2001 Open Forum
Call for Abstracts
Deadline July 17

ORIGINAL CONTRIBUTIONS
Effects of Continuous, Expiratory, Reverse, and Bi-Directional TGI on Delivered Tidal Volume, Total-PEEP, and CO₂ Elimination

High-Frequency Flow Interruption Ventilation vs Hyperventilation in Persistent Pulmonary Hypertension of the Newborn

Improved Quality of Life Among Patients Completing a Pulmonary Rehabilitation Program

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ORIGINAL CONTRIBUTIONS

Effects of Continuous, Expiratory, Reverse, and Bi-Directional Tracheal Gas Insufflation in Conjunction with a Flow Relief Valve on Delivered Tidal Volume, Total Positive End-Expiratory Pressure, and Carbon Dioxide Elimination: A Bench Study
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Bronchial Atresia with Relapsing Pulmonary Infection in a Middle-Aged Man
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SPECIAL ARTICLES

A New System for Understanding Modes of Mechanical Ventilation
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Frank P Premiano Jr—Philadelphia, Pennsylvania

BOOKS, FILMS, TAPES, & SOFTWARE

Pulmonary Diseases (Grassi C, editor)
reviewed by Duncan Powrie—London, United Kingdom

Bone's Atlas of Pulmonary and Critical Care Medicine (Bone RC, Campbell GD Jr, Payne DK, editors)
reviewed by William Thompson—Boise, Idaho

Fungal Diseases of the Lung, 3rd ed (Sarosi GA, Davies SF)
reviewed by Kieren A Marr—Seattle, Washington

Allergic Diseases: Diagnosis and Treatment, 2nd ed (Lieberman P, Anderson JA, editors)
reviewed by Rick Lane Johnson—Seattle, Washington

Principles and Practice of Sleep Medicine, 3rd ed (Kryger MHI, Roth T, Dement WC, editors)
reviewed by Noel T Johnson—Seattle, Washington
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COMING IN NOVEMBER & DECEMBER:
Proceedings from the 28th RESPIRATORY CARE Journal Conference on

Evidence-Based Medicine in Respiratory Care

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Taking the Mystery Out of Weaning the Pediatric Patient from the Ventilator

Peter Beint, BS, RRT, FAARC, and Richard D. Branson, BA, RRT, FAARC

Learn when to begin the process and how to recognize critical events in weaning a pediatric patient. Also teaches the physiological differences between the adult and pediatric patient and why weaning of the pediatric patient is different. The presentation confronts participants with options in providing assisted ventilation and the correct selection of options that expedite weaning.

Videotape available

Noninvasive Ventilation: The Latest Word

Dean R. Hess, PhD, RRT, FAARC, and Richard D. Branson, BA, RRT, FAARC

Learn how to avoid intubation in the acutely ill patient through identification of patients most likely to benefit from noninvasive ventilation. Learn selection and proper fit of full masks or nasal masks and how to select the proper ventilator based on the patient's condition and desired outcomes. Also learn when to make adjustments to achieve the goals of unloading respiratory muscles and achieving good patient/ventilator synchrony.

Live Videoconference - June 26, 11:30 a.m. - 1:00 p.m. Central Time
Teleconference with Videotape - July 17, 11:30 a.m. - 12:00 Noon Central Time

ARDS: The Disease and Its Management

Leonard D. Hudson, MD, and David J. Pierson, MD, FAARC

Presents the four diagnostic criteria for ARDS and the six clinical risk factors that place patients at increased likelihood for developing ARDS. The program will help viewers understand the implications of the lower and upper inflection points on the pressure-volume curve of the respiratory system and instruct them in the calculation of estimated required tidal volume.

Live Videoconference - Sept. 25, 11:30 a.m. - 1:00 p.m. Central Time
Teleconference with Videotape - Oct. 16, 11:30 a.m. - 12:00 Noon Central Time

Invasive Ventilation: The Latest Word

Richard Kallet, MS, RRT and Richard D. Branson, BA, RRT, FAARC

Learn how proper ventilator management can preclude inflicting harm on the patient and why it is essential for the clinician to understand the function and mechanics of newer mechanical ventilators. Also learn how reducing the patient's work of breathing is essential in reducing the additional load on ventilatory musculature, and how reinfusing lungs and enhancing the functional area of the lungs demands extraordinary means.

Live Videoconference - Sept. 25, 11:30 a.m. - 1:00 p.m. Central Time
Teleconference with Videotape - Oct. 16, 11:30 a.m. - 12:00 Noon Central Time

Pulmonary Rehabilitation: Standard Care for Chronic Lung Disease Patients

Trina Limberg, BS, RRT, and Thomas J. Kallstrom, RRT, FAARC

Presentation details when to refer a patient for pulmonary rehabilitation and the four elements necessary for the successful operation of a rehabilitation service. Details how to prepare a treatment plan during assessment and how to modify it based on subsequent evaluations as well as how to incorporate rehabilitation techniques into routine bedside therapy sessions.

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Education of the Patient with Asthma

Tracey Mitchell, RRT, RPFT, and Thomas J. Kallstrom, RRT, FAARC

This program teaches how to ensure that patients understand the disease process of asthma and their care plan for effective disease management. And, it details what patient education materials are available, their content, where to find them, and the best methods of presentation, including new terminologies, analogies, and techniques.

Sponsored in part by an educational grant from Sepracor, Inc.

Live Videoconference - May 22, 11:30 a.m. - 1:00 p.m. Central Time
Teleconference with Videotape - June 19, 11:30 a.m. - 12:00 Noon Central Time

New Respiratory Drugs: What, When, and How?

Joseph L. Rau, PhD, RRT, FAARC, and Patrick J. Dunne, MD, RRT, FAARC

Introduces participants to new formulations such as racemic drug mixtures and single isomers and their effective duration and how they lead to fewer costs with improved patient responses. Viewers will learn the use of improved anticholinergics in the treatment of asthma patients and learn the uses and effects of inhaled anti-inflammatory agents.

Sponsored in part by an educational grant from Sepracor, Inc.

Live Videoconference - Aug. 14, 11:30 a.m. - 1:00 p.m. Central Time
Teleconference with Videotape - Sept. 11, 11:30 a.m. - 12:00 Noon Central Time

Test Your Lungs, Know Your Numbers, Prevent Emphysema

Thomas L. Petty, MD, FAARC and David J. Pierson, MD, FAARC

Reviews the classic signs of COPD with an emphasis on emphysema and a discussion on the measures used to relieve symptoms and slow disease progression. Covers the importance of pulmonary function tests to determine VC, FVC, and FEV1; and why getting patients to know their numbers is the key to early diagnosis and successful treatment.

Live Videoconference - Oct. 23, 11:30 a.m. - 1:00 p.m. Central Time
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BACKGROUND: Chronic obstructive pulmonary disease (COPD) results from a progressive decline in lung function, which is thought to be the consequence of airway inflammation. We hypothesized that antiinflammatory therapy with inhaled corticosteroids would slow this decline. METHODS: We enrolled 1116 persons with COPD whose forced expiratory volume in one second (FEV1) was 30 to 90 percent of the predicted value in a 10-center, placebo-controlled, randomized trial of inhaled triamcinolone acetonide administered at a dose of 600 microg twice daily. The primary outcome measure was the rate of decline in FEV1 after the administration of a bronchodilator. The secondary outcome measures included respiratory symptoms, use of health care services, and airway reactivity. In a substudy of 412 participants, we measured bone density in the lumbar spine and femur at baseline and one and three years after the beginning of treatment. RESULTS: The mean duration of follow-up was 40 months. The rate of decline in the FEV1 after bronchodilator use was similar in the 539 participants in the triamcinolone group and the 557 participants in the placebo group (mean [±SE], 44.2 ± 2.9 vs. 47.0 ± 3.0 mL per year, p=0.50). Members of the triamcinolone group had fewer respiratory symptoms during the course of the study (21.1 per 100 person-years vs. 28.2 per 100 person-years, p=0.005) and had fewer visits to a physician because of a respiratory illness (1.2 per 100 person-years vs. 2.1 per 100 person-years, p=0.03). Those taking triamcinolone also had lower airway reactivity in response to methacholine challenge at 9 months and 33 months (p=0.02 for both comparisons). After three years, the bone density of the lumbar spine (p=0.007) and the femur (p<0.001) was significantly lower in the triamcinolone group. CONCLUSIONS: Inhaled triamcinolone does not slow the rate of decline in lung function in people with COPD, but it improves airway reactivity and respiratory symptoms and decreases the use of health care services for respiratory problems. These benefits should be weighed against the potential long-term adverse effects of triamcinolone on bone mineral density.

Medical education is often a frustrating endeavor, particularly when it attempts to change practice behavior. Traditional lecture-based educational methods are limited in their ability to sustain concentration and interest and to promote learner adherence to best-practice guidelines. Marketing techniques have been very effective in changing consumer behavior and physician behavior. However, the techniques of social marketing—goal identification, audience segmentation, and market research—have not been harnessed and applied to medical education. Social marketing can be applied to medical education in the effort to go beyond inoculation of learners with information and actually change behaviors. The tremendous potential of social marketing for medical education should be pilot-tested and systematically evaluated.


BACKGROUND: The lightwand may be useful as an alternative for tracheal intubation during a rapid-sequence induction of anesthesia in the presence of a full stomach. This study was undertaken to assess the effect of application of cricoid pressure on the success of lightwand intubation. METHODS: Sixty adult female patients presenting for abdominal hysterectomy were randomly allocated to lightwand intubation with and without cricoid pressure. The time to successful intubation and number of attempts were recorded. RESULTS: All 30 patients allocated to intubation without cricoid pressure were intubated successfully at the first attempt within a median time of 28 s (95% confidence interval, 18-77 s). Lightwand intubation with cricoid pressure was successful in 26 of 30 patients at the first attempt, but the median time to successful intubation was significantly longer at 48.5 s (95% confidence interval, 36-78 s; p = 0.001). Three patients required two attempts for successful intubation, and one could not be intubated with the lightwand while cricoid pressure was being applied. CONCLUSIONS: The lightwand cannot be recommended for the first attempt at intubation where cricoid pressure is being applied because the time to successful intubation is significantly prolonged, and the failure rate for the first attempt at lightwand intubation is 13%.
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CONTEXT: Focal pulmonary lesions are commonly encountered in clinical practice, and positron emission tomography (PET) with the glucose analog 18-fluorodeoxyglucose (FDG) may be an accurate test for identifying malignant lesions. OBJECTIVE: To estimate the diagnostic accuracy of FDG-PET for malignant focal pulmonary lesions. DATA SOURCES: Studies published between January 1966 and September 2000 in the MEDLINE and CANCERLIT databases; reference lists of identified studies; abstracts from recent conference proceedings; and direct contact with investigators. STUDY SELECTION: Studies that examined FDG-PET or FDG with a modified gamma camera in coincidence mode for diagnosis of focal pulmonary lesions; enrolled at least 10 participants with pulmonary nodules or masses, including at least 5 participants with malignant lesions; and presented sufficient data to permit calculation of sensitivity and specificity were included in the analysis. DATA EXTRATION: Two reviewers independently assessed study quality and abstracted data regarding prevalence of malignancy and sensitivity and specificity of the imaging test. Disagreements were resolved by discussion. DATA SYNTHESIS: We used a meta-analytic method to construct summary receiver operating characteristic curves. Forty studies met inclusion criteria. Study methodological quality was fair. Sample sizes were small and blinding was often incomplete. For 1474 focal pulmonary lesions of any size, the maximum joint sensitivity and specificity (the upper left point on the receiver operating characteristic curve at which sensitivity and specificity are equal) of FDG-PET was 91.2% (95% confidence interval, 89.1%–92.9%). In current practice, FDG-PET operates at a point on the summary receiver operating characteristic curve that corresponds approximately to a sensitivity and specificity of 96.8% and 77.8%, respectively. There was no difference in diagnostic accuracy for pulmonary nodules compared with lesions of any size (p = 0.43), for semiquantitative methods of image interpretation compared with qualitative methods (p = 0.52), or for FDG-PET compared with FDG imaging with a modified gamma camera in coincidence mode (p = 0.19). CONCLUSIONS: Positron emission tomography with 18-fluorodeoxyglucose is an accurate noninvasive imaging test for diagnosis of pulmonary nodules and larger mass lesions, although few data exist for nodules smaller than 1 cm in diameter. In current practice, FDG-PET has high sensitivity and intermediate specificity for malignancy.


The aim of the study was to compare the lung sounds in patients with asbestos related pulmonary disorders with findings in high-resolution computed tomography (HRCT), and with lung function variables, in order to find out associations of acoustic changes with radiological fibrosis, emphysema, or with pulmonary gas transfer factors. Sixty-four patients with asbestos related pleural disease, with or without pulmonary disease, were studied. Lung sound recording and analysis was carried out with a computerized lung sound analyzer, and HRCT of the chest, as well as forced spirometry and diffusing capacity measurement were performed. The fibrosis score correlated positively with the quartile frequencies of the power spectrum of lung sounds in inspiration (f50) and expiration (f50) and crackle count in inspiration, as well as negatively with diffusing capacity. When the patients with crackling sounds and significant fibrosis were excluded (n = 18), emphysenema correlated positively with respiratory quartile frequencies of the power spectrum, with f25 and f50. Furthermore, diffusing capacity correlated with inspiratory f25 and forced expiratory volume in one second with inspiratory f50 when crackles and
fibrosis were excluded. Changes in lung sounds were significantly associated with radiologically verified abnormalities and gas transfer of pulmonary tissue. High sound frequencies were associated with fibrotic changes of the lung while low sound frequencies with pulmonary emphysema. Acoustic analysis gives complementary clinical information for evaluation of asbestosis-related pulmonary disorders.

**Effect of Moderate Alcohol upon Obstructive Sleep Apnoea—Scan-**


Moderate-to-large quantities of alcohol are known to aggravate severe obstructive sleep apnoea (OSA), however, the reported effects of moderate alcohol consumption upon mild-to-moderate OSA are inconsistent. Given the reported benefits of moderate alcohol consumption on cardiovascular mortality, recommendations regarding the management of patients with OSA are difficult to formulate. The aim of this study was to evaluate the effects of moderate alcohol on sleep and breathing in subjects with mild-to-moderate OSA. Twenty-one male volunteers, who snored habitually, underwent polysomnography with and without 0.5 g alcohol x kg body weight (BW) consumed 90 min prior to sleep time, in random order. The mean blood alcohol concentration (BAC) following alcohol at the time of lights out was 0.07 g x dl^-1. The distribution amongst the various sleep stages was not significantly altered by alcohol. The mean apnoea/hypopnoea index rose from 7.1 ± 1.9 to 9.7 ± 2.1 events x h^-1 (mean ± SEM, p = 0.017); however, there was no significant change in the minimum arterial oxygen saturation measured by pulse oximetry SpO₂, apnoea length or snoring intensity. Mean sleep cardiac frequency rose significantly from 53.9 ± 1.4 to 59.0 ± 1.9 beats x min^-1 (p < 0.001) and overnight urinary noradrenaline increased from 14.9 ± 2.3 to 18.8 ± 2.3 nmol x min creatinine^-1 (p = 0.061) on the alcohol night compared to the nonalcohol night. To conclude, moderate alcohol consumption, giving a mean alcohol blood concentration of 0.07 g x dl^-1, significantly increases both obstructive sleep apnoea frequency and mean sleep cardiac frequency.


Asthma management guidelines provide recommendations for the optimum control of asthma. This survey assessed the current levels of asthma control as reported by patients, which partly reflect the extent to which guideline recommendations are implemented. Current asthma patients were identified by telephone by screening 73,880 households in seven European countries. Designated respondents were interviewed on healthcare utilization, symptom severity, activity limitations and asthma control. Current asthma patients were identified in 3,488 households, and 2,803 patients (80.4%) completed the survey. Forty-six per cent of patients reported daytime symptoms, and 30% reported asthma-related sleep disturbances, at least once a week. In the past 12 months, 25% of patients reported an unscheduled urgent care visit, 10% reported one or more emergency room visits and 7% reported overnight hospitalization due to asthma. In the past 4 weeks, more patients had used prescription quick-relief medication (63%) than inhaled corticosteroids (23%). Patient perception of asthma control did not match their symptom severity; approximately, 50% of patients reporting severe persistent symptoms also considered their asthma to be completely or well controlled. The current level of asthma control in Europe falls far short of the goals for long-term asthma management. Patients’ perception of asthma control is different from their actual asthma control.

**An Auto-Continuous Positive Airway Pressure Device Controlled Exclusively by the Forced Oscillation Technique—Fiecker HJ, Fuchs**


The forced oscillation technique (FOT) has been demonstrated to be a very sensitive tool for the assessment of upper airway obstruction during nasal continuous positive airway pressure (CPAP) therapy for obstructive sleep apnoea (OSA). The present study was designed to evaluate the therapeutic efficacy of a novel auto-CPAP device based exclusively on the FOT. Following manual CPAP titration, 18 patients with OSA (mean apnoea/hypopnoea index (AHI) 48.0 ± 28.1) were allocated to conventional CPAP and auto-CPAP treatment under polysomnographic control in randomized order. The patients were asked to assess their subjective daytime sleepiness using the Epworth Sleepiness Scale (ESS). The mean AHI during auto-CPAP treatment was 3.4 ± 3.4 and was comparable with that obtained during conventional CPAP treatment (4.2 ± 3.6). The analysis of sleep architecture, the arousal index (6.6 ± 2.1 versus 7.3 ± 4.4) or the ESS (5.6 ± 1.8 versus 7.3 ± 4.4) did not reveal any significant differences. However, the mean CPAP pressure during auto-CPAP treatment (8.4 ± 2.6 kPa) and in particular the pressure applied in the lateral body position (7.4 ± 0.35 kPa), was significantly lower than that employed in conventional CPAP treatment (9.9 ± 0.16 kPa; both comparisons: p < 0.05). The auto-continuous positive airway pressure device proved equally as effective as conventional continuous positive airway pressure. However, the mean treatment pressure was significantly reduced, especially when patients were sleeping in the lateral position.


Correct assessment of the overall treatment effectiveness requires knowledge about therapy compliance and efficacy. This study aimed to determine overall long-term apnoea alleviation after continuous positive airway pressure (CPAP) in a complete sleep laboratory cohort. Out of 209 consecutive CPAP candidates (mean age 57 ± 12 yrs, body mass index (BMI) 30.9 ± 5.1 kg m^-2, respiratory disturbance index (RDI) 32.9 ± 29 h), follow-up treatment was performed in 149 of them at 9, 18 and 30 months after CPAP prescription. Compliance with CPAP (machine run time/days CPAP available) was adjusted for the individual subjective sleep-time. Apnoea alleviation was defined as adjusted compliance multiplied by the CPAP effect (RDI with CPAP applied), remaining RDI was calculated. The baseline RDI, age or BMI in 75 patients, who did not tolerate nasal continuous positive airway pressure (nCPAP), did not differ from those accepting CPAP (acceptors, n = 74). In acceptors at 9 months follow-up RDI with CPAP applied was 1.4 ± 2.6 (CPAP effect, n = 66), mean CPAP use was 3.6 ± 2.5 x 24 h^-1 (n = 68), mean apnoea alleviation was 52.4 ± 32.0% (range 1–100%), n = 47), the average remaining whole-night RDI was 17.8 ± 26. At 9, 18 and 30 months (n = 47), the mean daily CPAP use increased from 3.6 ± 2.5 to 4.1 ± 2.5 and 4.4 ± 2.4 h (p < 0.01). Effectiveness of continuous positive airway pressure is potentially high but acceptance was low. When accounting for sleep-time, its actual effect and use, only 50% adjusted continuous positive airway pressure effectiveness was observed.


Mandibular advancing devices are proposed as nonsurgical treatment for certain patients with an obstructive sleep apnoea syndrome. Since they act by increasing the upper airway calibre, the aim of the present study was to investigate the changes in respiratory resistance (Rt) resulting from mandibular advancement. Rt was measured at the nose by the forced oscillation technique (4–32 Hz). Ten normal subjects were studied under three conditions: resting mandibular position, passive mandibular
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advancement stealed by a wax buc, and voluntary advancement, in random order. Respiratory resistance was extrapolated to 0 Hz (R0) and estimated at 16 Hz (R16) by linear regression analysis of respiratory
resistive impedance versus frequency. R0 (mean±SEM=3.5±0.2 cm
H2O×L−1×s in the resting position) decreased significantly with
passive advancement (2.9±0.2 cm H2O×L−1×s, p<0.001), but re-
mained unchanged with voluntary mandibular advancement (3.6±0.2 cm
H2O×L−1×s). Similar results were obtained for R16. The results of this
study demonstrate that the effects of mandibular advancement on upper
airway resistance differ, depending on whether advancement is passive or
active, and suggest that in order to simulate the actual effects of ther-
apeutic devices, mandibular advancement should be passive.

A Nationwide Survey in Germany on Fatal Asthma and Near-Fatal
Asthma in Children: Different Entities?—Schmitz T, von Kries R,

In adults fatal and near-fatal asthma have similar clinical characteristics.
Therefore, near-fatal asthma in adults can be used as a model for fatal
asthma. A nationwide study on fatal and near-fatal asthma in children<16 yrs was performed in order to assess whether, as in adults, near-fatal
asthma can be used as a model for fatal asthma. From 1996 to 1998, all
paediatric hospitals and paediatric pulmonologists in Germany were asked
to report cases of fatal asthma and near-fatal asthma to a central survey
unit (Erhebungseinheit für seltene padiatrische Erkrankungen in
Deutschland (ESPED)) on a monthly basis. All reports were followed by
detailed questionnaires. Sixteen fatal and 45 near-fatal asthma cases were
analysed. Fatal asthma patients were older than near-fatal asthma pa-
tients. Respiratory tract infections were frequently reported only in near-
fatal asthma (47 versus 0%). The proportion of cases with rapid-type
onset (duration of symptoms ≤1 h) was higher in fatal asthma (53 versus
14%). Long-term regular treatment with short acting β2-agonists was
common in both groups, but the use of concomitant inhaled corticoste-
roids was significantly lower in fatal asthma cases. A high proportion
of poor compliance was observed in both groups. As fatal and near-fatal
asthma differ significantly in important clinical aspects, analysis of near-
fatal asthma might be of limited value in elucidating the causes of fatal
asthma in children.

Aerosol Delivery from Spacers in Wheezy Infants: A Daily Life
Study—Janssens HM, Heijmen EM, de Jong VM, Hop WC, Holland WP,

The aims of this study were to assess and compare dose delivery and dose
variability of pressurized metered dose inhalers (pMDIs)/spacers in wheezy
infants in daily life and to investigate factors influencing aerosol delivery.
In an open randomized crossover study in 25 wheezy infants aged 5–26 months, a metal spacer (Nebuhaler), a detergent coated (DC)
and a non-detergent coated (nonDC) plastic spacer (Babyhaler) were
tested at home for 7 days each. Budesonide (200 mcg b.i.d) was
administered via a Nebuhaler or fluticasone (125 microg b.i.d) via a
Babyhaler. Aerosol was trapped in filters, positioned between the spacer
and face mask. Cooperation was scored on diary cards. Electrostatic
charge (ESC) of the spacers was measured. Evaluations of the admin-
istration technique were made from video recordings. Median (range)
dose delivery of the filters expressed as per cent (% of nominal dose, was
34% (3–59), 23% (11–49), and 41% (12–55) for the Nebuhaler, nonDC,
Babyhaler, and DC-Babyhaler respectively. Considerable dose variabil-
ity was found, median (range) within-subject dose variability, expressed
comment higher when compared with both nonDC (36% (12–325))
and DC-Babyhalers (27% (10–122)), for which dose variabilities were
similar. Detergent coating was effective to reduce electrostatic charge,
and to increase dose delivery, but had no effect on dose variability. Bad
cooperation was an important cause for high dose variability for all
spacers (r=0.5–0.6, p<0.02). Many mistakes were made during the ad-
ministration procedure.

Success and Safety of Sputum Induction in the Clinical Setting—
Vlahos-Maycr R, Leigh R, Sharon RF, Hussack P, Hargrave FE. Eur

It has previously been reported that sputum induction is successful and
safe in the clinical research setting. The authors examined the success
and safety of sputum induction in routine clinical practice in patients with
asthma or chronic airflow limitation of varying severity. Records of 304
patients with asthma and 25 with smoking related chronic airflow limi-
tation were examined retrospectively. All had sputum induced as part of
their routine clinical evaluation. When the baseline post salbutamol forced
expiratory volume in one second (FEV1) was ≥70% predicted, the in-
ductions consisted of inhalation of an aerosol of 3%, 4% and 5% saline,
each given for 7 min. If the FEV1 was <70%, or there were other reasons
for concern, the inductions were initiated with normal saline for shorter
periods. Inhalations were discontinued when sputum was obtained or
when there was a fall in FEV1 ≥20%. Success was identified by obtain-
ing nonsquamous total and differential cell counts containing macro-
phages, and safety by the fall in FEV1. The overall success was 93%. The
procedure was safe even amongst patients with an FEV1 of ≤60% and
<1 L. Of 77 patients with an FEV1 between 40–59%, 8% fell by ≥20%
and of 35 patients with an FEV1 <40%, 6% fell by 20%. Carefully
standardized sputum induction can be successful and safe in patients with
asthma or chronic airflow limitation in clinical practice, even when moder-
ate or severe airflow limitation is present.

Direct Medical Cost of Chronic Obstructive Pulmonary Disease in
the U.S.A.—Ward MM, Javitz HS, Smith WM, Bakst A. Respir Med

The aim of this study was to estimate the direct medical costs of chronic
obstructive pulmonary disease (COPD) in the United States using a pub-
clic-payor perspective. Cost estimates were derived separately for 10 com-
ponents of care using national survey databases and valued using Medi-
care and Medicaid reimbursement rates. COPD affects 15 million people
in the U.S.A. and the total annual U.S. payment for care is $6.6 billion.
Approximately one-third ($2.3 billion) is due to the cost of long-term
oxygen therapy, one-quarter is attributed to hospitalizations and inpatient
physician services ($1.9 billion), and one-seventh ($0.94 billion) is due
to nursing home stays. Other annual costs are outpatient physician visits
($480 million), prescription medications ($462 million), home healthcare
($309 million), emergency department visits ($148 million), outpatient
diagnostic procedures ($55 million) and hospice care ($28 million).
The cost of COPD is therefore considerable. The significant expenditure
for long-term oxygen therapy indicates that disease severity is a major driver
of costs. However, the cost of hospitalizations, nursing home stays, emer-
genous department and physician visits are not insignificant.

Changes in Carbon Monoxide Transfer over 22 Years in Middle-

We measured single breath CO transfer (TCO2), single breath alveolar
volume (VA), CO transfer coefficient (KCO), and forced expiratory
volume in 1 sec (FEV1) in 84 men, mean age 40.5 years at recruitment,
in 1975 and in 1997. At recruitment, 42 men were cigarette smokers and 42
were not smoking. Mean annual decline in FEV1 was similar in never-
(34.2 mL yr−1) and ex-smokers (33.1 mL yr−1) smokers and faster (51.0 mL yr−1)
in continuing smokers. In contrast to predictions from cross-sectional
reference values, there was no fall in TCO2 or KCO in men who did not
smoke over the period of follow-up. In the 16 men who smoked through-
out follow-up there was a 10% fall in TCO2 (p = 0.043) but most of this

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was due to a significant fall in VA \( p = 0.017 \), presumably reflecting uneven gas mixing. These results indicate the need for population-based longitudinal studies of \( T_{1/2}O \) and \( K_{1/2}O \). If single breath estimates of VA are used in subjects with even mild airflow obstruction, \( K_{1/2}O \) rather than \( T_{1/2}O \) should be used to assess alveolar function.

