effect on phospholipase A₂ activation and probably on its formation as well.

The study of inflammatory mediators has revealed an increasing number and variety of compounds that may contribute to the pathogenesis of asthma. Some 25-30 of these substances may be implicated in bronchial inflammation, posing an almost infinite number of potential attack points for researchers. This is the predicament now faced by pharmaceutical companies—identifying the most promising possibilities for drug intervention.

It is important to note that the action of corticosteroids in asthma therapy is also multifactorial. The overall suppression of protein synthesis by glucocorticoids accounts for the reduction of many reaction sequences that form inflammatory compounds. However, this action is indiscriminate, and other biosynthetic processes, such as growth and repair, are similarly inhibited. The retardation of growth is a principal hazard in the use of steroids in asthmatic children. Other undesirable actions, such as adrenal suppression, osteoporosis, and hematologic changes, demonstrate that prolonged steroid regimens are far from ideal. If the more noxious mediators can be singly identified, then agents that suppress mediator formation or occupy mediator bronchial-receptor sites may provide safer and more effective approaches to asthma control.

In this discussion, I focus primarily on compounds inhibiting lipid mediators and lymphocyte-derived cytokines. Another important line of investigation involves the development of agents designed to augment the intracellular action of cyclic adenosine monophosphate (cAMP), which favors the relaxation of bronchial smooth muscle.

Agents Targeting Leukotrienes

The effects of so-called cysteiny1 leukotrienes, which are designated as LTD₄, LTE₄, and LTF₄, mimic the pathologic changes seen in asthma. They have been shown to be produced by eosinophils, mast cells, basophils, macrophages, and monocytes. The role of cysteiny1 leukotrienes in asthma has been summarized in a recent review. Cysteiny1 leukotrienes cause bronchoconstriction, increase capillary permeability, and are potent mucus secretagogues. A large body of evidence has accumulated to demonstrate increased production of leukotrienes in asthmatic patients. For example, bronchoalveolar lavage fluid from a majority of asthmatic volunteers was shown to contain LTE₄, whereas control subjects showed none.

Therapeutic intervention aimed at leukotrienes has followed two main approaches: (1) competitive antagonists have been synthesized to block cysteiny1-leukotriene receptors, and (2) inhibitors of the enzyme 5-lipoxygenase have been...
developed to reduce leukotriene synthesis. To date, there have been about six reports of the use of a leukotriene-receptor antagonist in clinical asthma. The first appeared in 1981, using an agent (FPL-55712) that was only weakly effective. Subsequent studies have employed compounds named verlukast, tomelukast, and MK-571. The most potent antagonist appears to be IC-204219, which had a recent clinical trial involving 215 patients. This was a double-blind randomized placebo-controlled study; in 20-mg doses the drug produced significant improvement.

Inhibitors of 5-lipoxygenase can be divided into two classes: (1) those that inhibit the enzyme directly, and (2) those that bind to a transmembrane protein (called FLAP) that combines with 5-lipoxygenase before leukotriene biosynthesis can begin. Among the direct enzyme inhibitors, a recently developed compound, zileuton, has shown definite promise. An extended study is now (1994) in progress that thus far suggests that the drug significantly attenuates bronchial inflammation. A new FLAP binding agent, MK-0591, appears to be superior to previously tested drugs of this class. In these studies, the effects of both leukotriene-receptor antagonists and synthesis inhibitors are cumulative, suggesting that the maximum benefits may not have been reached at the end of the trial periods.

Agents Targeting PAF

Exogenous PAF causes marked bronchospasm and bronchial hyperreactivity, increases recruitment and activation of inflammatory cells, increases vascular permeability, and induces epithelial damage. PAF is released in largest quantities from eosinophils, but it is also extractable from macrophages, neutrophils, and platelets. It promotes the release of other mediators, including leukotrienes. Thus, PAF suppression has become a focus of study by a number of pharmaceutical companies. Several potent and selective antagonists of PAF receptors have been developed and are being clinically evaluated. To date, however, clinical studies have been disappointing. Apafant (WEB-2086), MK-287, and UK-74505 are examples of compounds subjected to trials that failed to show significant benefit. Data from longer studies are forthcoming.

Agents Targeting Thromboxane

The thromboxanes are another class of arachidonic acid derivatives whose receptors are abundant in bronchial smooth muscle. One thromboxane, TXA2, is a potent bronchoconstrictor. In a manner parallelizing research on leukotrienes, TXA2 suppressors are under study, both thromboxane-receptor blockers and thromboxane-synthetase inhibitors. Two receptor antagonists, GX-32191 and ICI-92604, have apparently been found ineffective, but a more potent agent, BAY-u305, is currently being evaluated. The thromboxane-synthetase inhibitor OKY-0046 (ozagrel) has been reported to show inhibition of bronchoconstriction in allergen-challenge studies, and is being investigated in Japan for the treatment of asthma.

Inhibitors of Lymphocyte-Derived Cytokines

This is a heterogenous group of compounds that can be divided into at least three distinct categories: (1) compounds that inhibit IgE production, (2) those that block or inhibit the formation of interleukin-4 (IL-4), and (3) those that modulate the action of interleukin-5 (IL-5). Attempts are in progress to determine the rate-limiting step in the synthesis of IgE, the antibody type that is an etiologic factor in asthma and other allergic diseases. Interleukin-4 is a cytokine that initiates IgE synthesis in human lymphocytes. It also induces expression of vascular-cell adhesion molecules on endothelial cells, thus promoting the migration of eosinophils into pulmonary tissues. Cromollyn partially blocks IgE production induced by IL-4 in human subjects. To date, no small molecules have been shown to antagonize IL-4 receptors. IL-5 prolongs survival of eosinophils in cell culture and is an active promoter of eosinophil responses in lung tissue. Although interleukin inhibition holds great research interest, its main correlation with inflammatory processes is nonspecific. No trials in asthmatic patients have yet been attempted.

Modulators of Intracellular cAMP

Because bronchodilation is promoted via the intracellular messenger cAMP, agents designed to augment cAMP activity are under widespread study. Theophylline and corticosteroids are known to stimulate cAMP pathways, but other mechanisms are probably more important in determining their antiasthmatic efficacy. Recent work has been directed toward compounds that inhibit phosphodiesterase type IV (PDE IV), the enzyme that catalyzes the breakdown of cAMP, and on cAMP-dependent protein-kinase agonists (cA-PKA), which activate cAMP intracellularly. Several selective PDE IV inhibitors now have names instead of experimental numbers, but not one of them has received clinical trials beyond studies on human volunteers. The cA-PKA compounds have not advanced beyond molecular biology experiments.

It is now obvious that many chemical substances are involved in the inflammatory process that leads to the clinical manifestations of asthma. This discussion has omitted several important inflammatory sequences in which bronchoactive mediators are produced, eg, Bradykinsins. The modest success of leukotriene antagonists would suggest that these lipid derivatives are just another group of mediators that happened to be identified early in the biochemical studies of asthma. Nevertheless, with the broadening applications of inflammatory suppressants, future researches are likely to reveal increasing numbers of compounds with therapeutic potential for asthma prophylaxis and treatment.
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RETROSPECTROSCOPE

Whodunit (And Why)¹

Part I

The scientific method depends on the careful accumulation of data by objective means. However, when medical scientists testify before congressional appropriations committees, they support their case with anecdotal evidence (the “let-me-give-you-an-example” approach). In fairness to them, they’ve had little choice, because the scientific method has never really been used to determine the genesis of crucial scientific discoveries. Yet national biomedical science policy should be directed mainly toward creating conditions that favor discovery, and a little objectivity might help.

About five years ago, Robert Dripps (then Vice President for Health Affairs at the University of Pennsylvania)² and I wondered whether we could obtain objective data on how critical discoveries have come about in the past, to serve as a historical basis for predicting how equally important discoveries are likely to be made in the future.

We decided to study the ten most important clinical advances in cardiovascular-pulmonary medicine and surgery of the past 30 years and see how much first had to be learned by how many scientists working in how many fields before someone could take the final step of successful clinical application (1). In our analysis we started with more than 4,000 scientific articles, whittled them down to about 2,500, and then, with the help of consultants, reduced these to 529 key articles. A key article was defined as one that had an important effect on the direction of subsequent research and development, which in turn proved to be important for one of the ten clinical advances. It reported new data, new ways of looking at old data, a new concept or hypothesis, a new method, a new drug, new apparatus, or a new technique that was either essential for the full development of a clinical advance or greatly accelerated it. The key article might represent basic laboratory investigation, clinical investigation, development of apparatus or essential components, synthesis of data or ideas of others, or wholly theoretic work.

