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CONTENTS
October 1990
Volume 35, Number 10

EDITORIALS
945 Pressure-Controlled Inverse-Ratio Ventilation: Panacea or Auto-PEEP?
by Robert M Kacmarek—Boston, Massachusetts; and Dean Hess—York, Pennsylvania

949 Malignant Hyperthermia: The Respiratory Care Practitioner's Critical Role
by Marilyn Green Larach—Hershey, Pennsylvania

ORIGINAL CONTRIBUTIONS
952 Laboratory and Clinical Evaluation of the MAX Transport Ventilator
by Jay A Johannigman, Richard D Branson, Robert S Campbell, and James M Hurst—San Antonio, Texas, and Cincinnati, Ohio

960 Evaluation of Ten Disposable Manual Resuscitators
by Thomas A Barnes and William P McGarry III—Boston, Massachusetts

969 Comparing RCPs to Physicians for the Description of Lung Sounds: Are We Accurate and Can We Communicate?
by Robert L Wilkins and James R Dexter—Loma Linda, California

REVIEWS, OVERVIEWS, AND UPDATES
977 Malignant Hyperthermia: A Review of Current Concepts
by Narendra Vakharia and Richard Hall—Halifax, Nova Scotia, Canada

DRUG CAPSULE
987 Systemic Antifungal Agents
by Hugh S Mathewson—Kansas City, Kansas

REPORTS
990 Report of Third Consensus Conference on Home Oxygen Therapy
(held in Washington DC, March 15-16, 1990)
by Thomas L Petty and Walter J O'Donohue, Co-Chairmen

PFT CORNER
997 PFT Corner #38—The Case of the Supranormal FEV1
by Christopher G Green and Ronald F Rankin—Madison, Wisconsin

BLOOD GAS CORNER
1001 Blood Gas Corner #27—Nonventilatory Cause of Hypercapnia during Weaning
by Robert S Campbell, Richard D Branson, and James M Hurst—Cincinnati, Ohio
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CONTENTS, Continued

TEST YOUR RADIOLOGIC SKILL
1003 Exertional Dyspnea in a Patient with Chronic Urinary Tract Infection by Dominic P Coppolo and James I Couser—Cooperstown, New York

BOOKS, FILMS, TAPES, & SOFTWARE
1008 Respiratory Infections: Diagnosis and Management, 2nd ed, edited by James E Pennington MD reviewed by John W Shigeoka—Salt Lake City, Utah

CORRECTION
989 Correction to CRE through the Journal: Answer Key, Respir Care 1990;35:906

ABSTRACTS
934 Summaries of Pertinent Articles in Other Journals

CALENDAR OF EVENTS
1011 Meeting Dates, Locations, Themes

NOTICES
1012 Examination Dates, Notices, Prizes

INFORMATION FOR AUTHORS
1013 Instructions for Authors and Typists

NEW PRODUCTS
1015 Pulse Oximeter
1015 Powder-Free Latex Gloves
1015 Dust Mite Test and Treatment Products
1015 Bite Block/Tube Holder

INDEXES
1016 Authors in This Issue
1016 Advertisers in This Issue

RESPIRATORY CARE • OCTOBER '90 Vol 35 No 10

Norepinephrine, an $\alpha_{1,2};\beta_{1,2}$-adrenergic agonist, seems to be an alternative to epinephrine, an $\alpha_{1,2};\beta_{2}$-agonist, for restoration of spontaneous circulation in ventricular fibrillation (VF). We therefore studied the effect of epinephrine and norepinephrine on myocardial oxygen delivery ($\text{MDO}_{2}$) and myocardial oxygen consumption ($\text{MVCO}_{2}$) using open-chest cardiac massage (OCCM) after 5 min of cardiopulmonary arrest in 21 pigs. After OCCM of 3 min, seven animals each received placebo (controls), epinephrine (45 µg/kg), or norepinephrine (45 µg/kg). All drugs were given blindly. At 90 sec after epinephrine or norepinephrine, mean arterial blood pressure was significantly higher than in the control group. Prior to cardiac arrest, myocardial blood flow (MBF) measured with radioactive microspheres, was 193 ± 30 mL/min/100 g. During CPR but before drug administration, MBF was 51 ± 23 in the control group, 71 ± 10 in the group with epinephrine, and 74 ± 11 mL/min/100 g in the group with norepinephrine. At 90 s after epinephrine, MBF increased to 126 ± 18 and after norepinephrine to 107 ± 30 mL/min/100 g (p < 0.05). Compared to OCCM
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alone, MDₜₒ₂ increased from 9.6 ± 1.7 to 17.1 ± 3.2 mL/min/100 g after epinephrine and from 9.4 ± 1.8 to 13.6 ± 4.2 mL/min/100 g after norepinephrine (p < 0.05). There was an increase in MVₒ₂ from 4.0 ± 1.5 to 9.4 ± 3.0 mL/min/100 g after epinephrine (p < 0.05), whereas MVₒ₂ increased only from 4.2 ± 0.8 to 5.1 ± 2.0 mL/min/100 g after norepinephrine. Because epinephrine led to a greater increase in MVₒ₂ than norepinephrine, the myocardial oxygen extraction ratio remained unchanged. The oxygen requirements of the fibrillating heart seemed to be increased via β₂-adrenergic stimulation. In both the control and epinephrine-treated groups, only three of the seven animals could be successfully resuscitated, whereas all of the animals in the group with norepinephrine survived the 15-min period of observation. In this model, norepinephrine, in contrast to epinephrine, improves the balance between MDₜₒ₂ and MVₒ₂, and eases restoration of spontaneous circulation.


Psychosocial assets of 37 adults with cystic fibrosis (CF) and 46 of their healthy peers were assessed by mailed questionnaire. Major sociodemographic variables did not differ significantly between the two groups, nor did indices of emotional social support, social network density, self-esteem, or current life satisfaction. This study revealed adults with CF to function on a par with their healthy peers in nearly all respects, a finding at odds with those from uncontrolled studies and which suggests to us that many previous conclusions about the psychosocial health of adults with CF have been unwarranted. Future psychosocial studies involving patients with CF should include control groups, and inferences about the effect of these patients' physical illness on their psychosocial health should not be made in the absence of normative data.


We designed a randomized controlled study to evaluate the benefit of upper-limb exercise training, alone and in combination with walking training, in patients with severe CAO. In an outpatient department supervised by a physiotherapist, we evaluated 28 patients with severe stable CAO (FEV₁ 32% of predicted). Patients were randomly allocated to either a control (8), upper-limb (6), lower-limb (7), or combined (7) exercise group. The upper-limb group trained with a circuit of upper-limb exercises, the lower-limb group by walking, and the combined group with both. Exercise was for 1 h three times/wk for 8 wk. Assessment before and after training included pulmonary function, mouth pressures, respiratory muscle endurance, maximal bicycle exercise test, maximal and submaximal arm ergometer, 6-min walking distance, and a scale of well-being (Bandura scale). Twenty-six patients completed the program. There was a significant improvement (Wilcoxon rank sum test) in the following: 6-min walking distance in the lower-limb (p < 0.005) and combined (p < 0.003) groups; arm ergometer in the upper-limb (p < 0.005) and combined (p < 0.04) groups; and the scale of well-being in the combined (p < 0.005) group. There was no significant change in any other parameter measured. We conclude that exercise training improves exercise performance in severe CAO, that the training is specific for the muscle group trained, and that upper-limb exercises should be included in training programs for these patients.


The Minneapolis Department of Veterans Affairs Medical Center began an intervention for tobacco use in its inpatient substance dependency treatment program on June 19, 1988, including an institutional smoke-free policy and a smoking cessation program. Sequential substance-dependent patients admitted before institution of the policy (n = 455) were compared with patients admitted after institution of the policy (n = 457). Patients completed self-administered questionnaires regarding smoking practices and attitudes. Seventy-six percent of pa-
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patients were current cigarette smokers. Fifty-eight percent of patients after institution of the policy described themselves as "not smoking regularly," compared with 19% of patients admitted before the institution of the policy. Similar proportions of patients admitted before and after the institution of the policy believed that quitting smoking would threaten recovery. Forty-one percent of patients hospitalized after institution of the policy abstained from smoking for more than 1 wk during their hospital stay. Rates of early termination of treatment did not change. We conclude that concurrent intervention for nicotine addiction during inpatient treatment of substance dependence is associated with a temporary reduction in smoking and increased motivation to quit smoking.


We measured forced expiratory volume in 1 s (FEV₁), respiratory impedance (Z_{rs}) from 4 to 60 Hz, and a multibreath N₂-washout (MBNW) in 6 normal, 10 asthmatic, and 5 cystic fibrosis (CF) subjects. The MBNW were characterized by the mean dilution number (MDN) derived by a moment analysis. The Z_{rs} spectra were characterized by the minimum resistance (R_{min}), the drop in resistance (R_{drop}) from 4 Hz to R_{min}, and the first resonance frequency (F₁). Measurements were repeated after bronchodilation in three normal and all asthmatic subjects. Before bronchodilation, six of the asthmatic subjects showed close to normal FEV₁. The Z_{rs} in the normal subjects showed low R_{min} (1.9 ± 0.7 cm H₂O·s·L⁻¹), R_{drop} (0.4 ± 0.4), and F₁ (10 ± 2 Hz). Four of the mildly obstructed asthmatic subjects had normal Z_{rs} but elevated MDNs (ie, abnormal ventilation distribution). The other six asthmatic subjects had significantly elevated R_{min} (4.1 ± 0.8), R_{drop} (6.3 ± 5.8), and F₁ (34 ± 0.4 Hz) and elevated MDNs. The CF patients had elevated Z_{rs} features and MDNs. After bronchodilation, no changes in FEV₁, MDN, or Z_{rs} occurred in the four asthmatic subjects whose baseline Z_{rs} was normal. For the other six asthmatic subjects, there were large decreases in the R_{min}, R_{drop}, and F₁. Finally, there was a poor correlation between the MDN and the Z_{rs} features but high correlation between the Z_{rs} features.
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ABSTRACTS


Forty patients with cystic fibrosis (CF) (mean age 13 ± 2.5 y) were studied with transcutaneous (tc) blood gas monitoring (TCM) during sleep and exercise. By comparing arterial blood samples and TCM in 24 of them (27 samples), a mean bias of $P_{a}CO_2$ = 15.91 torr with a precision of 8.4 torr was found. The mean bias of $P_{a}CO_2$ was 7.21 torr with a precision of 3.9 torr.

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References:
1. Hendler J, Shapiro S, Ogilvie R, Aldrich T: Accuracy of new and used portable peak flow meters. Abstract presented at the 49th Annual Meeting of the American College of Allergy and Immunology, Orlando, FL, November 30-December 3, 1990
3. Williams MH: Expiratory flow rates: Their role in asthma therapy. Hospital Practice 20, October, 1985
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Pressure-Controlled Inverse-Ratio Ventilation: Panacea or Auto-PEEP?

Pressure-controlled inverse-ratio ventilation (PCIRV) is a form of mechanical ventilation in which airway pressure during inspiration is limited, and the inspiratory time is longer than the expiratory time. This is in contrast to conventional mechanical ventilation in which the inspiratory phase is volume-limited, and expiration is longer than inspiration. PCIRV was popularized by Reynolds et al. in the early 1970s as a means of improving arterial oxygenation of neonates with infant respiratory distress syndrome. Although PCIRV has become less fashionable in neonatal mechanical ventilation, its use has been recently advocated in the treatment of ARDS.

Although PCIRV can be provided by a number of mechanical ventilators designed for adults, its current popularity in adults can probably be traced to the introduction of the Siemens Servo 900C ventilator. With the Servo 900C, PCIRV can be provided at an inspiratory-to-expiratory time ratio (I:E) as great as 4:1 (80% inspiratory time). Because this mode of ventilation is very uncomfortable and unnatural, paralysis and sedation are required.

Several recent reports on the use of PCIRV have claimed improvements in gas exchange at lower peak airway pressures and lower positive end-expiratory pressure (PEEP) levels. Proponents also suggest that PCIRV results in decreases in F_{1\text{O}_{2}} requirement, increases in P_{\text{O}_{2}}, and decreases in risk of barotrauma. However, four questions can be posed relative to PCIRV:

- How does this technique improve oxygenation?
- Without PEEP, how does improved recruitment of lung units occur?
- Are the claims of the proponents of this technique true?
- Is PCIRV a panacea for the management of acute lung injury or is it simply a method of applying auto-PEEP?

A summary of recent reports by Tharratt et al., Lain et al., and Abraham et al. is provided in Table 1. Two of these papers are seriously flawed due to their retrospective design. None of these reports states the authors' outcome criteria relative to increases in P_{\text{O}_{2}}, decreases in intrapulmonary shunt (Q_{s}/Q_{t}), decreases in peak inspiratory pressure, or changes in PEEP. None of these papers has a control group!

Perhaps most disturbing is that each of these papers advocating the use of PCIRV fails to acknowledge that the short expiratory times produced by PCIRV result in the development of air trapping and auto-PEEP. Only one report actually documents the development of auto-PEEP during PCIRV. The Servo 900C ventilator was used in each of these 'studies'—a ventilator with which auto-PEEP can be easily measured by using the expiratory-hold control. We speculate that PCIRV caused air trapping and auto-PEEP, and that the application of high levels of PEEP via this mechanism explains the reported improvement in gas exchange. Further, we believe that the same effect might have occurred if the authors had used higher PEEP levels with conventional volume-controlled ventilation!

For passive exhalation to be complete, adequate time is required. The exact amount of time is determined by the time constants of individual lung units. A time constant is the product of compliance and resistance. Expiratory times equal to 3-4 time constants are necessary for complete passive exhalation.
Table 1. Comparisons of Conventional Ventilation (CMV) to Pressure-Controlled Inverse-Ratio Ventilation (PCIRV) in Three Papers

<table>
<thead>
<tr>
<th></th>
<th>Tharratt(^5) (n = 31)</th>
<th>Lain(^7) (n = 19)</th>
<th>Abraham(^3) (n = 9)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CMV</td>
<td>PCIRV</td>
<td>CMV</td>
</tr>
<tr>
<td>(P_{A\text{O}_2}) (torr)</td>
<td>69 (4)*</td>
<td>80 (5)</td>
<td>66 (14)</td>
</tr>
<tr>
<td>(F_{\text{IO}_2})</td>
<td></td>
<td></td>
<td>0.65</td>
</tr>
<tr>
<td>PIP (cm H(_2)O)</td>
<td>66 (2)</td>
<td>46 (2)</td>
<td>48 (13)</td>
</tr>
<tr>
<td>PEEP(_{AP}) (cm H(_2)O)</td>
<td>15 (1)</td>
<td>2.5 (0.5)</td>
<td>7</td>
</tr>
<tr>
<td>(P_{aw}) (cm H(_2)O)</td>
<td>30 (2)</td>
<td>35 (2)</td>
<td>13</td>
</tr>
<tr>
<td>(V_E) (L/min)</td>
<td>22 (1)</td>
<td>15 (1)</td>
<td>15 (4)</td>
</tr>
<tr>
<td>Rate (breaths/min)</td>
<td></td>
<td></td>
<td>20 (7)</td>
</tr>
<tr>
<td>(V_T) (mL)</td>
<td></td>
<td></td>
<td>720 (123)</td>
</tr>
<tr>
<td>MAP (torr)</td>
<td>85 (3)</td>
<td>87 (3)</td>
<td></td>
</tr>
<tr>
<td>Mean PAP</td>
<td>38 (2)</td>
<td>36 (2)</td>
<td></td>
</tr>
<tr>
<td>PAP (torr)</td>
<td></td>
<td></td>
<td>44/23 (4/3)</td>
</tr>
<tr>
<td>PCWP (torr)</td>
<td>19 (1)</td>
<td>17 (1)</td>
<td>13 (1)</td>
</tr>
<tr>
<td>CI</td>
<td></td>
<td></td>
<td>4.8 (1)</td>
</tr>
</tbody>
</table>

*Values are mean (SD) throughout table.