**Abstracts**


The aim of this study was to analyse the correlates of reduced bone mineral density in patients with chronic obstructive pulmonary disease (COPD), with special regard to a possible protective role of hypercapnia. One hundred and four consecutive COPD inpatients in stabilized respiratory condition underwent a comprehensive assessment of their health status. Bone mineral density was measured by x-ray absorptiometry at the lumbar site and at the femoral neck site. Differences in health-related variables between patients with \( \text{group O, n}=62 \) and without \( \text{group N, n}=42 \) lumbar and/or femoral neck osteoporosis were assessed first by univariate analysis and then by logistic regression analysis aimed to identify independent correlates of osteoporosis. Group \( \text{O} \) was characterized by worse nutritional status, as reflected by indices expressing either lean or fat mass, and by a trend towards lower forced expiratory volume in 1 sec/forced vital capacity ratio. Arterial tension of carbon dioxide lacked any correlation with bone mineral density. According to the logistic regression analysis, body mass index \( \leq 22 \text{ kg m}^{-2} \) qualified as the only and positive independent correlate of osteoporosis (odds ratio = 4.18; 95% confidence intervals = 1.19–14.71). In conclusion, malnutrition characterizes COPD patients with osteoporosis, while mild to moderate hypercapnia lacks either a positive or negative effect on bone mineral density. Longitudinal studies are needed to identify predictors rather than correlates of bone mineral density.


Patient satisfaction with general practitioners (GP) and pulmonary outpatient clinics has not been previously compared in patients with asthma and chronic obstructive pulmonary disease (COPD) in addition to the effect of patient education on this satisfaction. We randomly allocated 78 asthmatics and 62 patients with COPD after ordinary outpatient management to a control or an intervention group. Intervention consisted of educational group sessions and individual sessions administered by a trained nurse and physiotherapist. A self-management plan was developed. A patient satisfaction questionnaire was answered at baseline and at the 1-year follow-up. Before randomization, a higher proportion of asthmatics were satisfied with the overall handling of their disease by the outpatient clinic (86%) compared with their GPs (72%, \( p=0.027 \), chi²-test). Equal and high proportions of patients with COPD were satisfied with both their GPs (85%) and the outpatient clinic (87%) and in general seemed more satisfied with their GP than asthmatics \( (p=0.064) \). At the 1 year follow-up, 100% of the educated patients with COPD reported overall satisfaction with GPs compared with 78% in the control group \( (p=0.023) \), but not for asthmatics (75% and 78%, respectively, \( p=0.381 \)). We conclude that before being given education, asthmatics are more satisfied with the pulmonary outpatient clinic than with GPs, regarding the overall handling of their disease. Patients with COPD seemed more satisfied with GPs than asthmatics. For patients with COPD, patient education seemed to improve overall patient satisfaction with GPs, but this was not true for asthmatics. At baseline, overall satisfaction with the outpatient clinic was so beneficial that we had little chance of detecting any improvement.


The objective of this study was to describe asthma exacerbation self-management in children and adolescents. We used a cross-sectional study population enrolled in the International Study of Asthma and Allergies in Childhood (ISAAC) in Bordeaux. Subjects answered an additional questionnaire on utilization of health services, self-evaluation of usual asthma exacerbation severity and home management of asthma exacerbation. Criteria used for selecting patients were both having asthma confirmed by a physician and having had suffered from symptoms during the past year. Children and adolescents attended similar health services for managing their asthma but compliance to anti-asthmatic treatment was better in children than in adolescents. Among the children 4.8% had asthma and 6.2% of adolescents had asthma, as diagnosed by a doctor. Of the children, 72.3% and of the adolescents 54.7% had less than one asthma attack per month. In cases of mild asthma exacerbation, 38.7% of adolescents and 9.3% of children waited until the end of exacerbation without taking any medication. The proportion of children not receiving any treatment was lower when symptoms were more severe but this was not the case in adolescents. Although most of the patients used were taking \( \beta_2 \)-agonists, we found that 21–43% of children or adolescents did not receive appropriate medication in the event of asthma exacerbation. These results demonstrate that (i) asthma exacerbation self-management is related to self-assessed severity of symptoms and that (ii) a large proportion of asthmatic children in the community, and particularly adolescents, do not therefore receive appropriate treatment in the event of asthma exacerbation.


Oestrogen and progesterone have been shown to have impact on cystic fibrosis transmembrane conductance regulator (CFTR) gene expression, tone of smooth muscle in the airways, immune response, exhaled nitric oxide and cytology in the tracheobronchial epithelium. The aim of this investigation was to study the influence of menstrual cyclicity on airway symptoms among cystic fibrosis (CF) females. Twelve CF women (mean age 30 years, mean Shwachman score 85) kept daily records during three menstrual cycles of lung function, sputum quality and need for intravenous antibiotics. Paired t-test was used as a statistical method to compare the airway symptoms between the time of ovulation (high levels of oestrogen and low levels of progesterone), the luteal phase (high levels of oestrogen and progesterone) and menstruation (low levels of oestrogens and progesterone). Forced expiratory volume in 1 sec (FEV1) was significantly higher during the luteal phase \( (p=0.027) \) compared to during ovulation \( (p=0.027) \) and menstruation \( (p=0.01) \). Forced vital capacity (FVC) showed the same pattern, being significantly higher during the luteal phase compared with during menstruation \( (p=0.01) \). In conclusion, lung function changes were found during menstrual cycles in women with cystic fibrosis. These changes are probably related to changes in progesterone levels during the menstrual cycles. This result warrants further studies to understand the complexity of CF lung disease in women.


BACKGROUND: Induction of hypothermia in patients with brain injury was shown to improve outcomes in small clinical studies, but the results were not definitive. To study this issue, we conducted a multicenter trial comparing the effects of hypothermia with those of normothermia in...
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Written by faculty from UCSD, one of the country's leading institutions in the development of protocols. Authors include Jan Phillips-Clar, BS, RRT; Richard Ford, BS, RRT; Timothy Morris, MD; and David Burns, MD. Softcover. 257 pages. Second Edition. Published in 2001.

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patients with acute brain injury. METHODS: The study subjects were 302 patients 16 to 65 years of age with coma after sustaining closed head injuries who were randomly assigned to be treated with hypothermia (body temperature, 33 degrees C), which was initiated within 6 hours after injury and maintained for 48 hours by means of surface cooling, or normothermia. All patients otherwise received standard treatment. The primary outcome measure was functional status six months after the injury. RESULTS: The mean age of the patients and the type and severity of injury in the two treatment groups were similar. The mean (±SD) time from injury to randomization was 4.3±1.1 hours in the hypothermia group and 4.1±1.2 hours in the normothermia group, and the mean time from injury to the achievement of the target temperature was 33 degrees C in the hypothermia group was 8.4±3.0 hours. The outcome was poor (defined as severe disability, a vegetative state, or death) in 57 percent of the patients in both groups. Mortality was 28 percent in the hypothermia group and 27 percent in the normothermia group (p=0.79). The patients in the hypothermia group had more hospital days with complications than the patients in the normothermia group. Fewer patients in the hypothermia group had high intracranial pressure than in the normothermia group. CONCLUSIONS: Treatment with hypothermia, with the body temperature reaching 33 degrees C within eight hours after injury, is not effective in improving outcomes in patients with severe brain injury.


Many patients with suspected pulmonary tuberculosis (PTB) do not produce sputum spontaneously or are smear-negative for acid-fast bacilli (AFB). We prospectively compared the yield of sputum induction (SI) and fiberoptic bronchoscopy with bronchoalveolar lavage (BAL) for the diagnosis of PTB in a region with a high prevalence of tuberculosis and human immunodeficiency virus (HIV) infection. Fifty seven percent (143 of 251) of patients had diagnoses of PTB, of whom 17% (25 of 143) were HIV seropositive. There were no significant differences in the yields of AFB smears or cultures whether obtained via SI or BAL. Among 207 HIV-seropositive patients, the AFB smear and mycobacterial culture results from specimens obtained by SI and BAL were in agreement in 97% (202 of 207) (kappa test = 0.92) and 90% (186 of 207) (kappa test = 0.78), respectively. Among HIV-seropositive patients the agreements between AFB smear and culture results for SI and BAL specimens were 98% (43 of 44) (kappa test = 0.93) and 86% (38 of 44) (kappa test = 0.69), respectively. We conclude that SI is a safe procedure with a high diagnostic yield and high agreement with the results of fiberoptic bronchoscopy for the diagnosis of PTB in both HIV-seronegative and HIV-seropositive patients.


Between the lower and the upper inflection point of a quasistatic pressure-volume (PV) curve, a segment usually appears in which the PV relationship is steep and linear (i.e., compliance is high, with minimal volume change per pressure change, and is constant). Traditionally it is assumed that when positive end-expiratory pressure (PEEP) and tidal volume (Vt) are titrated such that the end-inspiratory volume is positioned at this linear segment of the PV curve, compliance is constant over Vt during ongoing ventilation. The validity of this assumption was addressed in this study. In 14 surfactant-deficient piglets, PEEP was increased from 3 cm H2O to 24 cm H2O, and the compliance associated with a modified multiple-occlusion method at the different PEEP levels. With PEEP at approximately the lower inflection point, compliance was minimal in most lungs and decreased markedly over Vt, indicating overdistension. Compliance both increased and decreased within the same breath at intermediate PEEP levels. It is concluded that a PEEP that results in constant compliance over the full Vt range is difficult to find, and cannot be derived from conventional respiratory-mechanical analyses; nor does this PEEP level coincide with maximal gas exchange.


We compare two commonly used diagnostic approaches, one relying on plasma bicarbonate concentration and "anion gap," the other on "base excess," with a third method based on physicochemical principles, for their value in detecting complex metabolic acid-base disturbances. We analyzed arterial blood samples from 152 patients and nine normal subjects for pH, Pco2, and concentrations of plasma electrolytes and proteins. Ninety-six percent of the patients had serum albumin concentration <3 SD below the mean of the control subjects. In about one-sixth of the patients, base excess and plasma bicarbonate were normal. In a great majority of these apparently normal samples, the third method detected simultaneous presence of acidifying and alkalizing disturbances, many of them grave. The almost ubiquitous hypobulminemia confounded the interpretation of acid-base data when the customary approaches were applied. Base excess missed serious acid-base abnormalities in about one-sixth of the patients; this method fails when the plasma concentrations of the nonbicarbonate buffers (mainly albumin) are abnormal. Anion gap detected a hidden "gap acidosi" in only 31% of those samples with normal plasma bicarbonate in which such acidosis was diagnosed by the third method; when adjusted for hypobulminemia, it reliably detected the hidden abnormal anions. The proposed third method identifies and quantifies individual components of complex acid-base abnormalities and provides insights in their pathogenesis.


Permissive hypercapnia, involving tolerance to elevated Pco2, is associated with reduced acute lung injury (ALI), thought to result from reduced mechanical stretch, and improved outcome in ARDS. However, elaborately delivering inspired CO2 concentration alone (therapeutic hypercapnia) (TH) protects against ALI in ex vivo models. We investigated whether TH would protect against ALI in an in vivo model of lung ischemia-reperfusion (IR). Anesthetized open chest rabbits were ventilated (standard euparic settings), and were randomized to TH (Pco2 0.12) versus control (Pco2 0.00). Pco2 and arterial pH values achieved in the TH versus CON groups were 101 ± 3 versus 44.4 ± 4 mm Hg and 7.10 ± 0.03 versus 7.37 ± 0.03, respectively. Following left lung ischemia and reperfusion, TH versus control was associated with preservation of lung mechanics, attenuation of protein leakage, reduction in pulmonary edema, and improved oxygenation. Indices of systemic protection included improved acid-base and lactate profile, in the absence of systemic hypoxemia. In the TH group, mean BALF TNF-alpha levels were 3.5% of CON levels (p < 0.01), and mean 8-isoprostane levels were 30% of CON levels (p = 0.02). Western blot analyses demonstrated reduced lung tissue nitrotyrosine in TH, indicating attenuation of tissue nitration. Finally, preliminary data suggest that TH may attenuate apoptosis following lung IR. We conclude that in the current model TH is protective versus IR lung injury and mechanisms of protection include preservation of lung mechanics, attenuation of pulmonary inflammation, and reduction of free radical mediated injury. If these findings are confirmed in
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Arousal from sleep produces transient increases in systemic blood pressure, leading to the suggestion that repeated arousals are associated with a sustained increase in daytime blood pressure. Using data from the Wisconsin Sleep Cohort Study, a population-based study, we tested the hypothesis that sleep fragmentation is associated with elevated awake blood pressure. Sleep, breathing, and seated blood pressure measurements from 1,021 participants (age 42 ± 8 yr; 590 males) were analyzed. Sleep fragmentation was defined as the total number of awakenings and shifts to Stage 1 sleep divided by the total sleep time (sleep fragmentation index: SFI). To reduce the confounding influence of sleep-disordered breathing, which is related to both increased daytime blood pressure and sleep fragmentation, all participants with an apnea-hypopnea index (AHI) ≥ 1 were analyzed separately. Accounting for the influences of sex, age, body mass index, and antihypertensive medication use, the SFI was significantly associated with higher levels of awake systolic blood pressure in people with an AHI < 1; a 2 standard deviation increase in the SFI was associated with a 3.1 mm Hg rise in awake systolic blood pressure. In participants with an AHI ≥ 1, there was no independent association between the SFI and awake blood pressure after controlling for the influence of the AHI.


Epidemiological studies have implicated obstructive sleep apnea (OSA) as an independent comorbid factor in cardiovascular and cerebrovascular diseases. The recurrent episodes of occlusion of upper airways during the sleep result in pathophysiological changes that may predispose to vascular diseases, and we postulate that nitric oxide may be one of the mediators involved. This study investigates the levels of circulating nitric oxide (NO), measured as serum nitrites and nitrates, in the early morning in OSA subjects compared with control subjects, and the effect of overnight nasal continuous positive airway pressure (nCPAP) in OSA subjects. Thirty men with moderate to severe OSA (age = 41.9 ± 9.0; apnea-hypopnea index, AHI = 48.0 ± 18.1) were compared with 40 healthy men (age = 40.6 ± 5.4; AHI = 1.4 ± 1.2). Serum nitrite/nitrate levels were significantly lower in OSA subjects (OSA = 38.9 ± 22.9 microM, control subjects = 63.1 ± 47.5 microM, p = 0.015). There was significant negative correlation between serum nitrites/nitrates and the following parameters: AHI (r = -0.389, p = 0.001), oxygen desaturation time (r = -0.346, p = 0.004), and systolic blood pressure (BP) (r = -0.335, p = 0.005). Stepwise multiple linear regression with systolic or diastolic BP as the dependent variable identified serum nitrites/nitrates as the only significant correlate. Twenty-two OSA subjects had measurements of serum NO at baseline and after an overnight application of nCPAP. There was significant increase in serum NO after nCPAP (baseline = 30.5 ± 14.4 microM, after nCPAP = 81.0 ± 82.1 microM, p = 0.01). This study demonstrates, for the first time, that circulating NO is suppressed in OSA, and this is promptly reversible with the use of nCPAP. The findings offer support for nitric oxide being one of the mediators involved in the acute hemodynamic regulation and long-term vascular remodeling in OSA.


Nocturnal polysomnography is the standard diagnostic test for sleep apnea syndrome (SAS) but is both expensive and time-consuming. We developed a predictive index for SAS based on pulmonary function data, including respiratory resistance determined by the forced oscillation technique, from 168 obese snorers with suspected SAS. Our model used logistic regression to obtain case-by-case predictions of the probability of SAS, defined as an apnea-hypopnea index (AHI) ≥ 15 during overnight polysomnography. We then tested our model in a prospective group of 101 similar patients. Specific respiratory conductance and daytime oxygen saturation contributed significantly to the model. Sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) of the index computed from these parameters were 98%, 86%, 98%, and 97%, respectively. In the prospective group, the model proved repeatable, with 100% sensitivity, 84% specificity, 86% PPV, and 100% NPV. The high NPV may help to identify obese snorers with a SAS risk that is so low as to make polysomnography unnecessary. Based on the 50% prevalence of SAS in our study and on the fact that polysomnography is required in all patients with daytime somnolence, we calculated that using our model would have obviated the need for polysomnography in 38% of our patients.


Inhaled corticosteroids have become the mainstay treatment of bronchial asthma. However, simultaneous evaluations of efficacy and side effects are few. This study aimed to compare the relative effect of fluticasone propionate (FP) and budesonide (BUD) on bronchial responsiveness and endogenous cortisol secretion in adults with asthma. The study was double-blind and included 66 adults with asthma, who were randomized to FP (n = 33) or BUD (n = 33). Prestudy, all participants were clinically stable, using inhaled corticosteroids and hyperresponsive to methacholine. Eligible patients were randomized to three consecutive 2-wk periods with either FP 250 microg twice daily, FP 500 microg twice daily, and FP 1,000 microg twice daily, or BUD 400 microg twice daily, BUD 800 microg twice daily, and BUD 1,600 microg twice daily, delivered by Diskhaler and Turbuhaler, respectively. Before randomization and at the end of each treatment, bronchial methacholine PD24h, 24-h urinary cortisol excretion (24-h UC), plasma cortisol, serum osteocalcin, and blood eosinophils were determined. The relative PD24h potency between FP and BUD was 2.51 (95% CI, 1.05–5.99; p < 0.05), while the relative 24-h UC potency was 0.60 (95% CI, 0.44–0.83; p < 0.01). The differential therapeutic ratio (FP/BUD) based on PD24h potency and 24-h UC was 4.18 (95% CI, 1.16–15.03; p < 0.05). The difference in systemic potency was also seen for plasma cortisol, serum osteocalcin, and blood eosinophils. Therapeutic ratio over a wide dose range, determined by impact on bronchial responsiveness and endogenous corticosteroid production, seems to favor FP.


Pulmonary embolism (PE) is a common and lethal yet treatable condition. Several authors have reported on the diagnostic value of combinations of arterial blood gas (ABG) and other clinical data (i.e., prediction rules), and have claimed that these combinations can be safely used to exclude PE. The purpose of this investigation was to evaluate the diag-
NEW ADVANCEMENTS IN ASTHMA:

ONE BREATH AT A TIME Asthma is a serious, allergy-related breathing difficulty characterized by gasping, coughing, wheezing and sometimes a frightening inability to take a breath. Children suffer greatly with asthma, but many people are unaware that this disease affects adults as well. The number of asthma sufferers in the U.S. has increased to unprecedented numbers and has many physicians and researchers working hard to get at the cause. While we wrestle with air pollutants, some experts are faulting American’s fetish with cleanliness.

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In patients with chronic obstructive pulmonary disease (COPD), differentiating a pulmonary embolism (PE) from an exacerbation of COPD can be difficult, since clinical signs and symptoms of the two conditions overlap. Development of reliable noninvasive or minimally invasive techniques for the diagnosis of PE is, especially in these patients, necessary. In this study we assessed the effect of COPD on the accuracy of the clinical probability estimate (CPE), spiral computed tomographic angiography (SCTA), D-dimer analysis, ventilation perfusion (V/Q) scintigraphy, and pulmonary angiography for the diagnosis of PE. From May 1997 through March 1998, 627 consecutive patients with suspected PE were investigated in six teaching hospitals. In these patients, D-dimer testing, CPE, V/Q scintigraphy, and SCTA and/or pulmonary angiography were performed according to a strict diagnostic protocol. The patients were also independently categorized as having COPD or not. A diagnosis of COPD was established in 91 patients (15%). The prevalence of PE was similar in patients with and without COPD (29% and 31%, respectively), notwithstanding the larger proportion of nondiagnostic V/Q scan results in patients with COPD (46% versus 21%, p < 0.001). The distribution of CPEs, diagnostic value of the D-dimer assay and SCTA, and reproducibility of pulmonary angiography were comparable among patients with and without COPD. The presence of COPD does not affect the diagnostic performance of CPE, D-dimer testing, SCTA, or pulmonary angiography. Furthermore, although more nondiagnostic V/Q scan results can be expected in the presence of COPD, V/Q scintigraphy remains a valuable screening test in patients with COPD.


Nebulized aerosols are commonly used to deliver drugs into the lungs of patients with cystic fibrosis (CF). The aim of this study was to assess the effectiveness of pressure-support (PS) ventilation in increasing aerosol deposition within the lungs of children with CF. An in vitro study demonstrated the feasibility of coupling a breath-actuated nebulizer to a PS device. An in vivo study was done with 18 children (ages 6 to 21 yr) with clinically stable CF, each of whom underwent both a standard and a PS-driven ventilation scan (control session and PS session, respectively). In addition, a perfusion scan was used to determine lung outlines and to construct a geometric model for quantifying aerosol deposition by radioactive counting in MBq. Homogeneity of nebulization was evaluated from the four first-order moments of aerosol distribution in the peripheral and central lung regions. The time-activity nebulization curve was linear in all patients, with higher slopes during the PS than during the control session (0.43 ± 0.07 [mean ± SD] MBq/min and 0.32 ± 0.23 MBq/min, respectively; p < 0.018). Quantitatively, aerosol deposition was about 30% greater after the PS session (4.2 ± 2.7 MBq) than after the control session (3.4 ± 2.1 MBq; p < 0.05). Similarly, deposition efficacy (as a percentage of nebulizer output) was significantly better during the PS session than during the control session (15.3 ± 8.3% versus 11.5 ± 5.7%, p < 0.05). No differences in the regional deposition pattern or in homogeneity of uptake were observed. In conclusion, our data show that driving the delivery of a nebulized aerosol by noninvasive PS ventilation enhances total lung aerosol deposition without increasing particle impact in the proximal airways.


Many ventilators measure expired tidal volume (V'T) without compensation either for the compliance of the ventilator circuit or for variations in the circuit setup. We hypothesized that the exhaled V'T measured with a conventional ventilator at the expiratory valve would differ significantly from the exhaled V'E measured with a pneumotachometer placed at the endotracheal tube. To investigate this we studied 98 infants and children requiring conventional ventilation. We used linear regression analysis to compare the V'T obtained with the pneumotachometer with the ventilator-measured volume. An additional comparison was made between the pneumotachometer volume and a calculated effective V'T. For infant circuits (n = 70), our analysis revealed a poor correlation between the expiratory V'T measured with the pneumotachometer and the ventilator-measured volume (r² = 0.54). Similarly, the expiratory V'E measured with the pneumotachometer did not correlate with the calculated effective volume (r² = 0.58). For pediatric circuits (n = 28), there was improved correlation between the expiratory V'T measured with the pneumotachometer and both the expiratory V'E measured with the pneumotachometer and the calculated effective V'T (r² = 0.84 and r² = 0.85, respectively). The data demonstrate a significant discrepancy between expiratory V'E measured at a ventilator port and that measured with a pneumotachometer placed at the endotracheal tube in infants. Correcting for the compliance of the ventilator circuit by calculating the effective V'T did not alter this discrepancy. In conventionally ventilated infants, exhaled V'E should be determined with a pneumotachometer placed at the airway.


The use of esophageal and gastric balloons limits measurement of the tension-time index of inspiratory muscles (TTI) during exercise. The aim of this study was to assess whether a noninvasive tension-time index, TTI 0.1, given by P 0.1/PEmax × TTI 0.1 (where P 0.1 is mouth occlusion pressure, PEmax is maximal inspiratory pressure, and TTI 0.1 is the open cycle) could reliably assess TTI during exercise. In seven healthy young men and nine patients with COPD we measured TTI 0.1 and TTI (i.e., PES × TTI 0.1/PEmax × TTI) where PES is mean esophageal pressure and PEmax is maximal static PES) at rest and during an incremental exercise test. A significant linear correlation (p < 0.02) was found between TTI 0.1 and TTI in all normal subjects and patients with COPD. An equation for estimating TTI from TTI 0.1 was established for each group. In the normal subjects there was good agreement between estimated and observed data. In five additional normal males studied prospectively, the agreement was also satisfactory and reproducible. In the COPD patients the agreement was poor. In conclusion, in young healthy subjects the changes in TTI 0.1 during exercise reflect the changes in TTI, allowing satisfactory estimation of TTI from noninvasive measurements of TTI 0.1.
Effects of Continuous, Expiratory, Reverse, and Bi-Directional Tracheal Gas Insufflation in Conjunction with a Flow Relief Valve on Delivered Tidal Volume, Total Positive End-Expiratory Pressure, and Carbon Dioxide Elimination: A Bench Study

Edgar Delgado RRT, Bernard Hete PhD, Leslie A Hoffman RN PhD, Frederick J Tasota RN MSN, and Michael R Pinsky MD

INTRODUCTION: Tracheal gas insufflation (TGI) can increase total positive end-expiratory pressure (total-PEEP) when flow is delivered in a forward direction, necessitating adjustments to maintain total-PEEP constant. When TGI is delivered throughout the respiratory cycle, additional adjustments are needed to maintain tidal volume (Vt) constant. OBJECTIVE: Determine if bi-directional TGI (bi-TGI) (simultaneous flows toward the lungs and upper airway) in combination with a flow relief valve eliminates the increase in total-PEEP and maintains a constant Vt, thus simplifying TGI administration. METHODS: Using an artificial lung model and pressure control ventilation, we studied the effect of TGI at 10 L/min on inspired Vt, total-PEEP, and CO2 elimination during 6 conditions: (1) control (no TGI, no catheter in the airway), (2) baseline (catheter in the airway but no TGI), (3) continuous TGI, (4) expiratory TGI, (5) reverse TGI, and (6) bi-TGI. Each condition was studied under 3 inspiration-expiration ratios (1:1, 1:2, and 2:1). A preset flow relief valve was inserted into the ventilator circuit during all TGI conditions with continuous flow. SETTING: University research laboratory. RESULTS: CO2 elimination efficiency was similar under all conditions. Total-PEEP increased with continuous TGI and expiratory TGI, decreased during reverse TGI, and was unchanged during bi-TGI. With the flow relief valve in place, and no adjustment in mechanical ventilation, the change in minute ventilation ranged from 0% to 10%, with the least change during bi-TGI (0–5%). During bi-TGI, gas flow was equivalent in both directions during dynamic conditions and the flow relief valve consistently removed gas at 10 L/min under various pressures. CONCLUSIONS: Our data from an artificial lung model support that continuous bi-TGI minimizes the change in total-PEEP seen during other TGI modalities. The flow relief valve compensated for the extra gas volume delivered by the TGI catheter, thereby eliminating the need to make ventilator adjustments. Used in combination with a flow relief valve, bi-TGI appears to offer unique advantages by providing a simpler method to deliver TGI. Further testing is indicated to determine if similar benefits occur in the clinical setting. Key words: tracheal gas insufflation, mechanical ventilation, permissive hypercapnia, gas exchange, acute lung injury, acute respiratory distress syndrome. [Respir Care 2001;46(6):577–585]

Introduction

Tracheal gas insufflation (TGI) has been proposed as an adjunctive ventilation technique to enhance carbon dioxide elimination (CO2) in the presence of permissive hypercapnia. TGI involves the use of a catheter placed inside or alongside the endotracheal tube and advanced to a position just above the carina. Alternatively,
TGI can be provided through an endotracheal tube that incorporates fine capillaries molded into the lumen\cite{2,13} or a double-lumen tube.\cite{25,26,28} The major mechanism by which TGI is believed to improve CO\(_2\) elimination is washout of CO\(_2\) from the anatomic dead space proximal to the catheter tip.\cite{23,31} A second contributing mechanism involves distal effects caused by jet-induced turbulence.\cite{23}

TGI can be delivered by several methods. One method involves continuous TGI (c-TGI), in which the flow of gas is delivered continuously throughout the respiratory cycle.\cite{1,14,23,28} TGI can also be delivered during a specific phase of the respiratory cycle (inspiration, expiration, or a fraction of expiration), a technique termed phasic TGI. The most common phasic approach is expiratory TGI (e-TGI) implemented with a solenoid valve synchronized with the ventilator’s expiratory valve.\cite{21,30} Several catheter configurations have also been studied, including a straight-tip (directing flow toward the lungs)\cite{14} and a reverse-tip (directing flow toward the upper airways).\cite{15,20,23,29,30}

Irrespective of the delivery mode, there are interactions between mechanical ventilation and TGI that influence minute ventilation (V\(_E\)), airway pressure, and total positive end-expiratory pressure (total-PEEP).\cite{11} When TGI is delivered in a forward flow direction, both c-TGI and e-TGI can produce intrinsic positive end-expiratory pressure (auto-PEEP).\cite{11,21,28} Reverse TGI (r-TGI) offers the potential to prevent the auto-PEEP increase associated with other TGI modes.\cite{15,17,19,23,29,30} However, r-TGI may reduce PEEP levels to lower than baseline\cite{17,29,30} and in some cases can create negative end-expiratory pressure as a result of the air-entrainment (“Venturi”) effect created by the reverse flow.\cite{15}

We postulated that during c-TGI and e-TGI with forward flow a major cause of expiratory resistance and increased auto-PEEP is the back pressure effect that develops because of the collision of opposing flows from the lung and TGI catheter. The source of this back pressure may be the static pressure created by the absorption of kinetic energy from high velocity gas molecules decelerating as they collide. We hypothesized that bi-TGI (simultaneous flows toward and away from the carina) might therefore reduce the back pressure created by TGI with forward flow and thereby minimize the increase in total-PEEP caused by the opposing flows.