Having selected these key articles, we then examined them carefully to see why their authors did the work in the first place. One question we wanted to answer was, how many of these studies were done to solve a specific clinical problem related to the prevention, diagnosis, or treatment of a specific disease, and how many were done simply to learn something completely unrelated to clinical medicine or surgery or to the genesis or course of a disease?

Our analysis showed that 217 of the 529 key articles (41 per cent) reported work that, at the time it was done, had no relation whatever to the disease that it later helped to prevent, diagnose, treat, or alleviate (2). Because this is a surprisingly large percentage—enough to have a real impact on national biomedical science policy—I think it is important to list some of the 217 for you.

In previous Retrospectroscope columns, I have already mentioned some of these:

X-rays. When, in 1895, Roentgen discovered that rays from a Crookes’ tube could pass through the human hand and darken photographic plates, he was a physicist studying a basic problem dealing with the electrical nature of matter. He did not have a “mission” to look inside people and examine their lungs or hearts or coronary arteries (3a).

Motion pictures. When Muybridge devised the first motion pictures in 1887, he was a photographer interested only in determining whether all four hoofs of a horse ever left the ground at the same time (3b); he had no thought of devising an apparatus that was essential to the advance of angiocardiography—x-rays weren’t even discovered until December 1895. But in 1896, Macintyre combined Roentgen’s and Muybridge’s “pure” research and made the first ciné roentgenograms.

Lung volume. When Davy measured his own lung volume

¹ Studies on which this article is based were supported by Contract I-HO-1-2327 from the National Heart and Lung Institute, and by grants from the Commonwealth Fund and the Burroughs-Wellcome Fund.

² Dr. Dripps died October 30, 1973.

in 1800 by rebreathing hydrogen, he had no thought whatever of using it as a test to help in the diagnosis or evaluation of pulmonary disease (3c); he was simply curious.

Microvascular surgery. When two pharmacologists wanted to solve a basic problem that required complete denervation of a dog's artery in vivo, they went to a young research surgeon, Julius Jacobson, for advice. Jacobson knew that the only way he could be sure that he had severed every single nerve fiber was to excise a segment of the artery and sew it back in place. In trying to do this, he found that the problem was "not in the ability of the hand to sew but rather the eye to see"; to solve the problem, he applied a dissection microscope and special instruments to the task. He then went on to use the technique in clinical microvascular surgery (3d), including operations on coronary and intracranial vessels.

Pulmonary diffusing capacity. When Marie Krogh, in 1915, developed the carbon monoxide technique of measuring diffusing capacity in man, she wanted only to answer a basic physiologic question—did oxygen cross the alveolar capillary membrane only by diffusion or was oxygen secreted, at least in part, by an active process? She did answer it unequivocally (it was by diffusion alone) but she never applied her test to the clinical diagnosis of pulmonary disorders (3e).

Nitrogen meter. When John Lilly devised this instrument, it was to help the Air Force during World War II to determine when and why air leaked into oxygen masks worn by aviators (3f). Lilly reasoned that, since air contained 79 per cent N2 and oxygen contained none, he could determine the precise moment of the leak if he had a method for continuous, almost instantaneous, measurement of the nitrogen concentration in small samples of gas drawn from the mask. With his background in physics, it took him little time to devise the nitrogen meter. He soon saw the clinical usefulness of a nitrogen meter to determine uneven distribution of inspired gas to the lungs, but this came later, after he had solved the Air Force's problem.

Intracellular recordings of transmembrane potential differences. When, in 1936, J. Z. Young came across the giant axone of the squid, he had been studying the process of degeneration of the central stump after complete section of a nerve. In his first report on these huge axones (up to 1 mm in diameter—50 to 1,000 times thicker than mammalian nerve fibers), he mentioned that they might prove useful in studying the structure of nerve membranes and the location of inorganic ions involved in depolarization of membranes. He did not mention using the technique for measuring electrical activity in normal or diseased heart muscle fibers to obtain transcellular potential differences (3b).

Influence of calcium and potassium on cardiac contraction. When Sidney Ringer suddenly realized that calcium and potassium ions, in proper proportions, were essential for the normal rhythm and contraction of heart muscle, he was studying a basic physiologic problem in frogs (3d). He had no idea at that time that his observations would be clinically important in cardiac defibrillation, in elective cardiac arrest during open heart surgery, and in understanding the mechanism of cardiac malfunction in electrolyte disturbances in man.

Antimicrobial action of penicillin. When, in 1941, Chain re-investigated penicillin, first studied by Fleming in 1929, he considered his study to be of purely scientific interest; he had no thought of developing an antimicrobial drug for clinical use. As Chain put it in 1971, "a substance of the degree of instability which penicillin seemed to possess according to the published facts does not hold out much promise for practical application. If my working hypothesis had been correct and penicillin had been a protein, its practical use as a chemotherapeutic agent would have been out of the question because of anaphylactic phenomena which inevitably would have followed its repeated use" (3d).

Heparin. I have already mentioned that when McLean, a medical student working in the physiologic laboratory at Johns Hopkins Medical School, discovered heparin he was working on a basic problem (assigned to him by Professor Howell) on the biochemistry of factors favoring coagulation of blood; neither he nor Howell had an anticoagulant in mind (3d). Professor Howell continued McLean's work, and, in 1916-18 (4, 5) speculated that hemophilia might be due to abnormal amounts of heparin circulating in blood, but even then he did not suggest that heparin might be used clinically.

Who was the clinician who decided that it was important to purify and concentrate heparin for clinical work? There was none. The decision to purify it came from a physiologist. Best (a co-discoverer of insulin in 1922) relates that in 1926, when he was working with Sir Henry Dale in London on problems related to histamine, he had more than his share of problems with blood that clotted in his pressure recording system. The crude heparin available then was practically useless and Best determined that on his return to Toronto he would organize a group to purify heparin (Best, C. H.: Personal communication, Nov. 23, 1972). He did, and in 1933 Charles and Scott reported that, "because of the importance of heparin in certain physiologic experiments," they had tackled the problem of making heparin useful for biologic work (6). They succeeded and soon John Gibbon was using it in his physiologic experiments on the artificial heart-lung and Murray was using it clinically to manage thrombosis. But neither those who discovered heparin or purified it had a clinical problem in mind. It seems that neither physicians nor the pharmaceutical industry in the 1920s or 1930s saw a need to purify it for clinical use, just as no one earlier saw a need to purify hirudin, an anticoagulant that Abel, Rowntree, and Turner extracted from medicinal leeches (obtained from French cupping barbers) to prevent blood coagulation in their 1914 artificial kidney (7).

Cardiac catheterization. I have already mentioned (1) that when Courmand and Richards performed their first cardiac catheterizations in man, it was not to develop a new method for diagnosing or evaluating congenital or acquired heart disease. They were then pulmonary physiologists who wanted to
learn more about a basic physiologic problem about how blood and air were distributed to air sacs in the lungs. To do that, they needed to know the oxygen content of mixed blood in the right atrium and the only way to get such blood was to catheterize the human heart. Soon thereafter they used their new technique to study patients with shock and congestive heart failure, as did McMichael and Sharples-Schafer in England. And a few years later, Bing in Baltimore, Dexter in Boston, and Warren in Atlanta began to use the cardiac catheter to diagnose specific defects in patients with congenital cardiac disease.

Recently, Courand has written about the first cardiac catheterization in animals (8), which he attributes to Claude Bernard, the famous French physiologist. Bernard wanted to settle a then-controversial matter: Was animal heat produced in the lungs by the chemical reactions involving exchange of O₂ and CO₂ (as Lavoisier maintained) or was it produced by combustion occurring in all tissues of the body (as Magnus maintained)? Bernard decided to learn whether the temperature of left ventricular blood (that had just passed through the lungs) was greater than that of right ventricular blood. In 1844, he passed a long mercury thermometer down a carotid artery of a horse into its left ventricle and one down the jugular vein into the right ventricle and settled the matter in favor of Magnus (9). In 1847, Bernard passed a glass tube through the right jugular vein of a dog and measured right ventricular pressure.