CI = cardiac index.

MAP = mean arterial pressure.

PAP = pulmonary artery pressure.

PEEP\(_{AP}\) = applied PEEP.

PIP = peak inspiratory pressure.

PCWP = pulmonary capillary wedge pressure.

\(V_E\) = minute volume.

\(V_T\) = tidal volume.

The effect on air trapping of reducing expiratory time was described by Bergman,\(^11\) in 1972, in anesthetized postoperative patients with normal lungs. Bergman found an inverse relationship between volume of air trapped and available time constants, with as much as two liters (above 'normal' FRC) trapped as expiratory time was limited to one time constant.

Others have also suggested that the primary effect of PCIRV is the development of auto-PEEP.\(^12,13\) In addition, Marini et al.,\(^14\) using mathematical modeling, and one of us,\(^15\) in a laboratory model, have demonstrated the establishment of auto-PEEP during PCIRV. Not only does PCIRV result in auto-PEEP, but it also produces potential problems with tidal volume delivery. The tidal volume during this mode of ventilation is determined by the complex interplay of inspiratory pressure, inspiratory time, auto-PEEP, and lung characteristics (resistance and compliance). Unlike volume ventilation, tidal volume (and, therefore, alveolar ventilation) fluctuates during PCIRV as the patient’s lung function changes.

The only study to date that attempts to document the relationship between PCIRV and air trapping in patients with ARDS has been published by Cole et al.\(^16\) In 10 adult patients, they compared the effects of intermittent positive-pressure ventilation (IPPV) with an I:E of 1:2 to PCIRV with an I:E of 4:1. The effect of PCIRV on air trapping was evaluated by respiratory inductive plethysmography (RIP). They noted a mean 1200-mL increase in external end-expiratory volume (EEEV) during PCIRV.
compared to EEEV with IPPV. When the same EEEV was established with PEEP (12.8 cm H₂O) during IPPV, no statistically significant differences between \( Q_s/Q_t \), dead-space-to-tidal volume ratio, mean airway pressure, cardiac output, or compliance were noted between the two approaches. Although Cole et al did not measure auto-PEEP,\(^6\) Hoffman et al\(^7\) recently showed that RIP can be employed to successfully measure auto-PEEP. They demonstrated that the EEEV of the chest wall and abdomen does not increase with the application of PEEP until the auto-PEEP level is exceeded. The Hoffman data imply that the 12.8 cm H₂O PEEP that Cole et al needed to re-establish EEEV was equivalent to the auto-PEEP level established with PCIRV.

In general, advocates of PCIRV fail to acknowledge the effects of auto-PEEP. Only two reports, one published in abstract form,\(^8\) have documented auto-PEEP with PCIRV. Anderson\(^9\) noted from 7- to 22-cm H₂O auto-PEEP with I:E of 2:1 to 4:1 in a series of 105 patients with ARDS. This resulted in total PEEP levels between 15 and 30 cm H₂O, while Conoscenti et al\(^1\) measured between 10- and 23-cm H₂O auto-PEEP at ratios of 4:1 in three patients with ARDS. The most disturbing aspect of the lack of attention to the auto-PEEP effect of PCIRV is the assumption by its proponents that the PEEP level is lower with this technique, when, in fact, the total PEEP (applied PEEP + auto-PEEP) may be well over 20 cm H₂O.

A clinical scenario for mismanagement is created if auto-PEEP is not measured and documented. It is well established that high levels of PEEP may markedly alter pulmonary hemodynamics, affecting central venous pressure, pulmonary capillary wedge pressure, left-ventricular filling, and cardiac output. These changes may result in inappropriate fluid, vasopressor, or inotropic therapy\(^1\) if the presence of auto-PEEP is not recognized. The establishment of PEEP by PCIRV is not innately wrong. However, because of the potential deleterious effects of PEEP, auto-PEEP should only be knowingly administered in quantifiable and precisely monitored amounts.

Perhaps the most disturbing feature of PCIRV is its deceptiveness. The actual alveolar PEEP levels may be very high, even though the external PEEP set on the ventilator may be zero. If this is not appreciated, as is often the case, then adverse effects, such as unstable hemodynamics and barotrauma, might be attributed to causes other than auto-PEEP. Because of the complex interaction of the ventilator and the patient’s lungs, which determines tidal volume during PCIRV, clinically important changes in alveolar ventilation can occur without being obvious to those caring for the patient. Also, calculations of compliance based upon set PEEP levels may be considerably different from the actual compliance if auto-PEEP is present.\(^2\) The presence of auto-PEEP also requires increased patient effort to trigger assisted breaths, which might be part of the reason that sedation and paralysis are often necessary during PCIRV. Finally, because of the presence of auto-PEEP, mean airway pressure measured at the proximal endotracheal tube underestimates actual mean pressure unless inspiratory and expiratory airway resistance are equal.\(^3\)

If it is reasonable to assume that the development of auto-PEEP accounts for the effects of PCIRV, the incidence of barotrauma during IPPV and PCIRV should be similar. In fact, Tharratt et al\(^4\) noted a 26% incidence of barotrauma with PCIRV, which is similar to the levels of barotrauma commonly noted with the application of high levels of PEEP.\(^2\) From the data published by Tharratt et al,\(^4\) a case might be made that PCIRV is actually dangerous! Two patients did not tolerate attempts to institute PCIRV, and both died. Overall mortality for the 31 patients in this study was 77%! It is unlikely that PCIRV had any beneficial effect on outcome in this study.

Is PCIRV the new panacea for the treatment of ARDS? Absolutely not! At best, it may be equivalent to conventional ventilation with properly applied PEEP. However, it requires sedation and paralysis and, more importantly, may lead to misinterpretation of the total PEEP level and its resultant effects on hemodynamics and barotrauma. On the other hand, pressure control ventilation may prevent increased peak airway pressures, which may be beneficial in some patients. However, the effect of peak airway pressure on lung parenchyma in ARDS is not clearly established. Much of the peak airway pressure is dissipated at the endotracheal tube and large central airways, and it is questionable whether the shear forces established affect lung parenchyma more than a sustained plateau pressure.

There is currently no scientific evidence to support the use of PCIRV. Until controlled clinical trials are conducted, PCIRV must be considered investiga-
tional. Because no scientific studies exist to guide the application of PCIRV, because the potential for misuse of this technique is high, and because there are no scientifically supportable advantages over conventional volume ventilation, we urge caution in the use of this therapy. At the least, auto-PEEP levels must be measured and clearly documented when this therapy is employed.

Robert M Kacmarek PhD RRT
Assistant Professor
Department of Anesthesiology
Harvard Medical School
Director, Respiratory Care
Massachusetts General Hospital
Boston, Massachusetts

Dean Hess MEd RRT
Assistant Director of Clinical Research
York Hospital
Instructor, Respiratory Care Program
York Hospital
and York College of Pennsylvania
York, Pennsylvania

Reprints: Robert M Kacmarek, Respiratory Care—Ellison 4, Massachusetts General Hospital, Boston MA 02114.

REFERENCES

Malignant Hyperthermia: The Respiratory Care Practitioner’s Critical Role

In conjunction with the appearance of Vakharia and Hall’s review1 of current concepts in malignant hyperthermia in this issue of Respiratory Care, I want to emphasize the critical role of the respiratory care practitioner in the detection and treatment of malignant hyperthermia.

Susceptibility to malignant hyperthermia is an inherited disorder of skeletal muscle in which some medications in common use trigger sustained skeletal muscle hypermetabolism and/or contracture. If untreated, the hypermetabolism results in an acute fulminant episode of malignant hyperthermia (MH) that is fatal 70% of the time.2 This disease, which may present in previously asymptomatic subjects, continues to kill even young, healthy individuals during routine, elective surgery.3

Common medications that trigger MH include potent inhalational anesthetic agents (eg, halothane, isoflurane, enflurane) and depolarizing muscle relaxants (eg, succinylcholine). Every hour in the U.S., more than 2,000 individuals, who may be MH susceptible, are anesthetized with MH-triggering medications (Personal communication, Karen Allenstein, Associate Director Marketing Services—Anaquest, 1990). MH-susceptible individuals are usually asymptomatic and otherwise healthy and may be of any age. Their susceptibility to MH is frequently unknown until they are given a triggering medication to induce and maintain anesthesia or to facilitate emergency endotracheal intubation. The respiratory care practitioner may encounter patients with an acute MH episode in emergency, postanesthesia recovery, or intensive care units.

An acute MH episode presents with multiple, nonspecific signs and laboratory findings of variable intensity and time course during or after exposure to triggers.4,5 These abnormal signs and laboratory findings may include tachypnea, hypercarbia, respiratory and/or metabolic acidosis, tachycardia, masseter and/or generalized muscle rigidity, myoglobinuria, increased creatine kinase, skin mottling, cyanosis, hyperkalemia, arrhythmias including ventricular tachycardia and fibrillation, cardiac arrest, hypertension, diaphoresis, rapid temperature rise, and excessive bleeding.6–8

The respiratory care practitioner can play a key role in the early diagnosis of MH because unexplained hypercarbia due to increased carbon dioxide production ($V_{CO_2}$) in the absence of shivering or alveolar hypoventilation is an early and sensitive sign of MH. Alert respiratory care practitioners may be the first medical professionals to detect the onset of MH in emergency or intensive care settings. Additionally, they may be the first to detect potentially fatal MH relapse (recrudescence) in mechanically ventilated patients in the recovery room or intensive care unit. Early diagnosis of acute MH is important because incorrect or late treatment may result in death in 70% of cases.1 Even when appropriately treated, MH may have a 10% death rate.9

Once the diagnosis of a probable MH episode is established, treatment must be instituted rapidly (within minutes). The cornerstone of treatment is rapid administration of intravenous dantrolene. The respiratory care practitioner plays an important role in providing sufficient ventilation and oxygenation to these severely hypermetabolic patients who may have a threefold increase in oxygen uptake and massively elevated $V_{CO_2}$.10 In addition, patients undergoing fulminant MH may be difficult to ventilate because of severe generalized muscular rigidity despite the administration of nondepolarizing muscle relaxant drugs. After therapy has begun, the respiratory care practitioner can help monitor the effectiveness of
treatment by documenting decreasing ventilatory requirements. Weaning patients from mechanical ventilation once their MH episode has been well controlled may be complicated by muscular weakness after dantrolene, particularly in patients with underlying myopathies.

Survivors of acute fulminant MH may suffer serious sequelae including acute renal failure, pulmonary edema, disseminated intravascular coagulation, cerebral edema, and permanent neurologic sequelae including paralysis and coma. 11

Currently, the diagnosis of MH susceptibility is made with certainty only when a patient survives a fulminant MH episode after exposure to known MH-triggering agents. Fulminant MH is now rare because new monitoring modalities permit the early detection and treatment of nongenetic metabolic abnormalities that may represent an early MH episode. 12 Once therapeutic intervention for an early MH episode is undertaken, a definitive diagnosis of MH susceptibility cannot be made on clinical grounds. 6

The differential diagnosis of early (non-fulminant) MH is extensive and includes light (ie, inadequate) anesthesia, hypoxia and hypercarbia secondary to alveolar hypoventilation, iatrogenic hyperthermia, heat stroke, 13 sepsis, radiologic contrast material within the central nervous system, 14 thyrotoxicosis, 15 pheochromocytoma, 16 and neuroleptic malignant syndrome. 17-19 Current medical practice utilizes the in-vitro caffeine halothane contracture test (CHCT) to establish a diagnosis of MH susceptibility in individuals who have previously experienced an early (nonfulminant) MH episode and in family members of known MH-susceptible persons.

The Malignant Hyperthermia Association of the United States (MHAUS) (203-655-3007) is dedicated to the control of MH: the Association provides excellent educational materials to health care personnel, MH patients, and families. Because MH is the subject of active research, once an MH patient has been stabilized with initial treatment, it is recommended that medical personnel contact the MHAUS Hotline consultant (call 209-634-4917; ask for Index Zero) for the most up-to-date treatment recommendations. The Hotline consultants will also facilitate patient referral to an MH diagnostic center for further evaluation and counseling.

The North American Malignant Hyperthermia Registry provides patient-specific clinical information to physicians caring for registered MH-susceptible patients. In addition, the Registry accumulates comparative data to support its MH epidemiologic studies, including the prediction of MH susceptibility. The Registry's computerized database of 1,000 individuals represents the world's largest collection of clinical and laboratory data on MH-susceptible individuals, as well as control subjects who have undergone MH diagnostic evaluation (including CHCT). Respiratory care practitioners may contact the Registry (717-531-6936) to report acute MH episodes.

Respiratory care practitioners who facilitate patient referral to MHAUS and the Registry will help improve the care of MH-susceptible patients and contribute to MH research. The well-informed respiratory care practitioner can play a critical role in the diagnosis and management of MH episodes.

Marilyn Green Larach MD
Director
The North American Malignant Hyperthermia Registry
Assistant Professor
Department of Anesthesia
The Pennsylvania State University
Hershey, Pennsylvania

Reprints: Marilyn Larach MD, Dept of Anesthesia, The Milton S. Hershey Medical Ctr, PO Box 850, Hershey PA 17033.

REFERENCES

Laboratory and Clinical Evaluation of the MAX Transport Ventilator

Jay A Johannigman MD, Richard D Branson RRT, Robert Campbell RRT, and James M Hurst MD

BACKGROUND: Transport of critically ill, mechanically ventilated patients from intensive care units for diagnostic and therapeutic procedures has become common in the last decade. Maintenance of adequate oxygenation and ventilation during transport is mandatory. We evaluated the Hamilton MAX transport ventilator in the laboratory and in the clinical arena to determine its usefulness during in-hospital transport.

METHODS: In the laboratory, we determined the MAX's ability to assure tidal volume \( V_T \) delivery in the face of decreasing compliance of a test lung, and we tested the alarm system. Using a two-compartment lung model modified to simulate spontaneous breathing, we also evaluated the responsiveness of the demand valve. The clinical evaluation was accomplished by comparing arterial blood gases and ventilator settings in the intensive care unit to those during transport.