Other considerations relate to changes in V\(_E\). When TGI is administered continuously throughout the respiratory cycle, modifications are required to compensate for the additional gas delivered by the catheter.\cite{1,1,2,3,4,11,14,28} If TGI is used with pressure control ventilation, it is necessary to insert a pressure relief valve to keep total (ventilator-derived + TGI-derived) V\(_E\) constant.\cite{1,11} The pressure relief valve must be adjusted to maintain peak airway pressure at set inspiratory pressure (P\(_{set}\)) and readjusted if there are changes in patient condition. An alternative solution involves use of a flow relief valve set to release gas at a flow equivalent to that delivered by the TGI catheter. We hypothesized that use of this valve could eliminate the need to make adjustments to compensate for the additional gas delivered by the catheter.

To test our hypothesis, we used a single-compartment artificial lung model, which provided the ability to accurately measure volume and pressure without the variability that a human or animal model would exhibit. We assessed the function of an inspiratory flow relief valve to maintain constant V\(_E\) with and without ventilator adjustment and compared total-PEEP levels among c-TGI and e-TGI (gas flow toward the carina), r-TGI (gas flow cephalad, away from the carina), and bi-TGI (gas flow both toward and away from the carina). Additionally, we determined CO\(_2\) elimination efficiency for all conditions with V\(_E\) kept constant.

**Methods**

The apparatus consisted of a single-compartment artificial lung (Training Test Lung 2600, Michigan Instruments, Grand Rapids, Michigan), linear resistor (112275 7100R20, Hans Rudolph, Kansas City, Missouri), pneumotachograph (No. 2 Fleisch, Lausanne, Switzerland) for V\(_E\) measurement, and mechanical ventilator (Puritan-Bennett 7200ac, Mallinckrodt, Carlsbad, California), as previously described.\cite{9} TGI was delivered through a catheter with a prototype “H” tip (Fig. 1) (patent #6,102,042, Respionics, Murrysville, Pennsylvania) to permit varying the direction of flow while maintaining a constant airway restriction for all TGI testing conditions. A prototype preset flow relief valve (patent pending, Respionics, Murrysville, Pennsylvania) was inserted into the expiratory limb of the ventilator circuit to remove gas at 10 L/min during those TGI modalities that use continuous flow.

**Lung Model**

The lung model was operated in the single-lung mode. The calibrated pneumotachograph interfaced with a differential pressure transducer (MP45, Validyne Engineering, Northridge, California) was placed within the system. 14.5 cm distal to the tip of the TGI catheter, to record total V\(_E\) (ventilator-derived + TGI-derived) entering the lung. Volume calibration measurements were performed by injection of a known volume of air (0.5 L), using the calibration syringe. Volume calibration measurements were repeated until a ≤ 3% variation point was achieved. Pressure transducers were calibrated using a known pressure, as verified by the calibration analyser (Timeter Calibration Analyser, Series RT 200, Allied Healthcare Products, St Louis, Missouri). Resistance of the lung was set at 20 cm
Effects of Tracheal Gas Insufflation

TGI Delivery Mode

- Continuous
- Expiratory
- Reverse
- Bi-directional

Fig. 1. Catheter tip configurations and flow patterns with 4 different tracheal gas insufflation modes. Insp = inspiration, Exp = expiration. (From Delgado E, Hoffman LA, Tasota FJ, Pinsky MR. Monitoring and humidification during tracheal gas insufflation. Respir Care 2001; 46[2]:185–192.)

H₂O/L/s by insertion of a linear resistor, and a compliance of 0.02 L/cm H₂O was implemented by adjusting the compliance spring of the artificial lung model.

Tracheal Gas Insufflation System

A TGI catheter of 1.67 mm inner diameter and 2.0 mm outer diameter was inserted through a TGI catheter swivel adapter (adapter #600101, Concord/Portex, Keene, New Hampshire) into a #8 endotracheal tube in line with the pneumotachograph, resistor, and capnograph sensor. For all conditions, tracheal gas (fraction of inspired oxygen = 0.21) was insufflated at 10 L/min, the flow most commonly used clinically at our institution. To simulate CO₂ production, a continuous flow of CO₂ was bled into the artificial lung at 0.38 L/min and was measured by the capnograph (CO₂SMO Plus, Model 8100, Novametrix Medical Systems, Wallingford, Connecticut), calibrated as recommended by the manufacturer. Flows were verified prior to each condition of the experiment, using a calibration analyzer. The calibration analyzer was also used to test equivalency of forward and reverse flow during bi-TGI under dynamic conditions and to test the consistency of the flow relief valve over a range of pressures.

During e-TGI, r-TGI, and bi-TGI, a prototype flow relief valve was inserted into the expiratory limb of the ventilator circuit. The flow relief valve was set to the flow used for TGI (10 L/min) and vented this volume of gas from the ventilator circuit continuously. During e-TGI an external solenoid valve (A3314-S8, Precision Dynamics, New Britain, Connecticut) was electronically synchronized with the exhalation valve of the mechanical ventilator to provide TGI only during exhalation. The flow relief valve was not used during e-TGI. Identical catheter “H” tips were used for all TGI conditions, but either the reverse or straight lumen was plugged, depending on the condition being tested (see Fig. 1).

Measurements

Total-PEEP and peak intrapulmonary pressure measurements were obtained from the internal pressure transducer of the artificial lung. The tidal volume entering the artificial lung (V₁) was measured with the pneumotachograph in line, distal to the TGI catheter tip. Exhaled volume, measured by the ventilator, was recorded for comparison to a clinical scenario. Partial pressure of carbon dioxide (P<sub>CO₂</sub>) values were obtained at the lung with a mainstream capnograph.

To produce control values, the mechanical ventilator was set in the pressure control mode with an initial P<sub>set</sub> of 30 cm H₂O, a frequency of 16 breaths/min, PEEP of 5 cm H₂O, and no TGI catheter.
Table 1. Ventilator Parameters Under Varying Tracheal Gas Insufflation Conditions*

<table>
<thead>
<tr>
<th>EE = 1:2</th>
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<th>Baseline</th>
<th>c-TGI</th>
<th>e-TGI</th>
<th>r-TGI</th>
<th>bi-TGI</th>
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<td>33</td>
<td>34.9</td>
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<td>33</td>
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<td>$V_t$ (L)</td>
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<td>7.4</td>
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<td>$P_{CO_2}$ (mm Hg)</td>
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<td>54</td>
<td>45</td>
<td>45</td>
<td>47</td>
<td>46</td>
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<tr>
<td>total-PEEP (cm H$_2$O)</td>
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<td>5.5</td>
<td>7.3</td>
<td>7.7</td>
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<th>e-TGI</th>
<th>r-TGI</th>
<th>bi-TGI</th>
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<td>31</td>
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<tr>
<td>$P_{peak}$ (cm H$_2$O)</td>
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<td>34.1</td>
<td>35.1</td>
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<tr>
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<td>33.6</td>
<td>35.5</td>
<td>36.4</td>
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<tr>
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<td>$V_t$ (L)</td>
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<td>7.4</td>
<td>7.3</td>
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<td>48</td>
<td>50</td>
<td>49</td>
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<td>total-PEEP (cm H$_2$O)</td>
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<td>9.5</td>
<td>11.9</td>
<td>12.4</td>
<td>6.3</td>
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</table>

*Tracheal gas insufflation (TGI) conditions with set inspiratory pressure ($P_{set}$) adjusted as needed to maintain constant minute ventilation when compared to baseline (no TGI).

Control = no catheter and no TGI
Baseline = catheter in but no TGI
C-TGI = continuous TGI
e-TGI = expiratory TGI
r-TGI = reverse TGI
bi-TGI = bi-directional TGI
I:E = ratio of inspiratory time to expiratory time
$P_{peak}$ = peak airway pressure
$P_{aw peak}$ = peak alveolar pressure
$V_t$ = minute ventilation
$P_{CO_2}$ = partial pressure of carbon dioxide
total-PEEP = total positive end-expiratory pressure

To produce baseline values, the aforementioned control settings were used and the TGI catheter was inserted into the airway, to account for occlusion created by the presence of the TGI catheter. Three inspiration-expiration (I:E) ratios were tested: 1:2, 1:1, and 2:1. All measurements were made under ambient temperature and pressure, dry conditions.

Experimental Protocol

The following experimental conditions were studied:
1. Control: no catheter in the airway and no TGI.
2. Baseline: catheter in the airway but no TGI.
3. c-TGI with the flow relief valve and continuous forward flow.
4. e-TGI with forward flow synchronized to occur during expiration.
5. r-TGI with the flow relief valve and continuous reverse flow.
6. bi-TGI with the flow relief valve and continuous forward and backward flow.

During all TGI conditions, data were collected with no change in $P_{set}$ from baseline. When necessary, $P_{set}$ was increased to match baseline $V_t$, and both corrected and uncorrected $V_t$ data were obtained. During each experimental condition, measurements were made of $P_{set}$, peak intrapulmonary pressure, peak airway pressure, $V_t$, expired volume, total-PEEP, and CO$_2$.

To determine consistency under various conditions, forward and reverse flows from the bi-TGI catheter were isolated and exposed to different pressure gradients. In addition, gas vented to the atmosphere by the flow relief valve was measured under different pressures to verify that flow was constant as a function of pressure.

Repetitive trials at various settings were conducted after calibration of the equipment. In all instances, pressures were constant within ± 0.2 cm H$_2$O and volumes were accurate to ± 1%. Since no measurable variance occurred
on multiple observations, any difference between the experimental conditions was taken to be an absolute change, and no statistical analysis was done on the study data.

**Results**

**Carbon Dioxide**

For all I-E ratios, $P_{CO_2}$ was lower during control (no catheter, no TGI) than during baseline (catheter in airway, no TGI). Under constant $V_E$ for all conditions, $P_{CO_2}$ levels were similar for c-TGI, e-TGI, r-TGI and bi-TGI, and lower than baseline (Table 1).

**Total Positive End-Expiratory Pressure**

The addition of the catheter into the #8 endotracheal tube increased total-PEEP, compared to control (no catheter in the airway) (see Table 1). Specifically:

- 5.4 cm H$_2$O vs 5.5 cm H$_2$O at I-E ratio 1:2.
- 6.2 cm H$_2$O vs 6.4 cm H$_2$O at I-E ratio 1:1.
- 8.8 cm H$_2$O vs 9.5 cm H$_2$O at I-E ratio 2:1.

Compared to baseline (catheter in the airway), when c-TGI and e-TGI were administered with forward flow and a straight-tip catheter, total-PEEP was 25–33% and 31–40% greater, respectively (Fig. 2). During r-TGI, total-PEEP was 34–84% lower than baseline. Total-PEEP during bi-TGI was consistent (±7%) with baseline (no TGI) conditions at all I-E ratios.

**Minute Ventilation**

Without correction, $V_E$ decreased 2–10% from baseline, depending on the TGI delivery mode and the I-E ratio. Expiratory TGI required the most correction and bi-TGI the least. Uncorrected $V_E$ values were also compared to control (no catheter) to simulate a clinical scenario with initiation of TGI. Average percent decrease from control for the 3 I-E ratios studied ranged from 5.3% for bi-TGI to 12.3% for e-TGI (Fig. 3).

By experimental protocol, $V_E$ was maintained constant in relation to baseline (no TGI, catheter in the airway) during c-TGI, e-TGI, r-TGI, and bi-TGI conditions at the 3 I-E ratios. It was necessary to increase $P_{aw}$ by 1–3 cm H$_2$O under most conditions to maintain $V_E$ constant.

**Tidal Volume**

Exhaled volumes recorded from the ventilator were no more than 9% greater than those obtained with the pneu-
Fig. 4. Mean tidal volume at baseline (no tracheal gas insufflation) and during 4 tracheal gas insufflation modes, measured by in-line pneumotachograph and mechanical ventilator for the combined inspiratory time to expiratory time ratios. c-TGI = continuous tracheal gas insufflation. e-TGI = expiratory tracheal gas insufflation. r-TGI = reverse tracheal gas insufflation. bi-TGI = bi-directional tracheal gas insufflation.

motachograph for baseline and during all forms of TGI with continuous flow when using the flow relief valve (Fig. 4). Exhaled volumes with e-TGI (without the flow relief valve) were as much as 50% greater.

Stability Under Dynamic Conditions

When forward flow from the bi-TGI catheter was exposed to various pressure gradients (−20 to +20 cm H2O), it remained essentially unchanged at 5 L/min (Fig. 5). When system pressures were changed from 5 cm H2O to 60 cm H2O, the linearity of the flow relief valve remained unchanged (Fig. 6).

Discussion

TGI is an effective tool in promoting CO2 washout from the anatomical dead space in mechanically ventilated patients. However, one factor limiting successful application of this adjunctive technique is increased expiratory resistance and production of dynamic hyperinflation, known as auto-PEEP.6-8,11,15,16,20,29 Several factors facilitate the development of TGI-induced auto-PEEP. The cross-sectional area of the endotracheal tube is reduced because of the addition of the TGI catheter. Expiratory time is also a factor: expiratory time is inversely proportional to the total-PEEP generated, regardless of the TGI method used.6,8

In addition, we reasoned that a significant amount of back pressure may occur when the opposing gas flow exiting the lung encounters the incoming flow introduced by a forward flow (straight-tip) TGI catheter. To test this hypothesis, we designed an "H" tip catheter. With this catheter tip configuration, equal amounts of flow could be directed toward the lung and upper airway, an arrangement that, according to our data, maintains end-expiratory lung volume and, hence, total-PEEP constant.

This study supports previous studies by demonstrating that all modes of TGI increase gas exchange efficiency equally. However, the effects of TGI therapies on intrinsic PEEP are not similar. Both c-TGI and e-TGI induce hyperinflation, which becomes more pronounced with higher I:E ratios. Conversely, r-TGI decreases PEEP, which, at high I:E ratios, according to our data, resulted in almost total loss of extrinsic PEEP. Contrary to these opposing effects, bi-TGI flow caused no change in intrinsic PEEP at any of the I:E ratios tested.

Furthermore, since the flow relief valve keeps the pressure constant during the various system pressures, no adjustments in VT or driving pressure were needed during bi-TGI, compared to all other forms of TGI. Importantly, the equality of flow in both directions during bi-TGI was unaltered by differing downstream and upstream airway resistance. Presumably, this is because the driving pressure for flow out of the 2 TGI openings in the bi-TGI system is greater than airway pressure by "n" order of magnitude. The simplicity of bi-TGI in combination with the flow relief valve was reflected in the ability to rely on the exhaled measurements from the mechanical ventilator without the need for adjustments.

Prior studies have described a variety of interactions between mechanical ventilation and TGI that influence VT, airway pressure, and total-PEEP.1-30 During c-TGI and pressure control ventilation, increases in peak airway pressure have been observed because of the ventilator’s inability to recognize the extra gas delivered by the TGI catheter.8,11,15,20,28 If e-TGI is delivered with a straight-tip catheter (forward flow) during pressure control ventilation without a pressure relief valve to control VT, both peak airway pressure and the amount of total-PEEP may increase.8 These phenomena occur because of continued TGI flow during inspiration after flow from the ventilator has ceased. Accordingly, it is necessary to use a pressure relief valve to vent TGI flow and limit peak airway pressure after the flow delivered by the ventilator decreases to zero.8,9-11

Constraining TGI to occur only during expiration (e-TGI) avoids the problem of excess gas delivery during inspiration. However, when using e-TGI during pressure control ventilation, the volume delivered to the airway is dependent on the pressure gradient between the peak intrapulmonary and end-expiratory lung pressures. Therefore, the inspiratory pressure setting on the ventilator should be increased to maintain this gradient and avoid a loss of Vr created by the increase in total-PEEP generated by TGI. Expiratory TGI may also pose other disadvantages. When delivered in a forward flow direction, e-TGI may
produce auto-PEEP.\textsuperscript{9,21,25–28} Furthermore, the use of e-TGI is not simple, since it requires a special timing device to deliver TGI only during expiration.\textsuperscript{9,21,26} Thus, e-TGI can be conducted only with specialized equipment.

Several studies have also evaluated the benefits of r-TGI alone and in combination with e-TGI.\textsuperscript{15–20,29,30} When TGI was administered at 10 L/min, the combination of r-TGI plus e-TGI decreased total-PEEP measured at the carina at the 2 I:E ratios tested (1:2, 2:1), thus reducing lung volume.\textsuperscript{29} The reverse-thrust catheter created PEEP equivalent to conventional ventilation at low flows, but when $V_E$ was doubled or tripled for 10–20 seconds, PEEP at the carina decreased to less than zero, creating negative end-expiratory pressure.\textsuperscript{15} Our r-TGI results support those findings by demonstrating that with increasing expiratory time, PEEP is lost. Since arterial oxygenation in patients with acute lung injury is highly dependent on maintaining high mean airway pressures, and since derecruitment at end-expiration promotes ventilator-induced lung injury, these data collectively suggest that r-TGI should be judiciously applied, with close monitoring of total-PEEP. If total-PEEP is decreased, then adjustment of mechanically-applied PEEP should be considered to prevent derecruitment.

Our data demonstrate that the bi-TGI catheter is capable of maintaining equivalent forward and backward flow over a range of pressures. The quantity in either direction of the flow from a bi-directional catheter tip is not a function of the pressure at the catheter tip. Although the aggregate flow can decrease slightly with increased pressure at the tip, the upstream feed pressure is usually greater than 10 psi, which results in a choked flow situation. This arrangement was made specifically to reduce the impact of pressure increases (at the catheter tip) on flow magnitude. Furthermore, forward and backward jet flows should not change as long as the pressure near the catheter tip is fairly homogenous, because the flow ratio is determined by the geometry of the catheter tip. The external pressure at the tip can only alter the sum total of flow (and only slightly over a range of 5–60 cm H$_2$O), not the flow ratio. The only way the flow ratio can be altered is if an occlusion occurs between the forward and backward jet, causing the pressure at the exits of the forward and backward jets to be different. We think that scenario would be very unlikely in practice.

The flow relief valve used in this study was designed to eliminate excess gas flow delivered by the TGI catheter during inspiration. When system pressures were varied, the flow relief valve demonstrated the ability to remove a constant volume of gas from the system, regardless of system pressure (5–60 cm H$_2$O). Since the volume of gas...
insufflated by the catheter and removed by the flow relief valve was equivalent, it was possible to monitor delivered \( V_t \) using the ventilator control panel. Importantly, the flow relief valve functioned in an equivalent manner during c-TGI, r-TGI, and bi-TGI. Thus, the coupling of bi-TGI with a flow-relief valve makes the delivery of TGI safe, simple, and effective (Table 2).

**Limitations**

The lung model used in this study presented certain limitations. We used a single-compartment model, which is remotely different from the complex animal model or human subject. However, this model permitted us to accurately measure true end-expiratory lung pressure (total-PEEP), which may be impossible in the human lung.

Second, an artificial lung model provides a constant compliance and thus does not model the viscoelastic forces, various time constants, and gas exchange properties of the human lung. Therefore, regional volume and pressure changes and preferential distribution of ventilation could not be assessed in this model.

Third, there are safety issues inherent in any method of pulmonary insufflation (e.g., drying of airway secretions, mucus ball formation, and damage to tracheal mucosa), which were not addressed in this study, and require further clinical evaluation.

**Conclusions**

A major benefit of bi-TGI in combination with a flow relief valve is the potential to maintain CO\(_2\) elimination efficiency (as compared to other TGI modalities) and eliminate changes in total-PEEP without the need to use a complex delivery system or make frequent ventilator adjustments. Our data, obtained with an artificial lung model, show that bi-TGI minimizes the change in total-PEEP seen during other forms of TGI. The flow relief valve compensated for the extra gas volume delivered by the TGI catheter, thereby eliminating the need to make ventilator ad-

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**REFERENCES**


Comparison of High-Frequency Flow Interruption Ventilation and Hyperventilation in Persistent Pulmonary Hypertension of the Newborn

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INTRODUCTION: Because of the high mortality, potential limitations, and inherent adverse effects associated with conventional therapies, as well as extracorporeal membrane oxygenation, for persistent pulmonary hypertension of the newborn (PPHN), alternative modes of ventilatory support have been researched. There is anecdotal evidence that high-frequency flow interruption ventilation (HFFI) benefits neonates with severe air leak and lung diseases unresponsive to conventional ventilation, so we conducted a study to compare the hospital course, survival rate, and incidence of chronic lung disease of neonates with PPHN treated with hyperventilation (HV) and HFFI. METHODS: Enrolled in the study were 36 neonates who (1) were treated with HV and a fraction of inspired oxygen of 1.0 for PPHN, (2) had arterial partial pressure of oxygen (P_{O2}) values ≤ 60 mm Hg, and (3) met the inclusion criteria. Neonates were assigned to either HV or HFFI treatment and there were 18 neonates in each treatment group. RESULTS: HFFI did not statistically increase survival (78% vs 44%, p = 0.087). Compared to the HV group, the HFFI group had: (1) fewer neonates requiring vasopressor support (7 vs 14, p = 0.042); (2) lower mean pH (7.37 vs 7.52, p < 0.001) and higher mean P_{CO2} (37.7 vs 22.1 mm Hg, p < 0.001) for neonates with P_{O2} ≥ 120 mm Hg; (3) shorter mean time to P_{O2} ≥ 120 mm Hg (13.5 vs 50.2 h, p = 0.001); (4) shorter mean time to reduced fraction of inspired oxygen (16 vs 84 h, p < 0.001); (5) shorter mean time to fraction of inspired oxygen 0.70 (53 vs 187 h, p < 0.001); (6) shorter mean time to extubation (8.1 vs 18.7 d, p = 0.033); (7) shorter length of hospitalization (22.7 vs 50.6 d, p = 0.025); and (8) fewer neonates with chronic lung disease (1 vs 5, p = 0.018). CONCLUSIONS: HFFI with the ventilation strategy we describe accomplishes sustained hyperoxygenation without hypocarbia and alkalanosis, and response to HFFI can predict outcomes. HFFI does not significantly reduce mortality, but it does reduce the length of mechanical ventilation, the length of hospitalization, and the incidence of chronic lung disease in neonates with PPHN. The nonrandomized design of our study precludes firm conclusions about the potential benefits of HFFI. The results may be biased by practice variations. Additional randomized controlled trials are warranted to determine the efficacy of HFFI in neonates with PPHN. Key words: persistent pulmonary hypertension of the newborn, high-frequency flow interruption, high-frequency ventilation, hyperventilation, practice variation. [Respir Care 2001; 46(6):586–594]

Introduction

Conventional therapies, both pharmacologic (intravenously administered vasodilators) and ventilatory, for per-
ther morbidity and mortality of PPHN. Moreover, responses to vasodilators and hyperventilation (HV) do not predict the survival or mean successful neurodevelopmental outcome. Hyperventilation, which has been a standard therapy for a decade, is associated with lung hyperinflation, barotrauma, impairment of venous return and cardiac output, and high mortality. Complications, including hypotension, gastrointestinal hemorrhage and renal insufficiency, occur in up to two thirds of patients receiving tolazoline. Extracorporeal membrane oxygenation is labor-intensive, expensive, and associated with adverse neurologic sequelae. The reported overall mortality rate in PPHN patients treated with HV was 44%, and 46% for patients treated with tolazoline. Because of the high mortality, potential limitations, and inherent adverse effects of conventional therapies and extracorporeal membrane oxygenation, we have sought more effective and less traumatic modes of ventilatory support. One promising approach is high-frequency ventilation (HFV), which has been shown to be a useful means of respiratory support in pulmonary disease, because it accomplishes adequate gas exchange and oxygenation with small tidal volume, which may be an important advantage in neonates with PPHN. Anecdotal evidence suggests that high-frequency flow interruption ventilation (HFFI) has been employed safely in neonates with severe air leak and lung diseases unresponsive to conventional ventilation. However, there was no published study of the use of HFFI in a defined population of neonates, so we performed a prospective nonrandomized study to compare the hospital course and clinical outcomes of PPHN patients treated with HV to those treated with HFFI. The primary outcome measured was survival. Other outcomes and measurements of the clinical course included vasopressor support, gas exchange, oxygenation, air leak, chronic lung disease (CLD), and length of ventilator support and hospitalization. We hypothesized that among newborns with PPHN, HFFI would improve survival and pulmonary morbidity and decrease length of intubation and hospitalization.

**Methods**

The study was conducted at a level III neonatal intensive care unit (NICU), Faculty of Medicine, Siriraj Hospital, Bangkok, Thailand, and was approved by the Human Rights Committee of the Faculty.

**Diagnosis and Participation Criteria**

The diagnosis of PPHN was confirmed by two or more of the following criteria:

- Responded to HV by an increase in arterial partial pressure of oxygen (Pao2) of ≥ 60 mm Hg
- Echocardiographic evidence of right-to-left shunt at the atrial level
- Pre ductal and post ductal Pao2 gradient of at least 20 mm Hg

Neonates were eligible if they were treated with HV and a fraction of inspired oxygen (FiO2) of 1.0 for PPHN, had Pao2 ≤ 60 mm Hg, and met 2 or more of the following 3 criteria:

- Ventilator rate ≥ 60 cycles/min
- Peak inspiratory pressure (PIP) ≥ 35 cm H2O
- Arterial partial pressure of carbon dioxide (Paco2) ≤ 30 mm Hg

Neonates were enrolled in the study after informed consent had been obtained from parents.

**Treatment Assignment**

Neonates were assigned to HV or HFFI treatment based on the investigator, who was the attending physician of the month. Neonates cared for by investigators numbers 3 and 4 continued on HV, and those cared for by investigators number 1 and 2 received HFFI. After assignment, crossover between methods of ventilation was not allowed.

**Hyperventilation**

All neonates were hyperventilated with a Bear Cub Infant Ventilator BP2001 (Bear Medical Systems, Riverside, California). Subjects were hyperventilated with PIP that provided adequate chest movement, positive end-expiratory pressure (PEEP) of 2–4 cm H2O, inspiratory time of 0.3–0.5 s, inspiratory-expiratory ratio between 1:1 and 1:2, and expiratory time of not less than 0.25 s. The ventilator rate was first increased by 10 cycles/min from the intermittent mandatory ventilation (IMV) rate used before starting HV and incrementally increased in increments of 10 cycles/min. The patient was hyperventilated until an adequate decrease in Paco2 (critical Paco2), was achieved, to increase Paco2 to above 100 mm Hg. The initial target Paco2 was 30 mm Hg. If the target Paco2 was not thereby achieved, Paco2 was further reduced to 25 mm Hg, then 20 mm Hg or lower if necessary. When initial stability was achieved, changes in FiO2 were limited to 0.02 and changes in PIP were limited to 1 cm H2O maximum per change. PIP was decreased if Paco2 was below the critical Paco2. FiO2 was reduced if Paco2 was higher than 120 mm Hg. When FiO2 was decreased to 0.70, Paco2 was maintained at 45 mm Hg. Exubation was performed when the ventilator settings were FiO2 < 0.40, PIP < 16 cm H2O, PEEP 2–3 cm H2O, and ventilator rate of 10 cycles/min.
If the target $P_{aco}$ and $P_{ao}$ could not be achieved at a ventilator rate of 150 cycles/min, PIP was increased in increments of 1 cm H$_2$O until the target $P_{aco}$ and $P_{ao}$ were achieved.