Most physiologists credit Chauveau and Marey with being the first (in 1861) to use cardiac catheterization in animals to measure intracardiac pressures (10); certainly they were the first to make systematic and continuous measurements of atrial and ventricular pressures and were the first to design and use a double-lumen catheter for simultaneous recordings from two intracardiac locations (figure 1).

None of this research, from Bernard to Chauveau and Marey to Courand and Richards, was clinically oriented; it was all done to solve basic physiologic problems.

Some additional key discoveries that were not clinically oriented were:

Flow-directed cardiac catheter. The "Swan-Ganz catheter" (11), now widely used in the management of acutely ill cardiac patients, is usually listed as a clinically oriented venture, although the investigators credit Lategola and Rahn (12) with the development and use of a quite similar catheter 17 years earlier. Lategola and Rahn were pulmonary physiologists interested in solving physiologic problems in dogs. Since the title of their 1953 paper, "A self-guiding catheter for cardiac and pulmonary arterial catheterization and occlusion" didn't mention dogs, it should have brought cardiologists to their laboratory in droves—but no one applied it clinically until 1970.

Bronchospirometer. While discussing catheters, let us not forget that Henry Head in 1889 (13) devised a double-lumen tube for separating gas entering and leaving the right lung from that ventilating the left lung (figure 2). He was a physiologist who worked on animals. His work was not oriented toward the diagnosis of clinical problems, although his appa-

 Fig. 1. Chauveau and Marey’s double-lumen tube for cardiac catheterization and measurement of intracardiac pressures in dogs (1861). (Top) Complete instrument. (Bottom) Enlarged drawings of three sections of it (10).

ratus was later widely used for several decades in determining differential lung function.

Direct measurement of arterial blood pressure. Although the indirect (Riva-Rocci) method of estimating arterial blood pressure in man is by far the most widely used in clinical medicine (and the apparatus for making indirect measurements is now a standard item in drugstores and mail order catalogs), direct measurement is routine in intensive care units and many operating rooms. The first direct measurement was made in 1733 by an English clergyman, Stephen Hales. He used a 9-foot-long glass tube attached by a brass connection to a flexible windpipe of a goose and measured blood pressure in the femoral and carotid arteries of horses (14). Why did he do it? In his dedication "to the King's Most Excellent Majesty," Hales wrote:

As the beautiful fabric of this world was chiefly framed for and adapted to the use of man, so the greater insight we get into the nature and properties of things, so much the more beneficial will they be to us, the more will our real riches thereby increase, the more also will man's original grant of dominion over the creatures be enlarged. [This must have been one of the earliest statements on cost:benefit analysis of pure research.]
In brief, Hales was curious about nature. Earlier, he had been intrigued by the fact that sap rises to the very top of even tall trees and he made measurements of sap pressure at various distances from the ground (15). I suppose a logical next step was to measure how high the sap that circulated in the tubes of animals would rise. Hypertension was not yet recognized as a disease in Hales' time, and attaching a 9-foot glass tube to a man's carotid artery was not a very attractive way of beginning an inquiry into blood pressure in man—so Hales' discovery was surely not clinically oriented.

Poiseuille, in 1828, made Hales' 9-foot tube practical by using a U-tube filled with mercury (to reduce the height of the apparatus by a factor of 13.6). Why did Poiseuille want to measure blood pressure? Because physiologists (except for Poiseuille) believed that arterial blood pressure fell linearly from the beginning of the aorta to the smallest arteriole. Poiseuille, using his new transducer, measured precisely measurements from the ascending aorta down to the finest branch that he could cannulate; he proved that there was little decrease in pressure between the beginning and end of the arterial system and that the main drop (and therefore greatest resistance to flow) was in small arterioles (16). He then went on to his classic study of the flow of liquids through small tubes, now summarized in Poiseuille's law (17). Clinically oriented? No, actual measurements of blood pressure in man (as opposed to estimation of "hardness" of the pulse) waited until the end of his century.

Incidentally, Poiseuille didn't invent the U-tube. Hales' 1727 volume (Vegetable Staticks) shows U-tubes at various locations in trees, measuring sap pressure (figure 3) (15).

Strain gauge. This is now the standard transducer used to translate intra-arterial blood pressure into continuous, visible records. Whether you're a Tomlinson man or a Lambert and Wood man, neither used the strain wire or gauge to solve a clinical problem. Tomlinson, who in 1876 recorded in the Proceedings of the Royal Society of London that "the temporary increase per centimeter of resistance of a wire when stretched in the same direction as the line of flow of the current is exactly proportional to the stretching force" (18), was then a Demonstrator in Natural Philosophy at King's College, London, and pretty far removed from clinical medicine. Lambert and Wood (19), who in 1947 first reported the use of strain
wires to measure intra-arterial blood pressure, were not clinically motivated. As part of the war effort, they had built a human centrifuge at the Mayo Foundation and were measuring the response of man's cardiovascular system to increased gravitational forces. Because Lambert and Wood had to record at a distance from the subject, they could not use the Hamilton manometer because it had to be rigidly fixed close to its recording apparatus. This meant that they had to translate blood pressure into electrical signals and send these over wires to the central pole of the centrifuge and then to another room. The answer was the strain gauge manometer.

[To be continued in the July issue.]

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Respiratory care practitioners have established themselves as experts in equipment assembly and troubleshooting. This is due in part to dedicated professionals such as Steven McPherson. His text, Respiratory Care Equipment, now in its fifth edition has provided a foundation for students and tenure RCPs for over a decade. The new edition continues with the tradition of providing the essential skills necessary in the ever-changing field of respiratory care.

The text format has been left unchanged, with 15 chapters. The author has introduced learning objectives and summaries at the end of each chapter to facilitate increased retention. The bold content boxes located throughout previous editions have been deleted and replaced with highlighted boxes that have key points to emphasize pertinent information. A glossary has been included to list relevant terminology for the reader to use as a quick reference.

Chapter 1: Gas Physics begins with a very detailed and thorough discussion of various states of matter and gas laws. Chapter 2—Primary Systems: Cylinders and Piping Systems, offers an in-depth look at cylinder markings, safety systems, and station outlets. NFPA and CGA recommendations and regulations for cylinders, bulk oxygen, and liquid systems have been placed in appendices at the end of the chapter. Chapter 3: Gas Regulation, Administration, and Controlling Devices remains intact with a thorough discussion of oxygen devices, flowmeters, and regulators. Chapter 4: Humidifiers and Nebulizers has been updated to include content on metered-dose inhalers and special considerations for neonatal and pediatric patients. Chapter 5: Artificial Airways and Airway Adjuncts has received revision with the deletion of esophageal obturator airways. The chapter is still complete with a discussion on assorted airway maintenance devices. Chapter 6: Manual Resuscitators provides information on several different types of resuscitators. However, the author fails to include resuscitators that are not self-inflating, commonly used in the neonatal and pediatric population. Chapter 7: Bedside Measuring and Monitoring Devices offers an introduction into the various types of oxygen analyzers, transcutaneous electrodes, oximeters, capnograph, and bedside pulmonary function devices. More information could be included on oximeters and capnograph due to their wide clinical application.

Chapters 8-15 provide an intensive review of mechanical ventilators. Chapter 8 begins with an introduction into the classifications of ventilators and continues with the types of ventilatory modes. Chapters 9 and 10: Bird and Bennett Respirators have received extensive review with the deletion of several outdated machines. These chapters still offer an in-depth discussion on the mechanics of positive pressure ventilation. Chapters 11, 12, and 13, discuss a diverse number of adult ventilators. Several older machines have been replaced with new additions that contribute to the revisions in these chapters. Chapter 14: Pediatric Ventilators has remained intact with a listing of several pediatric ventilators. I was disappointed to see the author fail to include several new neonatal/pediatric ventilators such as the SensorMedics high frequency oscillator, Bunnell jet ventilator, VIP Bird, and Newport Breeze. The text concludes with Chapter 15: Transport and Homecare Ventilators. This chapter has received revisions in the deletion of several out-dated homecare ventilators, while discussing, numerous ventilators commonly seen in today's homecare market.

Also, included with this text is a quick reference pocket handbook by Christian Blazier. The handbook offers assembly and troubleshooting tips for the practitioner at the bedside. The pocket reference follows the same format as the text providing recommendations for each corresponding chapter.