RESULTS: As lung compliance was reduced from 0.1 to 0.02 L/cm H_2O [1.0 to 0.20 L/kPa], delivered \( V_T \) fell significantly at each set \( V_T \). The alarm systems performed according to manufacturer's specifications. The demand valve triggered appropriately without positive end-expiratory pressure (PEEP), but as PEEP was increased, triggering became more difficult. The demand valve is referenced to ambient pressure and cannot compensate for elevated end-expiratory pressures. During patient transport, arterial blood gases were comparable to those achieved in the ICU. Because an inspired oxygen concentration of 1.0 was used during transport, arterial oxygenation \( P_a(O_2) \) was significantly greater \([123 \pm 75 \text{ vs } 402 \pm 85 \text{ torr} [16.4 \pm 10 \text{ vs } 53.6 \pm 11 \text{ kPa}]]\). A higher ventilator rate was required during transport to prevent tachypnea \((7 \pm 3 \text{ vs } 12 \pm 6 \text{ breaths/min})\), and peak inspiratory pressure (PIP) was higher during transport \((40 \pm 8 \text{ vs } 52 \pm 11 \text{ cm H}_2\text{O}[3.9 \pm 0.8 \text{ vs } 5.1 \pm 1.1 \text{ kPa}])\).

CONCLUSIONS: The MAX is a reliable transport ventilator, capable of maintaining adequate ventilation and oxygenation in a majority of mechanically ventilated patients. Care should be taken to assure adequate \( V_T \) delivery at high PIP, and ventilator rate may require adjustment to prevent tachypnea associated with triggering the non-PEEP-compensated demand valve when PEEP > 8 cm H_2O [0.8 kPa] is used. (Respir Care 1990;35:952-959.)

Dr. Johannigman is Captain and Director, Intensive Care Services, Department of Surgery, Wilford Hall, United States Air Force, San Antonio, Texas. At the time this study was performed, Dr. Johannigman was Surgery Fellow at University of Cincinnati Medical Center. Mr Campbell is Critical Care Coordinator, Respiratory Care Dept; Mr Branson is Clinical Instructor, Department of Surgery; and Dr Hurst is Associate Professor of Surgery and Anesthesia, and Director, Department of Surgery, Division of Trauma and Critical Care—University of Cincinnati Medical Center, Cincinnati, Ohio.

None of the authors has a financial interest in the MAX transport ventilator or its manufacturer. The views expressed in this article are those of the authors and do not reflect the official policy or position of the Air Force, Department of Defense, or U.S. Government.

Reprints: Richard D Branson RRT, Dept of Surgery, ML 558, Univ of Cincinnati Medical Center, 231 Bethesda Ave, Cincinnati OH 45267.
Introduction

Several papers published recently have demonstrated the superiority and improved safety of ventilatory support with a portable ventilator during transport, compared to manual ventilation with a self-inflating bag. Successful use of a portable or transport ventilator, however, depends upon its capabilities and characteristics. We studied the MAX transport ventilator* in the laboratory and then evaluated its performance during transportation of mechanically ventilated patients from the surgical intensive care unit for diagnostic studies.

Description of Device

The MAX (Fig. 1) is an electronically controlled, pneumatically powered, time-cycled, constant flow ventilator. Specifications for controlled variables are listed in Table 1. The MAX weighs 2.5 kg, and its dimensions are 29 cm W × 16 cm D × 8 cm H. Electric power is supplied by four 1.5-volt, AA rechargeable or alkaline batteries. A low-battery alarm warns the operator when less than 30 minutes of battery life remains. Respiratory frequency and tidal volume are the only adjustable controls. Airway pressure is displayed by an analog gauge.

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

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Abbreviations Used in this Paper

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>ABG</td>
<td>Arterial blood gas</td>
</tr>
<tr>
<td>f</td>
<td>Breathing frequency</td>
</tr>
<tr>
<td>Fr&lt;sub&gt;2&lt;/sub&gt;</td>
<td>Fractional inspired oxygen concentration</td>
</tr>
<tr>
<td>I:E</td>
<td>Inspiratory-time-to-expiratory-time ratio</td>
</tr>
<tr>
<td>ICU</td>
<td>Intensive care unit</td>
</tr>
<tr>
<td>IMV</td>
<td>Intermittent mandatory ventilation</td>
</tr>
<tr>
<td>IPPB</td>
<td>Intermittent positive-pressure breathing</td>
</tr>
<tr>
<td>P&lt;sub&gt;ACO&lt;/sub&gt;</td>
<td>Arterial carbon dioxide tension</td>
</tr>
<tr>
<td>P&lt;sub&gt;A&lt;/sub&gt;O&lt;sub&gt;2&lt;/sub&gt;</td>
<td>Arterial oxygen tension</td>
</tr>
<tr>
<td>PEEP</td>
<td>Positive end-expiratory pressure</td>
</tr>
<tr>
<td>PIP</td>
<td>Peak inspiratory pressure</td>
</tr>
<tr>
<td>PSV</td>
<td>Pressure support ventilation</td>
</tr>
<tr>
<td>SIB</td>
<td>Self-inflating resuscitation bag</td>
</tr>
<tr>
<td>SICU</td>
<td>Surgical ICU</td>
</tr>
<tr>
<td>TTL</td>
<td>Training test lung</td>
</tr>
<tr>
<td>V&lt;sub&gt;F&lt;/sub&gt;</td>
<td>Minute volume</td>
</tr>
<tr>
<td>V&lt;sub&gt;T&lt;/sub&gt;</td>
<td>Tidal volume</td>
</tr>
</tbody>
</table>

A Guide to the Use of SI in This Paper*

The SI unit for pressure is kilopascal (kPa).

\[
(\text{torr})\times(0.133\,\text{3}) = \text{kPa}.
\]

\[
(\text{cm H}_2\text{O})\times(0.098\,\text{06}) = \text{kPa}.
\]

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

\[
(L/\text{cm H}_2\text{O})\times(10.20) = \text{L/kPa}
\]

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

Table 1. Specifications for Controlled Variables of the MAX Transport Ventilator

<table>
<thead>
<tr>
<th>Variable</th>
<th>Range of Operation</th>
</tr>
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<tbody>
<tr>
<td>Rate</td>
<td>2-30 breaths/min</td>
</tr>
<tr>
<td>Tidal volume</td>
<td>50-1500 mL</td>
</tr>
<tr>
<td>Inspiratory time</td>
<td>1 second</td>
</tr>
<tr>
<td>Fr&lt;sub&gt;2&lt;/sub&gt;</td>
<td>100% source gas</td>
</tr>
<tr>
<td>Modes</td>
<td>IMV</td>
</tr>
<tr>
<td>Peak flow (max)</td>
<td>90 L/min—mandatory breath</td>
</tr>
<tr>
<td></td>
<td>145 L/min—spontaneous breath</td>
</tr>
<tr>
<td>Pressure limit</td>
<td>45-90 cm H&lt;sub&gt;2&lt;/sub&gt;O—adjustable internally</td>
</tr>
<tr>
<td>Maximum pressure relief</td>
<td>120 cm H&lt;sub&gt;2&lt;/sub&gt;O fixed</td>
</tr>
</tbody>
</table>
A schematic of the internal components is shown in Figure 2. Compressed gas at 50-90 psi enters the ventilator and passes through a pressure switch. The pressure switch is activated when the rate control is in any position other than off. If source-gas pressure fails or falls below 27 psi, the ‘oxygen failure’ alarm sounds, warning the operator. Gas then travels bi-directionally to the demand valve and to a pressure regulator. Gas diverted to the demand valve is at source-gas pressure, thus allowing maximal flow for spontaneous breathing. The demand valve is pneumatic, is triggered on by a -2 cm H2O [-0.2 kPa] deflection in circuit pressure, and is referenced to ambient pressure. PEEP can be added using an external PEEP valve, but triggering will not be PEEP-compensated. An adjustable screw on the back of the demand valve allows the diaphragm to be biased. When it is biased, the demand valve will ‘overshoot’ the patient’s inspiratory flowrate and increase airway pressure slightly above baseline. This does not improve triggering response of the demand valve, but it does serve as an ‘inspiratory boost’ that may help to reduce the work of breathing. The pressure regulator reduces inlet pressure to 50 psi, which is optimal operating pressure for the ventilator. From the regulator, gas travels to an electronically controlled solenoid valve. The solenoid has a fixed inspiratory time of 1 s. Expiratory time is adjusted according to the respiratory rate setting, and I:E is limited at 1:1. Gas at a constant pressure and constant inspiratory time then passes through a flow control that regulates tidal volume. Airway pressure is monitored just proximal to the gas outlet of the ventilator. Peak airway pressure (PAP) can be limited by an adjustable pressure regulator from 45 to 90 cm H2O [4.4 to 8.8 kPa]. This pressure regulator controls the amount of gas that travels through the expiratory drive line of a disposable circuit to close a mushroom-type exhalation valve. At present, this control is accessible only by removing the ventilator’s cover. A nonadjustable safety pressure-relief valve limits maximum peak pressure to 120 cm H2O [11.8 kPa]. The gas consumption of the ventilator is equal to the delivered minute volume.

Proximal to the solenoid is a second gas pathway, which is normally closed. This pathway is opened by depressing the manual breath switch. In this fashion, a manual breath will continue as long as the button is depressed, but will be delivered at the set flowrate. A standard intermittent positive pressure breathing (IPPB) circuit is used to deliver gas to the patient. The ventilator is capable of delivering only 100% source gas. Positive end-expiratory pressure (PEEP) may be added by an external valve. The MAX ventilator is intended for use during inter- and intrahospital transport of mechanically ventilated patients and as initial ventilatory support in the emergency department.

Materials and Methods

Laboratory Evaluation

In the laboratory we evaluated the MAX’s ability to maintain set tidal volume at varying compliances, the responsiveness of the demand valve with and without PEEP, and the alarm functions. The ventilator was set up according to the manufacturer’s specifications and powered with oxygen at 50 psi. We set the ventilator to deliver tidal volumes of 0.05, 0.5, 1.0, and 1.5 L to a test lung; and we varied compliance from 0.1 to 0.04 to 0.02 L/cm H2O [1.0 to 0.4 to 0.2 L/kPa]. A pneumotachograph was placed between the test lung and ventilator circuit to record actual delivered tidal volume. At each tidal volume and compliance, ten consecutive breaths were recorded to determine mean tidal volume for that setting. We used the same tubing for all measurements and determined its compressible volume. Compressible volume was measured by connecting one end of the circuit to a calibration syringe while plugging all other open ports and monitoring airway pressure as a 1.5-L volume was injected in 0.1-L increments. Measured volume was compared to set volume using a paired t test, and V\text{̄}\text{T} at different compliances was compared by analysis of variance for repeated
measures. Additionally, the ventilator was set at 1.0 L and respiratory rate was increased by available increments while delivered tidal volume was measured. This test was done to determine whether $V_T$ remained constant as $f$ changed. A compliance of 0.02 L/cm H$_2$O [0.2 L/kPa] was used during this test. Measured volumes were compared using ANOVA for repeated measures.

We tested the oxygen failure alarm by interrupting oxygen supply and by decreasing inlet pressure below 27 psi and recording the alarm response.

Using the TTL modified to simulate spontaneous breathing, we evaluated the responsiveness of the demand valve. One side of the TTL was ventilated with a Hamilton Veolar ventilator at a $V_T$ of 0.5 L, $f$ of 10 breaths/min, peak flowrate of 1.0 L/s, and a sine-wave flow pattern. The MAX ventilator was attached to the other side of the TTL, which simulates spontaneous breathing. We recorded airway pressure at the end of the MAX patient circuit during simulated spontaneous breathing at 0, 2.5, 5, 7.5, and 10 cm H$_2$O [0, 0.25, 0.5, 0.74, 0.98 kPa] PEEP. PEEP was produced by the appropriate PEEP valve.

**Clinical Evaluation**

We used the MAX to provide ventilatory support to 25 patients requiring transport from the Surgical Intensive Care Unit (SICU) for diagnostic studies. The patient population included 19 males and 6 females, with a mean age of 39 ± 14 y. There were 15 trauma patients and 10 postoperative patients. Four patients (one trauma patient and three postoperative patients) had a history of chronic lung disease. Patients had required a mean ± SD duration of ventilation prior to study of 7 ± 3 days. Prior to transfer, ventilator settings and spontaneous respiratory frequency were recorded and an arterial blood gas analysis was performed. Patients were then placed on the MAX using two schemes. In the first, any patient on IMV and less than 8 cm H$_2$O [0.8 kPa] PEEP was placed on the MAX at settings identical to those of the ICU ventilator. Patients receiving pressure support ventilation (PSV) and/or greater than 8 cm H$_2$O [0.8 kPa] PEEP were ventilated at the same $V_T$ as before, but $f$ was increased to deliver a minute volume ($V_E$) of approximately 70% of the ICU $V_E$. Our previous experience has led us to use this formula when transporting patients with a ventilator capable only of IMV.

During transport, the respiratory care practitioner was allowed to modify settings according to patient response. Any problems or mishaps were noted, and prior to return to the SICU, transport ventilator settings were recorded and an arterial blood gas (ABG) analysis was performed. We compared ABGs and ventilator settings during transport to those done in the SICU, by analysis of variance. Statistical analyses were performed with commercial software.

**Results**

**Laboratory Evaluation**

Means ± SD for the four tidal volumes at each compliance are shown in Table 2. As compliance was reduced, there were significant differences in measured $V_T$ compared to set $V_T$. This was particularly evident at $V_T$ 5 of 1.0 and 1.5 L when compliance was 0.04 or 0.02 L/cm H$_2$O [0.41 or 0.20 L/kPa]. In both these instances, delivered $V_T$ was less than 90% of set tidal volume. Compressible volume of our circuit was found to be 1.4 mL/cm

<table>
<thead>
<tr>
<th>Table 2. Comparison of Set and Measured Tidal Volumes</th>
</tr>
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<tbody>
<tr>
<td><strong>Set $V_T$</strong> (mL)</td>
</tr>
<tr>
<td>---------------------</td>
</tr>
<tr>
<td>0.1 L/cm H$_2$O</td>
</tr>
<tr>
<td>0.04 L/cm H$_2$O</td>
</tr>
<tr>
<td>0.02 L/cm H$_2$O</td>
</tr>
<tr>
<td>1400 ± 63</td>
</tr>
</tbody>
</table>

* = p < 0.05 compared to set $V_T$, using a paired $t$ test.
† = p < 0.05 compared to $V_T$ at 0.1 L/cm H$_2$O, using ANOVA for repeated measures.
‡ = p < 0.01 compared to $V_T$ at 0.02 L/cm H$_2$O, using ANOVA for repeated measures.
§ = p < 0.01 compared to $V_T$ at 0.04 L/cm H$_2$O, using ANOVA for repeated measures.
|| = p < 0.01 compared to $V_T$ at 0.1 L/cm H$_2$O, using ANOVA for repeated measures.
** = p < 0.01 compared to set $V_T$, using a paired $t$ test.
H₂O [14 mL/kPa]. Based upon PAP, compressible volume lost in the tubing accounted for approximately 50% of the VT discrepancy. For example, at a set VT of 1500 mL, mean measured VT was 1268 mL and peak inspiratory pressure was 68 cm H₂O [6.7 kPa]. In this instance, compressible volume was approximately 95 mL, which accounts for 41% of the difference between set and measured VT (1500 - 1268 = 232 mL). At lower VTs this problem was not as evident. The statistically significant differences between VTs at varying compliance can also be attributed to increased PAP and compressible volume. Changing f at a VT of 1.0 L did not affect delivered VT (Table 3).