If $P_{ao}$ was still < 60 mm Hg when $P_{aco}$ was lower than 20 mm Hg, sodium nitroprusside was given intravenously, with an initial dose of 0.05–0.1 µg/kg/min, and increased by 0.05–0.1 µg/kg/min every 15 minutes until $P_{ao}$ was more than 120 mm Hg. The maximum dose of sodium nitroprusside was 6 µg/kg/min. If the target $P_{ao}$ could not be achieved within 2 hours of the maximum dose, the infusion was discontinued. If the target $P_{ao}$ was achieved and $F_{10}$ could be reduced to 0.70, sodium nitroprusside was then used gradually at 0.05 µg/kg/min every 6 hours and discontinued at a dose of 0.1 µg/kg/min.

**High-Frequency Flow Interruption Ventilation**

HFFI was delivered by the Infant Star Neonatal Ventilator (InfraSonics, San Diego, California). HFFI was set at a fixed frequency of 900 cycles/min and a pressure amplitude sufficient to produce visible chest wall motion. The $F_{10}$ was the same as that required during HV (ie, 1.0). The temperature of the respiratory gas from the ventilator was set at 36°C. Once HFFI was started, the IMV rate was decreased to 60 cycles/min to prevent sudden deterioration. The IMV rate was then reduced in decrements of 5 cycles/min every 5 minutes, guided by the pulse-oximetry-measured oxygen saturation reading ($S_{po}$), measured with a pulse oximeter, Criticare 503, Criticare Systems, Milwaukee, Wisconsin), as long as $S_{po}$ was more than 95%, until a final rate of 5 cycles/min was reached. Decreases in mean airway pressure ($P_{aw}$) caused by the reduction of IMV rate were compensated for by increasing PEEP to maintain $P_{aw}$ at 1–2 cm H$_2$O less than or equal to that used during HV at time of study entry. Lower $P_{aw}$ values were used when pre-HFFI $S_{po}$ could be maintained on HFFI. Minimum accepted $S_{po}$ was 85%.

An arterial blood gas (ABG) sample was obtained 15 minutes after the IMV rate had been reduced to a minimum of 5 cycles/min. If the IMV rate could not be decreased because $S_{po}$ decreased to < 85%, the IMV rate was returned to the previous rate (before $S_{po}$ decreased to less than 85%) and no further reduction in IMV rate was attempted. A blood sample was obtained for blood gas measurement when $S_{po}$ was > 85% to assess ventilation (Fig. 1).

The $P_{aco}$ target value was 45 mm Hg. If $P_{aco}$ was more than 45 mm Hg and $P_{ao}$ was < 120 mm Hg, the amplitude was increased in increments of 2 cm H$_2$O. If $P_{aco}$ was between 45 and 55 mm Hg and $P_{ao}$ was > 120 mm Hg, no further increase in the amplitude was made. If $P_{aco}$ was less than 45 mm Hg, the IMV rate was further reduced, in decrements of 5 cycles/min, with a final minimum rate of 5 cycles/min. Then the amplitude was gradually reduced in decrements of 2 cm H$_2$O.

The $P_{ao}$ target was 120 mm Hg. If $P_{ao}$ was less than 50 mm Hg, PEEP was increased in increments of 1 cm H$_2$O, until $P_{ao}$ was above 50 mm Hg, whereupon the settings were held and the patient was closely monitored with pulse oximetry until $S_{po}$ was greater than 98%. An ABG sample was then obtained.

In both interventions, $F_{10}$ was reduced when $P_{ao}$ was greater than 120 mm Hg. $F_{10}$ was decreased by 0.02, each time followed by an ABG measurement. If at any time a further reduction of $F_{10}$ could not be made, an ABG measurement was obtained at least every 4 hours. When $F_{10}$ had been reduced to 0.70, then $P_{ao}$ was maintained at 80 mm Hg and $F_{10}$ was decreased gradually, in decrements of 0.05, until $F_{10}$ reached 0.40.

In the HFFI group, PEEP was reduced in decrements of 1 cm H$_2$O when $P_{ao}$ was > 60 mm Hg (once $F_{10}$ was reduced to 0.40), to a minimum PEEP of 3 cm H$_2$O. The amplitude was reduced in decrements of 2 cm H$_2$O when $P_{aco}$ was < 45 mm Hg, until no chest movement was seen. During the reduction of HFFI settings, PIP was gradually reduced until a PIP was reached that adequately moved the chest equivalent to the movement observed with hand bagging or a minimum of 12 cm H$_2$O. HFFI was turned off when the HFFI settings were $F_{10}$ 0.40, PEEP 3 cm H$_2$O, and an amplitude that gave no chest motion. The patient would then be on IMV only, at a rate of 5 cycles/min, $F_{10}$ 0.40, PEEP 3 cm H$_2$O, and PIP of < 18 cm H$_2$O. The patient was extubated at this IMV setting if there was no substantial distress with spontaneous respiration and ABG values were acceptable. If the patient had respiratory difficulty and/or unacceptable ABG values, the IMV settings were increased as necessary.

Chest radiographs were taken to evaluate the degree of lung inflation. If they showed lung overinflation (flat right diaphragm dome with inflation more than 9 posterior ribs), $P_{aw}$ was decreased in decrements of 1–2 cm H$_2$O until the right diaphragm dome was at the eighth rib or between the eighth and ninth ribs.

**Supportive and Symptomatic Treatment**

All neonates enrolled in the study also received therapy consisting of intravenous fluid and correction of acid-base abnormalities, anemia, and hypoglycemia. Dopamine and/or dobutamine was administered to maintain systolic pressure in the upper limit of normal range according to birthweight and postnatal age.23 Dopamine was weaned first, at a decrement of 2 µg/kg/min every 10 minutes when systolic pressure was higher than the upper limit, and then dobutamine at the same rate and frequency. Dobutamine was discontinued when the infusion rate reached 2 µg/kg/min.
Continuous \( S_{\text{PaO}_2} \) monitoring and noninvasive measurement of blood pressure (using a Dinamap Vital Signs Monitor, Johnson & Johnson Medical) were mandatory in all neonates. Pancuronium bromide was given every 2–3 hours in the HV group. Neonates in the HFFI group were allowed to breathe spontaneously during HFFI. In our study, neither alkali infusion (to keep pH greater than 7.5) nor surfactant was administered. The use of fluids and nutrition was practiced under the same protocols. Neonates were discharged home when oxygen therapy was not required, and were on full breast-feeding or a minimum of 150 mL/kg of formula.

**Data Collection**

Data were collected at the time of enrollment, and included demographics, age at starting conventional mechanical ventilation, age at enrollment, duration of conventional mechanical ventilation when enrolled into the study, and vasopressor use. Ventilator settings (\( F_{\text{IO}_2} \), rate, PIP, PEEP, \( P_{\text{aw}} \)) and ABG values at enrollment were also recorded. The oxygenation index at study entry was calculated by the formula \( (P_{\text{aw}} \times F_{\text{IO}_2} \times 100)/P_{\text{aO}_2} \), and obtained from the mean of 2 values from 2 blood gas measurements 1–2 hours apart, prior to study entry. The \( P_{\text{aw}} \) measurement was that displayed by the ventilator.

The next data collection was completed at discharge and provided summary information concerning the clinical course and hospitalization. Data collected at discharge included length of mechanical ventilation, ventilator settings, ABG values when \( P_{\text{aO}_2} \) was \( > 120 \text{ mm Hg} \), duration of mechanical ventilation when \( F_{\text{IO}_2} \) could be reduced and when it was decreased to 0.70, length of intubation, length of hospitalization, survival, and condition at discharge. In the HFFI group, if vasopressors used before study entry were discontinued within 12 hours of starting HFFI, the patient was considered "no vasopressor support needed."

CLD was defined as oxygen dependence for \( \geq 28 \) days with persistently abnormal chest radiographs (cystic changes, hyperinflation, and/or fibrosis). Causes of death were identified by additional evidence from postmortem studies.
Statistical Analysis

The sample size was calculated with the Epi Info 6 public domain software package (available at http://www.cdc.gov/epiinfo), using alpha of 0.05, power of 0.80, and the previous mortality rate of infants with PPHN at our center: 77% with a reduction in the mortality of 65%.24 The calculated total sample size was 36 neonates.

Data were analyzed with the SPSS/PC+ software package (SPSS, Chicago, Illinois) and standard methodology.25 Data variables were tested for normality by the Kolmogorov-Smirnov goodness-of-fit test and were found to be normal. Clinical characteristics data are expressed as mean ± SD and range. The two-tailed independent t test was used to compare means between groups. Dichotomous outcome variables were assessed with the two-tailed Fisher’s exact test or Yate’s correction, as appropriate. Acceptable error probability was ≤ 0.05.

Results

From March 1990 to May 1997 there were 49 neonates with PPHN admitted to the NICU. Of these, 11 neonates responded to IMV and 2 to synchronized assist/control ventilation (Bear Cub 750v, Allied Healthcare Products, Riverside, California) without HV and were therefore not eligible for enrollment. Thirty-six neonates met study entry criteria. Table 1 shows patient data and diagnoses. All air leak syndromes developed before study entry. The ventilator settings required at enrollment were comparable between the 2 groups (Table 2). At study entry, 4 neonates in the HFFI group and 5 in the HV group had oxygenation index values ≥ 40.

The highest mean airway pressures used in the HFFI group, compared with pre-entry Paw values, were 2 cm H2O lower than pre-entry Paw values in 12 neonates, equal in 3, and higher by 1, 2, and 3 cm H2O than pre-entry Paw values in 3 neonates.

Table 3 shows the hospital courses and clinical outcomes. All patients in the HFFI group could be oxygenated when HFFI was first started, and all but one responded to HFFI with Paco2 increases to > 120 mm Hg. One patient, who finally died, recovered from hypoxemia with pre-entry Paco2 values of 33–47 mm Hg and had Paco2 values of only 50–65 mm Hg during HFFI. One patient, enrolled into the study at 16 hours of life, responded to HFFI with a Paco2 increase to 141 mm Hg, but suffered sudden deterioration 8 hours after initiating HFFI. He was then switched

<table>
<thead>
<tr>
<th>Table 1. Patient Data and Diagnoses</th>
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<tr>
<td><strong>Ventilation Group</strong></td>
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<tr>
<td><strong>HV (n = 18)</strong></td>
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<tr>
<td>Outborn neonate</td>
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<tr>
<td>Birth weight (g)</td>
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<td>Male, n (%)</td>
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<tr>
<td>Age at intubation (min)</td>
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<td>Oxygenation index at enrollment</td>
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<td>Age at enrollment (h)</td>
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<tr>
<td>Length of mechanical ventilation at enrollment (h)</td>
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<tr>
<td>Diagnoses (n)</td>
</tr>
<tr>
<td>Meconium aspiration syndrome</td>
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<tr>
<td>Respiratory distress syndrome</td>
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<tr>
<td>Transient tachypnea of the newborn</td>
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<tr>
<td>Congenital pneumonia</td>
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<tr>
<td>Clear amniotic aspiration</td>
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<tr>
<td>Asphyxia: 1 min Apgar scores:</td>
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<td>= 2</td>
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<td>= 3–4</td>
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<td>= 5–6</td>
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<tr>
<td>Polycythemia</td>
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<tr>
<td>Hypothermia</td>
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<tr>
<td>Patent ductus arteriosus</td>
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<tr>
<td>Cardiac arrest of unknown cause</td>
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<td>Air leak syndrome: At entry</td>
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<td>After entry</td>
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HFFI = high-frequency flow interruption ventilation
HV = hyperventilation
Values are mean ± SD where applicable
to IMV, with PIP of 30 cm H$_2$O (pre-entry PIP 35 cm H$_2$O), to stabilize without HV. His P$_{aO_2}$ could be maintained between 53 and 63 mm Hg while the P$_{aCO_2}$ was between 49 and 63 mm Hg and pH was between 7.37 and 7.44. He was on IMV for 5½ hours before starting HFFI again at 30 hours of life, with a sustained response.

Of 17 patients who responded to HFFI with hyperoxegenation, whose Fi$_O_2$ could be reduced to ≤ 0.40, and who could be weaned to IMV only, 14 could be extubated. The mean ± SD duration of HFFI was 78.6 ± 45.6 hours.

<table>
<thead>
<tr>
<th>Table 2. Ventilator Settings on Conventional Ventilation at Enrollment</th>
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<tr>
<td><strong>Ventilator Settings</strong></td>
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</tr>
<tr>
<td>Fi$_O_2$</td>
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<tr>
<td>f (cycles/min)</td>
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<tr>
<td>PIP (cm H$_2$O)</td>
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<tr>
<td>P$_{aCO_2}$ (cm H$_2$O)</td>
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<td>PEEP (cm H$_2$O)</td>
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Values are mean ± SD
HV = hyperventilation
HFFI = high-frequency flow interruption ventilation
Fi$_O_2$ = fraction of inspired oxygen
f = respiratory frequency
PIP = peak inspiratory pressure
P$_{aCO_2}$ = mean arterial pressure
PEEP = positive end expiratory pressure

Sixteen neonates in the HV group recovered from hypoxemia and had P$_{aO_2}$ values greater than 120 mm Hg, and 14 of these had sustained responses allowing Fi$_O_2$ to be reduced. Ten had Fi$_O_2$ reduced to 0.70, but only 8 could be extubated. Eight died while still on mechanical ventilation. Two neonates did not respond to HV and sodium nitroprusside infusion and finally died while on Fi$_O_2$ of 1.0.

Neonates in the HFFI group developed more bacterial infections at earlier ages than those in the HV group, but the difference was not significant. Significantly more (9 vs 1, p = 0.009) neonates in the HFFI group developed pneumonia and/or lung abscess. Table 4 shows the ages at which diagnoses of infections were made, and the responsible organisms. Three HV group neonates had systemic candidiasis at a mean age of 17 days of life (range 11–20 d). Three HFFI group patients who had sustained responses to HFFI died from pneumonia and/or lung abscess after weaning to IMV. One died from respiratory failure unresponsive to HFFI. In the HV group, 5 died from respiratory failure, unresponsive or transiently responsive to HV, and another 5 died from bacterial infection while on mechanical ventilation.

**Discussion**

The HFFI strategy used in this study provides adequate ventilation at the lowest possible proximal airway pressure.
and prevents deterioration when initiating HFFI. The results of this study show that HFFI with the described strategy is better than HV in improving oxygenation and removing carbon dioxide in neonates with PPHN. None of the HFFI neonates developed sudden and marked deterioration when HFFI was started. All neonates recovered from hypoxemia, but 17 of them had rapid sustained responses with hyperoxygenation. Interestingly, hyperoxygenation developed when P_{\text{aCO}_2} values were still higher than 45 mm Hg (n = 5) or within the normal range (n = 7). Furthermore, hyperoxygenation could also be achieved both when pH was less than 7.35 (n = 7) and within the normal range (n = 8). We postulate that HFFI, by increasing recruitment of atelectatic areas and decreasing intrapulmonary shunt, is markedly more effective than HV in improving oxygenation and pulmonary gas exchange. As a result, hyperoxygenation without ventilator-induced hypocarbia and alkalosis develops and, consequently, pulmonary vascular resistance decreases and cardiovascular status improves rapidly. Moreover, while ventilating the lungs, HFFI prevents further parenchymal lung damage and airway injury to the initial lesion, as evidenced by the fact that more HFFI patients could have F_{\text{IO}_2}, reduced and be extubated and fewer developed CLD. Hyperoxygenation achieved without hypocarbia or alkalosis can prevent detrimental neurodevelopmental consequences of alkalosis, which reduces cerebral blood flow.25,26

Although each high-frequency ventilator has functional characteristics that are design-related, it now appears that when used with similar treatment strategies and within their functional limitations, similar clinical outcomes can be realized.27 Our postulation is supported by previously reported data. Gerstman et al27 have stated that HFV supports adequate gas exchange with small tidal volume and that the cycle of inflation and deflation associated with conventional mechanical ventilation is greatly reduced with HFV. Hamilton et al28 found that gas exchange in high-frequency oscillatory ventilation is more effective than conventional mechanical ventilation and that there is less histologic evidence of trauma to small airways with high-frequency oscillatory ventilation than with conventional mechanical ventilation. The pathophysiologic progression of the underlying disease is markedly less or slower in infants treated with high-frequency oscillatory ventilation.

Cardiovascular status of HFFI patients improved more rapidly, as evidenced by fewer HFFI patients requiring vasopressor use and more HFFI patients having vasopressors weaned more rapidly. This observation indicates no adverse effect of HFFI on cardiac output, with the described HFFI strategy. Moreover, the resulting rapid sustained response in oxygenation improves myocardial function. This agrees with previous reports, which support that HFV has no adverse effects on the heart or blood pressure20,29,30 and improves cardiovascular status more rapidly.31

Seventeen neonates who responded to HFFI with hyperoxygenation all had sustained responses that allowed reduction of F_{\text{IO}_2} to 0.70 and allowed extubation in 14 patients (82%). In contrast, only 10 of 16 HV patients who responded to HV had sustained responses that allowed reduction of F_{\text{IO}_2} to 0.70 and allowed extubation in only 8 (50%). These findings confirm 2 previous studies on the use of high-frequency oscillation in comparable patient

### Table 4. Infection Complications

<table>
<thead>
<tr>
<th>Bacterial infection (n)</th>
<th>Ventilation Group</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>HV (n = 9)</td>
</tr>
<tr>
<td>Age when diagnosed (d)</td>
<td></td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>6.7 ± 1.3</td>
</tr>
<tr>
<td>Range</td>
<td>2-12</td>
</tr>
<tr>
<td>Type of infection</td>
<td></td>
</tr>
<tr>
<td>Pneumonia</td>
<td>Enterobacter aerogenes (1)</td>
</tr>
<tr>
<td></td>
<td>Staphylococcus epidermidis (5)</td>
</tr>
<tr>
<td></td>
<td>Group D Streptococcus (1)</td>
</tr>
<tr>
<td></td>
<td>Klebsiella pneumonia (1)</td>
</tr>
<tr>
<td></td>
<td>Pseudomonas aeruginosa (1)</td>
</tr>
<tr>
<td></td>
<td>Pseudomonas aeruginosa (1)</td>
</tr>
</tbody>
</table>

HV = hyperventilation
HFFI = high-frequency flow interruption ventilation
*Number of infants in parentheses.
†One infant died.
populations, which suggested that high-frequency oscillation improves ventilation and is a more effective rescue tool than conventional mechanical ventilation. Furthermore, it also supports a report that found that response to HV does not predict outcome or survival.

More neonates in the HFFI group developed pneumonia and/or lung abscess, among whom 3 died after weaning to IMV. The responsible microorganisms were Gram-negative bacilli, which are typically nosocomial organisms and contaminants of the ventilator circuit and humidification apparatus (Fisher & Paykel MR600 humidifier, Fisher & Paykel HealthCare, Auckland, New Zealand). We think that the chance of contamination in our setting is high because we use reusable circuits and have only one exhalation block and diaphragm for the Infant Star Ventilator. As a result, it is impossible to change the exhalation block and diaphragm when routinely changing the circuit every 3 days. In the HV group, 5 of the 7 responsible organisms were coagulase-negative staphylococci. The risk factors are iatrogenic, including manipulative, diagnostic, and therapeutic procedures, prolonged administration of nutrient lipids, and central line placement. All risk factors were present for these neonates because of prolonged intubation and long hospital stay.

Neonates in the HFFI group developed no air leaks during HFFI ventilation, suggesting that HFFI is safe with respect to acute volutrauma. The development of air leak syndrome in both groups before enrollment and the unsustainable responses to HV, with fewer neonates extubated and more with CLD in the HV group, are consistent with previous studies that found that HV superimposes additional damage on the underlying disease process. Moreover, with the use of the HV approach, there are potential detrimental effects on the cardiovascular system. Wung et al stated that the HV technique, which uses whatever inflating pressure necessary to decrease P\(_{\text{aco}}\), to a critical level, impedes venous blood return to the heart, which reduces cardiac output and hypotension, both of which further reduce oxygenation. Dreyfuss and Saumon and Hamilton et al found that high end-inspiratory lung volume and the large phasic pressure-volume fluctuations of conventional mechanical ventilation contribute to the development of pulmonary edema and lung injury. Parker et al reported that in animals with healthy lungs, large tidal volume ventilation can damage the pulmonary capillary endothelium, alveolar and airway epithelium, and basement membranes. This mechanical damage allows fluid, protein, and blood to leak into the airways, alveoli, and lung interstitium. The mortality rate in the HV group (56%) is higher than previous reports of an overall mortality of 44% and 46% in PPHN patients treated with HV and tolazoline, respectively.

Our study suggests that HFFI, used with the strategy we describe, is safer and more effective than HV in improving gas exchange and oxygenation in neonates with PPHN and significantly decreases the incidence of CLD and the length of mechanical ventilation and hospitalization. In addition, neurodevelopmental consequences from hypoxemia and alkalosis can be prevented.

Ideally, a controlled trial should be done to test the efficacy of HFFI. Our study was not controlled, and the justification was that not all our attending physicians were familiar with and capable of using HFFI effectively, along with the limitations of our schedule of attendings’ rotation. Thus, the results may be biased by many factors, including diversity of diagnoses, severity of illness, variations in practice, and practice change among individual physicians during the 7-year study period. Comparisons of the diagnosis diversity and severity of illness in this study population did not seem to affect the outcomes of treatments. Meconium aspiration syndrome was the most common diagnosis in both groups, and the distribution of diagnoses did not differ between groups. The measure of severity of illness—oxygenation index—was also not statistically different between the two groups.

Practice variations among individual physicians are the only important variable in this study that may have created a bias. Walsh-Sukys et al found wide variability in the treatments of PPHN in the 12 academic NICUs included in their study. An analysis of the variations in practice among different modalities in both groups, and the distribution of diagnoses did not differ between groups. The measure of severity of illness—oxygenation index—was also not statistically different between the two groups.

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Improved Quality of Life Among Patients Completing a Pulmonary Rehabilitation Program: One Center’s Early Experience

Shirin Shafazand MD, James Canfield CCPT, and Ware G Kuschner MD

INTRODUCTION: The conclusion of previous investigations that pulmonary rehabilitation (PR) is an effective intervention for the management of chronic lung disease may not be generalizable to PR programs with limited experience delivering this complex, interdisciplinary service. OBJECTIVE: Determine whether PR is effective for the first group of patients treated in a newly formed interdisciplinary PR program. METHODS: We conducted a longitudinal analysis of changes in health-related quality of life and 6-minute walk test for the first group of patients completing our newly formed 8-week outpatient PR program. We studied 6 men, age 65–77 years, with stable severe chronic obstructive pulmonary disease. Patients completed the Chronic Respiratory Disease Questionnaire immediately before and 1 year after participation in our PR program. RESULTS: Four patients completed the PR 6-minute walk test both before and after the program. We found improvement in all Chronic Respiratory Disease Questionnaire domains at follow-up (mean ± SD) before and after: dyspnea 1.67 ± 0.82 vs 4.92 ± 0.49; emotional function 2.33 ± 0.82 vs 5.50 ± 0.55; fatigue 2.00 ± 0.63 vs 5.00 ± 0.63; feeling of mastery over disease 1.83 ± 0.41 vs 5.83 ± 1.17. The interval improvements in all health-related quality of life domains were statistically significant (p < 0.02 for all comparisons). There was a trend toward improvement in exercise tolerance: 231 ± 213 ft before PR vs 353 ± 66 ft at the 1-year follow-up (p = 0.2). CONCLUSIONS: PR can result in sustained improvement in the quality of life of patients with severe chronic obstructive pulmonary disease, even when this complex, interdisciplinary service is delivered by a newly formed and inexperienced PR program. Key words: pulmonary rehabilitation, chronic obstructive pulmonary disease, health-related quality of life, chronic respiratory disease questionnaire. [Respir Care 2001;46(6):595–600]

Introduction

Chronic obstructive pulmonary disease (COPD) is the fourth leading cause of death in the United States and its prevalence is increasing.1 The psychological impact of this disorder, the overall loss of physical ability, and the increased dependence on others for completing even simple tasks have contributed to the considerable morbidity of this disease. Annually, up to $40 billion is spent in the care of COPD patients.2 Pulmonary rehabilitation (PR) is receiving increasing recognition as an important component in the overall management of many COPD patients. Multiple groups have reported improved outcomes among patients completing PR programs.2-11 In 1996, Lacasse et al published a meta-analysis of 14 randomized controlled trials of PR.2 All PR programs included in the analysis were at least 4 weeks in duration. The majority of patients in the trials had severe COPD. The meta-analysis showed that PR programs result in clinically important improvement in dyspnea and feeling of mastery over disease among COPD patients.

Most published reports have looked at the effect of rehabilitation in patients with moderate and severe COPD. In a recent study, however, Berry et al12 found that even patients with mild COPD who participate in PR programs show improvements in dyspnea and fatigue. There is little doubt that, in the short term, PR has an important impact on COPD patients’ quality of life. However, the costs of
establishing and maintaining an effective program, lack of reimbursement by some insurance companies, limited health care resources, and the perceived need for a large dedicated health care team are all potential barriers that may discourage many centers from initiating PR programs. Moreover, centers offering PR may face a learning curve associated with optimizing delivery of this complex interdisciplinary service. Accordingly, early cohorts of patients may not derive the same benefits from PR that later patient cohorts experience.

To our knowledge, no reports in the literature have analyzed the efficacy of PR conducted by a newly formed team of health care specialists lacking prior cooperative PR experience. The findings of previous investigations of PR outcomes may not be generalizable to programs with limited experience delivering this interdisciplinary service. The potential lack of an early "payoff" from a newly formed PR program may discourage some centers from dedicating resources to developing PR programs. The purpose of this investigation was to determine whether a newly formed interdisciplinary team, consisting of respiratory therapists and other health care professionals, could deliver effective PR to its first group of patients. We assessed the effectiveness of our program by measuring health-related quality of life (HRQoL) and exercise capacity before and 1 year after the PR program.

The PR program at the Veterans Affairs Palo Alto Health Care System (VAPAHCs) was established in April 1998 under the medical direction of one of the authors (WK) and the technical management of another author (JC). Herein we present our experience with the first class of patients completing the outpatient PR program. We describe the elements of our PR program, including the methods of participant selection, the content of instructional and counseling sessions, and exercise prescription. Standard instruments such as the Chronic Respiratory Disease Questionnaire and 6-minute walk test (6-MWT) were used to assess patient outcomes.

Methods

Overview of the Program

The objectives of the VAPAHCs PR program are to optimize the patient's capacity to carry out activities of daily living, to improve exercise tolerance, to decrease the rate of progression of disability, and to reduce the frequency of hospitalization. The program faculty and staff consist of a medical director/pulmonologist, 2 respiratory therapists, a psychologist, a pharmacist, and a dietitian. All faculty and staff have competing clinical responsibilities and deliver PR on a part-time basis. The program is 8 weeks in duration, with the first 2 weeks dedicated to screening and obtaining baseline patient history, physical exam data, and exercise capacity and pulmonary function studies. For the remaining 6 weeks, patients meet twice weekly for 4 hours per session. Sessions are divided to include exercise therapy, respiratory therapy, and educational lectures. Patients are given educational literature describing the goals and techniques of PR. This educational material complements therapist-led teaching.