I highly recommend this text to respiratory care practitioners regardless of their clinical experience. It is an excellent resource for practitioners who need an in-depth understanding of a subject area or just a review. The content is clear and concise with extensive high-quality illustrations throughout the text. Overall, this text has few weaknesses and I believe it is superior to other related books on respiratory care equipment.

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Progress in Domiciliary Respiratory Care: Current Status and Perspective, edited by Dr S Kira, Department of Respiratory Medicine, Juntendo University School of Medicine and the Respiratory Failure Research Committee, Japan, and Dr TL Petty, Director of Academic and Research Affairs, Presbyterian/St Lukes' Center for Health Sciences Education, Denver, Colorado. 372 pages, hardcover, illustrated. Amsterdam: Excerpta Medica, 1994. $182.75.

This book is a summary of the proceedings of the International Symposium on Domiciliary Respiratory Care held in Tokyo, Japan in September 1993. The Symposium was an invitational conference organized by Japanese pulmonologists and attended by pulmonary specialists (predominantly, but not exclusively physicians) from North America, eastern and western Europe, several Pacific Rim countries, and Australia. Participants were selected for their interest and expertise in long-term oxygen and noninvasive ventilation therapy. They address a broad spectrum of technologic, economic, and psychosocial issues involved in this rapidly developing facet of clinical practice. Some chapters focus on a review of known information, others present new research data.

This book is useful for physicians, nurses and therapists with experience in long-term oxygen and noninvasive ventilation and an interest in international trends and practice patterns. For those of us with that experience and interest who were unable to attend the conference, it is probably the next best thing to having been there. This book is of limited value for the nonspecialist because the various papers assume and expand on a solid understanding of the therapies discussed.

Organized into sections, the book begins with a description of long-term oxygen therapy as practiced in the 14 countries represented. Following the descriptions are sections about physiologic effects of home oxygen; guidelines for its use; psychological, economic, and life-quality concerns; newer technologies; prognosis for patients using long-term oxygen therapy; and a brief section on noninvasive ventilatory support for patients with sleep apnea or chronic ventilatory failure. Questions and answers at the end of each section help to clarify important points, and reinforce the reader's sense of participation in the conference.
A major strength of this book is its fascinating international perspective on the topic. Rarely does one have an opportunity to read, in one publication, clinical practice information from many widely differing cultures. The chapter by Shim describing the current status of home oxygen in Korea was especially interesting as an example of a country just beginning to develop home care technologies. Several authors present innovative approaches and ideas that could be useful in North American practice (eg, Miwa et al’s description of telephone-transmitted oximetry data; Ishihara et al’s use of ineffective gastric tubes for administration of transtracheal oxygen; Sano’s telephone monitoring of oxygen concentrator outputs). Also, information about the level of care and availability of home care technologies in a particular area may help patients traveling or relocating to one of the countries represented. As I reviewed this book, I found remarkable similarities from different countries in the criteria for use of long-term oxygen therapy—a testimony to the efficacy of just this kind of international scientific conference. The book reinforces that we do, indeed, live in a “global village.”

Although the text is interesting overall, there is considerable variability in the quality of writing, the relevance of content to a North American audience, and general readability of specific chapters. Pierson’s review of criteria for home oxygen, and Petty’s review of new technologies, for example, are clear, succinct and easy to follow, as is Grunstein et al’s discussion of noninvasive ventilation for sleep apnea and kyphoscoliosis. Manresa et al’s study of continuous pulse oximetry includes several specific and useful recommendations, and Zielinski presents an excellent review of the effect of long-term oxygen therapy on the pulmonary vasculature. Luhdensuo’s brief chapter on the psychosocial effects of home oxygen therapy contains no new information but is easy to follow and reinforces the importance of pulmonary rehabilitation and the appropriate treatment of depression.

In contrast, the discussion of nocturnal desaturation during sleep is obscure, and the section on psychological and socioeconomic concerns is generally weak. This may be in part a reflection of the general paucity of good data on improved quality of life attributable to home care technologies. Muramatsu et al’s data is especially difficult to follow. The paper includes terms (eg, alexithymic state), tests (eg, Tree test) and figures (eg, Figure 1, Page 295 discussing male and female “egograms”) that are neither defined nor explained.

Readability could have been improved by better editing of some of the papers written by non-English speaking presenters. Translations are often awkward (for example, “It is a difficult environment to walk outdoors.” on Page 297; and “Patient discomfort occurs frequently because of the need to lie near- ly supine and cannot be moved during use.” on Page 230). Translations also include odd word choices (for example, the term “ag- gravation” for “exacerbation,” Page 262, “shortpoints” for “shortcomings,” Page 230, and “administrated” for “admitted,” Page 293). Though these translations are charming, comprehension of the differences in phrasing requires slower and more careful reading.

Readability could also have been improved by more vigilance for typographical errors and for unexplained or overused abbreviations. This “alphabet soup” approach is quite distracting.

In most instances tables and graphs help to clarify data, although in some chapters they are confusing (eg, Tables 2 and 3, and Figures 3 and 4 in the chapter about transtracheal oxygen). Muramatsu et al’s Table 2 (Page 295) is particularly troublesome in that it describes a sample size of 28 patients when Table 1 notes 70 patients; states a room air Pao2 in the home oxygen therapy group that is markedly greater than that in the non-home oxygen therapy group (are these figures reversed, perhaps?); and omits p-values for differences between groups. Photographs in some chapters are useful. The editorial decision to omit the titles of papers from references is frustrating to me.

In summary, this book is a worthy compilation of international presentations on the topic of home oxygen therapy and noninvasive ventilation. However, inherent in a book of this nature is the dilemma that one must continually consider socioeconomic and health policy differences between countries in order to evaluate the relevance of its content to one’s own practice setting. This tends to make the book more ‘interesting’ than ‘useful.’ It is expensive, but if what one seeks is a good international perspective on this topic, I recommend it.


Drs Müller and von Wichert have assembled the contribution of a superb international group of scientists and physicians who met to review the state-of-the-art in surfactant research. The authors have expertise in current lung surfactant research, with particular emphasis on the pathogenesis of pulmonary disorders. An excellent summary of selected topics of research is presented. There is an in-depth review of the state-of-the-art research on the function of surfactant in normal and disease states, addressing the current concepts about surfactant metabolism, including regulation of gene expression, synthesis, and secretion as well as reuptake versus degradation and clearance. The book consists of 41 chapters that deal particularly with in-vitro and in-vivo laboratory-based research. The majority of the chapters are excellent reports of original data, summary of various studies, or review of the literature. However, there is some heterogeneity in the styles and quality of the chapters and lack of integration or cohesion among the chapters, a problem common to books that result from independent contributions by multiple authors. The book appears to be intended for clinical scientists and other investigators interested in surfactant research. This book would be of interest to selected respiratory therapists, respiratory technicians, nurses, physicians, and others involved in laboratory-based surfactant research. The six chapters that deal with the pathogenic role of surfactant in lung diseases will appeal to clinicians.

In summary, I think this book will be useful for those with strong interest in surfactant research.

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Save time and money by making your department more efficient with the Policy and Procedure Manual. Its 130 pages of policies and procedures cover the aspects of administrative and clinical respiratory care for both adult and pediatric practice. Sections on administrative policies, therapeutic, clinical monitoring, and mechanical ventilation. Standardized formats include objectives, indications, equipment, policies, contraindications, troubleshooting, procedures hazards, and assessment of effectiveness.

Item BK6 $60 ($70 nonmembers)

Respiratory Home Care Equipment


Item BK7 $59 ($12 nonmembers)

Orders with credit cards or P.O. numbers may call (214) 243-2272, or Fax to (214) 484-2730. If ordering by mail, send coupon to: AARC Order Department, 11030 Ables Lane, Dallas, Texas 75229-4893.

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□ Please send me the items I have indicated below.

Item Description Quantity Price Each Total Price
New Products & Services

AIR QUALITY MONITOR. Young Environmental Systems Inc, a Canadian manufacturer, announces the YES-204 Carbon-Dioxide Logger. The device monitors carbon dioxide, temperature and relative humidity for use in structures from commercial buildings to greenhouses. Using proven nondispersive infrared (NDR), the monitor can store up to 22 months of data for further downloading and analysis on a personal computer, according to the company. YES analysis software is also available. Contact Young Environmental Systems Inc, Dept RC, #140, 8771 Douglas Street, Richmond BC V6X 1V2 Canada, or call (604) 276-9923. Don’t forget to mention RESPIRATORY CARE when you call.