Table 3. Changes in Measured Tidal Volume as Rate Changed*

<table>
<thead>
<tr>
<th>Rate (breaths/min)</th>
<th>Measured VT (mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>865 ± 17</td>
</tr>
<tr>
<td>4</td>
<td>870 ± 12</td>
</tr>
<tr>
<td>6</td>
<td>870 ± 15</td>
</tr>
<tr>
<td>8</td>
<td>882 ± 36</td>
</tr>
<tr>
<td>10</td>
<td>868 ± 13</td>
</tr>
<tr>
<td>12</td>
<td>887 ± 18</td>
</tr>
<tr>
<td>15</td>
<td>866 ± 12</td>
</tr>
<tr>
<td>20</td>
<td>869 ± 18</td>
</tr>
<tr>
<td>25</td>
<td>891 ± 21</td>
</tr>
<tr>
<td>30</td>
<td>894 ± 19</td>
</tr>
</tbody>
</table>

*All measurements were made at a compliance of 0.02 L/cm H₂O and with a set tidal volume of 1.0 L.

Low-battery and oxygen-failure alarms performed appropriately in response to our testing. Figure 3 depicts airway pressure at the end of the patient circuit during simulated spontaneous breathing. At zero PEEP, the demand valve triggered on at -3 cm H₂O [-0.3 kPa]. As PEEP was increased, trigger sensitivity remained the same, although uncompensated for PEEP, but the time for the demand valve to restore pressure to baseline lengthened. The tracings in Figure 3 also show the slight positive pressure developed by the biased demand valve. The second slightly positive aspect of the tracing represents exhalation.

Clinical Evaluation

Results of our clinical evaluation are summarized in Table 4. The only significant difference in blood gases was in PₐO₂, because during transport an F₁O₂...
of 1.0 was used. By design, the mean ventilator \( f \) was greater during transport, to prevent patients on > 8 cm \( \text{H}_2\text{O} [0.8 \text{ kPa}] \) PEEP and PSV from having to trigger the uncompensated demand valve. Peak inspiratory pressure (PIP) was also significantly greater during ventilation with MAX. On two occasions, the exhalation drive line was inadvertently disconnected from the ventilator by patient movement. Both patients were undergoing radiologic procedures, and disconnection went unnoticed until desaturation was detected by pulse oximetry. No prolonged adverse effects were caused by these mishaps. Once, the ventilator was kicked from the bed to the floor, but it remained connected and continued to operate. Mean transport time was 48 ± 36 minutes. Median transport was 39 minutes.

### Discussion

The necessity of providing safe and effective ventilation during patient transport remains an issue of paramount importance in critical care medicine. As technologic sophistication advances, so too does the complexity of the patient-equipment interface. The need to transport critically ill patients within and between hospitals has steadily increased over the past decade. Diagnostic facilities for CT scanning, radionuclide imaging, and diagnostic angiography are often distant from the Intensive Care Unit and require that the patient be safely maintained outside the SICU for significant periods of time. The frequency of inter-hospital transport has been promoted by the regionalization and specialization of care that has been fostered by the proliferation of technologic advances. Transport ventilation must be capable of providing safe and efficient ventilation in a patient population that often displays marked pulmonary dysfunction. A transport ventilator must meet patient needs while simultaneously fulfilling criteria of portability, ease of monitoring, maintenance, and setup.

A number of investigators have evaluated the respiratory changes that occur during in-hospital transports.\(^1\)\(^3,6\) Several studies have compared manual ventilation with a self-inflating resuscitation bag (SIB) to ventilation provided by a transport ventilator.\(^1\)\(^3\) Significant respiratory alkalosis was documented to occur during manual ventilation in separate studies by Gervais et al.\(^5\) Braman et al.\(^7\) and Hurst et al.\(^1\) This change was noted to be accompanied by episodic hypotension and cardiac arrhythmias. The respiratory alkalosis that typically accompanies manual ventilation with a SIB may be avoided by continuously monitoring \( V_T \) with a spirometer,\(^3\) but this becomes labor-intensive and requires the undivided attention of one member of the transport team. A properly operating transport ventilator is ideally capable of providing consistent ventilation that is independent of inter-operator variability, attention, fatigue, or interruption.\(^1\) With respect to cost, most ventilators are less expensive than personnel time, when considered over the life of the transport ventilator.

Based on a review of the literature and a survey conducted in 1985,\(^7\) an ideal transport ventilator should: (1) provide IMV, (2) have a variable tidal volume, (3) offer a variable rate (2-30 breaths/min), (4) provide a minute ventilation of 4-20 L/min, (5) have low- and high-pressure alarms, (6) provide PEEP (0-20 cm \( \text{H}_2\text{O} [0-2.0 \text{ kPa}] \)), (7) have a demand valve, and (8) monitor airway pressure. Additionally, PEEP-compensation of the demand valve is desirable. With the exception of providing PEEP (which can be provided by adding an external PEEP valve) and the alarm system, the MAX meets these requirements.

There was a significant difference in the actual vs set \( V_T \) at 0.04 and 0.02 L/cm \( \text{H}_2\text{O} [0.41 \) and
The loss of \( V_T \) with decreased compliance can be attributed to two factors. Approximately 40 to 50\% of the lost \( V_T \) is attributable to the compliance of the circuit. As with any ventilator that utilizes a compliant circuit, this loss becomes more prominent as airway pressures increase. The second source of decreased measured \( V_T \) is attributable to loss of delivered volume as the ventilator flow stalls secondary to decreased compliance and the accompanying elevated airway pressure. We do not feel that this is an important problem with the MAX ventilator (< 10\%). Rather, it emphasizes the need to directly measure delivered \( V_T \) anytime airway pressures are elevated or PIP exceeds 50 cm H\(_2\)O [4.9 kPa].

Evaluation of the performance of the demand valve demonstrated a typical characteristic of a non-PEEP-compensated system. At a PEEP of zero, the demand valve promptly triggered on at -3 cm H\(_2\)O [0.3 kPa]. At levels of PEEP above 5 cm H\(_2\)O [0.5 kPa], the demand valve required greater negative pressures before triggering to restore pressure to baseline (Fig. 3). This observation is of obvious clinical importance for patients who are on elevated levels of PEEP, because they may not be capable of consistently triggering the ventilator during transport. Because the MAX utilizes only the IMV mode, a situation may result in which the patient experiences a significant fall in \( V_E \) when transferred to the transport ventilator. For this reason, at our institution we empirically set the transport IMV to deliver 70\% of the patient's total \( V_E \). In the clinical arm of the evaluation, this formula provided an easy means of establishing the IMV rate for transport that resulted in a mean \( P_{aCO_2} \) of 38 torr [5.1 kPa].

In the clinical evaluation of the MAX, the ventilator proved capable of providing adequate and reliable ventilation during transport. When ventilator variables and ABGs from the ICU were compared to those during transport, there were three areas of significant difference. Ventilator rate during transport was intentionally increased to adjust for the non-PEEP-compensated demand valve (as discussed above). The significant increase in \( P_{aO_2} \) reflects our policy of routinely transporting patients on an \( FIO_2 \) of 1.0. (Because of our transport policy, we do not view the capability of the MAX to deliver only 100\% source gas as a shortcoming, although some practitioners may view it as such.) The one significant difference during ventilation with MAX was an increase in PIP (40 ± 8 vs 52 ± 11 cm H\(_2\)O [3.9 ± 0.8 vs 5.1 ± 1.1 kPa]). This is most probably a reflection of the MAX’s fixed 1-s inspiratory time.

**Conclusions**

We found the MAX to be a reliable, easy-to-operate transport ventilator that meets most of the demands of ventilation during transport. The results of our evaluation suggests that \( V_T \) should be checked at the airway prior to leaving the intensive care unit anytime airway pressures are elevated (> 50 cm H\(_2\)O [4.9 kPa]) The characteristics of a non-PEEP-compensated demand valve make it necessary to increase \( f \) to provide adequate \( V_E \) whenever patients are on assisted modes of ventilation or greater than 8 cm H\(_2\)O [0.8 kPa] PEEP. In patients with severe respiratory muscle dysfunction, smaller increments of PEEP may also prevent triggering of the demand valve. Finally, the fixed 1-s inspiratory time may result in significantly higher PIP during transport if \( V_T \) is held constant. We would suggest the following improvements to the current MAX design: (1) external access to the adjustable pressure-relief valve, (2) addition of a disconnect alarm, and (3) PEEP-compensation of the demand valve. At present, if careful attention is paid to the limitations we have discussed, the MAX can provide adequate ventilation and oxygenation to a majority of patients.

**PRODUCT SOURCES**

**Calibration Syringe:**
- Hans Rudolph Inc, Kansas City MO

**PEEP Valve:**
- Vital Signs Inc, Totowa NJ

**Pneumotachograph:**
- Hans Rudolph 3200, Hans Rudolph Inc, Kansas City MO

**Statistical Software:**
- Micro Stat, Ecosoft Inc, St Joseph MI

**Test Lung:**
- TTL, Michigan Instruments Inc, Grand Rapids MI

**Ventilators:**
- MAX, Hamilton Medical, Reno NV
- Veolar, Hamilton Medical, Reno NV
REFERENCES

Evaluation of Ten Disposable Manual Resuscitators

Thomas A Barnes EdD RRT and William P McGarry III BS RRT

We evaluated the performance and safety of 10 disposable resuscitators—six adult units: SPUR, Code Blue, 1st Response, Hospitak MPR, CPR Bag, and Pulmanex; and four pediatric units: CPR Bag, 1st Response, Hospitak MPR, and LSP Bag Mask. METHOD: We tested the devices against the American Society for Testing and Materials (ASTM) Standard F-920. We tested each resuscitator by using a lung model, the Bio-Tek VT-1 Ventilator Tester. RESULTS: All resuscitators met the ventilation requirements for $V_F$ and $f$ (adult: $600 \text{ mL } \times 12/\text{min}; \text{child: } 300 \text{ mL } \times 20/\text{min} \text{ and } 70 \text{ mL } \times 30/\text{min} \text{ and } I:E < 1:1$). Standard F-920 specifies a fractional delivered $O_2$ concentration $(F_{DO_2}) \geq 0.85$ with attachments and $\geq 0.40$ without attachments, at oxygen flow of 15 L/min, and $V_F$ of 7.2 L (600 mL × 12/ min) for adult units and $V_F$ of 6 L (300 mL × 20/min) for pediatric units. All 10 resuscitators met standard F-920 for $F_{DO_2}$ without attachments. Nine resuscitators met the $F_{DO_2}$ standard without attachments. The 10 resuscitators passed the test for valve function after contamination with simulated vomitus, at an oxygen flow of 30 L/min, and for backward leakage. Three pediatric resuscitators (1st Response, Hospitak MPR, and LSP Bag Mask) did not pass the pressure-limit requirement of 40 ± 10 cm H.O. Four resuscitators, Hospitak MPR (adult and pediatric) and CPR Bag (adult and pediatric), were unable to pass the test for mechanical shock (a fall from a height of at least 1 meter). CONCLUSION: We conclude that only Code Blue, 1st response, Pulmanex (with tube-type reservoir), and SPUR meet ASTM Standard F-920 and are acceptable replacements for permanent resuscitators. (Respir Care 1990;35:960-968.)

Introduction

We evaluated 10 disposable resuscitators against eight requirements of Standard F-920 published by the American Society for Testing and Materials (ASTM). The first disposable manual resuscitators were introduced in the United States in 1985. The first on the market were Stat Blue and Pulmanex devices.* During 1989, four new disposable manual resuscitators were introduced. By the Spring of 1990, 11 adult disposable resuscitators were available in the United States, with most manufacturers providing smaller versions for children and infants. In its 1987 product literature, Vital Signs, one of the first on the market with a disposable resuscitator and supplier of the Stat Blue and Code Blue devices, claimed that disposable resuscitators reduce cost by avoiding (1) disassembly and washing, (2) sterilizing and reassembly, (3) the need for spare parts, and (4) in-process inventory. The purchase price of a disposable resuscitator is approximately one sixth the cost of similar permanent resuscitators. The lower cost of disposable units allows them to be placed in areas

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Dr Barnes is Director of Clinical Education and Associate Professor of Respiratory Therapy, College of Pharmacy and Allied Health Professions, Northeastern University; and Mr McGarry is Clinical Instructor of Respiratory Therapy, College of Pharmacy and Allied Health Professions, Northeastern University, and Respiratory Therapy Supervisor, Beth Israel Hospital—Boston, Massachusetts.

This study was completed at Northeastern University, and neither of the authors has a financial interest in any of the products tested. Dr Barnes and Mr McGarry presented some of the material in this paper during the Respiratory Care Open Forum at the 35th Annual Convention of the American Association for Respiratory Care, December 2-5, 1989, in Anaheim, California.

*Suppliers are identified in the Product Sources section at the end of the text.
of the hospital, such as clinics, where the cost of permanent resuscitators has been prohibitive.

The number of disposable resuscitators has increased steadily—raising hard questions about safety and performance among those available. Thus, we followed work already reported on the performance of BagEasy, 1st Response, LSP Bag Mask, Pulmanex, and Stat Blue, with an evaluation of 4 new adult disposable resuscitators: SPUR, Hospitak MPR, Code Blue, and CPR Bag. We also re-evaluated the Pulmanex and 1st Response to assess their performance with tube-type oxygen reservoirs. Three adult resuscitators had bag-type oxygen reservoirs (Fig. 1) and three had tube reservoirs (Fig. 2). Four pediatric disposable resuscitators were included in the study—1st Response, LSP Bag Mask, CPR Bag, and Hospitak MPR (Figs. 3 & 4).

Materials and Methods

For all the resuscitators, we investigated: (1) $F_{DO_2}$ with oxygen reservoir attached, (2) $F_{DO_2}$ without oxygen reservoir attached, (3) valve function in the presence of simulated vomitus, (4) valve function during high flow (30 L/min), (5) backward leakage of exhaled gas, (6) tolerance of mechanical shock

Fig. 1. Three of the six adult disposable manual resuscitators tested. A—Hospitak MPR, B—CPR Bag, C—SPUR. (See Fig. 2 for the other adult resuscitators tested.)

Fig. 2. Three of the six adult disposable manual resuscitators tested. A—Code Blue, B—Pulmanex, C—1st Response. (See Fig. 1 for the other adult resuscitators tested.)
Tek VT-1 Ventilator Tester. A sampling probe from a Beckman OM-15 polarographic oxygen analyzer was connected to the gas port of the VT-1 to measure $F_{DO_2}$. A 3-point calibration of the oxygen monitor was performed immediately prior to testing each resuscitator, with test gases having oxygen concentrations of 0.21, 0.80, and 1.00 to assure linearity across the entire scale.