Components of the Program

Prior to initiating the exercise program, participants in our PR program (including all patients in this investigation) undergo a 6-MWT conducted by a respiratory therapist. Heart rate and a dyspnea score based on the Borg scale are recorded throughout the test. Aerobic exercises are then tailored to each patient's needs and capabilities. Exercise sessions include stretch and warm-up exercises followed by aerobic exercises such as walking on a treadmill, using a stationary bicycle, and walking around a track. Other exercises include bench press and weight lifting. Heart rate and dyspnea are monitored, and the intensity of the exercise is varied to maintain work load at 60% of maximum heart rate achieved on the initial 6-MWT. During each exercise session, work load is varied until the participant reaches the target heart rate and maximum Borg score (determined during the initial 6-MWT). A 5-minute rest stop is then permitted, followed by another 2–3 minutes of exercise to maximum intensity. Participants are able to exercise at higher work loads during consecutive exercise sessions while achieving maximum Borg score and target heart rate. These supervised exercise sessions last 1 hour per week during the first 3 weeks and are extended to two 1-hour sessions per week for the remainder of the program. In addition, patients are encouraged to continue with their predetermined home exercises every day for 30 minutes.

During respiratory therapy sessions, which follow the exercise sessions and are 30 minutes in duration, patients are instructed in the correct use and maintenance of the metered-dose inhaler, spacer device, nebulizer equipment, positive-pressure breathing devices, and suctioning equipment. Chest physical therapy, postural drainage, and percussion techniques are discussed in class. Effective coughing and proper breathing techniques, such as pursed lip breathing, are demonstrated and practiced. Also taught and performed are respiratory muscle exercises, including inspiratory muscle training using a handheld pressure threshold breathing device. Upper body, neck, and arm resistance movements using sandbags are reviewed. Relaxation techniques discussed include biofeedback for heart rate and respiratory control, calming imagery, and meditation.

The final component of our PR program includes educational lectures offered to family members and patients.
Family involvement is strongly encouraged. Caregivers of patients with severe COPD may be overwhelmed with the increased responsibilities and feelings of anger at the patient or situation. The aims of these sessions are to provide a basic understanding of the underlying disease process, promote medical compliance, emphasize preventive measures, and provide a stronger social support network for patients. Lectures are devoted to basic anatomy and physiology of the respiratory system and to understanding COPD. Participants are taught to recognize the signs and symptoms of respiratory infections, cope with exacerbations, and understand the indications for and potential side effects of medications. Lectures are conducted once a week and are 2 hours in duration.

In the initial 2 weeks of the PR program, the team psychologist offers sessions on stress management and smoking cessation. The remaining 4 weeks are devoted to 1-hour-long psychosocial group meetings where coping skills are reviewed and patients’ concerns addressed. The team nutritionist provides nutrition and dietary guidance in four 45-minute sessions. An individualized dietary plan is provided for each participant, and key principles are reviewed.

Each patient is provided a workbook, exercise instruction manual, and diary. Home exercises such as pelvic tilts, alternate leg raises, single knee-to-shoulder bending, forward bending, sideward bends, elbow and shoulder circling, and various other movements are described in the manual. Patients are asked to perform these exercises for 30 minutes daily and to keep a log of these exercises in their diaries. Prior to completion of the PR program, the home exercises are reviewed in class, and patients are encouraged to incorporate them in their daily activities beyond the PR program.

Study Design

We conducted a prospective analysis of HRQOL and exercise capacity for the first class of participants completing the 8-week outpatient PR program at VAPAHCS. The study was approved by the Stanford University Administrative Panel on Human Subjects in Medical Research.

Participants were referred from general medicine clinics, pulmonary, and other subspecialty clinics. Each potential candidate completed a screening questionnaire to assess health status, perceived dyspnea with activities of daily living, and goals and motivation for undertaking a PR program. The patients selected were clinically stable, COPD Class II-IV, and motivated as assessed by a formal psychological evaluation. Exclusion criteria included cor pulmonale, active coronary artery disease, recent myocardial infarction, cardiac arrhythmia, or any condition that precluded active participation in the PR program. In a screening period prior to initiation of the PR program, history, physical examination, and pulmonary function tests were obtained. The initial 6-MWT was conducted in the week prior to initiation of the PR program. Patients walked along a hospital corridor with predetermined distances in feet marked at various points along the corridor. They were instructed to walk at a pace comfortable to them and allowed as many stops as necessary. The distance in feet, number of rest pauses, dyspnea score, oxygen saturation, and maximum heart rate achieved were recorded. The measurements were repeated on a second day during the screening period and the average of 2 results was considered. The 6-MWT was repeated at the 1-year follow-up. Exercise prescription was based on the subject’s performance in the 6-MWT before the PR program.

Outcome Measurements

HRQOL was evaluated by responses to the Chronic Respiratory Disease Questionnaire (CRDQ), previously validated in the literature. The questionnaire was completed at baseline and 1-year follow-up. The CRDQ is a condition-specific HRQOL instrument administered by an interviewer in 15–25 minutes. Questions are divided into 4 domains, assessing dyspnea, fatigue, emotional function, and feeling of mastery over disease. Feeling of mastery over disease refers to how much the patient feels the disease is ruling his or her life. It attempts to quantify the patient’s feeling of control over the disease. The questionnaire is scored on a 7-point scale, larger values indicating improvement. The minimum important difference is a 0.5 change on a 7-point scale. All questionnaires were administered by one of the authors (JC).

Changes in exercise capacity were measured using the 6-MWT before the PR program and at the 1-year follow-up. Although timed walk tests are less reproducible than formal cardiopulmonary exercise testing, they are more convenient to conduct and are well accepted by patients. The ability to walk is functionally important in performing the activities of daily living, and performance in timed walked tests correlates well with self-reports on functional status questionnaires. The minimum clinically important difference for the 6-MWT has been shown to be around 54 m.

Statistical Analysis

Paired t tests were used to compare mean scores before and 1 year after the PR program for the 4 items in the CRDQ. The Wilcoxon matched pairs test was used to compare the mean 6-MWT results.

Results

Six participants started and completed the first PR class. All were men between 65 and 77 years old. All had COPD...
improved Quality of Life with a Pulmonary Rehabilitation Program

with forced expiratory volume in the first second values ranging from 15% to 39% of predicted. All were available for follow-up HRQOL assessments at 1 year. Table 1 reports the participants’ baseline characteristics.

Table 2 reports the CRDQ results. At the 1-year follow-up, there was statistically significant improvement in all 4 HRQOL domains. The mean dyspnea score improved from 1.67 at baseline to 0.49, emotional function score improved from 2.33 to 1.35, fatigue score improved from 2.00 to 1.00, and feeling of mastery of disease score improved from 1.83 to 0.83 (p < 0.02 for all comparisons).

All participants underwent exercise assessment at baseline, which included a 6-MWT. Two patients performed a 12-MWT at the 1-year follow-up. We did not collect 6-MWT information on those 2 subjects and accordingly cannot make interval comparisons of their exercise capacities. Those 2 patients were not available to return for formal 6-MWTs within an appropriate time frame. Table 3 presents baseline and 1-year follow-up results for 4 participants. Among those evaluated at the 1-year follow-up, participants exercised at home for a greater number of minutes per day following the PR program than at baseline, and there was a trend toward participants exercising more frequently following the PR program. Table 3 also shows a trend toward improvement in mean 6-MWT, though the difference was not statistically significant.

Discussion

This study demonstrates that an interdisciplinary team consisting of respiratory therapists and other health care professionals without prior cooperative PR experience can conduct effective PR. Despite the small sample size, we found that the first VAPAHCS PR program participant class experienced statistically significant improvement in all HRQOL indices measured.

The improvement in HRQOL noted at 1-year follow-up is encouraging; however, previous reports in the literature have indicated that these effects are not sustained and tend to diminish over time. Though our PR program did not have a structured maintenance phase, all 6 patients were seen periodically at the VAPAHCS for routine medical appointments, including appointments in our pulmonary clinic. The general principles learned in the PR program, including breathing techniques, principles of medication use, the importance of regular exercise, and strategies to improve well-being, were reinforced at these office visits. Reinforcement during clinic visits was unstructured and brief, but we speculate that this limited reinforcement had a positive role in contributing to the improved HRQOL at 1-year follow-up, insofar as it offered encouragement and served to remind patients to continue self-help health strategies. It remains to be determined whether regular maintenance programs that reinforce education and offer structured exercise sessions lead to additional benefits for PR program participants with respect to long-term outcomes.

Though there was a trend toward improvement in the 6-MWT, the analysis was underpowered to achieve statistical significance. However, significant improvement in 6-MWT has been reported. We chose the 6-MWT as a measure of exercise capacity. The chief advantage of this test is its ease of administration and good correlation with self-reports of functional capacity. Time walked tests are frequently used to evaluate the impact of PR programs. A recent study by Elpers et al highlights the considerable

Table 1. Baseline Characteristics of Participants*

<table>
<thead>
<tr>
<th>Domain</th>
<th>Baseline</th>
<th>1-Year Follow-Up</th>
<th>p*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyspnea</td>
<td>1.67 ± 0.82</td>
<td>0.49 ± 0.49</td>
<td>0.001</td>
</tr>
<tr>
<td>Emotional function</td>
<td>2.33 ± 0.82</td>
<td>1.50 ± 0.55</td>
<td>0.0005</td>
</tr>
<tr>
<td>Fatigue</td>
<td>2.00 ± 0.63</td>
<td>5.00 ± 0.63</td>
<td>0.002</td>
</tr>
<tr>
<td>Feeling of mastery over disease</td>
<td>1.83 ± 0.41</td>
<td>5.83 ± 1.17</td>
<td>0.001</td>
</tr>
</tbody>
</table>

*Assessed by the Chronic Respiratory Disease Questionnaire (12–16).
For all questionnaire items, higher score indicates better health-related quality of life.
* Baseline questionnaires were administered 2 weeks prior to initiation of the pulmonary rehabilitation program.
* Calculated by paired t test.
Values are mean ± SD.

Table 2. Exercise at Baseline and at 1-Year Follow-Up*

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Baseline</th>
<th>1-Year Follow-Up</th>
<th>p*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exercise duration (min/d)</td>
<td>13.00 ± 4.76</td>
<td>22.50 ± 8.88</td>
<td>0.031</td>
</tr>
<tr>
<td>Exercise frequency (d/wk)</td>
<td>2.00 ± 0.00</td>
<td>2.75 ± 0.50</td>
<td>0.058</td>
</tr>
<tr>
<td>6-minute walk test (ft)</td>
<td>231.25 ± 213.48</td>
<td>325.50 ± 66.02</td>
<td>0.201</td>
</tr>
</tbody>
</table>

*n = 4
Values are mean ± SD.
* Baseline data obtained during 1-week period prior to initiation of the rehabilitation program.
* Two patients did not return to the study site for exercise assessment at 1-year follow-up.
* Calculated by Wilcoxon matched pairs test.
* Patient reported average time spent exercising on those days performing any exercise.
* Patient reported number of days per week engaged in exercise on average.
variability among rehabilitation centers across North America in conducting these tests. The 6-MWT in our program was standardized such that all 6 participants walked down the same hallway while receiving similar instruction and encouragement by one of the authors (JC).

Baseline 6-MWT, including measurement of dyspnea and maximum heart rate achieved, were used to determine the appropriate exercise prescription for each participant. Formal cardiopulmonary exercise testing would have given a more precise picture of participants’ exercise capabilities. This may be an important limitation for smaller programs that do not have the resources to perform cardiopulmonary exercise testing on all PR program participants.

The most recent American Thoracic Society guidelines recommend using a percentage of maximum work rate, such as 60% of maximum oxygen consumption, as an appropriate target for exercise prescription. However, it is acknowledged that, despite several limitations, using a percentage of maximum heart rate as an estimate of training intensity is appropriate. In our program we set the maximum target exercise intensity at 60% of maximum heart rate achieved during baseline 6-MWT.

We carried out baseline pulmonary function test studies to stage participants’ disease. We intentionally did not perform follow-up pulmonary function tests. Prior investigators have failed to show improvement in pulmonary function test results following completion of a PR program.

We did not carry out health care utilization and cost-effectiveness analyses and therefore cannot determine whether a relatively inexperienced PR program can yield health care cost savings. Previous studies have, however, shown a trend toward decreased health care utilization by patients completing PR programs.

In 1995, Bickford et al. conducted a national PR survey that reviewed the variety of rehabilitation programs offered across the United States. The survey indicated that programs differed in patient selection criteria, duration, exercise prescription, educational component, size and time commitment of health care team, and costs. Nevertheless, most programs report some benefit for participants.

Many questions in the field of pulmonary rehabilitation remain unanswered. More studies are needed to determine the optimal staffing composition of the rehabilitation team, as well as the relative importance of the various elements of the PR program, including education, psychosocial support, and exercise. Finally, additional work is needed to establish how cost-efficiency can be maximized.

Conclusions

We conclude that an interdisciplinary team of respiratory therapists and other health care specialists lacking prior cooperative experience in PR can conduct PR that results in sustained improvement in HRQOL for PR program participants. This study demonstrates that PR can be delivered effectively without a prolonged learning curve for newly formed PR programs. Centers initiating this complex interdisciplinary service can expect benefits for the very first group of patients.

REFERENCES

Improved Quality of Life with a Pulmonary Rehabilitation Program


47th International Respiratory Congress
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Bronchial Atresia with Relapsing Pulmonary Infection in a Middle-Aged Man

Carla R Nordstrom MD, Gregory C Kane MD, Richard J Wechsler MD, Ana M Salazar MD, Herbert E Cohn MD, and John L Farber MD

Congenital bronchial atresia (CBA) is a rare disorder, first reported in 1953. Less than 100 cases are reported in the literature, mostly in young, asymptomatic male patients with involvement of the apical-posterior segment of the left upper lobe. Patients may complain of fever, cough, or shortness of breath, symptoms that result from post-obstructive, sometimes recurrent, infections. Chest radiography and computed tomography reveal a tubular branching density representing mucus impaction or mucocele with surrounding focal hyperinflation. Surgical excision is reserved for symptomatic cases. We report an unusual case of CBA in a middle-aged man with a history of relapsing infections, who was found to have an atretic superior segment of the left lower lobe, with surrounding areas of organizing pneumonia. *Key words: atresia, bronchial, congenital, mucocele, hyperinflation.* [Respir Care 2001;46(6):601–603]

Introduction

In a description of 3 cases of cystic changes in the lungs, Ramsay and Byron in 1953 reported the case of a 17-year-old female patient with a mucocele who was found at surgery to have an atretic superior segment of the left lower lobe.1 Simon and Reid reported 3 additional cases in 1963 and termed the disorder congenital bronchial atresia (CBA).2 Since then, less than 100 cases have been reported. Most affected persons have been asymptomatic men in the second or third decade of life. Several patients presented with fever, cough, and shortness of breath—symptoms that resulted from post-obstructive, sometimes recurrent infection. Radiography and computed tomography findings usually revealed mucus impaction or a mucocele with surrounding focal hyperinflation. Surgery was reserved for symptomatic cases. We report here an unusual case of this rare disorder in order to emphasize that CBA should also be considered in older patients with histories of relapsing infections and otherwise typical radiologic findings.

Case Report

A 39-year-old white man presented with low-grade fever, dry cough, pleuritic chest pain, and generalized fatigue refractory to prolonged courses of antibiotic therapy. He had been in excellent health until one year prior to presentation, when he developed an upper respiratory infection. He experienced a sudden, shaking chill, low-grade fever, sweats, productive cough, and left-sided pleuritic chest pain. Antibiotic therapy was given initially as an outpatient and then as an inpatient. Despite prolonged treatment, the fever and fatigue persisted for 8 weeks. Evaluation at another institution resulted in another prolonged course of antibiotics for a presumed lung abscess. Bronchoscopy with transbronchial biopsy was not diagnostic. The fever and fatigue improved but recurred and prompted evaluation at our institution. The patient had continued to work, but he could tolerate only local business trips and could not participate in his usual sports activities. He had
no prior medical problems and was taking no medications. He was a lifelong non-smoker.

On physical examination, the patient was a well developed, well nourished, fit but pale, middle-aged man in no apparent distress. Vital signs: temperature 98.3° F, pulse 70 beats/min and regular, respiratory rate 12 breaths/min, and blood pressure 110/60 mm Hg in both arms. The nasal mucosa was slightly boggy. Scattered wheezes were heard throughout both lung fields. A chest radiograph revealed a branching, tubular density in the left middle lung field, with surrounding hyperinflation. Computed tomography with contrast revealed abrupt termination of the superior segment of the left lower lobe bronchus, with distal mucoid impaction and surrounding air trapping, which are indicative of CBA (Figs. 1 and 2). Pulmonary function tests revealed mild obstructive disease, with forced vital capacity 5.47 L (108% of predicted), forced expiratory volume in the first second 3.29 L (80% of predicted), ratio of forced expiratory volume in the first second to forced vital capacity 60%, normal lung volumes, and normal diffusing capacity. Laboratory data were all normal. There was no eosinophilia. The immunoglobulin E level was normal. Skin testing with Aspergillus antigen and purified protein derivative was negative. Fiberoptic bronchoscopy failed to visualize the superior segment of the left lower lobe—an observation supporting the diagnosis of bronchial atresia. Surgical resection was performed to prevent further episodes of pneumonia. The postoperative course was uncomplicated. On examination 4 months later, the patient had no new fever, cough, or fatigue. He had returned to work and resumed full activity, including sports.

Examination of the lobectomy specimen revealed bronchial stenosis with distal bronchocele. At the site of the narrowed lumen, the cartilage plates of the bronchus were abnormal in shape and distribution. In addition, they were surrounded by considerable fibrosis (Fig. 3). The distal bronchus was dilated, chronically inflamed, and impacted with mucus. The surrounding pulmonary parenchyma displayed chronic pneumonia with organizing pneumonia and fibrosis. The findings confirmed the diagnosis of CBA with persistent, post-obstructive inflammation.

**Discussion**

Formation of the bronchial bud and segmental bronchi takes place at 4 and 5 weeks gestation, respectively. Complete bronchial ramification occurs at 16 weeks gestation.
Ultrasonography has suggested that CBA occurs after this ramification, at about 24–25 weeks gestation. Similar to congenital intestinal atresia, CBA is thought to result from an earlier, probably vascular, insult. It is not known why the left upper lobe seems to be more vulnerable to such developmental trauma. There is no direct aeration of the atretic segment. Collateral ventilation occurs via the interalveolar pores of Kohn, the bronchovascular channels of Lambert, and the interbronchial channels, resulting in progressive distal air trapping (hyperinflation). Bronchial mucus secretion distal to the atretic segment results in mucus impaction and obstruction with bronchial dilation and then formation of the mucocele. The affected bronchi are often tubular and branching in appearance, but can also be round and mimic a neoplasm. Infection distal to the atresia occurs via the collateral ventilatory channels.

Most CBA patients are asymptomatic at the time of incidental diagnosis. Less than a third of patients present with fever, cough, or shortness of breath, and even fewer present with a history of recurrent infections. Affected individuals are usually in their teens and twenties, with a slight predominance of occurrence in males. Physical findings are limited to decreased breath sounds and sometimes wheezing in the affected area. Spirometry is usually restricted, owing to the complete absence of direct ventilation of the affected segment. Chest radiography and computed tomography reveal an extralobar mass or mucocele with distal hyperinflation.

In addition to CBA, the differential diagnosis of a hilar or extralobar mass with peripheral hyperinflation produced by internal or external bronchial obstruction includes bronchial adenoma or carcinoma, mucoid bronchial impaction, vascular compression, pulmonary sequestration, or bronchogenic cyst. Unilateral perihilar masses usually represent a neoplasm. Central mucoid impaction is most often the result of allergic bronchopulmonary aspergillosis, but that condition is not associated with distal hyperinflation. Lobular hyperinflation can occur with bronchial neoplasm, foreign body, infantile emphysema, and Swyer-James syndrome.

A diagnosis of CBA is supported by failure of bronchoscopy to visualize the affected segment. In the past, bronchography was helpful. However, today, radiographic and computed tomographic characteristics make those procedures unnecessary. Surgery is not required for diagnosis and is reserved for symptomatic cases. Excision by segmentectomy or lobectomy is considered curative.

This patient’s presentation with CBA was unusual because of his age, location of atretic segment, and history of relapsing infections. Few cases have been reported in middle-age patients. In the largest review, of 86 patients, Jederlinic et al reported one case involving a 44-year-old woman. An earlier, smaller review of 17 patients by Montague and Shaw in 1974 reported 1 patient age 41 years. CBA typically involves the apical-posterior segment of the left upper lobe. Involvement of the left lower lobe is much less common. Only 14 cases (16%) involving the left lower lobe were reported in the Jederlinic et al series and only 1 case (6%) with left lower lobe involvement was reported in the Montague and Shaw series.

The diagnosis in the present case was suggested by the typical computed tomography findings of a mucocele with surrounding hyperinflation. The resected specimen demonstrated not only the classic bronchial stenosis, but disorganization of the bronchial cartilaginous plates was also evident. CBA should be considered in middle-aged patients with recurrent or relapsing pneumonia and characteristic radiologic findings.

REFERENCES
A New System for Understanding Modes of Mechanical Ventilation

Robert L Chatburn RRT FAARC and Frank P Primiano Jr PhD MBA

Numerous ventilation modes and ventilation options have become available as new mechanical ventilators have reached the market. Ventilator manufacturers have no standardized terminology for ventilator modes and ventilation options, and ventilator operator’s manuals do not help the clinician compare the modes of ventilators from different manufacturers. This article proposes a standardized system for classifying ventilation modes, based on general engineering principles and a small set of explicit definitions. Though there may be resistance by ventilator manufacturers to a standardized system of ventilation terminology, clinicians and health care equipment purchasers should adopt such a system in the interest of clear communication—the lack of which prevents clinicians from fully understanding the therapies they administer and could compromise the quality of patient care. Key words: mechanical ventilation, ventilation mode, ventilator terminology, ventilator functions, classification. [Respir Care 2001;46(6):604–621]

Introduction

New modes of ventilation are proliferating at an alarming rate. Whether these new features are effective remains to be shown. But an even more basic problem is that we have not developed a means to educate ourselves about how ventilators function in these modes. The time delay between editions of standard textbooks is too long to reflect current ventilator capabilities. Product information from manufacturers is biased and confusing. Not everyone can gain access to demonstration models to personally examine the various ventilator features. These factors and more conspire to create a serious knowledge gap in this important subject.

ECRI (formerly known as Emergency Care Research Institute), publisher of Health Devices reports, is one respected source for objective product comparisons. However, even their reports do not address the knowledge gap adequately. For example, a recent report1 contained an update of an earlier evaluation of intensive care ventilators.2 The purpose of the report was to examine the new features of 9 “comprehensive-capability” intensive care ventilators and provide recommendations on what ECR1 thinks we should buy. The 9 ventilators were: BEAR 1000 and TBird AVSIII (ThermoRespiratory Group, Palm Springs, California), Evita 2 dura and Evita 4 (Dräger Medical, Telford, Pennsylvania), Puritan Bennett 760 and 840 (Mallinckrodt, Carlsbad, California), Wave E200 (Newport Medical Instruments, Newport Beach, California), Galileo (Hamilton Medical, Reno, Nevada), and Servo 300 (Siemens Medical, Danvers, Massachusetts).

Each ventilator was judged on 10 characteristics. One of these, the “performance characteristic,” was based on which of the advanced modes and features the ventilator had. These modes and features were defined by ECR1 in a special glossary table. Unfortunately, ECR1 appears to have made up its own definitions rather than taking advantage of the standard classification system and terminology that has been adopted by the major textbooks covering mechanical ventilation3–5 or the documents generated by the Consensus Conference on the Essentials of Mechanical Ventilators, sponsored by the American Association for Respiratory Care.6 As a result, readers of the ECR1 report might find the subject bewildering.

The purpose of this article is to point out areas of potential confusion in the description of ventilation
modes and to introduce a ventilation mode classification system that extends the standard ventilator classification system.

Describing New Features

Twenty years ago, when ventilators had only 1 or 2 modes, there was no need for a classification system. But today a ventilator may have over a dozen modes, and there is no consistency in mode nomenclature from one manufacturer to the next. The need for a mode classification system becomes evident to anyone trying to understand and compare the new features of current ventilators. As an introduction to this need, we will examine some of the issues raised by the ECRI report.1

The ECRI report describes 2 of the most notable new ventilator capabilities. One they call the "responsive-valve feature" and the other they call "patient-responsive modes." The "responsive-valve feature" maintains a constant airway pressure during inspiration, even if the patient makes a sudden ventilatory motion, such as breathing in or coughing. Some earlier ventilators were unable to respond to those patient actions very well and would, for instance, cycle inspiration off with a high pressure alarm in the case of an expiratory effort or cough. In short, a "responsive valve feature" is simply a better pressure control mechanism, employing better software algorithms and what some manufacturers have called an "active exhalation valve." Such a valve usually does not close completely during inspiration but, rather, provides a dynamic resistance to the continuous flow through the patient circuit to maintain even pressure control at the airway opening. This improved form of pressure control is an advantage mainly during pressure-controlled continuous or intermittent mandatory ventilation (ie, wherein inspiration is pressure-limited and time-cycled). It is less meaningful in modes that are designed to improve synchrony with patient demands by other means.

The "patient responsive modes" are those that "adjust ventilator performance in response to measured changes in the patient's lung conditions."1 The ventilator measures respiratory system mechanics, which include effects of the lungs and chest wall. The ECRI report also mentions a "combination mode" that provides "not only the variable flow associated with pressure-controlled modes but also the volume guarantee associated with volume-controlled modes."1

The ECRI glossary table goes on to define some of the newer modes found on the 9 ventilators reviewed. Patient-responsive modes include adaptive pressure ventilation, pressure-regulated volume control, and adaptive support ventilation. Adaptive pressure ventilation and pressure-regulated volume control are given the same definition: "This pressure-controlled, variable-flow mode adjusts the inspiratory pressure to the lowest possible level while delivering the target volume." Adaptive support ventilation is defined, in part, as follows:

The delivered minute volume is based on the patient's ideal body weight and the percentage of the minute volume that is to be supplemented. During each breath, the ventilator determines the patient's lung mechanics, then sets the frequency, tidal volume, and inspiration-expiration ratio to minimize pressure while still ensuring the required minute volume.1

ECRI defines AutoFlow as a "responsive valve feature: in addition, though it is not an actual ventilation mode, it provides ventilation similar to adaptive pressure ventilation and pressure-regulated volume control, which are patient-responsive modes."1

Finally, the ECRI glossary states that some of the following are "combination modes": pressure augmentation, volume-assured pressure support, adaptive pressure ventilation, pressure-regulated volume control, and volume support.

Potential Problems

What message might be conveyed to readers with no prior knowledge of the above-mentioned modes? Well, adaptive support ventilation adjusts frequency, tidal volume, and inspiration-expiration ratio, but we don't know if it is with pressure control or volume control. AutoFlow "is not an actual mode of ventilation," which raises the question, "What is a mode?" AutoFlow "provides ventilation similar to adaptive pressure ventilation and pressure-regulated volume control," which makes us wonder how it differs from those 2 modes. ECRI defines modes in the 1998 paper2 (p. 309) as: "A mode of operation defines both the variable that the ventilator will control during gas delivery (normally pressure, flow or volume) and the algorithm that will be used to initiate a machine breath (called triggering) and to end the machine breath." As we will see, this is too simplistic a definition.

The ECRI report lists several "combination modes," but some are also called "patient-responsive" modes, with no indication of how to tell them apart. We recall that a "patient-responsive" mode adjusts inspiratory pressure according to lung mechanics, and a "combination mode" is a pressure-controlled mode with a volume guarantee. Upon reflection we realize that a volume guarantee cannot be enforced in pressure-control without monitoring lung mechanics and adjusting inspiratory pressure. Therefore, calling a mode both "patient-responsive" and a "combination mode" is redundant: it adds no new discriminating ability. And we are now even more confused about AutoFlow. It is called a "responsive-valve" feature rather than a mode.
and yet it is "... similar to patient-responsive combination modes."