POCKET MASK. Laerdal’s Pocket Mask now features a disposable filter and an updated one-way valve. According to the company, these changes offer better protection to both the rescuer and the victim. The filter is positioned on the underside of the mask to protect the valve from blockage by vomit and to allow expiratory gas free passage away from the rescuer and victim. The new one-way valve has a larger mouthpiece and may be cleaned for reuse during training. The enhancements are compatible with all Laerdal pocket masks. An update pack is available. Write to Laerdal, Dept RC, PO Box 190, Armonk NY 10504-0190 or call (800) 431-1055. When calling, please mention RESPIRATORY CARE.

PROBED MASK. BioSafety Systems now offers the Probed HEPA-Tech Mask, designed for quantitative fit testing of HEPA-Tech NIOSH-approved disposable HEPA respirators. According to the manufacturer, the probed mask is an actual HEPA-Tech respirator that is fitted with a metal tube for sampling air inside the mask during the fit test process. Properly fitted, HEPA-Tech respirators meet OSHA requirements for protection against tuberculosis in health-care settings. Write to BioSafety Systems, Dept RC, 10225 Willow Creek Road, San Diego CA 92131, or call (800) 421-6556 for information. Please mention RESPIRATORY CARE when you call.

LOW DEAD-SPACE ADAPTER. NELLCOR Inc introduces the ADAP-N low dead space adapter for use with the ULTRA CAP capnograph and pulse oximeter. For neonatal patients who weigh less than 10 kg and have small respiratory volumes, the adapter balances minimal dead space and low airflow resistance and provides accurate monitoring. According to the manufacturer, the adapter aids the current ULTRA CAP monitor, NELLCOR pulse oximetry, and CO2 technology in providing moisture resistance. Contact NELLCOR Inc, Dept RC, 4280 Hacienda Drive, Pleasanton CA 94588, (800) NELLCOR. Please mention RESPIRATORY CARE when calling.

DATA LOGGER/CONTROLLER. Onset Computer recently launched the Tatletale Model 8 for medical research and OEM applications. According to Onset, the Model 8 combines eight 12-bit analog channels, upgradeable with additional A-D converters; 25 digital I/O pins with 14 Time Processor Unit (TPU) Channels; 256K to 1 MB RAM; 128KB to 1 MB Flash EEPROM; real-time clock; dual RS-232; and two microprocessors—Motorola’s 68332 MPU and Microchip Technologies’ PIC16C64 controller. The Model 8 fits on a 2 in. by 3 in. by 0.5 in. board. Both C and BASIC development kits provide a complete
hardware and software package for portable and embedded instruments. Other options are available from Onset Computer, Dept RC, PO Box 3450, Pocasset MA (508) 563-9000. Please mention RESPIRATORY CARE when you call.

**CLOSED HUMIDIFICATION SYSTEM.** IPI Medical Products introduces a new closed humidification system—Therma-Trol. According to IPI, heat is conducted through the system’s heater assembly. Sterile water from a flexible container is absorbed by the cylinder liner and evaporated, producing molecular super-saturated gases. The system has virtually no resistance to gas flow through the ventilator. Combined with IPI’s single puncture feed set, the closed system provides a noninvasive, contamination-free circuit. Contact IPI Medical Products, Dept RC, 3217 N. Kilpatrick Avenue, Chicago IL 60641, (312) 777-0000. Please mention RESPIRATORY CARE.

**PFT BARRIER FILTER.** Pall Corporation now offers the Pall Pro-Tec Barrier Filter for pulmonary function testing. According to the manufacturer, the filter reduces (by a factor of 1 million) the risk of cross-contamination between patient and equipment by removing aerosolized droplets that may collect in pneumotachometers, valves, tubings, and reservoirs. In addition, the filter is made of a low-resistance hydrophobic membrane that removes bacterial and viral cross-contamination. The filter also provides minimal resistance to airflow and will not interfere with results of flow-dependent tests. The filter is completely disposable. Contact Pall Biomedical Products Company, Dept RC, 2200 Northern Boulevard, East Hills NY 11548, or call (800) 645-6532 [New York residents (516) 484-5400]. Please request B-PF310DATA and mention RESPIRATORY CARE when you call.

**MULTI-GAS ANALYZER.** Marquette Electronics introduces the Smart Anesthesia Multi-Gas module, SAM. For use in operating and recovery rooms, the analyzer offers on-line monitoring of respiratory and anesthetic gas concentrations. According to Marquette, the unit measures inspired and expired values for CO₂, N₂O, O₂, and identifies agents such as Desflurane, Isoflurane, Halothane, and Enflurane. Designed with no internal heaters or coolers, the unit eliminates long warm-up times. All gases use one path and one filter in the process, helping to eliminate drift and instability inherent with the use of multiple filters. In addition oxygen detection is provided with a paramagnetic oxygen sensor. When the SAM module is integrated with the Marquette Unity Network, the total system helps the health-care provider monitor patient data during anesthesia. For details, contact Marquette Electronics, Dept RC, 8200 West Tower Avenue, Milwaukee WI 53223, (414) 355-5000. Don’t forget to mention RESPIRATORY CARE when you call.

**PULSE OXIMETER.** Burdick Inc adds the PRO2 Pulse Oximeter. The portable, hand-held unit offers accurate noninvasive blood-oxygen saturation analysis and heart-rate monitoring. By simply inserting a patient’s finger into a small sensor, the pulse oximeter produces arterial oxygen saturation (SpO₂) results in seconds. According to Burdick, the unit also displays heart rate and pulse signal strength on an LED screen. Available with software programs, the battery-operated unit can be used with adults and children. Other options are available from Burdick Inc. Please mention RESPIRATORY CARE when you call (800) 955-5177.

**FREE GUIDE.** Fogg System Company Inc offers a free booklet, “Guide to Connecting Pressure Transducers.” The 16-page guide contains a list of manufacturer and model numbers that identify transducers, monitors, and recording systems. The codes are used to define cables in two easy steps. To receive the guide, write to Fogg System Company Inc, Dept RC, 15592 East Batavia Drive, Aurora CO 80011 or call (800) 525-0292. Please mention RESPIRATORY CARE when you call.
Videotapes...
The Latest In Technology And Techniques For Respiratory Care Practitioners

Pulmonary Rehabilitation
By John E. Hodgkin, MD. Provides an overview of the sequence for pulmonary rehabilitation. Learn candidate evaluation and selection, rehabilitation team establishment, identification of short- and long-term goals, program components, assessment of patients' progress, and long-term follow-up. Discusses aerosol therapy, IPPB, oxygen therapy, and chest physiotherapy in the treatment of COPD. 60 minutes.
Item VT7

Sleep Apnea
By Alan K. Pierce, MD. Explains how sleep stages are related to respiratory patterns and blood gas values in both normal and abnormal subjects. Includes a discussion of the criteria for defining the sleep apnea syndrome and the distinguishing features of central, obstructive, and mixed causes of apnea. Also addressed is the efficacy of medical treatment to correct specific types of sleep apnea. 60 minutes.
Item VT11

Practical Management of ARDS
By David J. Pierson, MD. Adult respiratory distress syndrome is defined in this informative tape and its clinical features described, including incidence of risk factors and clinical predictors. Also extensively discussed is the use of PEEP to treat ARDS, including goals, complications, best or optimal PEEP levels, PEEP trials, and PEEP withdrawal, as well as general treatment, prognosis, and sequelae of ARDS. 60 minutes.
Item VT16

Pressure Support Ventilation
By Neil MacIntyre, MD. This presentation defines and describes the physiologic considerations attributed to PSV and the clinical situations when PSV may be useful. Includes comparisons when low-pressure levels and high-pressure levels of PSV are offered. Discussion includes using PSV to help overcome resistance for intubated patients and as an augmented ventilatory mode of weaning. 60 minutes.
Item VT17

Drainage of the Pleural Space: Management of Chest Tubes and Bronchopleural Air Leak
By Martha L. Tyler, RN, RRT. Learn the physiologic effects of abnormal pleural space function and the potential problems associated with chest tube stripping, difficulties of bronchopleural air leaks with mechanical ventilation, the therapeutic goals of chest tube placement, and techniques for maintaining gas exchange with air leaks. 60 minutes.
Item VT21