For tests of adult resuscitators the VT-1 was set at a compliance of 0.02 L/cm H$_2$O [0.20 L/kPa] and a resistance of 20 cm H$_2$O $\cdot$ s $\cdot$ L$^{-1}$ [2.0 kPa $\cdot$ s $\cdot$ L$^{-1}$]. Pediatric resuscitators were tested with the VT-1 set at a compliance of 0.01 L/cm H$_2$O [0.10 L/kPa] and a resistance of 20 cm H$_2$O $\cdot$ s $\cdot$ L$^{-1}$ [2.0 kPa $\cdot$ s $\cdot$ L$^{-1}$]. The precision of the VT-1 display of tidal volume ($V_T$) was verified with a calibrated super syringe, and the precision of the display of ventilatory rate ($f$) was verified with a chronometer. A Wright Model L-D panel-mounted respirometer was placed between the VT-1 and the resuscitator to provide an approximate indication of the delivered $V_T$, which was monitored breath-by-breath by the VT-1 status display. The primary control of the ventilation pattern was the VT-1 display, which signalled the $V_T$, $f$, minute volume ($V_F$), and I:E. We ventilated the VT-1 by squeezing the resuscitator bag while observing the $V_T$ displayed by the Wright respirometer and using the chronometer to determine $f$. When the desired $V_T$ was reached, the bag was released and allowed to fill without restriction. Immediately after the bag had been released, the VT-1 status display was checked to verify that the $V_T$, $f$, and $V_F$ were correct. We were able to control the ventilatory

(wet and dry), (7) tidal volume capability, and (8) cycle-rate capability. The pressure-limiting systems of four pediatric resuscitators were also evaluated.

$F_{DO_2}$

We evaluated the performance of each resuscitator by means of the test apparatus shown in Figure 5. A pressure-compensated Thorpe-tube flowmeter supplied oxygen to the resuscitator. The oxygen flowrate was verified before and after each test run with a Timeter Model RT-200 Calibration Analyzer. The resuscitator was manipulated to ventilate a Bio-
pattern by making small adjustments in the $V_T$ and $f$ based on immediate feedback from the VT-1 display.

Each adult resuscitator was tested for $F_{DO_2}$ at a $V_E$ of 7.2 L/min (600 mL $\times$ 12) with oxygen flow to the unit of 15 L/min. The 4 pediatric resuscitators were tested for $F_{DO_2}$ at a $V_E$ of 6 L/min (300 mL $\times$ 20) with oxygen flow to the unit of 15 L/min. For each run, the VT-1 was ventilated until the $F_{DO_2}$ was constant (3 to 4 min). Five runs were made for each resuscitator using Ventilation Pattern 1 for adult units and Ventilation Pattern 3 for pediatric units (Table 1).

Valve Performance

High Supplemental Flows. Oxygen flow to the resuscitator was set at 30 L/min and the VT-1 was ventilated with Patterns 1 and 2 for adult resuscitators, and Pattern 3 for pediatric units to verify that the patient valve did not leak at high flow. The flow of 30 L/min was verified with a Timeter Model RT-200 Calibration Analyzer.

Patient-Valve Backward Leakage. The potential for rebreathing was tested by connecting the resuscitator (without attachments or oxygen flow) to a 2-L anesthetic bag supplied with an oxygen flow of 15 L/min. The resuscitator was cycled at f 30/min for 3 min, and then the $F_O_2$ in the resuscitator bag was measured with a Beckman OM-15 Oxygen Monitor. The ASTM backward leak requirement limits the increase of $F_O_2$ in the resuscitator bag to less than 10 percentage points (ie, to $F_O_2$ < 0.31).

Valve Function after Contamination by Vomitus. To evaluate this requirement, we poured 175 mL of simulated vomitus into the patient-connection port while cycling the resuscitator at f 12/min for 30 s. Vomitus was simulated by a mixture of two parts of baby food (Gerber Toddler Meal: Beef with Vegetables) and one part water. The patient valve was cleared of vomitus by squeezing the bag briskly and shaking any remaining obstructing material out of the exhalation port and patient-connection port. Immediately following removal of the vomitus from the patient-valve assembly, performance was assessed by using the resuscitator to ventilate the VT-1 with Patterns 1 and 2 for adult units and Pattern 3 for pediatric units.

Ventilation

Cycle Rate and Tidal Volume. These requirements were tested using Ventilation Pattern 2 for adult resuscitators and Ventilation Pattern 3 for pediatric units, and an oxygen flow of 15 L/min (Table 1). Each resuscitator was evaluated against ASTM specifications for tidal volume and cycle rate by ventilating the VT-1 for 4 min—Configuration 1 for adult units and Configuration 2 for pediatric units (Table 2).

### Table 1. Ventilation Patterns Used To Test Resuscitators

<table>
<thead>
<tr>
<th>Ventilation Pattern</th>
<th>Tidal Volume (mL)</th>
<th>Ventilatory Rate (cycles/min)</th>
<th>Minute Volume (L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>600</td>
<td>12</td>
<td>7.2</td>
</tr>
<tr>
<td>2</td>
<td>600</td>
<td>20</td>
<td>12.0</td>
</tr>
<tr>
<td>3</td>
<td>300</td>
<td>20</td>
<td>6.0</td>
</tr>
</tbody>
</table>

The mean and standard deviation (SD) were calculated for the $F_{DO_2}$ of each resuscitator at 15 L/min, both with and without oxygen reservoirs. The effect of resuscitator design on $F_{DO_2}$ was evaluated by one-way analysis of variance. A t test was used to evaluate the $F_{DO_2}$ difference among resuscitators; $p < 0.05$ was considered statistically significant. All statistical tests were performed with Exstatix version 1.0.1 software.

### Table 2. VT-1 Configuration Used To Test Resuscitators

<table>
<thead>
<tr>
<th>Configuration Pattern</th>
<th>Compliance $^*$</th>
<th>Resistance $^+$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.020 [0.20]</td>
<td>20 [2]</td>
</tr>
<tr>
<td>2</td>
<td>0.010 [0.10]</td>
<td>20 [2]</td>
</tr>
</tbody>
</table>

$^* = L/cm H_2O [L/kPa]$.

$^+ = cm H_2O \cdot s \cdot L^{-1} [kPa \cdot s \cdot L^{-1}]$. 
Table 3. Fractional Oxygen Concentration Delivered by Six Adult and Four Pediatric Disposable Resuscitators

<table>
<thead>
<tr>
<th>Type</th>
<th>With Reservoir</th>
<th>Without Reservoir</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adult*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SPUR†</td>
<td>0.98 ± 0.02</td>
<td>—</td>
</tr>
<tr>
<td>Code Blue</td>
<td>0.99 ± 0.01</td>
<td>0.40 ± 0.02</td>
</tr>
<tr>
<td>CPR Bag</td>
<td>0.95 ± 0.02</td>
<td>0.43 ± 0.02</td>
</tr>
<tr>
<td>1st Response</td>
<td>0.97 ± 0.01</td>
<td>0.40 ± 0.01</td>
</tr>
<tr>
<td>MPR</td>
<td>0.87 ± 0.01</td>
<td>0.48 ± 0.01</td>
</tr>
<tr>
<td>Pulmanex‡</td>
<td>0.99 ± 0.01</td>
<td>0.45 ± 0.01</td>
</tr>
<tr>
<td>Pediatric§</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1st Response</td>
<td>0.99 ± 0.02</td>
<td>0.43 ± 0.01</td>
</tr>
<tr>
<td>LSP Bag Mask</td>
<td>1.00 ± 0.01</td>
<td>0.38 ± 0.02</td>
</tr>
<tr>
<td>MPR</td>
<td>1.00 ± 0.01</td>
<td>0.48 ± 0.01</td>
</tr>
<tr>
<td>CPR Bag</td>
<td>0.98 ± 0.01</td>
<td>0.43 ± 0.01</td>
</tr>
</tbody>
</table>

*Midal volume 600 mL, cycle rate 12/min. Compliance 0.020 L/cm H2O [0.20 L/kPa]. Resistance 20 cm H2O s · L−1 [2.0 kPa s · L−1]. Oxygen flow 15 L/min.

†The Ambu SPUR reservoir is permanently attached.

‡The Pulmanex was tested with 3 sections of the tube-type reservoir extended.

§Tidal volume 300 mL, cycle rate 20/min. Compliance 0.010 L/cm H2O [0.10 L/kPa]. Resistance 20 cm H2O s · L−1 [2.0 kPa s · L−1]. Oxygen flow 15 L/min.

Mechanical Shock (Drop Test)

Each resuscitator was dropped five times from a height of 1 meter onto a concrete floor. The unit was dropped in a worst-case mode so that it landed on the patient-valve and gas-intake valve assemblies. Following the shock test, we inspected the resuscitator for damage and checked its performance by using it to ventilate the VT-1 with Configuration 1, Ventilation Patterns 1 and 2 for adult units; and Configuration 2, Ventilation Pattern 3 for pediatric units (Tables 1 & 2).

Results

Table 3 lists the mean and SD FDO2 values for the 10 resuscitators we studied. Tables 4 and 5 list the resuscitators' pass/fail results for the eight ASTM requirements for resuscitator performance and safety. We found that resuscitator design significantly affects FDO2 (p < 0.001 adult units, p < 0.01 pediatric units).

Code Blue

The Code Blue adult resuscitator with tube reservoir passed all eight ASTM standards. The problem with backward leakage into the bag observed with the earlier Stat Blue resuscitator was solved by using the common “duck bill” type nonrebreathing valve with the Code Blue.

Table 4. Performance of Six Adult Disposable Resuscitators against Eight Requirements of ASTM Standard F-920

<table>
<thead>
<tr>
<th>Resuscitator Tested</th>
<th>FDO2</th>
<th>Valve Function</th>
<th>Mechanical Shock</th>
<th>Ventilation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>With Reservoir</td>
<td>Without Reservoir</td>
<td>With Vomitus</td>
<td>With Flow of 30 L/min</td>
</tr>
<tr>
<td>Code Blue</td>
<td>•</td>
<td>•</td>
<td>•</td>
<td>•</td>
</tr>
<tr>
<td>1st Response</td>
<td>•</td>
<td>•</td>
<td>•</td>
<td>•</td>
</tr>
<tr>
<td>Pulmanex</td>
<td>•</td>
<td>•</td>
<td>•</td>
<td>•</td>
</tr>
<tr>
<td>SPUR</td>
<td>•</td>
<td>—</td>
<td>•</td>
<td>•</td>
</tr>
<tr>
<td>MPR</td>
<td>•</td>
<td>•</td>
<td>•</td>
<td>•</td>
</tr>
<tr>
<td>CPR Bag</td>
<td>•</td>
<td>•</td>
<td>•</td>
<td>•</td>
</tr>
</tbody>
</table>

• = Resuscitator met or exceeded ASTM standard.
O = Resuscitator did not meet ASTM standard.
Table 5. Performance of Four Pediatric Disposable Resuscitators against Eight Requirements of ASTM Standard F-920

<table>
<thead>
<tr>
<th>Resuscitator Tested</th>
<th>F_{DO2}</th>
<th>Valve Function</th>
<th>Mechanical Shock</th>
<th>Ventilation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>With Reservoir</td>
<td>With Vomitus</td>
<td>Flow of 30 L/min</td>
<td>Backward Leakage</td>
</tr>
<tr>
<td>1st Response</td>
<td>●</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>LSP Bag Mask</td>
<td>●</td>
<td>○</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>MPR</td>
<td>●</td>
<td>○</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>CPR Bag</td>
<td>●</td>
<td>●</td>
<td>●</td>
<td>●</td>
</tr>
</tbody>
</table>

● = Resuscitator met or exceeded ASTM standard.
○ = Resuscitator did not meet ASTM standard.
* = Pressure limit standard = 40 ± 10 cm H₂O.

**Pulmanex**

The Pulmanex adult resuscitator with tube-type oxygen reservoir (3 sections extended) passed all eight ASTM standards. The tube reservoir when fully extended provided a high F_{DO2} of 0.99 at an oxygen flow of 15 L/min.

**SPUR**

When tested in the Spring of 1989, the SPUR adult resuscitator passed all eight ASTM standards when dry; however, it failed the shock test when wet because the nonrebreathing valve was pushed into the bag when dropped from a height of 1 meter. The flange that holds the bag to the nonrebreathing valve was redesigned in the Fall of 1989 and the SPUR, wet or dry, now passes the shock test.

**1st Response**

The 1st Response adult resuscitator passed all eight ASTM performance standards. The resuscitator was tested using the tube reservoir supplied with the unit. The 1st Response pediatric resuscitator failed the pressure limit standard (40 ± 10 cm H₂O [4.0 ± 1.0 kPa]) because it allowed pressure to reach 57 cm H₂O [5.6 kPa] with Ventilation Pattern 3 (Table 1). The 1st Response pediatric resuscitator passed the other seven ASTM standards.

**Hospitak MPR**

The Hospitak MPR adult resuscitator with oxygen reservoir attached delivered a mean F_{DO2} of 0.87 at an oxygen flow of 15 L/min. The F_{DO2} in this configuration barely passed the ASTM minimum requirement of 0.85 and was significantly lower than the F_{DO2} in the other five adult resuscitators tested (p < 0.001). The Hospitak MPR adult resuscitator failed the shock test because the exhalation port was broken into several pieces when dropped from a height of 1 meter. The MPR adult model passed the other six ASTM performance standards. The MPR pediatric model failed the shock test because the exhalation port was broken into several pieces and the donut-shaped gas-intake valve fell into the bag when the resuscitator was dropped from 1 meter. The MPR pediatric resuscitator failed the pressure-limit standard because it had no pressure-release mechanism. The peak pressure observed with Ventilation Pattern 3 was 71 cm H₂O [7.0 kPa]. The Hospitak MPR pediatric resuscitator passed the other six ASTM performance standards.

**CPR Bag**

The CPR Bag adult and pediatric resuscitators passed seven of eight ASTM standards. They failed the shock test because the female coupling that attaches the oxygen reservoir to the gas-intake valve cracked and the reservoir no longer remained attached to the bag. Also, the protective covers for the auxiliary air-intake and oxygen-release valves flew off when the unit was dropped from 1 meter. Finally, the retainer ring that holds the donut-shaped valve in place in the nonrebreathing valve assembly broke...
away when the resuscitator was dropped. The override switch on the pressure-release valve of the pediatric model broke off when the resuscitator was dropped from 1 meter.

LSP Bag Mask

The LSP Bag Mask pediatric resuscitator passed seven of the eight ASTM standards tested. It failed the pressure-limit standard because the resuscitator had no provision for limiting pressure. The peak pressure observed with Ventilation Pattern 3 was 61 cm H₂O [6.0 kPa].

Discussion

There were clinically important differences in performance and safety among the resuscitators tested. Two adult resuscitators failed ASTM requirements for shock tolerance and one failed the FDO₂ requirement without a reservoir. Three of the pediatric resuscitators failed the pressure-limit requirement and two failed the shock test.