Ventilator operator manuals are often a source of confusion. For example, in the Evita 4 operator manual (preliminary version), in the section on setting ventilation modes, AutoFlow is mentioned as an optional setting for volume control modes. It is described as "... automatically optimizing inspiratory flow... Inspiratory flow is decelerated and controlled in such a way that the set tidal volume is delivered at a minimum airway pressure for a given patient lung compliance while avoiding pressure peaks." In the section on the theory of operation, there is a statement that "AutoFlow is a new supplement optimizing Inspiratory flow during mandatory ventilator breaths in the volume-controlled modes CMV [continuous mandatory ventilation], SIMV [synchronized intermittent mandatory ventilation], and MMV [mandatory minute ventilation]." The manual does not give the definition of volume control, pressure control, or mandatory breaths. The manual leads one to believe that AutoFlow and $P_{\text{max}}$ (maximum pressure) are simply features that one can activate during volume control modes, but, in fact, they switch the basic mode entirely by changing inspiratory phase variables from flow-limited and time-cycled to pressure-limited and time-cycled (i.e., changes inspiration from volume control to pressure control).

As an aside, during mechanical ventilation, we are not usually interested if flow decelerates or accelerates. Of more concern is if a volume of gas does. Consequently, the terms "accelerating flow" and "decelerating flow" to describe time-varying flow waveforms are inappropriate and invariably used incorrectly. If a car slows, we do not say that its velocity decelerates; we say that the car decelerates. We do not say that a cyclotron is a velocity accelerator but that it is a particle accelerator. The rate of change of position of an object is the velocity of the object; analogously, the rate of change of volume is flow. The rate of change of velocity of an object is the acceleration of the position of the object; likewise, the rate of change of flow is the acceleration of volume, not the acceleration of flow. So if we want to say that flow changes, we should simply talk about an increasing flow or a decreasing flow (or an accelerating volume or a decelerating volume), not an accelerating flow or a decelerating flow. The term "decelerating flow" is another one of those unfortunate cases in which some anonymous author of a ventilator manual used a word without much thought and it stuck.

**Definitions: Order from Chaos**

The problems illustrated above stem from the lack of standardization of terminology in the field. To remedy this, we need a classification system for ventilation modes. But before we can create a classification scheme, we must first define some basic terms, many of which have been presented elsewhere, and others are borrowed from engineering control theory.

**Objectives of mechanical ventilation:** Assure that the patient receives, via pulmonary ventilation, the minute volume of appropriate gases required to satisfy the patient's respiratory needs while not damaging the lungs, impairing circulatory function, or increasing the patient's discomfort.

**Mode of ventilation:** The manner in which a ventilator achieves the objectives of mechanical ventilation. A mode can be identified and/or classified by specifying a combination of:

- Breathing pattern produced: primary breath control variable and breath sequence
- Control type: classification of high-level control strategy: hierarchical set point control, hierarchical servo control, adaptive set point control
- Specific strategy: phase variables, and operational logic

**Transrespiratory pressure (difference):** The pressure difference across the lungs, airway (including all or part of a breathing circuit), and chest wall; airway pressure minus pressure on the body surface ($P_{aw} - P_{aw}$). Routinely referred to simply as "transrespiratory pressure."

**Control variable:** The variable (e.g., pressure, volume, or flow) that the ventilator manipulates to cause inspiration. According to the equation of motion for the respiratory system, if transrespiratory pressure is the control variable, then volume and flow are dependent on it: on lung, chest wall, and breathing circuit resistance and compliance; and on muscle effort. If volume or flow is the control variable, then transrespiratory pressure is dependent on them: on lung, chest wall, and breathing circuit resistance and compliance; and on muscle effort. Only one variable can be manipulated and serve as the control variable at one time during inspiration.

**Output variable(s):** The variable(s) associated with the specific control strategy. Common examples are peak inspiratory pressure, peak flow, flow waveform, tidal volume, and minute ventilation.

**Pressure control:** A control scheme in which pressure is the control variable. Specifically, the ventilator attempts to attain or maintain a specified transrespiratory pressure (usually by manipulating either airway or body surface pressure) waveform and/or amplitude for every inspiration. Delivered volume and flow are dependent on lung, chest wall, and breathing circuit mechanics; on ventilatory muscle activity; and on the transrespiratory pressure waveform.

**Volume control:** A control scheme in which volume (or, equivalently, flow) is the control variable. Specifically, the ventilator attempts to attain or maintain a specified volume or flow waveform and/or amplitude for every inspiration. Transrespiratory pressure is dependent on lung, chest wall...
and breathing circuit mechanics: ventilatory muscle activity; and the volume and flow waveforms. Note that because volume and flow are mathematically related (ie, volume is the integral of flow and flow is the derivative of volume), controlling one gives control of the other.

**Dual control:** A control scheme in which the ventilator can switch between volume control and pressure control. Examples include (a) inspiration is pressure-controlled within breaths but the pressure limit is automatically adjusted between breaths to achieve a target tidal volume in the presence of changing lung mechanics and ventilatory muscle activity, and (b) inspiration switches between pressure control and volume control within a breath, depending on lung mechanics and ventilatory muscle activity.

**Breath:** A positive airway flow (inspiration), relative to its contemporary baseline, paired with a negative airway flow (expiration) relative to its contemporary baseline, both associated with ventilation of the lungs. This definition excludes flow changes caused by hiccups or cardiogenic oscillations, but it allows the superimposition of, say, a spontaneous breath on a mandatory breath or vice versa (these breath types are defined below). Traditionally, the flow baseline is taken as flow = 0. However, since "breaths" can be superimposed on existing flow in various circumstances, the inspiratory and expiratory movements of gas must be judged relative to the level of flow existing when these movements occur. Typically, inspiration immediately precedes expiration. However, it is possible for the reverse to occur, such as during manual resuscitation or during assisted ventilation using a tilt bed.

**Spontaneous breath:** In the context of mechanical ventilation, a breath for which the patient determines both when to start and the tidal volume. That is, the patient both triggers and cycles the breath. A spontaneous breath may occur during a mandatory breath (Fig. 1).

**Mandatory breath:** In the context of mechanical ventilation, all breaths that are not spontaneous breaths: a breath for which the machine determines when to start and/or the tidal volume. That is, the machine triggers and/or cycles the breath (Fig. 2).

**Assisted breath (ie, a mechanically assisted breath):** A breath during which all or part of inspiratory and/or expiratory flow is generated by a change in transrespiratory pressure (ie, airway pressure minus body surface pressure, $P_{aw} - P_{bs}$) due to an external agent (eg, manual or mechanical ventilator).
Understanding Modes of Mechanical Ventilation

Breath sequence: There are 3 possible breath sequences, designated as follows:
- Continuous mandatory ventilation (CMV): all breaths are mandatory.
- Continuous spontaneous ventilation (CSV): all breaths are spontaneous.
- Intermittent mandatory ventilation (IMV): breaths can be either mandatory or spontaneous.

Breaths can occur separately, or breaths can be superimposed on each other (e.g., spontaneous breaths superimposed on mandatory breaths, as in bi-level or airway pressure release ventilation [APRV]; or mandatory breaths superimposed on spontaneous breaths, as in high-frequency ventilation administered during breathing). When the mandatory breath is patient-triggered, it is commonly referred to as SIMV. However, because the trigger variable can be specified in the description of phase variables, we will use IMV instead of SIMV to designate general breath sequences.

Control type: A categorization of the high-level control function of the ventilator. The control type specifies the control variable and how it is to be manipulated (e.g., which waveform is to be produced). There are a variety of ways that control variables are manipulated during mechanical ventilation (Table 1). The 2 basic categories are open loop and closed loop control.

Open loop control: Output is controlled by the operator-preset input (e.g., flow and inspiratory time) and disturbances in the environment (e.g., circuit leaks, changes in lung mechanics, respiratory muscle effort). The output is not measured and therefore is not used to make corrective adjustments.

Closed loop (negative feedback) control: Output is measured, providing a feedback signal that can be compared to the input value. In the classic negative feedback control system, when a difference between input and output is sensed, an error signal is generated that is used to adjust the output so that it matches the input. Feedback control forces the output to be stable in the presence of disturbances in the environment (e.g., circuit leaks, changes in lung mechanics, respiratory muscle effort). Note that the feedback signal may be electrical (e.g., from an electronic pressure transducer) or mechanical (e.g., some continuous positive airway pressure valves. In such mechanical devices, a spring provides the input setting, and the position of the diaphragm [a measure of the gas pressure] is the feedback signal. When the force caused by the pressure exceeds the spring load, the diaphragm deflects and vents gas to the atmosphere to relieve the pressure.)

Closed loop control strategies: Several types of closed loop, negative feedback control strategies have been used to control commercial mechanical ventilators. These can be divided into 2 groups, set point control and servo control, according to the dynamics of the input to the controller:

Set point control: The output is forced to match a constant, unvarying, operator-preset input value. The production of a constant tidal volume from breath to breath is an example of set point control.

Servo control: The output is forced to follow a dynamic, varying, operator-specified input. For example, the tube compensation mode of the Puritan Bennett 840 ventilator measures instantaneous flow and forces instantaneous pressure to be equal to flow multiplied by a constant (representing endotracheal tube resistance).

Two strategies have been used to implement set point and servo control in commercial ventilators: hierarchical control and adaptive control.

Hierarchical control: The output is forced to follow or match an operator preset input according to several layers of conditional logic. An example of this is pressure augment on the Bear 1000 ventilator. Inspiration starts out as pressure-controlled and is modified by a series of "if-then" statements: if the preset tidal volume is not met by the time flow decays to the set value, then inspiration switches to volume control. However, if airway pressure drops below the set value, then inspiration switches back to pressure control. (All commercial ventilators have some degree of
hierarchical control. This is embodied in control statements containing phase variables.)

Adaptive control: The output is forced to follow or match an operator-specified input as the operating conditions or characteristics (parameters) of the controlled system change. Control is achieved by using the values of the output and other system variables to adjust the phase variables so that some measure of the performance of the system is optimized (either maximized or minimized). Even though there is a hierarchical organization to adaptive control strategies used in mechanical ventilators, we refer to these strategies as simply “adaptive control.” Adaptive control is illustrated by pressure-regulated volume control on the Servo 300. Here, tidal volume is the output variable and there are multiple levels of feedback. Feedback of pressure is used to maintain the inspiratory pressure waveform and amplitude and, as the mechanics of the patient’s ventilatory system change, flow (and volume) feedback is used to adjust the pressure limit to the minimum value that will maintain the operator-prescribed tidal volume. Adaptive support ventilation on the Galileo is another example in which adaptive control is used to automatically adjust the pressure limit that will maintain a required tidal volume. However, the tidal volume is not operator preset. Rather, the ventilator determines the optimal tidal volume (based on current lung mechanics) that minimizes the work of breathing while producing a minute ventilation based on the patient’s physical characteristics. In this case, minute ventilation is the output variable.

Phase variable: A variable (ie, pressure, volume, flow, or time) that is measured and used by the ventilator to initiate some phase of the breath cycle. The trigger variable starts inspiration. The limit variable sets the maximum value (amplitude) the control variable can attain before inspiration ends. The cycle variable terminates inspiration. For example, a particular breath may be flow-triggered, pressure-limited, and time-cycled. Some of the newer ventilators allow the operator to set the cycle threshold value on flow-cycled breaths in the pressure-support mode. Alarm settings are often referred to as “limits,” which is inappropriate if they end inspiration. To avoid confusion, they should be called alarm settings or cycle thresholds.

Conditional variables: Variables that are used in control logic that can be structured as “if-then” statements. That is, if the value of a conditional variable does or does not match some preset criterion, then some action occurs to change the ventilatory pattern. For example, if the measured tidal volume is less than the preset tidal volume, then the pressure limit is increased by 2 cm H₂O.

Elastic (pneumatic) load: The pressure difference applied across a system (eg, a container) that sustains the system’s volume relative to some reference volume, and for the amount of its contents relative to some reference amount. For a linear system, elastance × volume, or volume/comp-

pliance. For a container, the overall effective elastance (compliance) includes the elastances (compliances) of its structural components and the compressibility of the fluid (gas or liquid) within it.

Restive (pneumatic) load: The pressure difference applied across a system (eg, a container) that sustains a rate of change of the system’s volume and/or sustains a flow of fluid within or through the system. For a linear system, resistance × flow, or resistance × rate of change of volume. For a container, the effective resistance includes the mechanical (usually viscous) resistances of its structural components and the flow resistance of the fluid (gas or liquid) within it.

Inertial (pneumatic) load: The pressure difference applied across a system (eg, a container) that sustains acceleration of the system’s volume and/or sustains a rate of change in the flow of fluid within or through the system. For a linear system, inertia × rate of change of flow, or inertia × acceleration of volume. For a container, the effective inertance includes the inertances of its structural components and the inertance of the fluid (gas or liquid) within it.

We will use these definitions to unravel the mystery of adaptive pressure ventilation, adaptive support ventilation, and other modes, but first it may be helpful to realize that there is an evolutionary scale that can be applied to ventilator control schemes. This will help us to view the new modes in their proper context.

The Evolution of Control Schemes

Table 1 shows that there have been 3 major types of ventilator control scheme (open loop control, closed loop control of a single control variable, and closed loop control of 2 control variables [dual control], with several subtypes. Open loop control is essentially no control. For example, early high-frequency ventilators simply generated pulses of gas flow without measurement or control of pressure, volume, or flow. Flow into the patient was a function of the relative impedances of the respiratory system and the exhalation manifold. Thus, delivered pressures and volumes were affected by any disturbances in the system (eg, changing lung mechanics, the patient’s ventilatory efforts, and leaks). Other examples are simple infant ventilators or transport ventilators in which the pressure limit was set at maximum and the only controls are inspiratory time and the flow meter.

Closed loop control was an improvement in that the delivered pressure, volume, and flow could be measured and used as feedback information to control the driving mechanism. Thus, inspiratory volumes, flows, and pressures could be made to match or follow specified input values despite disturbances such as changes in patient load and minor leaks in the system. All ventilators that deliver
### Understanding Modes of Mechanical Ventilation

<table>
<thead>
<tr>
<th>Control Type</th>
<th>Characteristics</th>
<th>Example Control Scheme</th>
<th>Example Mode</th>
<th>Example Ventilator</th>
</tr>
</thead>
<tbody>
<tr>
<td>Open loop</td>
<td>Output controlled by the operator-determined input and disturbances, but output is not measured</td>
<td>Pressure regulator and timer driving a solenoid valve</td>
<td>Early high-frequency jet ventilation</td>
<td>Healthy Impulse jet ventilator (Respironics, Pittsburgh, Pennsylvania)</td>
</tr>
<tr>
<td>Closed loop (feedback) Single control variable</td>
<td>Output measured and forced to match or follow an operator-specified input in the presence of disturbances</td>
<td>Value of target output variable (eg, peak inspiratory pressure, tidal volume, minute ventilation) held constant by manipulating control variable (eg, pressure or flow) according to conditional statements governing phases of the breath (eg, triggering, limiting, or cycling)</td>
<td>PC-IMV or CPAP</td>
<td>Bourns BP200 (Thermorespiratory Group, Palm Springs, California)</td>
</tr>
<tr>
<td>Hierarchical set point control</td>
<td>Output forced to match input according to several layers of conditional logic</td>
<td>The instantaneous value of pressure is proportional to the instantaneous volume and/or flow generated by the patient, with conditional statements governing phases of the breath (eg, triggering, or cycling)</td>
<td>Proportional assist</td>
<td>Servo 900c (Siemens Medical, Danvers, Massachusetts)</td>
</tr>
<tr>
<td>Hierarchical servo control</td>
<td>Output forced to follow a dynamic input according to several layers of conditional logic</td>
<td>Pressure-limited ventilation, pressure augment, Volume-assured pressure support</td>
<td>Automatic tube compensation, Tube compensation</td>
<td>Veolar (Hamilton Medical, Reno, Nevada)</td>
</tr>
<tr>
<td>Closed loop (feedback) Dual control variables</td>
<td>Output forced to match input according to several layers of conditional logic</td>
<td>Value of target output variable (eg, peak inspiratory pressure, tidal volume, minute ventilation) held constant by manipulating control variable (eg, pressure or flow) according to conditional statements governing phases of the breath (eg, triggering, limiting, or cycling)</td>
<td>Pressure-limited ventilation</td>
<td>(Not available in United States)</td>
</tr>
<tr>
<td>Adaptive set point control</td>
<td>Output forced to match a fixed input according to a control strategy that, as the condition of the controlled system changes, can be modified automatically or optimally (ie, in such a way that a measure of system performance is maximized or minimized)</td>
<td>Each breath is pressure-limited and pressure limit is automatically minimized between breaths (using ventilatory mechanics measurements) to be consistent with preset tidal volume Each breath is pressure-limited and pressure limit is automatically adjusted between breaths (using ventilatory mechanics measurements) in such a way that the work of breathing is minimized and a preset minute ventilation is produced</td>
<td>Pressure-regulated volume control, Adaptive support ventilation</td>
<td>Servo 300 (Thermo Respiratory Group, Palm Springs, California)</td>
</tr>
</tbody>
</table>

*See text for more detailed definitions.
1. Examples of disturbances include patient system leaks, changes in lung mechanics, and the patient's ventilatory efforts.
2. Either pressure or volume is controlled.
3. PC-IMV = pressure control intermittent mandatory ventilation, CPAP = continuous positive airway pressure, VC-CMV = volume control continuous mandatory ventilation.
4. Control switches between volume-based and pressure-based and/or combination criteria.
5. See page 604 for additional product sources.
set (constant output) levels of pressure, volume, or minute volume use set point control. Those that follow a varying input use servo control.

As our understanding of patient-ventilator interaction improved, our need for better control was recognized. We came to see that synchrony was important in an actively breathing patient. It became evident that the operator might set an inappropriate flow for volume control or an inappropriate inspiratory time for mandatory pressure-controlled breaths. Pressure control addressed the problem of deciding on flow because the ventilator supplies all that is demanded to maintain the pressure limit. The problem of matching the inspiratory time (and, hence, tidal volume) to the patient’s need was solved by inventing a spontaneous form of pressure-controlled breath. This appeared on the Siemens Servo 900c ventilator as pressure support, and was implemented using multilevel hierarchical control. In pressure support, the ventilator produces a constant airway pressure above baseline, which it turns on and off using a series of “if-then” conditional logic statements in the software algorithm. If inspiratory flow decays to a certain threshold (eg. 25% of peak flow), then end inspiration (ie. flow cycle); or if inspiratory time lasts longer than another threshold (eg. 80% of the set ventilatory period), then end inspiration (ie. time cycle).

Eventually, technologic advances allowed us to monitor lung resistance and elastance on a breath-by-breath basis, using the equation of motion. Not only did this afford the possibility that the operator could track changes in lung mechanics in response to ventilator or pharmacologic interventions, it also created the opportunity for the ventilator to control itself in response to the patient’s changing condition and needs.

The equation of motion has long been the model of choice for describing ventilatory mechanics: it is the framework that defines lung and chest wall mechanical parameters. Because this equation is so important in understanding ventilatory mechanics, ventilator control, and ventilator-patient interactions, the reader should have a basic familiarity with it. In words, the equation says that the forces necessary to inflate or deflate the lungs and chest wall are balanced at each moment in time by the forces opposing inflation or deflation. Expressed mathematically, the equation during mechanical ventilation is:

\[ P_{mus} + P_{vent} = E \times V + R \times \dot{V} \]  

(1)

where \( P_{mus} \) is the equivalent pressure change (relative to baseline) generated by the force of ventilatory muscle contraction, \( P_{vent} \) is the transrespiratory pressure change (relative to baseline) generated by the ventilator, \( E \) is respiratory (ventilatory) system elastance, \( V \) is volume, \( R \) is respiratory (ventilatory) system resistance, and \( \dot{V} \) is flow.

The expression \( E \times V \) is the elastic load and \( R \times \dot{V} \) is the resistive load (see definitions above), which both have units of pressure.

In conventional ventilators, \( P_{vent} \) is positive during inspiration and, with rare exceptions, falls to baseline during expiration. One of those exceptions is the exhalation assist mode on the Venturi ventilator (Cardiopulmonary Corporation, Milford, Connecticut), in which negative airway pressure is applied during expiration.

The advances in our understanding of patient-ventilator synchrony and continuous monitoring of ventilatory mechanics laid the foundation for the use of servo control of the transrespiratory pressure. An example of this is the proportional assist mode. Described in detail elsewhere, proportional assist mode is a type of closed loop pressure control. However, the ventilator does not attempt to maintain a preset limit value for inspiratory pressure, as with set point control. Rather, it adjusts pressure in proportion to the patient’s spontaneous inspiratory volume and flow to support an operator preset level of elastic and resistive load. The operator actually sets the amount of elastance and resistance the ventilator will compensate for, and what is left is the amount of elastance and resistance the patient feels. The basis for this can be seen by examining the equation of motion.

Assume that the resistance and elastance of the patient’s ventilatory system can each be expressed as the sum of a normal value and an abnormal value, designated by subscripts \( n \) and \( ab \), respectively. Then Equation 1 can be rewritten as:

\[ P_{mus} + P_{vent} = (E_n + E_ab)V + (R_n + R_ab)\dot{V} \]  

(2)

or

\[ P_{mus} + P_{vent} = (E_nV + R_n\dot{V}) + (E_abV + R_ab\dot{V}) \]  

(3)

If we want the patient’s respiratory muscles to feel only the load corresponding to a normal ventilatory system, then we can eliminate the abnormal portion of the load using the ventilator to produce \( P_{vent} \) such that:

\[ P_{vent} = (E_nV + R_n\dot{V}) \]  

(4)

When Equation 3 is subtracted from Equation 2 we are left with:

\[ P_{mus} = (E_nV + R_n\dot{V}) \]  

(5)

which says that the respiratory muscles have only to support the normal amounts of resistance and elastance if \( P_{vent} \),
can be made to be proportional to the patient's volume and flow. Currently, in the United States only a partial implementation of the proportional assist mode is available: on the Evita 4 and the Puritan Bennett 840 it is called tube compensation. This form of proportional assist only supports the resistive load of the endotracheal tube, based on the computer-estimated resistance of the operator-input endotracheal tube diameter.

All through the 1980s, various forms of high-frequency ventilation were studied as potential alternatives to the traditional high-volume low-frequency techniques. But for a variety of reasons, high-frequency ventilation never replaced conventional practice. In the shadow of this failure, much research was spawned in an attempt to understand the specific damage conventional ventilation does to the lungs and to develop compensatory strategies. It became clear that setting an arbitrary tidal volume could lead to over-distention of diseased lung units. Areas of low compliance quickly reach the flat portion of their volume-pressure curves and become at risk for rupture. To avoid these problems with volume control, pressure-controlled ventilation has been viewed as the preferred way to limit the maximum regional expansion of the lung.

Pressure control is often touted as being superior to volume control because it results in lower peak airway pressure and better distribution of ventilation. But these concepts are often misunderstood. Peak airway pressure during volume control is higher because of the resistive pressure drop (i.e., flow \times resistance in the equation of motion) but it is not airway pressure that leads to lung damage, but rather trans-alveolar pressure (i.e., volume \times elastance). If the tidal volumes are the same for pressure control and volume control, then both will produce the same alveolar pressure and presumably the same risk of over-distention. Regarding the distribution of ventilation, pressure control results in a more even distribution (compared to volume control) of volume among lung units with different time constants but equal compliances (e.g., with status asthmaticus). However, volume control results in a more even distribution of volume among lung units with different time constants but equal resistances (e.g., in ARDS). Pressure control has the added benefit of providing the patient whatever inspiratory flow is demanded to improve synchrony and comfort. The problem is that as lung impedance changes, volume delivery (and hence, minute ventilation) changes, leading to poor control of blood gases.

The comparison of pressure control and volume control shows that each has some benefits and some disadvantages. Recall that the equation of motion mandates that only one variable can be controlled at a time during inspiration (see definition of control variable above). As the relative advantages of these 2 approaches to inspiratory control became clear in the literature, engineers refined their concept of adaptive control. They wanted to achieve the better patient synchrony of pressure control with the guaranteed breath size of volume control. Thus, schemes evolved that switched between these 2 control variables in a variety of ways. Because both pressure and volume are used as control variables (but not simultaneously), this approach is called dual control.

The first approach that became popular was to control pressure within a breath but to control tidal volume over several breaths through automatic adjustment of the pressure limit to its minimum value consistent with the desired tidal volume. This is a form of adaptive control.

Another approach to dual control is to switch between pressure control and volume control within a breath, as with the volume-assured, pressure-support mode on the Bird 8400ST. Here, the operator presets both a pressure limit and a tidal volume. The breath starts using pressure control (i.e., pressure-limited and flow-cycled). But if the tidal volume is not reached by the time flow decays to the set flow, then the ventilator switches to volume control (i.e., flow-limited and volume-cycled). As a result, inspiratory pressure becomes dependent once again on ventilatory system impedance, but tidal volume is assured within that breath. This scheme is called hierarchical (set point) dual (variable) control, or just hierarchical dual control. The “hierarchy” is the various layers of conditional logic the system uses to make adjustments (e.g., the “if-then” statement in the above example).

Dual control schemes adjust to changes in lung mechanics and provide the theoretical advantages of both volume and pressure control. However, their weakness can be that the target tidal volume is still a matter of human judgment. Too often the tidal volume is set arbitrarily, without regard to the patient’s lung mechanics, just as it was before dual control was invented. And even if this were not true, it would be impractical to manually adjust the target volume to match changing mechanics on a moment-to-moment basis. In addition, there is evidence that automatic control of tidal volume without regard for other variables such as gas trapping can lead to patient asynchrony and tidal volume instability. These problems have led to the development of another example of adaptive control, the adaptive support ventilation mode on the Galileo ventilator. This employs a control strategy that automatically adjusts the target tidal volume in response to changes in lung compliance. The idea is to mimic the body's own control strategy. It has been known for decades that the neural response to decreased lung compliance is decreased tidal volume and increased breathing frequency. Also, when resistance increases, the person tends to breathe at a lower rate. It has been postulated by pulmonary physiologists that the body is attempting to minimize either the work of breathing or peak muscle force, and various mathematical
Models have been created that predict the optimum breathing frequency (to achieve a given minute ventilation) based on lung resistance and compliance.\textsuperscript{18} \textsuperscript{20}

The adaptive control strategy of the Galileo ventilator uses the Otis et al\textsuperscript{19} model to adjust tidal volume automatically in response to changing respiratory system mechanics.\textsuperscript{21} \textsuperscript{23} Specifically, the algorithm uses the patient weight (input by the operator) to estimate the minute ventilation and dead space volume. It also measures the expiratory time constant (ie, the time necessary to passively exhale 63\% of the tidal volume, mathematically equivalent to resistance \times \text{compliance}\textsuperscript{23}). Then, with the estimated minute ventilation, dead space, time constant, and an initial estimate for frequency, the algorithm uses an iterative procedure to converge on the optimum frequency (ie, one that minimizes ventilatory work according to the Otis equation). The target tidal volume is calculated as the estimated minute ventilation divided by the optimum frequency.\textsuperscript{25}

The ventilator then adjusts the inspiratory pressure limit over several breaths to achieve the target tidal volume. If the patient fails to draw enough spontaneous breaths (ie, flow-triggered, pressure-limited, and flow-cycled) to maintain the expected minute ventilation (the output), the ventilator provides mandatory breaths (ie, machine-triggered, pressure-limited, and time-cycled). This control scheme is an advanced form of mandatory minute ventilation, designed to minimize the risks and limitations of earlier forms of the mode.\textsuperscript{25}

The Otis et al model was originally derived for spontaneously breathing subjects. The rationale for using it to set a mechanical frequency and tidal volume is that:

\ldots if one wants to encourage the patients to breathe on their own as early in the disease process as possible, it does make sense to minimize the work of breathing. Choosing a breathing pattern with a low work of breathing was thought to encourage spontaneous breathing.\textsuperscript{21}

There are boundaries set on allowable frequency, tidal volume, and inspiration-expiration ratio, to implement lung-protective strategies. That is, tidal volume will not go too high (to prevent over-distention) or too low (to prevent excessive dead space ventilation or atelectasis), and frequency and inspiration-expiration ratio will not get high enough to cause alveolar gas trapping. Although this is a more sophisticated form of control than nonoptimizing methods, it is too early to tell if patient outcomes are improved with its use.