Clinical Use of the Swan-Ganz Catheter
By John Marini, MD. Clinical applications of data obtained by Swan-Ganz catheter placement and the situations in which SGC placement are useful are described in this video. The clinical value of the clinical variables monitored by SGC and the complications of its placement are detailed, including "damping" of the waveform, "overwedging," and optimal lung zone placement. 60 minutes.
Item VT22

Fetal Lung Development
By Charles Rosenfield, MD. Examines the four anatomical phases of fetal lung development along with a discussion of the biochemical development of the fetal lung through gestation. Includes a description of the substances in surfactant, the importance of their timely development, and the methods used to assess fetus survivability by using tracheal aspirant to identify the necessary ratios of the phospholipids making up surfactant. 60 minutes.
Item VT23

Theory and Application of Neonatal Ventilation
By Robert Chatburn, RRT. Knob-turning in the neonatal intensive care unit should be a profound activity because it often has profound consequences. Adjustment of a single control on a ventilator generally has multiple effects, and thorough consideration of how controls are interrelated is essential for optimum care. Presents a well-organized and systemic approach for managing mechanical ventilation. 60 minutes.
Item VT25

$35 each ($40 nonmembers)
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Item VT88 $420 ($480 nonmembers)
Tissue Oxygen Delivery
By David R. Dantzker, MD. Discusses the relationship between adequate tissue oxygenation and adequate energy production, the development of lactic acidosis, and the probable role of tissue hypoxia in the multiple-organ failure of ARDS. Also discusses the factors that govern oxygen transport to the tissues and the variables that reflect the adequacy of tissue oxygen transport. 60 minutes.
Item VT26

Managing the Work of Breathing During Mechanical Ventilation
By John Marini, MD. Learn about the work of breathing and ways in which it can be minimized in the clinical setting on patients receiving mechanical ventilation. Focuses on diminishing the breathing workload through quantifying the effort during mechanical ventilation via direct measures such as oxygen consumption, electromyography, pressure-time index, and external work of breathing, as well as indirect measures. 60 minutes.
Item VT27

Pressure Support Update
By Neil MacIntyre, MD. Reviews pressure support ventilation and how it is used. Explains waveforms for airway pressure, flow, and volume. Also illustrates the measurement of inspiratory muscle loads by pressure-time index and muscle VO2 and demonstrates how muscle efficiency changes as the character of work changes. Includes examples of low- and high-level PSV for elimination of the imposed work of breathing and as a weaning tool. 60 minutes.
Item VT28

The Hospitalized COPD Patient: 10 Commandments for the Clinician
By David J. Pierson, MD. Takes you inside the decision-making process of caring for a respiratory care patient with chronic obstructive pulmonary disease. Details the 10 rules for the clinician to follow when the COPD patient enters the hospital. Emphasis is on the "what not to do." 60 minutes.
Item VT29

Monitoring Respiratory Mechanics During Mechanical Ventilation
By Robert L. Chatburn, RRT. Explains how physical and mathematical models are developed and applied and how they are incorporated into ventilator design to provide estimates of mechanics. Also explains some of the problems that can develop because of the limitations of the models. 60 minutes.
Item VT30

ARDS Review
By Tony Dal Nogare, MD. Discusses the latest developments in risk factors and treatment. Also covers the five diagnostic criteria that must be present to make an accurate diagnosis of ARDS, including clinical, radiographic, and physiologic criteria. 60 minutes.
Item VT31

Sleep Disorders
By Brian Foresman, DO. A discussion of the physiology of sleep and the kinds of respiratory and nonrespiratory sleep disorders seen in the hospital. Discusses how to spot sleep apnea, the problems caused by the inpatient hospital setting, sleep disorder diagnosis, and treatment. 62 minutes.
Item VT32

Uses and Abuses of Noninvasive Monitors in Respiratory Care
By Dean Hess, MEd, RRT. Presents a discussion of how much noninvasive monitoring is needed, proof of false positives, its financial impact, and its future. Discusses the various methods: transcutaneous, pulse oximetry, and capnography. 54 minutes.
Item VT33

Nutrition and Respiratory Care
By Rich Branson, RRT. Explains the relationships and interactions of malnutrition on ventilatory drive, respiratory muscles, lung structure, and immunity. Further discusses the effects of nutrients on the respiratory system, particularly the nutritional support needed by mechanically ventilated and COPD patients. 60 minutes.
Item VT34

Smoking Cessation: Intervention Techniques for the Respiratory Care Practitioner
By Kathleen A. Smalky, MD, MPH. Covers four major aspects of smoking cessation — the impact of smoking on illness and mortality, behavioral components, current cessation programs, and effective intervention. Discusses identification of the physically addicted smoker and the effects of nicotine withdrawal. 43 minutes.
Item VT35

Hospital Acquired Pneumonia
By Galen B. Toews, MD. Provides information on the epidemiology, pathogenesis, and management of nosocomial pulmonary infections. 52 minutes.
Item VT36

New Approaches to the Treatment of Asthma
By Roger Bone, MD. This videotape discusses physician-directed adult patient education and self-management programs using a peak flow meter. Includes information on the morbidity and mortality of asthma, therapy goals from the National Asthma Education Program, and the beta-agonist controversy. Explains the "step care" treatment approach and the complications of mechanical ventilation in status asthmaticus. 60 minutes.
Item VT37
IISPs...

Individual Independent Study Packages Assist Practitioners in Expanding Their Respiratory Care Knowledge

Smoking Cessation

Nicotine Dependency Evaluation and Treatment
Helps you understand the physiologic effects of nicotine and the tests and questions used to evaluate dependency. Provides an understanding of nicotine, its effects on brain chemistry, its measurement in bodily fluids, and the value of self-tests to determine addiction level. Also teaches about nicotine replacement and how to enhance it with behavioral counseling.

Item SC1 $10 each ($15 nonmembers)

Bedside Counseling of the Hospitalized Smoker
Prepares you for the role of a smoking cessation counselor to hospital inpatients by teaching assessment and bedside counseling. Help patients cope with nicotine withdrawal, increase motivation for permanent cessation, and understand the factors contributing to relapse.

Item SC2 $10 each ($15 nonmembers)

Clinical Science

Transpulmonary Pressure Changes in Breathing
Discusses the pulmonary pressures and how they vary during normal breathing and in special situations. Introduces barometric, alveolar, intrapleural, transpulmonary, chest wall, and transthoracic pulmonary pressures.

Item CSS $10 each ($15 nonmembers)

Lung Mechanics
Assists you in understanding the pulmonary characteristics of compliance and resistance, how these characteristics can change, and their influence on mechanical ventilator performance.

Item SC6 $10 each ($15 nonmembers)

Practical Application of Gas Laws
A common-sense approach to the gas laws and how to apply them to respiratory care. Defines the laws of Boyle, Charles, Gay-Lussac, Dalton, Graham, and Henry.

Item CS7 $10 each ($15 nonmembers)

Toxicity of Solutions and the Respiratory Tract
Gives you an understanding of how to predict the effects of therapeutic solutions on the respiratory tract and how to utilize them in the treatment of various respiratory tract pathologies.

Item CS8 $7 each ($12 nonmembers)

Carbon Monoxide Inhalation: Introduction to Physiologic Effects and Respiratory Management
Explains the effects of carbon monoxide inhalation on oxygen transport mechanisms in the body and the use of CO in physiologic tests of pulmonary function. Also covers recognition signs and symptoms, treatment of CO poisoning, and the equipment used to administer therapy.

Item CS9 $10 each ($15 nonmembers)

Electrical Safety in Respiratory Therapy: Basic Electrical Circuitry
Enables you to relate the basic principles of electrical theory to commonly used equipment and procedures in the clinical environment and provides you with an understanding of electrical safety in respiratory care.

Item CS12 $7 each ($12 nonmembers)

Electrical Safety in Respiratory Therapy II: Identification of Electrical Hazards
Helps you identify and, where possible, minimize or eliminate common electrical hazards. Teaches differentiation between electrical hazards and which patients and hospital personnel are exposed.

Item CS13 $7 each ($12 nonmembers)

Bronchodilators: Sympathomimetic Amines
Understand the results of stimulating the autonomic nervous system and the use of sympathomimetic drugs to accomplish bronchodilation. Exposes you to basic aspects of adrenergic bronchodilators and the patient situations for which they are indicated.