Shock Test

Shock tolerance is an important requirement for resuscitators because the time lost to repair or replace a disabled resuscitator may be critical during emergency ventilation. The most likely accident would be dropping the unit onto a hard floor during a resuscitation or transport. Shock resistance is evaluated based on the premise that a resuscitator should still be functional after being dropped from 1 meter, which is the height of an average hospital bed.¹ Previously tested resuscitators have been reported to have clinically important problems with mechanical shock resistance.² When we dropped the Hospitak MPR adult or pediatric resuscitator in such a way that the patient valve assembly struck the floor first, the exhalation port shattered leaving a jagged orifice unsuitable for attachment of a PEEP valve. The sudden loss of PEEP during transport of a severely hypoxemic patient could cause a serious medical emergency if the resuscitator could not be replaced quickly. When the MPR pediatric unit was dropped the leaf of the gas intake valve fell into the bag and could not be reseated because the gas-intake and nonrebreathing valve assemblies are permanently sealed to the bag. Squeezing the bag with the leaf of the gas-intake valve missing resulted in no tidal volume being delivered to the VT-1. Hospitak improved the valve seat in October 1989, and the leaf remained in place when the new unit was retested.

The CPR Bag failed the shock test because the female connector of the oxygen reservoir cracked when the unit was dropped. The cracked connector prevented the reservoir from being attached to the male fitting of the gas-intake valve assembly. The plastic material used to mold the patient-valve and oxygen-reservoir assemblies would not tolerate a drop from 1 meter as evidenced by several components cracking or falling apart. The pediatric CPR Bag failed the shock test because of fractured oxygen-reservoir components and the complete destruction of the pressure-release valve and override switch, which broke apart when the resuscitator was dropped. Mercury Medical Corporation is studying the use of a more shock-resistant plastic for the CPR Bag.

The first time it was tested, the SPUR failed the shock test because the patient-valve assembly, when wet, was pushed into the bag when the bag was dropped from 1 meter. In September of 1989, Ambu redesigned the SPUR’s flange that attaches the patient valve to the bag, and when the unit was retested it passed the shock test.

FDO₂

Delivery of a minimum of 85% oxygen is often necessary for treatment of severely hypoxemic patients during resuscitations. This concentration should be obtained with oxygen flows ≤ 15 L/min, because to specify flows > 15 L/min would exceed the normal calibration of standard adult flowmeters and could potentially lead to extremely high input flows and valve lockup.⁵ Previous studies have reported that interaction between resuscitator design and oxygen flowrate significantly affected FDO₂, and that each factor independently affected FDO₂.⁶⁻¹²

The Hospitak MPR adult resuscitator with oxygen reservoir attached passed the FDO₂ test, but its FDO₂ was significantly lower (p < 0.001) than the other nine resuscitators (Table 3). The FDO₂ of 0.87 vs 1.00 may be clinically important when patients with severe hypoxemia are ventilated. The volume of the MPR oxygen reservoir was only 1,900 mL and the
reservoir was constructed of material that is stiffer than most oxygen reservoirs we have tested. A recent study reported that resuscitators with folding bag reservoirs with volume > 3,300 mL and manufactured of compliant material had an $F_{DO_2} > 0.97$; it was also reported that a bag reservoir with a volume of 1,850 mL and made of stiff material resulted in lowering the Pulmanex's $F_{DO_2}$ to < 0.85 at O$_2$ flow of 15 L/min. Replacing the stiff, small bag-type reservoir of the Pulmanex with Life Design System's tube-type reservoir resulted in an $F_{DO_2}$ of 0.99 (Table 3). The use of a tube reservoir or a larger, softer bag reservoir would probably also improve the $F_{DO_2}$ capability of the MPR.

Valve Performance

All 10 resuscitators passed the three valve performance requirements tested. During resuscitation, time is extremely important and a device requiring longer than 20 s to be sufficiently cleared of vomitus to continue functioning would be inappropriate for emergency use. Valve malfunction at high supplemental gas flows may lead to excessive airway pressures. Thus, resuscitators should be capable of functioning normally at flows of 30 L/min because the adjustment between 15 L/min and the 30 L/min portion of the flood setting is small.

Patient-valve backward leakage may cause rebreathing of exhaled gases that contain increased carbon dioxide. Exposure to even low levels of inspired CO$_2$ may lead to increased $P_{aCO_2}$ and to decreased arterial pH. Tidal volumes of 600 mL for adults and 300 mL for children are typically the maximum that can be delivered by a one-handed use of a manual resuscitator at the compliances and resistances found in diseased lungs. The requirements for frequency of ventilation represent the upper limits that are likely to be used with the tidal volumes specified in the F-920 standard.

Pressure Limit

The peak inspiratory pressure-limit requirement for the child resuscitators tested was 40 cm H$_2$O [3.9 kPa]. This level of pressure will probably not produce lung damage, but will permit adequate tidal volume delivery in most patients with diseased lungs. Three of the four pediatric resuscitators tested failed the ASTM F-920 pressure-limit requirement intended to reduce the incidence of barotrauma. The LSP Bag Mask and Hospitak MPR had no release valve, and 1st Response released pressure at a level higher than the requirement of 40 ± 10 cm H$_2$O [3.9 ± 1.0 kPa]. The pressure limit should not be exceeded under the conditions of Ventilation Pattern 3, and Test Lung Configuration 2 (Tables 1 & 2). However, an override mechanism may be provided if its operating mode (on or off) is readily apparent. The pressure-limit valves of the CPR Bag and the 1st Response both had an override mechanism.

Conclusions

Of the six adult resuscitators tested, only the Code Blue, 1st Response, Pulmanex (with tube-type reservoir), and SPUR met the ASTM standard for operator-powered adult resuscitators (Table 4). All of the pediatric resuscitators tested failed to meet one or more of the ASTM standards (Table 5) and cannot be recommended as replacements for permanent resuscitators. We recommend that practitioners use disposable resuscitators that meet the ASTM F-920 standard. Also, respiratory care practitioners should be aware that differences in design may affect the performance and safety of disposable manual resuscitators.

ACKNOWLEDGMENTS

We thank Ambu Inc, Hospitak Inc, Intertech Resources Inc, Life Design Systems Inc, Life Support Products Inc, Mercury Medical Inc, and Vital Signs Inc for donating samples of their products for use in this study.

PRODUCT SOURCES

Disposable manual resuscitators:
(Note: These are sold 6 per case; prices quoted are suppliers' list prices per unit as of October 1989.)

- Code Blue, Vital Signs Inc, Totowa NJ, Adult: $19.85
- CPR Bag, Mercury Medical Inc, St Petersburg FL, Adult: $22.72, Pediatric: $24.90
- 1st Response, Intertech Resources Inc, Bannockburn IL, Adult: $23.40, Pediatric: $26.00
- Hospitak MPR, Hospitak Inc, Lindenhurst NY, Adult: $17.00, Pediatric: $18.00
LSP Bag Mask, Life Support Products Inc, Irvine CA, Pediatric: $24.75
Pulmancx, Life Design Systems Inc, Carrollton TX, Adult: $19.20
SPUR, Ambu Inc, Hanover MD, Adult: $20.95

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

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

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

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

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

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

REFERENCES
Comparing RCPs to Physicians for the Description of Lung Sounds: Are We Accurate and Can We Communicate?

Robert L Wilkins MA RRT and James R Dexter MD

Precise communication among clinicians of chest-auscultation findings depends on use of standardized nomenclature for lung sounds. To identify the current practice of clinicians in describing lung sounds, we surveyed physicians and respiratory care practitioners (RCPs). MATERIALS AND METHOD: Surveys were specifically designed to identify: (1) whether RCPs and physicians use similar terms to describe adventitious lung sounds (ALS); (2) whether changes are occurring in response to the recommendations of the ATS-ACCP Ad Hoc Subcommittee on Pulmonary Nomenclature, and (3) whether RCPs and physicians differ in their ability to accurately recognize ALS. We surveyed 156 RCPs at the 1987 Annual Meeting of the American Association for Respiratory Care and 223 pulmonary physicians (PPs) and 54 nonpulmonary physicians (NPPs) at the 1988 Annual Meeting of the American College of Chest Physicians. Each survey participant was required to listen to five examples of ALS using earphones and an audiostream player and then to write ‘free-form’ descriptions of what they heard. (All participants listened to the same ALS.) RESULTS: Fine crackles and high-pitched monophonic and polyphonic wheezes were readily recognized by the majority of RCPs and physicians. Fine crackles were described as rales or crackles; high-pitched, monophonic wheezes were described as stridor or wheezes; however, high-pitched, polyphonic wheezes were usually described as wheezes. RCPs and physicians used a variety of terms to describe coarse crackles and rhonchi. The term rhonchi was frequently used inappropriately by all groups surveyed. There were no significant differences between PPs and RCPs in their ability to accurately recognize adventitious lung sounds; however, PPs were superior to NPPs (p < 0.05) in this regard. PPs were superior to RCPs and NPPs (p < 0.05) in appropriately using the term ‘fine’ for the description of crackles. CONCLUSION: All three groups of clinicians need to improve their ability to recognize and describe lung sounds. (Respir Care 1990;35:969-976.)

Mr Wilkins is Associate Professor and Program Director, Associate Degree Program, School of Allied Health Professions, Department of Respiratory Therapy; and Dr Dexter is Associate Professor, School of Medicine—Loma Linda University, Loma Linda, California.

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

Reprints: Robert L Wilkins, Room 1926 Nichol Hall, Loma Linda University, Loma Linda CA 92350.
DESCRIPTION OF LUNG SOUNDS

**Abbreviations Used in this Paper**

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>AARC</td>
<td>American Association for Respiratory Care</td>
</tr>
<tr>
<td>ALS</td>
<td>Adventitious lung sounds</td>
</tr>
<tr>
<td>ACCP</td>
<td>American College of Chest Physicians</td>
</tr>
<tr>
<td>ATS</td>
<td>American Thoracic Society</td>
</tr>
<tr>
<td>NPP</td>
<td>Nonpulmonary physician</td>
</tr>
<tr>
<td>PP</td>
<td>Pulmonary physician</td>
</tr>
<tr>
<td>RCP</td>
<td>Respiratory care practitioner</td>
</tr>
</tbody>
</table>

**Introduction**

The ability to accurately communicate the findings of chest auscultation requires competency in recognizing lung sounds and use of a standardized nomenclature. In an effort to standardize lung sound terminology, the American College of Chest Physicians (ACCP) and the American Thoracic Society (ATS) established an Ad Hoc Subcommittee on Pulmonary Nomenclature. This committee initially published its recommendations in *Chest* in 1975 and later with modifications in a 1977 ATS newsletter (Table 1). We believe that the relevance of future recommendations can be increased if knowledge is available on the current application of terms used to describe lung sounds.

Surveys of the terminology used to describe lung sounds in published case studies have been reported. While the latter of these investigations found the term crackles to be increasing in popularity, it is not clear whether this change reflects the preference of clinicians or journal editors. To identify the current practice of clinicians in describing lung sounds, we surveyed physicians and respiratory care practitioners (RCPs) at national meetings. These surveys were specifically designed to identify: (1) whether RCPs and physicians use similar terms to describe adventitious lung sounds (ALS), (2) whether changes are occurring in response to the recommendations of the ATS-ACCP Ad Hoc Subcommittee on Pulmonary Nomenclature, and (3) whether RCPs and physicians differ in their ability to accurately recognize ALS.

Our initial report published in this journal identified the terms RCPs prefer to use for describing ALS. Now that we have completed a survey of physicians, we are reporting a comparison between pulmonary physicians (PPs), nonpulmonary physicians (NPPs) (those with a specialty other than pulmonary), and RCPs.

**Materials and Method**

The survey of RCPs was conducted at the 1987 Annual Meeting of the American Association for Respiratory Care (AARC) in Las Vegas, Nevada; and the survey of physicians was conducted at the 1988 Annual Meeting of the American College of Chest Physicians (ACCP) in Anaheim, California. Participants in the survey completed a background data sheet to identify items such as age, region of practice, and primary job responsibilities, and then listened to five examples of ALS using earphones and an audiocassette player. (The same ALS were used for both surveys.) While listening to the ALS the participants wrote ‘free-form’ descriptions of what they heard. Prior to the survey, the recorded ALS were analyzed with the aid of two- and three-dimensional waveforms to identify the characteristics of each sound (Fig. 1). Interpretations of the waveforms are presented in Table 2. Chi-square analysis was used to identify the significant differences between the physicians and RCPs in their descriptions of the sound samples and to identify the influence of demographics on the terms selected.

**Results**

Approximately 90% of the physicians and RCPs used the terms crackles, rales, or both to describe Sound 1. While RCPs and NPPs preferred the term
DESCRIPTION OF LUNG SOUNDS

Sound 1

Sound 2

Fig. 1. Waveform analysis for the five lung sound samples used in the survey. (See Table 2 for interpretation of the tracings.) Time is represented on the horizontal axis and intensity on the vertical axis. "I" denotes the beginning of inspiration and "E" denotes the beginning of expiration. Each line on the horizontal axis represents 0.34 s, and the entire sound sample is approximately 4 s.

Sound 3

Sound 4

Sound 5

Wheeze/Stridor

rales over crackles, PPs used the two terms in approximately equal proportion (Fig. 2). For Sound 2, RCPs and NPPs preferred the term rhonchi over the terms rales or crackles, but PPs used all three terms in approximately equal proportion (Fig. 3). The term rhonchi was especially popular among RCPs for the description of Sound 2. For Sound 3, at least 70% of the participants in each group used the term wheeze in their description. RCPs and PPs were more precise in their description of this sound than were NPPs in that they more often noted the inspiratory crackles that preceded the loud polyphonic expiratory wheeze (Fig. 4). Only 30% of the participants in each group accurately described Sound 4 with the term rhonchi. Other terms that were used to describe this sound included wheeze, rub, bronchial breath sounds, and stridor (Fig. 5). Sound 5 was accurately described with the term stridor by approximately 50% of RCPs and PPs but only 37% of NPPs. The majority of those in each group who did not use the term stridor used the term wheeze to describe Sound 5 (Fig. 6).

Because it has been suggested by the Subcommittee on Pulmonary Nomenclature and other experts that the terms 'fine' and 'coarse' be used to qualify crackles,
Table 2. Descriptions of the Five Adventitious Lung Sounds

<table>
<thead>
<tr>
<th>Sound No.</th>
<th>Description</th>
<th>Pathophysiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Pan-inspiratory fine crackles</td>
<td>Diffuse pulmonary fibrosis</td>
</tr>
<tr>
<td>2</td>
<td>Coarse inspiratory &amp; expiratory crackles</td>
<td>Excessive airway secretion from severe pneumonia</td>
</tr>
<tr>
<td>3</td>
<td>Fine inspiratory crackles with severe expiratory polyphonic wheezes</td>
<td>Diffuse airway obstruction from acute asthma</td>
</tr>
<tr>
<td>4</td>
<td>expiratory rhonchi or low-pitched wheezes</td>
<td>Single bronchus obstruction from airway tumor</td>
</tr>
<tr>
<td>5</td>
<td>Inspiratory stridor and expiratory monophonic wheeze</td>
<td>Partial laryngeal obstruction post-extubation</td>
</tr>
</tbody>
</table>

we identified the percentage of participants in each group who appropriately used these adjectives for Sounds 1 and 2. We found that approximately 1% of RCPs, 4% of NPPs, and 11% of PPs appropriately used the term fine as part of their description for Sound 1 (Fig. 7). For Sound 2, 16% of RCPs, 17% of NPPs, and 20% of PPs appropriately used the term coarse in their description of the sound. We did not analyze the qualifying adjectives used to describe the

Fig. 2. Comparison between RCPs and physicians for the terms used to describe Sound 1, fine crackles—respiratory care practitioners , pulmonary physicians , nonpulmonary physicians .

continuous ALS because no generally accepted recommendations exist for these sounds.