A Conceptual Model for Understanding Modes of Ventilation

Having reviewed basic definitions and the conceptual foundations of control schemes, we are ready to establish a mode classification system. This system should help avoid the confusion evident in many areas. For example, in the course of co-editing 2 editions of \textit{Respiratory Care Equipment}, the first author of the present article has studied the operator manual of every intensive care ventilator sold in the United States. In general, the manuals are not well organized and they do a poor job of describing how modes operate. In addition, manufacturers have made no attempt to standardize terminology. Features that are identical on different ventilators are given different names, and features that operate differently are sometimes called the same thing. In order to "decode" marketing jargon, we need 2 items: (1) a set of definitions that can act as building blocks in a systematic classification system and (2) a conceptual model that can be used to organize ventilator functions in a logical, universally applicable format. The problem is similar to classifying plants in the study of botany. The basic definitions we need have been described in the sections above.

The goal in constructing a conceptual model is to link simple, defined terms in a way that allows us to build descriptions of varying complexity. This is much more practical than trying to memorize arbitrary names for every new feature a manufacturer wishes to promote. It is analogous to using an alphabet of several dozen letters to build words rather than memorizing thousands of separate ideographs that each represent a word. One need only compare the English language to Chinese to appreciate the analogy.

The essence of intelligence is the ability to make useful distinctions. A useful mode classification system should allow us to distinguish any unique mode from all the rest, and also to group modes that are similar. To classify modes we will use an outline structure similar to the one used to classify ventilators.\textsuperscript{3} The general idea in this case is to show how a mode description can be built up from general characteristics to a complete specification using only necessary and sufficient characteristics.

Mode Classification Outline

Table 2 shows that, as a first step, a mode can be classified simply on the basis of the breathing pattern it produces. This is identified by the variable manipulated by the system to effect control (that is, the primary breath control variable for mandatory breaths, unless only spontaneous breaths are allowed), and the permissible breath sequence. At the level of detail of the breath control variable, we can only distinguish among pressure control, volume control, and dual control modes. Often this is all we need to communicate. For example, at the bedside we might only have to indicate that the mode has been changed from volume control to dual control once we have realized that the condition of the lungs has become unstable. Note that when we say
pressure control or volume control, it can mean either open loop or closed loop control, but dual control can only be accomplished with closed loop (feedback) control. Also, remember that we might want to be a little more specific when talking about ventilators as opposed to modes. A ventilator may be a pressure, volume, or flow controller, whereas a mode of ventilation can be more simply classified as pressure control or volume control.

When we add the breath sequence to the control variable, we can distinguish between, say, pressure-controlled IMV and pressure-controlled CSV. By adding the type of control (see Table 2), we can extend this description to distinguish between various types of, say, pressure-controlled CSV: that using a set point control (eg, continuous positive airway pressure) and that using servo control (eg, proportional assist) (see Table 1).

If we confine ourselves to classifying based solely on the breathing pattern, we see that there are only 8 possibilities in 3 groups (Table 3). The utility of this system is immediately obvious. We can introduce a new mode, say, APRV, as simply a form of pressure-controlled IMV. Assuming we already understand the concept of pressure-controlled IMV, it takes little effort to understand the additional nuances of APRV (eg, different labels for control settings and alarms). At this level of description, we can avoid detailed ad hoc definitions such as the one for APRV in the ECRI report ("A mode that allows spontaneously breathing patients to breathe at a positive-pressure level, but drops briefly to a reduced pressure level for CO2 elimination during each breathing cycle") or the cryptic definition from other authors who explain APRV as 2 levels of continuous positive airway pressure.

We can also use pressure-controlled IMV and pressure-controlled CSV to clarify what "bi-level positive airway pressure ventilation (BiPAP)" means. For example, on the BiPAP ST-D ventilator (Respironics, Pittsburgh, Pennsylvania), the "timed" BiPAP mode is pressure-controlled IMV, whereas the "spontaneous" BiPAP mode is pressure-controlled CSV. "BiPAP ventilation" and "bi-level ventilation" are particularly ambiguous terms because any form of assisted ventilation can be thought of as using 2 levels of pressure (see the definition of assisted breath above). To make matters even more confusing, the Puritan Bennett 840 ventilator has a bi-level mode that allows for additional pressure support during a pressure-limited, time-cycled mandatory breath (see Figure 1). Therefore, with positive end-expiratory pressure (PEEP), the mandatory pressure limit, and the pressure-support limit, the mode actually provides "tri-level" ventilation.

Not only can we describe a new mode in terms of the prototypical breathing pattern it produces, we can also use this description to group together modes that function in the same way but are given different names. For example, both pressure augment (BEAR 1000) and volume-assured pressure support (Bird 8400ST) are dual control IMV. We could also add that they use hierarchical (set point) control. Another example is pressure-regulated volume control (Servo 300) and pressure-controlled assist control + adaptive pressure ventilation (Galileo), which are both adaptive dual control CMV. It is also possible to group ventilators in terms of the number of breathing patterns they offer: some offer only one or two; others offer all

| Table 2. | Ventilator Mode Classification Scheme*
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Breathing pattern</td>
<td>Breath Control Variable</td>
<td>Breath Sequence</td>
</tr>
<tr>
<td></td>
<td>a. Primary breath control variable</td>
<td>Continuous mandatory ventilation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Intermittent mandatory ventilation</td>
</tr>
<tr>
<td></td>
<td>b. Breath sequence</td>
<td>Withdrawal</td>
</tr>
<tr>
<td></td>
<td>i. Continuous mandatory ventilation (CMV)</td>
<td>Intermittent mandatory ventilation</td>
</tr>
<tr>
<td></td>
<td>ii. Intermittent mandatory ventilation (IMV)</td>
<td>Continuous spontaneous ventilation</td>
</tr>
<tr>
<td></td>
<td>iii. Continuous spontaneous ventilation (CSV)</td>
<td>Continuous mandatory ventilation</td>
</tr>
</tbody>
</table>

*These elements can be used to characterize modes of ventilation operation. If both mandatory and spontaneous breaths are possible in a given mode, the specification of that mode should begin with, and may just be limited to, a description of the mandatory breaths. However, a complete specification would include descriptions of both mandatory and spontaneous breaths.

| Table 3. | Possible Breathing Patterns |
|---|---|---|
| Breath Control Variable | Breath Sequence | Abbreviation |
| Volume (control) | Continuous mandatory ventilation | VC-CMV |
| | Intermittent mandatory ventilation | VC-IMV |
| Pressure (control) | Withdrawal | No VC |
| | Continuous mandatory ventilation | PC-CMV |
| | Intermittent mandatory ventilation | PC-IMV |
| | Continuous spontaneous ventilation | PC-CSV |
| Dual (control) | Continuous mandatory ventilation | DC-CMV |
| | Intermittent mandatory ventilation | DC-IMV |
| | Continuous spontaneous ventilation | DC-CSV |
eight. This might be useful as an initial screening tool when planning ventilator purchases.

Table 2 shows that a more detailed mode description would include the type of control used to manipulate the control variables to produce the permissible breaths. For single-variable closed loop control, hierarchical set point and hierarchical servo strategies have been employed. Dual-variable closed loop control has used hierarchical set point and adaptive set point strategies (see Table 1). Thus, adaptive support ventilation and pressure-regulated volume control can be classified as adaptive dual (set point) control CMV. (The term “set point” could be left out since all dual control schemes so far have been set point.)

Finally, we can fully characterize a mode by adding the specific strategy it employs. This begins with the naming of the phase variables, followed by describing the operational logic, and, if necessary, giving the parameter values used in the conditional statements. The specification of the breathing pattern that the mode can produce (i.e., breath control variable[s] and breath sequence), the type of control (hierarchical set point, hierarchical servo, or adaptive set point control), and the specific strategy (phase variable and operational logic) it uses, for both mandatory and spontaneous breaths, constitutes a complete classification for any mode of ventilation.

Figure 3 provides a simple example of how a mode can be described using this system.

This system helps us to distinguish modes that look the same on graphics monitors and suggests what the operator must do to set the controls. For example, pressure support (any ventilator) is pressure-controlled CSV for which the operator sets the sensitivity and pressure limit. In contrast, volume support (Servo 300) is dual control CSV and looks similar to pressure support on the graphics monitor, but the operator must set a tidal volume in addition to sensitivity and pressure limit.

We can also use this classification system to describe a mode even if the ventilator manufacturer does not treat it
like a mode. For example, Dräger calls AutoFlow a "mode extension," but it is more descriptive to say that it is a form of dual control that can be set up as CMV or IMV (see Tables 3 and 4). What makes AutoFlow an extension on the Evita 4 is that it can be combined with other modes to get different control types (eg, dual control CMV and dual control IMV). On another ventilator, dual control may be treated like a complete mode setting by itself (eg, volume support on the Servo 300). Similarly, the front panel layout of the Evita 4 would lead one to think that Pmax (maximum pressure) is just a pressure-limit control. But this feature is unique and allows for hierarchical dual control in CMV or IMV (see Table 4).

Specifying the phase variables and operational logic helps to distinguish among similar modes. For example, on the Bear 1000, assist control is volume control CMV but assist control + pressure augment is dual control IMV. Most clinicians think of "assist control" in traditional terms, wherein every breath is mandatory and volume-controlled (although the term says nothing about the control variable or the breathing sequence and only suggests that breaths may be patient- or machine-triggered). How can adding pressure augment change not only the control type but also the breath sequence designation? First of all, it is necessary to have a clear understanding of the definitions of mandatory versus spontaneous breaths and phase variables (see definitions above). Then we need a description of the phase variables for these 2 modes:

Assist control (without an inspiratory hold) is patient- or time-triggered, flow-limited, and volume-cycled. Because every breath is machine-cycled, every breath is mandatory, so the breath sequence is CMV.

Assist control + pressure augment is patient- or time-triggered, pressure- or flow-limited, and flow- or volume-cycled. If a breath happens to be patient-triggered and flow-cycled, it is spontaneous. If it is patient- or time-triggered and volume-cycled, it is mandatory. Because both types of breath are possible, the pattern is IMV.

In a similar manner, specifying the phase variables and operational logic helps to distinguish among the 4 types of dual control IMV on the Evita 4 (see Table 4).

**Application**

Table 4 gives detailed descriptions of all the modes found on five commonly used ventilators in the United States. It is a sort of "Rosetta stone" that allows us to interpret the different languages used by various manufacturers. This table has been simplified in that the phase variables for both mandatory and spontaneous breaths have been combined. Ideally, the mandatory and spontaneous phase variables should be separated for a complete and unambiguous description of a mode. The Puritan Bennett 840 illustrates one example of why it is important to separate mandatory from spontaneous breath descriptions. This ventilator offers both bi-level mode and pressure control SIMV mode. Both are forms of pressure-controlled IMV and both use the same trigger, limit, and cycle variables and conditional logic (as shown in Table 4). If that were all the detail we had, it would seem that the modes were identical. The subtle difference becomes evident when we examine the phase variables for the spontaneous breaths in the 2 modes. During both bi-level and pressure control SIMV, spontaneous breaths that occur between mandatory breaths are pressure- or flow-triggered, pressure-limited, and flow-cycled. Both modes also permit spontaneous breaths to occur during a mandatory breath cycle. However, during pressure control SIMV, these spontaneous breaths are only pressure-triggered, have a factory-set pressure limit, and are flow-cycled. In contrast, during bi-level, spontaneous breaths that are initiated during a mandatory breath cycle are flow- or pressure-triggered, have an adjustable pressure limit (i.e., pressure support can be added), and are flow-cycled.

Table 4 highlights 2 important facts about current ventilators. First, they offer far more complex modes than in the past: understanding how they work is no trivial task. Manufacturers are throwing together combinations of features that are not only difficult to comprehend but also strain rational justification, often with no clinical data to support the efficacy of the new modes.26-28

Second, there is absolutely no agreement among ventilator manufacturers when it comes to nomenclature. The names they create for modes are baffling. Table 4 shows 36 different names, and that is for only 5 ventilators.

Another utility of Table 4 is that it unmasks the identical modes. Notice all the different names for a basic mode such as volume control CMV. Recognizing redundancies can have simple, practical applications. For example, a table that appeared in a recent magazine entitled Ventilators at a Glance had ventilator models as column headings and specific features as row headings. Ventilators are thus quickly compared by the number of Xs in each column, representing the number of features each model has.

The table lists pressure ventilation SIMV and APRV, yet the two are functionally the same mode. Also, pressure ventilation with volume guarantee is listed as separate from adaptive pressure ventilation, yet they are both dual control. Also, it makes no sense to list a proprietary mode name, such as adaptive support ventilation, as a feature to be compared among all ventilators. Nomenclature problems like these limit the usefulness of such a table, the key function of which is to compare different ventilators by tallying features (ie, wrong tally, wrong comparison).

Once you become comfortable with this classification system, you begin to see problems everywhere with unstructured descriptions of modes. For example, we recently
### Table 4: Detailed Description of Modes Available on 5 Intensive Care Ventilators Used in the United States

<table>
<thead>
<tr>
<th>Mode Name</th>
<th>Breathing Pattern</th>
<th>Control Type</th>
<th>Phase Variables</th>
<th>Specific Control Strategy</th>
<th>Operational Logic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Evita 4 (Dräger Medical)</td>
<td></td>
<td></td>
<td>Trigger</td>
<td>Limit*</td>
<td>N/A</td>
</tr>
<tr>
<td>Continuous mandatory ventilation</td>
<td>VC-CMV</td>
<td>Hierarchical set point</td>
<td>T</td>
<td>F, V</td>
<td>N/A</td>
</tr>
<tr>
<td>Continuous mandatory ventilation + assist</td>
<td>VC-CMV</td>
<td>Hierarchical set point</td>
<td>T, F</td>
<td>F, V</td>
<td>N/A</td>
</tr>
<tr>
<td>Continuous mandatory ventilation + autoflow</td>
<td>DC-CMV</td>
<td>Adaptive set point</td>
<td>T, F</td>
<td>P, T</td>
<td>If set tidal volume is not achieved, then adjust pressure limit.</td>
</tr>
<tr>
<td>Continuous mandatory ventilation + pressure-limited ventilation</td>
<td>DC-CMV</td>
<td>Hierarchical set point</td>
<td>T, F, P, T</td>
<td>N/A</td>
<td>If pressure reaches set value (P_{max}), then switch from flow limit to pressure limit.</td>
</tr>
<tr>
<td>Synchronized intermittent mandatory ventilation + pressure support</td>
<td>DC-IMV</td>
<td>Adaptive set point</td>
<td>T, F</td>
<td>P, T</td>
<td>If set tidal volume is not achieved, then adjust pressure limit.</td>
</tr>
<tr>
<td>Synchronized intermittent mandatory ventilation + autoflow</td>
<td>DC-IMV</td>
<td>Hierarchical set point</td>
<td>T, F, P, T</td>
<td>N/A</td>
<td>Switch between mandatory and spontaneous breaths.</td>
</tr>
<tr>
<td>Synchronized intermittent mandatory ventilation + pressure-limited ventilation</td>
<td>PC-IMV</td>
<td>Hierarchical set point</td>
<td>T, F</td>
<td>P, T</td>
<td>If pressure reaches set value (P_{max}), then switch from flow limit to pressure limit.</td>
</tr>
<tr>
<td>Pressure-controlled ventilation + (bi-level positive airway pressure)/pressure support</td>
<td>PC-CMV</td>
<td>Hierarchical set point</td>
<td>T, F</td>
<td>P, T</td>
<td>Switch between mandatory and spontaneous breaths.</td>
</tr>
<tr>
<td>Pressure-controlled ventilation + assist</td>
<td>PC-CMV</td>
<td>Hierarchical set point</td>
<td>T, F</td>
<td>P, T</td>
<td>N/A</td>
</tr>
<tr>
<td>Mandatory minute volume ventilation + pressure support</td>
<td>VC-IMV</td>
<td>Adaptive set point</td>
<td>T, F, F, T</td>
<td>P, T</td>
<td>If set minute ventilation is not achieved by spontaneous breaths, then trigger mandatory breaths.</td>
</tr>
<tr>
<td>Mandatory minute volume ventilation + autoflow</td>
<td>DC-IMV</td>
<td>Adaptive set point</td>
<td>T, F, F, T</td>
<td>P, V</td>
<td>If set minute ventilation is not achieved by spontaneous breaths, then trigger mandatory breaths. If set tidal volume is not achieved, then adjust pressure limit.</td>
</tr>
<tr>
<td>Mandatory minute volume ventilation + pressure-limited ventilation</td>
<td>DC-IMV</td>
<td>Hierarchical set point</td>
<td>T, F</td>
<td>P, V</td>
<td>If set minute ventilation is not achieved by spontaneous breaths, then trigger mandatory breaths. If set tidal volume is not achieved, then adjust pressure limit.</td>
</tr>
<tr>
<td>Airway pressure release ventilation</td>
<td>PC-IMV</td>
<td>Hierarchical set point</td>
<td>T, F</td>
<td>P, T, F</td>
<td>N/A</td>
</tr>
<tr>
<td>Pressure-support</td>
<td>PC-CSV</td>
<td>Hierarchical set point</td>
<td>F</td>
<td>P, F</td>
<td>N/A</td>
</tr>
<tr>
<td>Continuous positive airway pressure</td>
<td>PC-CSV</td>
<td>Hierarchical set point</td>
<td>F</td>
<td>P, F</td>
<td>N/A</td>
</tr>
<tr>
<td>Automatic tube compensation</td>
<td>PC-CSV</td>
<td>Hierarchical servo</td>
<td>F</td>
<td>F</td>
<td>N/A</td>
</tr>
</tbody>
</table>

All possible breathing patterns shown in Table 3 are represented here.

*Multiple limit and cycle variables account for mandatory breaths, mandatory breaths with inspiratory hold, and spontaneous breaths. A more detailed description could separate each case.

*Additional variables for alarm conditions.

VC = volume control; PC = pressure control; DC = dual control; CMV = continuous mandatory ventilation; IMV = intermittent mandatory ventilation; CSV = continuous spontaneous ventilation; T = time; P = pressure; V = volume; F = flow; N/A = not applicable; P_{max} = preset maximum pressure.
<table>
<thead>
<tr>
<th>Mode Name</th>
<th>Breathing Pattern</th>
<th>Control Type</th>
<th>Phase Variables</th>
<th>Specific Control Strategy</th>
<th>Operational Logic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Puritan Bennett-840 (Mallinckrodt)</td>
<td>VC-CMV</td>
<td>Hierarchical set point</td>
<td>T, P, F, F, V</td>
<td>T, P</td>
<td>N/A</td>
</tr>
<tr>
<td>Volume control assist control</td>
<td>PC-CMV</td>
<td>Hierarchical set point</td>
<td>T, P, F, F</td>
<td>T, P</td>
<td>N/A</td>
</tr>
<tr>
<td>Pressure control assist control</td>
<td>PC-IMV</td>
<td>Hierarchical set point</td>
<td>T, P, F, F</td>
<td>T, P, T</td>
<td>Switch between mandatory and spontaneous breaths.</td>
</tr>
<tr>
<td>Pressure control synchronized intermittent mandatory ventilation</td>
<td>PC-IMV</td>
<td>Hierarchical set point</td>
<td>T, P, F, F</td>
<td>T, F, P</td>
<td>N/A</td>
</tr>
<tr>
<td>Bi-level</td>
<td>PC-IMV</td>
<td>Hierarchical set point</td>
<td>P, F, F</td>
<td>F, T, P</td>
<td>The instantaneous value of pressure is proportional to the instantaneous flow.</td>
</tr>
<tr>
<td>SPONT</td>
<td>PC-CSV</td>
<td>Hierarchical servo</td>
<td>F</td>
<td>F, T, P</td>
<td>N/A</td>
</tr>
<tr>
<td>Tube compensation</td>
<td>PC-CSV</td>
<td>Hierarchical servo</td>
<td>F</td>
<td>F, T, P</td>
<td>N/A</td>
</tr>
<tr>
<td>Gallileo (Hamilton Medical)</td>
<td>VC-CMV</td>
<td>Hierarchical set point</td>
<td>T, P, F, F, V</td>
<td>T, P</td>
<td>N/A</td>
</tr>
<tr>
<td>Assist control (synchronized controlled mandatory ventilation)</td>
<td>PC-CMV</td>
<td>Hierarchical set point</td>
<td>T, P, F, F</td>
<td>T, P</td>
<td>N/A</td>
</tr>
<tr>
<td>Pressure-controlled assist control (pressure-controlled, controlled mandatory ventilation)</td>
<td>DC-CMV</td>
<td>Adaptive set point</td>
<td>T, P, F, F</td>
<td>T, P</td>
<td>If set tidal volume is not achieved, then adjust pressure limit.</td>
</tr>
<tr>
<td>Synchronized intermittent mandatory ventilation</td>
<td>DC-CMV</td>
<td>Adaptive set point</td>
<td>T, P, F, F</td>
<td>T, P</td>
<td>N/A</td>
</tr>
<tr>
<td>Synchronized intermittent mandatory ventilation + pressure support</td>
<td>VC-IMV</td>
<td>Hierarchical set point</td>
<td>T, P, F, F, V</td>
<td>T, P</td>
<td>N/A</td>
</tr>
<tr>
<td>Pressure-controlled synchronized intermittent mandatory ventilation + pressure support</td>
<td>PC-IMV</td>
<td>Hierarchical set point</td>
<td>T, P, F, F</td>
<td>T, P, T</td>
<td>Switch between mandatory and spontaneous breaths.</td>
</tr>
<tr>
<td>Pressure-controlled synchronized intermittent mandatory ventilation + adaptive pressure ventilation</td>
<td>PC-IMV</td>
<td>Hierarchical set point</td>
<td>T, P, F, F</td>
<td>T, P, T</td>
<td>Switch between mandatory and spontaneous breaths.</td>
</tr>
<tr>
<td>Adaptive support ventilation</td>
<td>DC-IMV</td>
<td>Adaptive set point</td>
<td>T, P, F, F</td>
<td>T, F, P</td>
<td>If set tidal volume is not achieved then adjust pressure limit.</td>
</tr>
<tr>
<td>Spontaneous</td>
<td>PC-CSV</td>
<td>Hierarchical set point</td>
<td>P, F, F</td>
<td>F, T, P</td>
<td>N/A</td>
</tr>
</tbody>
</table>

All 8 possible breathing patterns shown in Table 4 are represented here.
*Multiple limit and cycle variables account for mandatory breaths, mandatory breaths with inspiratory hold, and spontaneous breaths. A more detailed description could separate each case.
†Additional variables for alarm conditions.
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Table 4. Continued

<table>
<thead>
<tr>
<th>Mode Name</th>
<th>Breathing Pattern</th>
<th>Control Type</th>
<th>Phase Variables</th>
<th>Specific Control Strategy</th>
<th>Operational Logic</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Servo 300 (Siemens Medical)</strong></td>
<td></td>
<td></td>
<td>Trigger</td>
<td>Limit*</td>
<td>Cycle*</td>
</tr>
<tr>
<td>Pressure control</td>
<td>PC-CMV</td>
<td>Hierarchical set point</td>
<td>T, P, F, P</td>
<td>T</td>
<td>N/A</td>
</tr>
<tr>
<td>Volume control</td>
<td>VC-CMV</td>
<td>Hierarchical set point</td>
<td>T, P, F, F, V</td>
<td>T</td>
<td>P</td>
</tr>
<tr>
<td>Pressure-regulated volume control</td>
<td>DC-CMV</td>
<td>Adaptive set point</td>
<td>T, P, F, P</td>
<td>T</td>
<td>N/A</td>
</tr>
<tr>
<td>Pressure support/continuous positive airway pressure</td>
<td>PC-CSV</td>
<td>Hierarchical set point</td>
<td>P, F, F, F, P</td>
<td>P, T</td>
<td></td>
</tr>
<tr>
<td><strong>B-EAR 1000 (Thermo Respiratory Group)</strong></td>
<td></td>
<td></td>
<td>Trigger</td>
<td>Limit*</td>
<td>Cycle*</td>
</tr>
<tr>
<td>Assist control mechanical ventilation</td>
<td>VC-CMV</td>
<td>Hierarchical set point</td>
<td>T, P, F, F, V</td>
<td>V, T</td>
<td>P</td>
</tr>
<tr>
<td>Assist control mechanical ventilation + pressure augment</td>
<td>DC-IMV</td>
<td>Hierarchical set point</td>
<td>T, P, F, F, V</td>
<td>V, T</td>
<td>P</td>
</tr>
<tr>
<td>Pressure control</td>
<td>PC-CMV</td>
<td>Hierarchical set point</td>
<td>T, P, F, F, V</td>
<td>V, T</td>
<td>P</td>
</tr>
<tr>
<td>Synchronized intermittent mandatory ventilation + pressure support</td>
<td>DC-IMV</td>
<td>Hierarchical set point</td>
<td>T, P, F, F, V</td>
<td>V, T</td>
<td>P, T</td>
</tr>
<tr>
<td>Pressure support/continuous positive airway pressure</td>
<td>PC-CSV</td>
<td>Hierarchical set point</td>
<td>P, F, F, F, P</td>
<td>P, T</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Trigger</td>
<td>Limit*</td>
<td>Cycle*</td>
</tr>
</tbody>
</table>

All possible breathing patterns shown in Table 4 are represented here.

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saw this description of a European ventilator on an Internet report of the world’s largest trade fair, Medica:

Blease’s new 8500 anesthesia device includes a new, feature-packed ventilator that is designed to be used both with Blease’s and other manufacturers’ anesthesia devices. The ventilator has individually-configurable screens and all the modes that comparable ICU ventilators include, such as CMV, SIMV, and PCV, as well as pressure support.

This shows how some authors resort to “hand waving” instead of conveying consistent, logical information. Note that the description refers to 2 modes (CMV and SIMV) in terms of permissible breath sequence only, one mode (PCV) in terms of control variable only, and one (pressure support) that is a specific name associated with a complete specification. Overall, this is a pretty poor description of “all the modes that comparable ICU ventilators include.”

There is some hint of progress, though. The Puritan Bennett 840 divides its control panel into sections for mandatory and spontaneous breaths. The operator manual for the Galileo actually has a table of all modes, listing the control and phase variables. Both manufacturers include a glossary of terms in the operator manual. Yet, of all the operator manuals we have read, only the Puritan Bennett 840 manual defines the term mandatory: “A breath whose settings and timing are preset; can be triggered by the ventilator, patient, or operator.” This is nearly identical in concept, if not in clarity, to the definition given above. The key idea is that the timing is preset (ie, when to trigger and/or when to cycle the breath). The other settings are irrelevant. Puritan Bennett also provides sort of a definition for spontaneous breath under the definition for its SPONT mode, which is a little less specific because it does not refer to the cycling mechanism.

The ideal operator manual would include a complete description of every mode, using a system like the one described here. Some of this information is already in the manuals, but you have to dig deep, cross-check numerous areas, and interpolate a bit. It should not be that hard to get the basic information we need to understand what the ventilator does and which settings the operator adjusts. Key terms such as volume control, pressure control, and mandatory and spontaneous breaths must be defined, as they are the very basis of describing how the ventilator operates. When this is not done, operators can be fooled into thinking they understand more than they do. Contradictory statements are made that mislead the operator, as with AutoFlow in the Exiva 4 operator manual. If the engineers at ECRI were confused, imagine the plight of the average clinician who has neither the background nor the time to work such puzzles.

If instructors cannot rely on published reports and manufacturer’s product information, what will become of the next generation of respiratory therapists? Standard terms must be defined in a standard way that everyone agrees on, and used in that way. Then terms for new modes or features must be explained clearly and related to the standard terms and concepts. If that is not possible, then and only then should a new term be coined, and it should be agreed upon and used appropriately. Of course, marketers at the ventilator manufacturers are not likely to go for this idea, but, then, we would argue that manufacturers should not drive the education format for our profession.