Item CS14 $10 each ($15 nonmembers)

Bronchodilators II: Anticholinergics and Xanthines
Identifies the three categories of drugs that promote bronchodilation and the mechanism of action for each. This package also provides examples of drugs in each category. Clinical situations are presented with methods of bronchodilator and rationale for method selected.

Item CS15 $10 each ($15 nonmembers)

Calculation and Preparation of Respiratory Medications
Teaches you how to calculate and prepare percentage solutions, ratio solutions, solutions prepared from solutes less than 100 percent in strength, and how to prepare povages from powdered drugs.

Item CS16 $10 each ($15 nonmembers)

Microbiology for Respiratory Therapy: A Review of Microbial Growth and Cross-Contamination
Provides an overview of microbiology in respiratory care, including groups and characteristics of microbes, requirements for microbial growth, cross-contamination, and prevention of transmission.

Item CS17 $9 each ($14 nonmembers)

Classification of Mechanical Ventilators I
Outlines the basic concepts of ventilator classification and a mathematical model of the respiratory system that provides the basis for classifying ventilator control systems. Also teaches the specific criteria for determining whether a ventilator primarily controls pressure, volume, or flow.

Item CS18 $10 each ($15 nonmembers)

Classification of Mechanical Ventilators II
You will learn detailed information about the control of ventilation in terms of switching from inspiration to expiration. You will also be introduced to the common drive mechanisms used in various mechanical devices.

Item CS19 $10 each ($15 nonmembers)

Neonatal

Recognition and Stabilization of the Premature Infant in Respiratory Distress
This IISP helps you identify primary risk factors associated with prematurity birth. Discusses and explains the importance of providing a neutral-thermal environment for the premature infant and the necessity of continuous noninvasive oxygen monitoring of the premature infant on supplemental oxygen. You will also learn to recognize aberrant blood gases in the neonate and oxygen therapy initiation with an oxygen hood.

Item NN1 $10 each ($15 nonmembers)

Order the complete set of 33 IISPs and

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Item CP10 $210 ($325.50 nonmembers)

To Order by Credit Card or Purchase Order, Call (214) 243-2272.
Classification of Mechanical Ventilators III
Explores the output waveforms that ventilators produce and their clinical implications. Instructs in the various modes of ventilation in terms of the specific combinations of control characteristics along with ventilator performance testing and the practical application of ventilator classification.
Item CS20 $10 each ($15 nonmembers)

Ventilation/Perfusion Relationships In Health and Disease
Covers the different degrees of hypoxia commonly encountered in patients with pulmonary disease. Focuses on ventilation/perfusion abnormalities, which account for much of the severe hypoxia in COPD, with alveolar hypventilation also a contributing factor.
Item CS21 $10 each ($15 nonmembers)

Patient Evaluation
Pulmonary Function Assessment I: Basic Screening Studies
Understand simple spirometry results, the definition of obstructive and restrictive pulmonary disorders, and the basic pathology involved in each. Covers the four reasons for simple spirometry tests, the common parameters of lung function, and describes Boyle's Law, thoracic volume, airway resistance and its normal values, and the measurement of airway resistance.
Item PE5 $7 each ($12 nonmembers)

Body Plethysmography
Explains the theory of body plethysmography and its use in the measurement of FRC and airway resistance. Defines and the results that are indicative of obstructive and restrictive pulmonary disorders.
Item PE3 $8 each ($13 nonmembers)

Pulmonary Function Assessment II: Bedside Studies
Familiarizes you with the use and interpretation of bedside pulmonary function tests. Learn how pulmonary function test results are used to assess ventilatory ability and which tests are commonly used. Also learn the measuring instrument required for each test, how it is performed, and what is significant about the results.
Item PE4 $7 each ($12 nonmembers)

Pulmonary Function Assessment III: Lung Volume Determination and Closing Volume Studies
Assists in the recall of the eight subdivisions of total lung volume and their individual characteristics in health and disease. Discusses the measurement of lung volume by nitrogen washout, helium dilution, and closing volume techniques.
Item PE6 $7 each ($12 nonmembers)

Sources of Error in the Determination of Blood Gas Values and pH
Familiarizes you with the errors that may occur in the analysis of blood gases and pH. These are often the most important laboratory data used in the diagnosis and treatment of pulmonary disease, and errors in these values can result in deleterious effects on patient care.
Item PE7 $10 each ($15 nonmembers)

Temperature Adjustment of Blood Gases and pH
Teaches you the effects of abnormal body temperature on blood gas and pH values. Blood gas values and pH are determined at 37 degrees Celsius, and this package teaches you how to adjust these values.
Item PE8 $10 each ($15 nonmembers)

Updated Plotting Static and Dynamic Compliance Curves During Mechanical Ventilation
Learn how to compute and record static compliance curves and dynamic characteristics. Also learn the procedure of obtaining pressure-volume measurements and interpret the compliance characteristics measurements.
Item PE9 $10 each ($15 nonmembers)

Arterial Blood Gas Interpretation
Teaches you ABG interpretation in order that the prescribed therapy can be administered in a knowledgeable manner. Describes a systematic method that allows you to correctly classify the acid/base dysfunction and to relate the diagnosis concisely and coherently.
Item PE10 $10 each ($15 nonmembers)

Clinical Practice
Chest Tubes and Pleural Drainage
Helps you understand the purpose of pleural drainage, how it might affect the patient's respiratory status, and what precautions you must take when working with patients who are receiving pleural drainage.
Item CP3 $10 each ($15 nonmembers)

Tracheal Intubation I: Upper Airway Anatomy and Goals of Intubation
After completing this IISP, you will understand the rationale for tracheal intubation and be able to identify the important landmarks of upper airway anatomy.
Item CP4 $7 each ($12 nonmembers)

Tracheal Intubation II: Routes of Intubation
Describes the four routes of tracheal intubation and some advantages and hazards of each. Also presents the process for selecting the most suitable route in a given situation.
Item CP5 $7 each ($12 nonmembers)

Tracheal Intubation III: Equipment Procedures for Intubation
Covers the selection of the proper equipment necessary to perform endotracheal intubation and to ensure that they are in working order.
Item CP6 $8 each ($13 nonmembers)

Respiratory Management of Neuromuscular Crisis
Teaches respiratory management of patients with ventilatory failure caused by a neuromuscular disorder. Provides a basic understanding of how neuromuscular conditions lead to respiratory insufficiency and the considerations to be taken when working with these patients.
Item CP7 $7 each ($12 nonmembers)

Respiratory Management of Flail Chest
This study package helps you increase your understanding of the pathophysiology of flail chest and the respiratory management of patients who have sustained chest wall trauma that results in a flail chest.
Item CP8 $10 each ($15 nonmembers)

Respiratory Management of Head Trauma
Teaches identification of the five physical signs indicative of head trauma and explains the development of respiratory failure secondary to trauma. Also discusses airway management, drug therapy, ventilator parameters, acid-base status, and measures to be taken to maintain the appropriate pH, PaCO2, and PaO2.
Item CP9 $10 each ($15 nonmembers)
Notices

**JUNE 30 DEADLINE FOR ARCF FELLOWSHIPS**

The Allen and Hanburys Fellowship for Asthma Education provides $3,500 to allow completion of a project encompassing asthma self-management or asthma awareness. The Fellowship also provides airfare and one night’s lodging to attend the Awards Ceremony at the AARC Annual Convention.

Three $1,000 fellowships are available. In addition to the cash award, each includes airfare and one night’s lodging to attend the Awards Ceremony at the AARC Annual Convention.

- **The LifeCare Fellowship** is designed to foster projects dealing with mechanical ventilation, especially outside of the ICU.
- **The Monaghan/Trudell Fellowship** is designed to support projects dealing with the development of cost-effective aerosol delivery.
- **The Respironics Fellowship** is designed to foster projects dealing with non-invasive ventilatory support. For details and specifications for application, contact Lynn Perkins or Joy Rea in the AARC Executive Office by phone (214) 243-2272 or FAX (214) 484-2720.