Table 3 identifies the number of correct responses for each group and each sound sample. We considered the following responses to be correct: Sound 1, “rales” or “crackles”; Sound 2, “rales,” “crackles,” “rales and rhonchi,” or “crackles and rhonchi” (see discussion); Sound 3, “rales and wheezes” or “crackles and wheezes”; Sound 4, “rhonchi” or “wheeze”; and Sound 5, “stridor” or “wheeze.” The table shows

RESPIRATORY CARE • OCTOBER '90 Vol 35 No 10
that Sounds 1, 3, and 5 were correctly identified by the large majority of physicians and RCPs, but Sounds 2 and 4 were correctly described by only about 50% of the participants. It should be mentioned that the polyphonic wheeze portion of Sound 3 was accurately described by 76% of all participants; however, only 34% reported hearing the inspiratory crackles as well as the wheeze when describing this sound sample. PPs were slightly superior to RCPs and significantly superior to NPPs (p < 0.05) in the ability to accurately identify the sounds. RCPs were slightly more accurate than NPPs, but the difference is not significant.

Analysis of the demographic data showed that 55% of RCPs and all but two of the physicians participated in patient care at the time of the survey. We found no significant differences between the RCPs currently participating in patient care and those whose job descriptions do not directly involve them in patient care. The influence of other demographic data on the terms selected has been detailed in our previous reports.5,6

Table 3. Percentage of Participants in Each Group Using Correct Answers To Describe the Five Adventitious Lung Sounds

<table>
<thead>
<tr>
<th></th>
<th>RCPs</th>
<th>PPs</th>
<th>NPPs</th>
<th>Total*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sound 1</td>
<td>88%</td>
<td>91%</td>
<td>89%</td>
<td>90%</td>
</tr>
<tr>
<td>Sound 2</td>
<td>42%</td>
<td>55%</td>
<td>44%</td>
<td>49%</td>
</tr>
<tr>
<td>Sound 3</td>
<td>33%/76%†</td>
<td>38%/77%†</td>
<td>19%/72%†</td>
<td>34%/76%†</td>
</tr>
<tr>
<td>Sound 4</td>
<td>46%</td>
<td>43%</td>
<td>41%</td>
<td>44%</td>
</tr>
<tr>
<td>Sound 5</td>
<td>86%</td>
<td>81%</td>
<td>80%</td>
<td>82%</td>
</tr>
<tr>
<td>Total†</td>
<td>59%</td>
<td>61%</td>
<td>54%</td>
<td></td>
</tr>
</tbody>
</table>

RCPs = 156 respiratory care practitioners; PPs = 223 pulmonary physicians; and NPPs = 54 nonpulmonary physicians.

*These totals represent the percentage of correct answers for all participants.
†Represents the percentage of those who at least used the term wheeze for Sound 3.
‡These totals represent the percentage of correct answers for each group.
Discussion

Our survey results suggest that fine crackles are readily recognized as discontinuous ALS by the majority of RCPs and physicians. Although this sound is not universally described by a single term, most (90%) use either rales or crackles to describe it. Because the terms rales and crackles were used only to describe discontinuous ALS, we feel justified in stating that RCPs and physicians easily recognize fine crackles as discontinuous ALS.

While it is clear that the terms rales and crackles are synonyms, it is not clear which term should be promoted for the description of discontinuous ALS. Even though the term crackles has excellent onomatopoeia, some clinicians feel that it should be reserved for describing the sound made by their breakfast cereal as milk is added and therefore consider the term unprofessional. This bias may partially explain the lack of uniform acceptance of the term crackles more than 10 years after its endorsement by the Subcommittee on Pulmonary Nomenclature.

The term rales was introduced by Laennec, the French physician who invented the stethoscope. He used the term rales with a rather elaborate list of qualifying adjectives to describe all adventitious lung sounds. Laennec chose the term rales because it is the French word for the rattling sounds he frequently heard while auscultating patients with his "cylinder." Because Laennec wrote all his case notes in Latin, he used the term rhonchi—the Latin equivalent of rales—in many of his writings. When his writings were later translated, the terms rales and rhonchi were mistakenly interpreted to mean two different types of sounds: rales for the discontinuous and rhonchi for the continuous type. The term wheeze was introduced when Laennec's writings were translated into English because it is the Anglo-Saxon equivalent for rales.

Later, in England, when the translation error was identified, Robertson suggested "crackling sounds" for discontinuous ALS and "wheeze" for continuous ALS. He thought it would be better to introduce a new term (crackles) rather than try to revise the definitions of old terms (rales and rhonchi). In this country, the Subcommittee initially recommended the term rales for discontinuous ALS and rhonchi for continuous ALS. In 1977, however, the Subcommittee suggested that clinicians use the term crackles for discontinuous ALS and abandon the term rales. Although our British counterparts have apparently embraced the term crackles, in this country only PPs and West Coast RCPs commonly use the term. We believe that the recommendation of the Subcommittee to use the term crackles is appropriate, but promotion of the term has been inadequate. More uniform acceptance of the term might be achieved if the recommendations were more widely publicized and if rationale for the recommendations were logically and clearly explained.

Inspiratory and expiratory coarse crackles (Sound 2) are not precisely described by clinicians. We were surprised at the number of those surveyed who used the term rhonchi alone to describe this sound. While use of the term rhonchi as part of the description for Sound 2 (ie, "coarse crackles and rhonchi") is defensible, the majority of this sound sample contained coarse discontinuous sounds, and it is not accurate to use the term rhonchi alone. It is not clear whether this inaccurate use of the term rhonchi represents clinicians' inability to recognize coarse discontinuous ALS or whether they were educated to describe all secretion sounds as rhonchi. In either case, more education is needed. Clearly, RCPs and physicians are not communicating well either among themselves or with each other regarding auscultatory findings when coarse discontinuous ALS are present in their patients.

Our surveys show that polyphonic high-pitched wheezes are well recognized and uniformly described by the term wheeze by RCPs and physicians. Communication regarding the findings of polyphonic wheezes appears to be good. However, if the expiratory wheeze is preceded by brief inspiratory crackles, most will not report hearing the crackles. We believe reporting the inspiratory crackles as well as the expiratory wheezes could be important because it may suggest that in addition to the bronchospasm other pathophysiology, such as atelectasis, may be present.

Rhonchi (low-pitched, monophonic continuous sounds) appear to be difficult to recognize as evidenced by the numerous miscellaneous terms or phrases that were used to describe this sound. We believe that most clinicians have trouble recognizing this sound because it occurs less frequently and its characteristics are less distinctive than other sounds.
such as the polyphonic wheezes of asthma. Educational efforts aimed at improving clinicians’ ability to recognize low-pitched continuous ALS might be helpful.

Rhonchi was the only term frequently used inappropriately by all groups surveyed. The term was used almost equally to describe both continuous and discontinuous ALS, and only a minority of any group used the term to describe the low-pitched wheeze (Sound 4). This suggests that the term rhonchi has little meaning and that there may be advantage to abandoning the term. Other support for abandoning the term rhonchi is found in the logic that Robertson used to suggest replacing the term rales with the term crackles. If applying new definitions to old terms is not appropriate, both of the original terms used by Laennec should be abandoned (rales and rhonchi). We believe the current confusion with the term rhonchi reflects the problems associated with giving old terms new definitions.

High-pitched, monophonic continuous ALS represent an important sound. When this sound originates from the larynx, a serious form of upper-airway obstruction may be present. In such cases, the term stridor is appropriate and suggests that careful attention to the patient’s airway is needed. Our Sound 5 was recorded from the neck of a patient with partial laryngeal obstruction. However, only about 55% of RCPs and PPs and 40% of NPPs used the term stridor in their description of this sound. While the majority of others used the term wheeze (which is technologically correct) to describe this sound, the “life-threatening” characteristics are not implied with this description.

The appropriate use of qualifying adjectives in describing ALS is another aspect that needs improvement. This is especially true for crackles in which the terms fine and coarse are very useful in suggesting the underlying pathologic changes in the lung.9,10 Table 4 contains a list of the adjectives we believe clinicians should use to describe ALS. We agree with Forgacs’s suggestion7 to use adjectives with an acoustic basis and to avoid historical terms that may be subject to a variety of translations.

We are encouraged to find that RCPs are nearly as accurate as PPs in the basic recognition of ALS. This suggests that RCPs and PPs are equally adept at using the stethoscope. It is not surprising that RCPs and PPs are superior to NPPs in the recognition of ALS because NPPs may not listen to the lung sounds of their patients or read related literature as frequently as RCPs or PPs.

Although persons attending a national meeting may not represent the typical practitioner within the profession, we feel our results accurately portray the groups surveyed. We base this on the findings that there were no differences among the participants based on job description (ie, managers vs patient care providers) and given the number of participants who indicated that they currently provided patient care. Given that this was a “self-selected” survey, there may be limitations based on a bias associated with persons accepting an invitation to participate in the survey.

In conclusion, we believe that educational efforts should be directed towards improving the ability of RCPs and physicians to recognize coarse crackles and rhonchi (low-pitched wheezes) and in the use of appropriate qualifying adjectives. It is also important for educators to collaborate with ATS-ACCP in promoting a standardized nomenclature. The availability of several teaching audiotapes and texts that appropriately use the recommended terminology should help.11,13 The ATS-ACCP Ad Hoc Subcommittee on Pulmonary Nomenclature should re-evaluate the recommendations suggested in the 1977 ATS Newsletter2 and should make their recommen-

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Table 4. Suggested Adjectives for Describing Adventitious Lung Sounds (ALS)*

<table>
<thead>
<tr>
<th>ALS</th>
<th>Adjectives</th>
<th>Identifiable Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crackles</td>
<td>Fine</td>
<td>High-pitched, shorter duration, less loud; typically heard only on inhalation</td>
</tr>
<tr>
<td></td>
<td>Coarse</td>
<td>Low-pitched, longer duration, louder; often heard on inhalation and exhalation</td>
</tr>
<tr>
<td>Wheezes</td>
<td>Monophonic</td>
<td>Single note; can be heard on inhalation or exhalation</td>
</tr>
<tr>
<td></td>
<td>Polyphonic</td>
<td>Multiple notes similar to a musical chord; most often heard on exhalation</td>
</tr>
</tbody>
</table>

*As suggested by authors of this paper.
Achieving a standardized nomenclature for lung sounds is a reasonable goal that should improve the clinical value of auscultation. In this age of soaring medical costs, optimizing the use of simple, inexpensive assessment techniques such as auscultation is important.  

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Malignant Hyperthermia:  
A Review of Current Concepts  
Narendra Vakharia BS RRT and Richard Hall MD  

Introduction  
Triggering Agents  
Clinical Signs and Symptoms  
Treatment of Acute Crisis  
Prophylaxis with Dantrolene  
Diagnosis of Susceptibility  
Genetics  
Pathophysiology  
Conclusion  

Previous uneventful exposure to an anaesthetic does not guarantee protection against the occurrence of MH during subsequent administration of anaesthetics. Respiratory therapists involved in the postoperative care of patients in the intensive care unit or employed as anaesthetic technologists may encounter patients with MH, or patients who are MH susceptible, during the course of their duties. We review advances in knowledge, recognition, and management of this syndrome.  

Triggering Agents  

Drugs known to precipitate an MH crisis are found predominantly in anaesthetic practice. Chief among these are the volatile anaesthetic agents especially halothane but including enflurane, isoflurane, methoxyflurane, diethylether, chloroform, and cyclopropane. Muscle relaxants, especially of the depolarizing type (eg, succinylcholine and decamethonium) have also been described as MH triggers (Table 1). The amide class of local anaesthetics (eg, lidocaine, bupivacaine, and mepivacaine) were originally felt to be potential triggers for MH attacks because of their ability to increase intracellular Ca++. However, recent studies have demonstrated the safety of amide local anaesthetics at concentrations currently
Table I. Agents Known or Suspected To Trigger Malignant Hyperthermia

<table>
<thead>
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<th>Known Triggers</th>
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<tbody>
<tr>
<td>Inhalation Anaesthetics</td>
</tr>
<tr>
<td>Halothane</td>
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<tr>
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<tr>
<td>Isoflurane</td>
</tr>
<tr>
<td>Cyclopropane</td>
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<tr>
<td>Methoxyflurane</td>
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<tr>
<td>Muscle Relaxants</td>
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<td>Succinylcholine</td>
</tr>
<tr>
<td>Decamethonium</td>
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<table>
<thead>
<tr>
<th>Suspected Triggers</th>
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<tbody>
<tr>
<td>Ethanol</td>
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<td>Cocaine</td>
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</table>

employed for therapeutic use. Although at least one investigator has suggested that drugs that elevate myoplasmic Ca\textsuperscript{++} concentrations (eg, cardiac glycosides) may exacerbate an active crisis or may reduce the threshold for the induction of an MH reaction, this concept has not been universally accepted. Reports of MH triggering by recreational cocaine and ethanol abuse have also been published.

Clinical Signs and Symptoms

Because of the seriousness and the rapidity with which an MH reaction may progress, early recognition of clinical signs indicative of MH is of paramount importance.

Clinical signs and symptoms have been widely reviewed in the literature. The earliest observable sign during anaesthesia is a rise in end-tidal carbon dioxide tension (P_{etCO}_2) secondary to increased metabolism and production of lactic acid and CO\textsubscript{2} (Fig. 1).

In one case, the P_{etCO}_2 increased from a baseline value of between 34-40 torr to 62 torr within minutes and reached 70 torr in 30 min despite a fourfold increase in ventilation. In another case described in the same article, P_{etCO}_2 increased from 45 torr to 80 torr in 10 min. In both cases, changes in P_{etCO}_2 preceded changes in other parameters. Dantrolene treatment was instituted based on the rising P_{etCO}_2 values and resulted in the successful resolution of the crises, with normal postoperative laboratory values. Thus, early institution of treatment improves the likelihood of a successful outcome. Causes of elevated P_{etCO}_2 other than MH, including inadequate ventilatory volumes, circuit disconnection, faulty CO\textsubscript{2} absorption due to defective soda lime, lung disease, increased dead-space-to-tidal-volume ratios, and ventilation/perfusion (V/Q) inequalities, must be kept in mind.