Final Thoughts

We are willing to bet that the majority of clinical training in mechanical ventilation comes at the hands of ventilator manufacturers’ employees rather than employees of hospitals, universities, or even from textbooks. No doubt there are many highly skilled product managers who are experts at describing their products, but, as a group, they do not provide a consistent and logical approach to understanding the technology in general. The problem that causes clinicians is obvious. The problem the ventilator manufacturers cause themselves is less obvious but just as damaging.

Marketing ventilator technology requires that field representatives communicate with their customers on a level that promotes mutual understanding, not just sales promotion. Field representatives must also be knowledgeable about competing devices. Both these tasks are hampered by a lack of consistent definitions and unifying principles. There is probably nothing more frustrating than trying to explain a feature to a customer, only to find that not only does the customer not understand the explanation, he or she does not even have the background knowledge needed to quickly bring them up to speed. Or perhaps it is more embarrassing for sales representatives to have it pointed out that they do not have the background to understand what the customer is saying. These situations could be avoided if we could move toward a shared understanding using a common language.

It seems unlikely that marketing departments will forgo product differentiation through use of creative and unique terminology. However, it is hoped that at least the engineers who design ventilators may see their own work and that of competitors in a new light. Indeed, adoption of some standards by engineering departments would drive acceptance of the standards throughout the profession. It would serve us all well to reach consensus about some general terms and concepts. The definitions and classification scheme presented here may provide a starting point.

Our profession is facing a staff development crisis. Cutbacks have all but eliminated staff educator positions. Staff
time for training is in direct conflict with increasing work demands, yet technology marches on. Mechanical ventilation is the most important skill respiratory therapists possess. No other profession is specifically trained to perform it. We must find a way to advance the common level of understanding among therapists and keep pace with new developments. But we have to be careful not to make educational materials more difficult by making them unnecessarily detailed and by using ad hoc terms. On the other hand, we must avoid the natural tendency toward oversimplification that just leads to more confusion. Perhaps that response to the constant pressure of marketing and technology is the most dangerous of all.

REFERENCES

28. MacIntyre NR. Innovations in mechanical ventilation: what are (and what should be) the drivers? Respir Care 2001;46(3):267-272.

Pulmonary Diseases is the most recent volume in McGraw-Hill's Clinical Medicine series. It claims to be a practical book on managing the patient. Its stated aim is to help the busy clinician, and its intention is to provide a text for general practitioners, primary care physicians, students, and postgraduates that is friendly, clearly written, succinct, and up to date.

As a general respiratory text, it is divided into 12 sections. The first of these, entitled "Approach to Respiratory Patients," begins with a chapter on history, symptoms, and physical examination and is followed by chapters on radiology, nuclear medicine techniques, microbiology, endoscopic techniques, pulmonary function tests, and genetics. Further sections deal with obstructive lung disease, pulmonary infections, interstitial lung disease, environmental lung disorders, lung tumors, vascular disorders, diseases of the pleura, diseases of the mediastinum, the lung in systemic diseases, respiratory failure, and treatment of pulmonary diseases.

Each section begins with a chapter on how to approach patients with the general category of disease in question (eg, lung cancer, interstitial lung disease), followed by further chapters dealing with specific diseases.

At 520 pages long, the text is of a reasonable length to be read as a summary of the subject. It is also not so heavy that it cannot easily be transported in one's briefcase and used as an easy reference book.

The book is aimed at a very broad readership, and this causes difficulties in meeting the needs of both students and specialists in training. Many of the chapters assume a fairly high level of prior knowledge on the part of the reader, and as such the text would be of more value to the postgraduate reader than to medical students. However, students would find the book useful as a reference text.

The book is comprehensive in its coverage of the field of respiratory medicine—the only disappointing omission being the lack of information regarding mechanical ventilation. Each chapter includes reviews of etiology, pathogenesis, clinical findings, diagnosis, and management, although often not in the same order. The level of detail to which each topic is covered is extremely variable and does not always seem in proportion to the importance of the subject. For example, only 10 pages are devoted to radiology, whereas beryllium disease received a chapter of its own (though of only 5 pages), and drug delivery is hardly mentioned at all.

As a book whose stated aim is to provide a practical guide for the clinician, often surprisingly little space is devoted to patient management, although many chapters have algorithms that are clear and easy to follow.

In any book, with multiple contributors, one has the problem of combining the styles and formats of the contributions. There is no set format for each chapter, and therefore each contribution has a different emphasis, some on pathophysiology or pathogenesis, and some on investigation or management. This is not a problem when referring to a specific topic, but does become frustrating when reading the text as a whole. The literary styles of the contributors range from clear and concise to wordy in the extreme.

The contents of the book are well laid out, and with the aid of a comprehensive index, the book is easy to use. The sections are in logical order, although the excellent chapter on principles of drug therapy may have benefited from being nearer the front of the book.

The biggest disappointment is the lack of illustrations and paucity of radiographic images. The images that are included are usually so small that it is very difficult to make out the radiographic features they are intended to illustrate. Illustrations, tables, and algorithms are in a rather uninspiring blue/black ink, which does little to attract the reader's attention. Though many of the chapters provide a clear and concise insight into their subject matter, the abundance of text and lack of diagrams are not immediately enticing to the reader. There are several irritating typographical errors, a couple of which render whole sentences incomprehensible.

Any text soon becomes out of date as new developments occur; however, the contributors to Pulmonary Diseases all included recent developments in their respective fields. They also included suggestions of areas where further research is warranted.

There are now many respiratory texts available. This new volume aims to improve on others by being a concise, readable summary of the field, accessible to a broad readership. I feel it succeeds in many of its aims. It is concise, comprehensive, and easy to use. The lack of illustrations and somewhat unattractive appearance detract slightly from the quality of the written contribution. Those with an interest in respiratory medicine could read the book in its entirety as a summary of the subject. Nonrespiratory specialists, nurses, and respiratory therapists would also find it useful as a reference text, particularly for less common diseases they might encounter.

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The explosion of new information in pulmonary medicine, as in other medical fields, has made it ever more challenging to stay abreast of recent advances in the pathophysiology, diagnosis, and treatment of lung diseases. The editors and authors of this book have set out to provide a concise yet complete review of the most common diseases in pulmonary and critical care medicine by compiling a collection of tables and figures in atlas form.

The book was conceived by the late Dr Roger C Bone and subsequently completed by the other 2 editors-in-chief. They are supported by 4 contributing editors and about 40 authors, most of whom are well known nationally and internationally in their fields. The 6 sections of the book are divided into 26 chapters, four of which cover pulmonary aspects of critical care medicine. The remainder review a broad range of other pulmonary diseases. The format of each chap-
That said, each figure is well referenced and provides the reader with a source of more thorough discussion on the subject. There are also individual topics that are more thoroughly reviewed and summarized, especially in the areas of pathophysiology, differential diagnoses, and mechanism of action for drugs. The prophylaxis and treatment of venous thromboembolic disease are covered in a thorough and practical manner. Similarly, a great deal of information on the microbiology and treatment of mycobacterial disease is presented very efficiently. The genetics and pathophysiology of cystic fibrosis are well described, and the discussion of the pulmonary manifestations of connective tissue diseases is one of the most useful I have seen, especially as it pertains to diagnostic criteria and differential diagnosis. In addition, the diagnostic work-up and surgical treatment of lung cancer are extensively reviewed.

The physical appearance of the book's hard cover is attractive: its large (approximately 10 × 12 inch) pages are uncluttered and allow for the clear presentation of larger charts and tables. The incidence of typographic errors is very low.

Taken as a whole, this atlas is a tribute to Dr. Roger Bone as a scholar and educator. It is a very clinically relevant and easy-to-read review of pulmonary medicine. It also serves as an excellent supplement to other, more standard textbooks of pulmonary medicine in that it consists of a unique collection of tables, figures, and diagrams adapted from the original references. Such, it makes a great resource for anyone teaching and lecturing on pulmonary diseases.

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Interest in fungal infections has increased since these pathogens have become a major cause of mortality in people immunocompromised by human immunodeficiency virus infection or chemotherapy. During the past 10 years, major advances in the field of clinical mycology have come from the introduction of new antifungal drugs, yet problems persist as organisms have developed resistance to existing antifungals and new pathogens have been uncovered. To this end, this new edition of Fungal Diseases of the Lung presents a comprehensive yet current review of the fungi that infect the lung. The intended readership encompasses all levels of clinicians caring for people with pulmonary lung infections; in fact, this volume might be appealing to any clinician with an interest in these increasingly recognized pathogens.

The book is organized according to a schema explained in the editors' preface. That is, organisms are classified as "T cell opportunists" or "phagocyte opportunists," according to susceptibility to non-immune phagocytes. This concept is based on classic studies that emphasize the importance of multiple arms of the immune system in fungal host defense. However, caution must be exercised in interpretation, as the importance of T cell immunity is now becoming apparent even for the classic phagocyte opportunists such as Aspergillus species. Even so, this book is unique in that it attempts to classify these pathogens according to a more sophisticated understanding of host defense. Perhaps it could have been strengthened by the addition of a chapter that focuses only on fungal host defense and immunomodulation.

Introductory chapters present overviews of diagnosis by the clinical laboratory and serologic techniques. Although many issues are reiterated in later chapters specific to each organism, these chapters are useful as they are both comprehensive and current. The second section of the book is organized according to specific fungal pathogens. These chapters are, in general, comprehensive. Particularly interesting features include sections on the history of the microorganisms and pathogenesis of the diseases. Although this format allows for the presentation of diagnosis and therapy of different clinical syndromes caused by a particular organism, it does not allow for a comprehensive discussion of features specific to each type of host. Instead, this approach is taken in the later "overview" chapters of infections in people with human immunodeficiency virus, neoplasms, or transplantation. The book ends with chapters that discuss antifungal therapies.

Overall, the editors have achieved their aims of presenting clinically useful information regarding fungal infections of the
lungs. However, I found some aspects of the book’s organization problematic. The inclusion of 2 chapters on bacterial infections that mimic fungal infections (actinomycosis and nocardiosis) could have been replaced with chapters featuring the important recently recognized fungus Pneumocystis carinii and some less common but important organisms such as Fusarium species, Dematiaceous molds, and Penicillium marneffei. It was also curious that the section on bone marrow transplantation was included in the chapter on transplantation instead of the one on neoplastic diseases. The result was a good comprehensive discussion on infections in solid organ transplant recipients that effectively mask the subtle host differences among bone marrow transplant recipients. Also, the chapter on human immunodeficiency virus-infected patients seemed brief relative to the other overview chapters, diminishing its clinical usefulness.

Despite these criticisms, the chapters are well written by noted experts in the field. The content is limited only by the dynamic nature of this field. This becomes apparent primarily in the sections that discuss therapy, as new antifungals have been introduced since the book’s publication. The illustrations and photographs are plentiful and clear, and the book is well produced, attractive, and a valuable reference. It should be useful for any clinician caring for people with fungal infections of the lung.

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I knew this book was a winner when I saw the new University of Washington allergy fellow’s copy, well underlined and annotated, carried to clinic with him and consulted. The rapidly expanding immunologic and clinical knowledge base for allergic diseases has required that the well-accepted first edition be followed by this second edition after a mere 3 years.

The editors, both active in academic teaching and clinical practice and both past presidents of the American Academy of Allergy, Asthma, and Immunology, have done a beautiful job for their intended readers. The intended audiences are those who care for allergic patients; primary care family practitioners; primary care internists; internal medicine specialists; ear, nose, and throat, and other specialists; and, yes, even practicing allergists. Nurses and respiratory therapists who care for hospitalized asthmatics may find certain chapters to be helpful reference as well. The book is attractive and sturdy bound and is a handy size for reference.

The format is concise, easy to read, and yet comprehensive, without excessive immunologic jargon and fluff. Pathophysiology is well explained and lays the basis for rational and evidence-based therapy. Main points, pearls, differential diagnoses, algorithms, guidelines, and summaries are boxed in bold print for easy return reference. Tables and figures are well placed and easy to understand. The index is useful, and the writing styles of the well chosen authors are remarkably consistent.

Controversies are well presented, but the readers are never left in a quandary about what is the best scientific evidence-based approach to diagnosis and treatment.

Basic allergic immunology is concisely but completely covered, with an illustrative case report used to good effect. Where immunologic knowledge is incomplete or confusing at the current state of research, this is clearly pointed out. References are limited in number but are excellently chosen for any reader needing a deeper background in a particular subject.

There is some inevitable repetition of basic allergic immunology, as each chapter devoted to asthma, rhinitis, anaphylaxis, etc., lays the background again. Because it is a clinically useful reference book, however, this is acceptable; the practitioner needs only to pick up a particular chapter for a particular problem to “get it all packed in.” background and all, without having to review introductory chapters as well.

A new chapter has been added on how to evaluate the patient with too many respiratory infections and is an excellent summary of how to approach the problem of immunologic deficiencies. New chapters on antihistamines and antileukotrienes sort out the useful information from marketer’s hype.

A few typographical errors were found, and I might quibble about a few of the statements made, but generally the safest, most conservative approaches have been recommended.

The chapters on childhood and adult asthma were very good. The criteria for categorizing asthma as mild intermittent or mild, moderate, or severe persistent could have been presented earlier to promote a better frame in which to discuss treatment guidelines and presented in a single-page format for quick reference.

The discussion of severe, life-threatening asthma in children is especially valuable for intensive care nurses and respiratory therapists, as well as for house staff and primary care physicians.

Asthma diagnosis and treatment is not simple and takes years of experience to master (if ever). However, these 2 chapters on childhood and adult asthma distill a hundred years’ worth of knowledge and experience into an hour of reading (even if high proof).

Some chapters have photos of fiberoptic screens, which show up poorly, are hard to interpret, and have little value. Conversely, the photos of ocular allergy and contact dermatitis are very helpful.

The chapter on atopic dermatitis devotes much space to discussing current palliative treatment measures, but only a brief paragraph to new emerging disease-altering therapies. This chapter will soon be outdated, but nearly all others will stand a longer test of time.

Contact dermatitis is well presented, but the extensive detail on how and what to patch test is useful only for the dermatologist or allergist, because of cost and time constraints. However, it may serve to acquaint the primary care physician with the difficulties inherent in these tests.

The chapters on classes of medications, such as antihistamines, β adrenergics, anti-cholinergics, anti-leukotrienes, and steroids, are straightforward and uniformly well done. Allergy immunotherapy is also well presented, with good evidence-based studies quoted.

I particularly enjoyed the chapter on controversies in allergy and allergy-like diseases, which was delightfully and concisely summarized by Dr. Abba Terr. Finally, the chapter on patients with too many infections was extremely well done.

Even after 30 years of allergy practice, I am going to come back to this book—for example to brush up on occupational allergies, drug allergies, food allergies and intolerances, and contact dermatitis, to name a few.
In summary, this is an improved 2nd edition of a very useful book for consultation in the care of allergic diseases. It is a handy size, up to date, concise yet comprehensive, focused on scientifically and clinically evidence-based diagnosis and treatment. The book is easy to read, and the summaries encourage review.

I am glad I have it and would recommend it to you.

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The 3rd edition of Principles and Practice of Sleep Medicine is an important update of this general reference text. Its strength, as in prior editions, lies in its comprehensive scope and the expertise of its contributors. Many of the 137 contributors have played pioneering roles in the development of the relatively new discipline of sleep medicine. The text is organized into 110 chapters that summarize the essence of current knowledge regarding sleep and sleep disorders.

The stated intention of this text is to assist practitioners of sleep medicine: physicians and dentists. The scope of the text, however, and the helpful synopsis preceding each basic science chapter will appeal as a reference resource to all who work in sleep medicine.

The editors endeavor to complement the expert-based management approach with an evidence-based presentation to assist the practitioner in patient management. Largely, they have been successful. The content is well organized, extensively indexed, and accompanied by a useful glossary of abbreviations. Useful Internet addresses are included in an appendix. Information is frequently tabulated for rapid access. Helpful tables include such topics as a treatment algorithm for idiopathic hypersomnia, normative values for the Epworth Sleepiness Scale, a table of psychometric assessments for sleep disorders patients, and a table of stimulants and dosages for treatment of narcolepsy.

The text is replete with illustrations. The figures include exquisite illustrations of surgical techniques for sleep apnea (Chapter 77), beautiful figures on the anatomy and physiology of upper airway obstruction (Chapter 72), and a gallery of oral appliance photos (Chapter 78).

The editors have indeed compiled an "in depth critical evaluation of the contemporary status of sleep disorders medicine, underlying scientific concepts to clinical approaches and the latest technical advances." Nevertheless, this edition has some notable shortcomings. Most importantly, the text largely ignores several prominent subjects of controversy within the discipline of sleep medicine, the most prominent of which is the scoring of respiratory events. The field of sleep medicine has been unable to achieve a meaningful consensus on a clinical definition for hypopnea. Defining efforts to date have been either (1) academy-sponsored recommendations for research applications only or (2) small, self-appointed expert panel recommendations. Even the definition of apnea can vary widely from lab to lab. The controversy is briefly mentioned, but the importance is understated, and the issue was not subjected to a critical review. Because of the continuing lack of consensus definitions, therapeutic trial results and epidemiologic data cannot be applied with confidence. This topic is of vital interest to the clinician wishing to apply meaningful consensus standards to scoring procedures in a given sleep lab. Accordingly, such a topic might warrant a chapter of its own. Optimally, it would include a table of recommended definitions/criteria, accompanying rationale, and literature references.

This edition also fails to address the controversy that accompanies the notion of an "upper airway resistance syndrome." Since the existence of this entity was originally proposed, precious few data have emerged to prove that it deserves a place in the nomenclature of sleep medicine apart from sleep apnea per se. Other than a rather arbitrarily defined respiratory disturbance in dex cut-off, no important clinical or laboratory features have surfaced to warrant a separation from obstructive sleep apnea. Failure to address this issue leaves a confusing body of literature for the clinician to dissect and interpret. A frank, critical review of this topic would have been most helpful.

Other less important shortcomings are encountered here and there. For instance, on page 539 the author states that "electrophysiologic radiography and pharyngoscopy may be useful in the preoperative identification of sites of obstruction..." but doesn't provide any specific or general guidelines for applying these tools. A table summarizing the various treatments for central sleep apnea in Chapter 72 would have been useful.

In Chapter 76 a graph illustrates the failure of automatically titrating nasal continuous positive airway pressure to maintain normal oxyhemoglobin saturation. The only explanation offered is: "presumably due to non-detection of any upper airway obstruction..." The author provides no supporting evidence or consideration of alternative explanations. In Chapter 77, "Surgical Treatment for Obstructive Sleep Apnea-Hypopnea Syndrome," an illustration of the new radiofrequency tissue ablation technique would have been helpful. Further, this chapter scarcely mentions treatment of obstructive sleep apnea by tonsillectomy, and it is only briefly discussed in Chapter 79, "Management of Obstructive Sleep Apnea-Hypopnea Syndrome: Overview." A concise critical review of indications for and outcomes of this common procedure would have been appropriate.

Such shortcomings, however, are inevitable in an undertaking of this scope. Forbodes aside, this updated edition will be treasured as a valued reference text for sleep medicine physicians, psychologists, respiratory therapists who work in this field, nurse practitioners, physician assistants, and sleep technologists.

Noel T Johnson DO
Sleep Disorders Center
Swedish Medical Center/Providence
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☐ Have the manufacturers and their locations been provided for all devices and equipment used?
**A. Patient information**

1. **Patient identifier**
2. **Age at time of event:**
   - [ ] male
   - [ ] female
3. **Weight**
   - [ ] lbs
   - [ ] kg
4. **Sex**
5. **Date of birth**

**B. Adverse event or product problem**

1. **Adverse event**
2. **Product problem**

2. **Outcomes attributed to adverse event**
   - [ ] death
   - [ ] life-threatening
   - [ ] hospitalization - initial or prolonged
   - [ ] other
   - [ ] disability
   - [ ] congenital anomaly
   - [ ] required intervention to prevent
   - [ ] permanent impairment damage

3. **Date of event**
4. **Date of this report**

5. **Describe event or problem**

**C. Suspect medication(s)**

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

**D. Suspect medical device**

1. **Brand name**
2. **Type of device**
3. **Manufacturer name & address**
4. **Operator of device**
   - [ ] health professional
   - [ ] lay user/patient
   - [ ] other
5. **Expiration date**
6. **Model #**
7. **Catalog #**
8. **Serial #**
9. **Lot #**
10. **Other #**

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

1. **Name & address**
2. **Phone #**
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.**

Submission of a report does not constitute an admission that medical personnel or the product caused or contributed to the event.
ADVICE ABOUT VOLUNTARY REPORTING

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

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

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

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

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

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

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

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

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Sleep Studies Screening Device. SensorMedics introduces the AlphaScreen, a screening device specifically designed for ambulatory screening of sleep-related breathing disorders. The company says the modular design and durable sensors are built for day-to-day data collection in many different environments, but that the device is ideally suited for sleep studies outside the hospital environment. According to SensorMedics, the AlphaScreen uses a simple graphical, icon-based, touch-screen interface where users can input and edit patient demographic data, including alphanumeric data. The company says the device is designed for unattended data collection and that preparing the AlphaScreen and attaching the sensors is easily done in the laboratory, a physician’s office, or at the patient’s home. SensorMedics also says the AlphaScreen’s Yoke Box design allows for simple, quality signal pre-processing and maximum expandability; the snoring microphone, body position sensor and pressure transducer for CPAP/bi-level positive airway pressure and nasal pressure signals are integrated into the Yoke and the color and key-coded sensors assure correct patient hook up. For more information from SensorMedics, circle number 159 on the reader service card in this issue, or send your request electronically via “Advertisers Online” at http://www.aarc.org/buyers_guide/

Handheld Spirometer. Futuremed America Inc now provides new data management software for its handheld spirometer, the Discovery. According to the company, the Windows-based software lets you review a patient’s PFT files and compare results of various tests. Futuremed says the Discovery is all-inclusive, does not require a base station, and meets the new American Thoracic Society standards for spirometry. The device offers an easy-to-read LCD monitor that displays tests in real time, allowing you to monitor patient performance and to have results immediately. For more information from Futuremed America, circle number 161 on the reader service card in this issue, or send your request electronically via “Advertisers Online” at http://www.aarc.org/buyers_guide/

Nebulizer Solution. The Food and Drug Administration has given approval to Dey for its dual-therapy nebulizer solution, Hyperinflation System. Mercury Medical® has introduced a latex-free hyperinflation system with a built-in manometer. Company press materials say the manometer is lightweight and disposable and that it is built directly into the patient elbow on select models which allows for more complete patient observation during positive pressure ventilation. Mercury Medical says the disposable feature eliminates misplaced, expensive manometers that can be out of calibration when ventilating patients. For more information from Mercury Medical, circle number 163 on the reader service card in this issue, or send your request electronically via “Advertisers Online” at http://www.aarc.org/buyers_guide/

Virtual Ventilator. Newport Medical Instruments has announced availability of a new Interactive Product Guide on CD for their Newport E100M ventilator. According to Newport, the guide is fully interactive, providing comprehensive product information in a format that is easy to navigate and convenient to access. Company press materials say the presentation includes a completely functional, “virtual” E100 M ventilator that allows viewers to adjust controls, set alarms, and test their skills in ventilator setup and patient use. For more information from Newport Medical Instruments, circle number 162 on the reader service card in this issue, or send your request electronically via “Advertisers Online” at http://www.aarc.org/buyers_guide/
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<td>June 6–8</td>
<td>FSRC State Convention; Fort Lauderdale, FL</td>
<td>Pat Nolan, (561) 546-1863, (800) 447-3772, <a href="mailto:fsfc@inew.net">fsfc@inew.net</a></td>
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<td>June 13–15</td>
<td>New Jersey Society for Respiratory Care’s 14th Annual NJ/NY Spring Forum, Round Top, NY</td>
<td>Ken Wyka, (201) 725-2528; or Bob Fluck, (315) 464-5580</td>
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<td>June 19</td>
<td>Professor’s Rounds 2001 Teleconference, Program 4</td>
<td>AARC, (972) 243-2272</td>
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<td>Professor’s Rounds 2001 Live Videoconference, Program 5</td>
<td>AARC, (972) 243-2272</td>
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<td>July 17</td>
<td>Professor’s Rounds 2001 Teleconference, Program 5</td>
<td>AARC, (972) 243-2272</td>
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<td>Aug. 29–31</td>
<td>West Virginia Society for Respiratory Care’s Respiratory Reunion; Charleston, WV (304) 357-4837 or <a href="mailto:AParkman@ucwv.edu">AParkman@ucwv.edu</a></td>
<td>Jay Wildt at (304) 442-7474 or <a href="mailto:jay.wildt@mghwv.org">jay.wildt@mghwv.org</a>; or Anna Parkman at</td>
</tr>
<tr>
<td>Sept. 12–14</td>
<td>MD/DC Society for Respiratory Care, 20th Annual Conference by the Sea, Ocean City, MD</td>
<td>Joe Lynott at (202) 877-6086 or <a href="mailto:joseph.p.lynott@medstar.net">joseph.p.lynott@medstar.net</a>; or Tom Striplin at (301) 784-5523 or <a href="mailto:tstriplin@mindspring.com">tstriplin@mindspring.com</a></td>
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<tr>
<td>Sept. 12–14</td>
<td>Alabama Society for Respiratory Care’s Annual Meeting, Birmingham, AL</td>
<td>Bill Pruitt, (334) 434-3405, <a href="mailto:wpruitt@jaguar1.usouthal.edu">wpruitt@jaguar1.usouthal.edu</a></td>
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<tr>
<td>Sept. 26–27</td>
<td>MSRC Annual Meeting, Sturbridge, MA <a href="mailto:O2val@aol.com">O2val@aol.com</a></td>
<td>Valeri-Ann Bolduc, (508) 429-7478,</td>
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<tr>
<td>Dec. 1–4</td>
<td>47th International Respiratory Congress; San Antonio, TX</td>
<td>AARC, (972) 243-2272, <a href="http://www.aarc.org">www.aarc.org</a></td>
</tr>
<tr>
<td>Feb. 6–8, 2002</td>
<td>West Virginia Society for Respiratory Care’s Winter Meeting, Davis, WV</td>
<td>Jay Wildt at (304) 442-7474 or <a href="mailto:jay.wildt@mghwv.org">jay.wildt@mghwv.org</a>; or Anna Parkman at (304) 357-4837 or <a href="mailto:AParkman@ucwv.edu">AParkman@ucwv.edu</a></td>
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<td>June 29–July 1</td>
<td>Current Concepts in Pediatric Respiratory Diseases: San Diego 2001; San Diego, CA</td>
<td>Children’s Hospital &amp; Health Center, Continuing Medical Education, (888) 892-9269, <a href="mailto:rwebb@chsd.org">rwebb@chsd.org</a></td>
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<td>Oct. 2–4</td>
<td>Cardiorespiratory Diagnostics 2001; Las Vegas, NV <a href="http://www.medgraph.com">www.medgraph.com</a></td>
<td>Medical Graphics Corporation, Mari Orke, (800) 950-5597, ext. 444,</td>
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Scheduled Professor's Rounds 2001

Program #4 Patient Education for the Asthmatic—Tracey Mitchell RRT; Host Thomas J Kallstrom RRT FAARC—Video May 22 Audio June 19

Program #5 ARDS: The Disease and Its Management—Leonard D Hudson MD; Host David J Pierson MD FAARC—Video June 26 Audio July 17


Program #7 Invasive Ventilation: The Latest Word—Richard H Kallet MS RRT; Host Richard D Branson BA RRT FAARC—Video September 25 Audio October 16

Program #8 Test Your Lungs-Know Your Numbers—Prevent Emphysema—Thomas L Petty MD FAARC; Host David J Pierson MD FAARC—Video October 23 Audio November 20
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