**1995 Publication Awards**

<table>
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<th>Award Description</th>
<th>Amount</th>
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<tr>
<td>Radiometer Awards for Best Feature Articles (Divided equally among three winners)</td>
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<tr>
<td>Best Papers by 1995 OPEN FORUM Participants Who Have Never Published as First Author (Two winners - $500 each)</td>
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<tr>
<td>Best Papers on OPEN FORUM Presentation Sponsored by Allen &amp; Hanburys (Two winners - $1000 each)</td>
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<td>Best Paper Published in RESPIRATORY CARE Sponsored by Allen &amp; Hanburys</td>
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<tr>
<td>Dr Allen DeVilbiss Technology Paper Award</td>
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**THE NATIONAL BOARD FOR RESPIRATORY CARE—1995 Examination and Fee Schedule**

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**AARC SUMMER FORUM**

Vail, Colorado July 14-16, 1995

**RESPIRATORY CARE WEEK**

October 1-7, 1995

**AARC 41st ANNUAL CONVENTION & EXHIBITION**

Orlando, Florida December 2-5, 1995
### A. Patient Information

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In confidence

### B. Adverse event or product problem

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<th>Outcomes attributed to adverse event (check all that apply)</th>
<th>Adverse event and/or Product problem (e.g., defects/malfunctions)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death</td>
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<tr>
<td>Congenital anomaly</td>
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<tr>
<td>Life-threatening</td>
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<tr>
<td>Hospitalization – initial or prolonged</td>
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### C. Suspect medication(s)

1. Name (give labeled strength & mfr/labeler, if known)

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2. Dose, frequency & route used

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3. Therapy dates (if unknown, give duration)

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4. Diagnosis for use (indication)

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5. Event abated after use stopped or dose reduced

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6. Lot # (if known)

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7. Exp. date (if known)

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8. Event reappeared after reintroduction

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9. NDC # (for product problems only)

### D. Suspect medical device

1. Brand name

2. Type of device

3. Manufacturer name & address

4. Operator of device
   - Health professional
   - Lay user/patient
   - Other:

5. Expiration date

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6. model #

7. catalog #

8. serial #

9. lot #

10. Device available for evaluation? (Do not send to FDA)

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<th>#1</th>
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<th>Returned to manufacturer on</th>
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Other relevant history, including preexisting medical conditions (e.g., allergies, race, pregnancy, smoking and alcohol use, hepatic/renal dysfunction, etc.)

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

1. Name, address & phone #

2. Health professional?

3. Occupation

4. Also reported to
   - manufacturer
   - user facility
   - distributor

5. If you do NOT want your identity disclosed to the manufacturer, place an "X" in this box.
ADVICE ABOUT VOLUNTARY REPORTING

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

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

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

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

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

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

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

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

The public reporting burden for this collection of information has been estimated to average 30 minutes per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. Send your comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden to:

Reports Clearance Officer, PHS
Hubert H. Humphrey Building
Room 721-B
200 Independence Avenue, S.W.
Washington, DC 20201
ATTN: PRA

and to:
Office of Management and Budget
Paperwork Reduction Project (0910-0230)
Washington, DC 20503

Please do NOT return this form to either of these addresses.

Please Use Address Provided Below - Just Fold In Thirds, Tape and Mail

Department of Health and Human Services
Public Health Service
Food and Drug Administration
Rockville, MD 20857

Official Business
Penalty for Private Use $300

BUSINESS REPLY MAIL
FIRST CLASS MAIL PERMIT NO. 946 ROCKVILLE, MD
POSTAGE WILL BE PAID BY FOOD AND DRUG ADMINISTRATION

MEDWATCH
The FDA Medical Products Reporting Program
Food and Drug Administration
5600 Fishers Lane
Rockville, MD 20852-9787
AARC & AFFILIATES

June 14-16 in St. Charles, Illinois. The ISRC hosts its 27th Annual State Convention, featuring workshops that examine respiratory protocols, point-of-care blood gas testing, essential assessment skills, and how to improve your professional image. Contact Doug McQueary at (312) 962-4060 or Jane Reynolds at (312) 883-6535.

June 20—AARC Videoconference. The AARC, in conjunction with VHA Satellite Network, presents the third installment of the seven-part “Professor’s Rounds in Respiratory Care” videoconference series. This presentation, entitled “The Multidisciplinary Team Approach and Respiratory Care,” focuses on what RCPs must do to demonstrate value and create opportunity in the changing health care environment. Each videoconference in the series runs from 12:30 to 2 pm. Eastern Time; viewers of the live presentation receive 1 CRCE credit hour. For information, call VHA Satellite Network at (214) 830-0061.

June 23-24 in Atlanta, Georgia. The GSRC and the Georgia Thoracic Society present a professional education conference, in conjunction with the American Lung Association of Georgia, at the Ritz Carlton Hotel in Buckhead. Topics include pulmonary vascular disease and reorganization of respiratory care. Contact Lynda Thomas Goodfellow or Vijay Deshpande at Georgia State University, Department of Cardiopulmonary Care Sciences, University Plaza, Atlanta GA 30303, (404) 651-3037, fax (404) 651-1531.

July 1—August 31, in Indianapolis, Indiana. The AARC and Indiana University present Module 3 of the Management Training Institute program; the enrollment deadline for the program is June 1. Those unable to attend the AARC Convention seminars can complete Modules 1-8 through this program to receive the MTI Certificate in Management Development. Module 3 consists of Human Resources Management Part 1 and Managerial Accounting Part 2. To enroll, fill out the MTI Enrollment Form in the May issue of AARC Times and mail it to Barbara Hakes, IUPUI Division of Continuing Studies, 620 Union Dr, Suite 318, Indianapolis IN 46202-5171, or call (317) 274-4475.

July 14-16 in Vail, Colorado. The AARC presents the Summer Forum, with emphasis on management and education topics. Consult the April issue of AARC Times for program details and registration information.

August 23-25 in Albuquerque, New Mexico. The NMSRC announces its Annual Summer Convention at the Albuquerque Convention Center. The program features an I.V. class and a bronchoalveolar lavage class; other topics include postacute care and pressure-control ventilation. Contact Brooke Patterson of THC Hospital at (505) 242-4444.

OTHER MEETINGS

June 16-19 in Toronto, Ontario, Canada. The Canadian Society of Respiratory Therapists (CSRT) announces its 30th Educational Forum at the Royal York Hotel. Workshops cover management, ventilation, cardiopulmonary diagnostics, lung transplantation, and research. Call (416) 368-2511 or fax (416) 368-2884 for more information.


October 12-15 in Minneapolis, Minnesota. The American Association of Cardiovascular and Pulmonary Rehabilitation hosts its 10th Annual Meeting, “Building on Success—A Decade of Progress,” at the Minneapolis Convention Center. For registration and hotel information, contact Michele Johnson (608) 831-6989.

### Authors in This Issue

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**RESPIRATORY CARE**

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Expires September 30, 1995

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**AARC Information Requests or Change of Address**

Please complete the card below

**AARC Membership No.**

**Old Address**

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Check the boxes below for information from the AARC

- Change of address
- AARC Membership Info
- AARC Catalog
- AARC Position Statement
Summer Forum Is Approaching

Start planning now to bring the whole family and join your RC colleagues in beautiful Vail, CO, July 14–16. Look for registration and program information in AARC Times.

△

See you there!
PULMONARY FUNCTION/VENTILATION MONITOR
Graphic Printouts...Multi-Patient Memory...and Easy to Use

Results-Oriented Features At Cost Effective Prices

- New Graphic Forced Vital Capacity (FVC) document printout of Flow vs Volume and Volume vs Time
- New 10 patient memory with 8 pre-bronchodilator and 8 post-bronchodilator tests per patient and automatic calculation of % change
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- New Slow Vital Capacity (SVC) monitoring
- Automatic determination of “best test”
- Knudson, ITS and ECCS reference nomograms
- Easy to operate

Performs A Complete Range Of Test Measurements

Forced Exhalation Parameters

- Forced Vital Capacity (FVC)
- Forced Expiratory Volume in One Second (FEV₁)
- FEV₁/FVC Ratio
- FVC Time
- Peak Flow
- Forced Expiratory Flow Between 25% and 75% of Vital Capacity (FEF 25-75%)
- Percent Extrapolated Volume (Vol. EXTRA)

Weaning/Extubation Parameters

- Respiratory Rate (RR)
- Tidal Volume (TV)
- Minute Volume (MV)
- Slow Vital Capacity (SVC)
- Maximum Voluntary Ventilation (MVV)
- Negative Inspiratory Force (NIF)

For further information, call:
1-800-325-7472 (outside Missouri)
1-800-392-7318 (in Missouri)

Circle 157 on reader service card