The availability and wider use of end-tidal CO\textsubscript{2} monitors has greatly aided in the early diagnosis of MH because a rising end-tidal CO\textsubscript{2} concentration can serve as an early sign of a developing MH reaction. In response to elevated CO\textsubscript{2} levels, tachypnea (in spontaneously breathing patients) and tachycardia develop as early signs. These signs can be misinterpreted as inadequate anaesthesia. A drop in hemoglobin oxygen saturation (S\textsubscript{aO}_2) has also been reported as occurring as much as 40 min prior to temperature elevation. Therefore, use of oximeters may be of some value; however, the differential diagnoses for oxygen desaturation must be kept in mind. Generalized muscle rigidity is often seen during an MH reaction and is associated with an 18-fold increased incidence in patients subsequently diagnosed as MH susceptible by halothane-caffeine contracture assay. Patients receiving succinylcholine may develop masster muscle rigidity with or without the administration of other MH-triggering agents. When present, masseter jaw rigidity can be a useful

\begin{itemize}
  \item Carbon dioxide production
  \item Respiratory rate
  \item Heart rate
  \item Muscle rigidity
  \item Acid-base disturbances
  \item Serum potassium and creatine phosphokinase levels
  \item Blood pressure
  \item Cyanosis
  \item Temperature
  \item Cardiac dysrhythmias
  \item Cardiac arrest
\end{itemize}

Fig. 1. Physiologic events that may occur following the onset of a malignant hyperthermic reaction. The sequence of physiologic changes is highly variable among patients and may not occur in the order depicted.
indication of MH susceptibility.\(^6,1^4\) However, muscle rigidity may or may not be present early in the course of a reaction.\(^1\)

When present, rigidity of muscles of the chest wall may occur early in the reaction\(^3,2^1\) and may be reflected as an increase in peak airway pressures and difficulty in mechanical ventilation of the patient.

Mottling of the skin and cyanosis, usually due to vascular spasm, may also be present even with an adequate \(P_{A\text{-}O_2}\).\(^5\) The alveolar-arterial oxygen gradient \(P_{A\text{-}a\text{-}O_2}\) is widened and, although administration of 100% oxygen increases arterial and venous \(P_{O_2}\), it may not relieve the cyanosis caused by vasospasm.\(^3\)

Other signs include sweating, hypertension with hypotension later, and cardiac dysrhythmias including ventricular extrasystoles (which may be multifocal), bigeminy, and ventricular tachycardia, that can progress to ventricular fibrillation.\(^1^4,1^5,2^2,2^3\) A rise in temperature, which is a dominant feature of MH, may or may not be present early in a reaction and is usually the result of biochemical disturbances occurring in muscles. Muscle rigidity may be present at some point during the course of the reaction. The degree of rigidity is usually dependent upon the triggering agents and the level of the inherited genetic defect.\(^1^9\)

Laboratory findings demonstrate disturbances in acid-base balance with metabolic and respiratory acidosis prevailing due to the elevated metabolic rate and inability to eliminate accumulating carbon dioxide. \(P_{A\text{-}CO_2}\) values > 100 torr and pH < 7.0 are not uncommon,\(^1^4\) and \(P_{A\text{-}CO_2}\) values as high as 179 torr (with pH 6.85) have been reported.\(^2^4\)

In addition, serum potassium and phosphate levels are elevated due to muscle-cell-membrane damage and breakdown of adenosine triphosphate (ATP) energy stores.

To summarize, early recognition of signs and symptoms diagnostic of MH is crucial because it allows the rapid institution of appropriate therapy and greatly enhances the chances of a successful resolution of the crisis with a minimum of deleterious sequelae. Currently this is best done by measuring changes in end-tidal \(CO_2\) concentrations and arterial blood gas values.

A condition similar to MH is the neuroleptic malignant syndrome (NMS)\(^2^5,2^6\) characterized by akinesia, hyperthermia, muscle rigidity, altered levels of consciousness, autonomic instability, and myoglobinuria. NMS is precipitated by the administration of neuroleptic drugs with symptoms manifesting within a few days to a number of weeks following drug administration.\(^2^6\) The clinical course of the condition spans 24-72 hours. NMS can also occur when neuroleptic drugs are used in combination with other drugs like lithium and tricyclic antidepressants or following cessation of dopaminergic drug therapy.\(^3,2^6\)

Both MH and NMS respond to dantrolene treatment; however, there are distinct differences in the two conditions. NMS muscle rigidity is of central nervous system origin, whereas MH muscle rigidity is of peripheral origin; and NMS reactions take place within days of drug administration, whereas MH occurs within minutes to hours of exposure.\(^3\) Thus, although NMS and MH have certain clinical features in common, the conditions may not be associated.\(^2^7\)

**Treatment of an Acute Crisis**

Once the diagnosis of MH is suspected, treatment must be instituted promptly (Table 2). First, the triggering agents must be discontinued and the patient hyperventilated with 100% oxygen with minute ventilation two to three times normal until normal \(P_{CO_2}\) levels are achieved.\(^2^8,2^9\)

Second, dantrolene sodium must be administered intravenously (initially, 2-3 mg/kg) with the dose titrated to patient response.

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**Table 2. Management of MH Reaction**

<table>
<thead>
<tr>
<th>Procedure</th>
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<tr>
<td>Terminate administration of triggering anaesthetic and hyperventilate at 2-3 times normal minute ventilation with 100% oxygen. (Change to clean vapour-free anaesthetic machine as quickly as practical.)</td>
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<tr>
<td>Administer dantrolene—2-3 mg/kg; repeat as needed, titrating dose to response.</td>
</tr>
<tr>
<td>Cool patient—surface cooling; gastric, rectal, peritoneal lavage with cold saline.</td>
</tr>
<tr>
<td>Treat cardiac dysrhythmias symptomatically.</td>
</tr>
<tr>
<td>Treat hyperkalemia with glucose, insulin, furosemide.</td>
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<tr>
<td>Promote diuresis with mannitol, fluids, furosemide to maintain urine output.</td>
</tr>
<tr>
<td>Administer sodium bicarbonate for acid-base disturbances.</td>
</tr>
<tr>
<td>Monitor cardiac activity, blood pressure, electrolytes, acid-base status, muscle-enzyme levels, (P_{A\text{-}CO_2}), (S_{O_2}), urine output, temperature, and level of consciousness.</td>
</tr>
</tbody>
</table>
and repeated as necessary. The anaesthesia tubing, absorber, bag, and, if practical, the entire anaesthesia machine should be changed because it may contain residual amounts of triggering agents, which can sustain the MH episode. $P_{CO_2}$ should be monitored both by end-tidal $CO_2$ determination and blood-gas analysis. Results of arterial and venous blood-gas analysis should be monitored and guide treatment of acid-base aberrations. Acidois should be treated with sodium bicarbonate. In addition, serum electrolytes and muscle enzyme levels should be checked to assess the degree and severity of the damage produced by the MH reaction. The hyperthermic patient should be cooled by any available means, including surface cooling with cooling blanket or ice packs. Internal cooling may be carried out through gastric, peritoneal, or rectal lavage with iced Ringer's lactate solution. Cardiac dysrhythmias should be treated symptomatically, and some experts recommend that procaine or procainamide be used and lidocaine be avoided because of its propensity for increasing myoplasmic calcium levels. In addition, to prevent renal damage or failure due to release of myoglobin from damaged muscle tissue, fluid and diuretic therapy should be implemented to maintain urine output at 2-3 mL $\cdot$ kg$^{-1}$ $\cdot$ h$^{-1}$. Diuresis must be balanced with adequate fluid replacement to prevent renal damage. Once the MH reaction has been satisfactorily controlled, the patient's status should be monitored closely, preferably in an intensive care environment for any signs of recurrence.

While the incidence is not known, perioperative MH recrudescence has been reported. Symptoms of recrudescence of MH are similar to those of the initial reaction. Patients become tachypneic and tachycardic and develop muscle rigidity. They can progress through the other characteristic signs and may die unless treated. Therefore, dantrolene sodium should be continued until the initial crisis resolves completely and may be required again to treat a recurrent episode.

The mortality rate for MH between 1975 and 1981 was 8.9% and dropped to 7% between 1981 and 1984. This decrease has been attributed to greater awareness of MH, the introduction of dantrolene, and its use for treatment of MH reactions.

**Prophylaxis with Dantrolene**

Dantrolene sodium is presently considered to be the most effective drug for the treatment of MH. Its first reported use in MH was in the effective management of reactions in pigs. Although dantrolene is essential for the treatment of the acute MH reaction, some controversy exists with regard to its use as a prophylactic agent in susceptible patients undergoing elective surgery. Although reports exist that describe the successful anaesthetic management of susceptible patients pretreated with dantrolene prior to anaesthesia, the need for such pretreatment has been questioned especially because susceptible patients should be managed with an anaesthetic protocol that avoids the use of triggering agents, uses a vapour-free anaesthetic machine, and follows other recognized precautions. In a retrospective study of 956 patients thought to be MH susceptible—patients undergoing elective muscle biopsy due to previous MH reaction, family history of MH, or muscle cramps of unknown origin—it was reported that only 0.62% of muscle-biopsy-positive patients developed symptoms of MH in the presence of trigger-free anaesthetics and without dantrolene pretreatment. Furthermore, there are reports of the occurrence of MH in patients who were treated with dantrolene prior to surgery. However, it has been argued that these patients exhibited signs of MH due to a failure to achieve adequate serum levels with dantrolene pretreatment.

Pretreatment of patients with oral dantrolene is associated with a number of side effects including nausea, dizziness, drowsiness, blurred vision, vomiting, diarrhea, epigastric cramps, and muscle weakness. Profound muscle weakness leading to pulmonary complications in a patient with pre-existing myopathy has been reported.

A 5½-year-old patient who was receiving oral dantrolene pretreatment (1 mg/kg, q 6 h for 3 doses) for suspected susceptibility to MH developed marked muscle weakness and noisy respiration following the second dose. The third dose was withheld, and the patient was operated on. The patient developed perioperative tachycardia and temperature increase with no acid-base disturbances and responded to surface cooling. The patient was mechanically ventilated postoperatively (IMV 12/min, PEEP 5 cm H$_2$O, exhale $V_T$ 300 mL) and also developed lower-lobe atelectasis. The patient required vigorous chest physiotherapy and tracheal suctioning, but was extubated 36 hours postoperatively, although her swallow and gag reflexes were still somewhat diminished. The patient subsequently recovered with no deficits.
Prolonged neuromuscular blockade with vecuronium following oral dantrolene pretreatment has also been reported. Muscle paralysis was measured using the evoked compound electromyographic (EMG) response and found to be prolonged in a pretreated patient compared to controls subjected to the same anaesthetic regimen without dantrolene pretreatment. While no overt postoperative respiratory complications have been described, it is important for medical personnel involved in respiratory care to be aware of the possibility of prolonged muscle paralysis following dantrolene pretreatment and to be prepared to institute ventilatory support if needed. While occurrence rates for these complications are not known, greater awareness of the possibility of their occurrence will allow for better patient management should they appear.

Most of the side effects described have been associated with the 2-4 day regimen of oral dantrolene prior to surgery. In order to minimize side effects, the use of intravenous dantrolene 2.4 mg/kg 1-4 hours prior to or during induction has been recommended. Because of its relatively high pH, dantrolene is irritating to veins, and intravenous administration should be through a fast-running infusion.

In spite of the ongoing debate, in cases in which the contemplated surgery is expected to be prolonged or injurious to muscle tissue, the patient is apprehensive, or acid-base imbalances are anticipated, preoperative intravenous dantrolene treatment appears to be a prudent precaution and is recommended.

Diagnosis of Susceptibility

Clinical Diagnosis

Identification of susceptible subjects prior to anaesthesia to avoid precipitating an MH crisis is the ideal. Because it is generally believed that the MH crisis originates in skeletal muscle, persons demonstrating abnormalities associated with skeletal muscle should be considered to be at risk. Such subjects would include patients suffering from Duchenne’s muscular dystrophy or central core disease. Other abnormalities identified in MH susceptible subjects include osteogenesis imperfecta, hernias, kyphosis or scoliosis, deformities of the feet, spinal deformities, pectus excavatum, squint, and cryptorchidism.

Caffeine-Halothane Contracture Test

The diagnostic use of caffeine and the potentiating effects of halothane on caffeine-induced contractures was first described by Kalow et al. To perform the caffeine-halothane contracture test, muscle fascicles are carefully dissected from skeletal muscle and divided into strips to allow replicate testing. Care must be exercised during dissection in order to avoid contraction or stretching of the muscle cells. The excised fascicles are transported immediately to the laboratory because delays in transport may influence test results. Each of the muscle strips is secured to an electrode housing with one end tied to a transducer to allow recording of changes in muscle tension. The strips are placed in Krebs-Ringer’s solution aerated with carbogen (95% O₂, 5% CO₂).

Under current North American standards, required testing is carried out using 3% halothane or caffeine in incremental doses of 0.5 mmol to 32 mmol. The parameter measured is the resting tension of the muscle and its contracture response to halothane and/or caffeine. A positive halothane contracture test is defined as a contracture of 0.2-0.7 g upon exposure to halothane for 10 min. The specific baseline normal value is determined by each testing center by testing 30 normal control subjects. Caffeine contracture testing is performed by exposing the muscle to caffeine concentrations of 0.5 mmol, 1.0 mmol, 2.0 mmol, and 32 mmol. The muscle is exposed to the caffeine for 4 minutes or until contracture achieves a plateau. The test is defined as positive if there is > 0.2 g tension with 2.0 mmol caffeine or increase of 1 g tension above baseline at < 4.0 mmol caffeine (caffeine-specific concentration) or if there is an increase of > 7% of maximal contracture (as measured at 32 mmol) at a caffeine concentration of 2 mmol. Additional optional tests include (1) the recommended 2% halothane test performed under the same conditions as the 3% halothane test but using 2% halothane and (2) the joint halothane-caffeine test in which muscle is exposed to 1% halothane for 10 min followed by addition of incremental doses of caffeine. There is controversy as to the validity of the latter test.

Age and muscle-fiber-types do not affect the results of tests. However, composition and pH of the Krebs solution and temperature of the bath do affect results (temperatures of 22 °C or 25 °C produce weaker contractures than does 37 °C).

Various drugs may influence test results. Dantrolene has been shown to decrease caffeine contractures,
and, therefore, should not be administered prior to
diagnostic biopsies. Calcium-blocking drugs such as
calmodulin and diltiazem have been found to alter
contracture responses to halothane and caffeine.\textsuperscript{52} In
order to eliminate differences in the testing protocol
among testing centers, the North American MH
Group has established standards for the performance
of the caffeine-halothane contracture test.\textsuperscript{53} The
standards specify all criteria that must be met to assure
uniformity in the results of the testing procedure.

**Succinylcholine-Induced Masseter Spasm**

Studies have shown a correlation between
succinylcholine-induced masseter muscle spasm and
MH susceptibility as confirmed by contracture
testing.\textsuperscript{7,54-58} However, not all MH-susceptible
persons exhibit masseter muscle spasm following
administration of succinylcholine. Predictive accuracy
ranges from 51%\textsuperscript{58} to 64%,\textsuperscript{57} suggesting that 36% to
49% of MH-susceptible subjects are not identified as
such by the masseter-muscle-spasm criterion.
Therefore, the occurrence of masseter muscle spasm
with succinylcholine administration should be viewed
as suggestive of MH susceptibility; however, the
absence of masseter spasm cannot be regarded as
indicating non-susceptibility. Succinylcholine is a
known triggering agent and may precipitate a full-
blown MH crisis.

**Noninvasive Tests under Development**

Noninvasive tests (including the Quin-2 lympho-
cyte test that utilizes levels of fluorescence to
determine cytoplasmic \textsuperscript{5+} concentrations, spin
labeling,\textsuperscript{59} and nuclear magnetic resonance spectro-
scopy\textsuperscript{60}) are under development and may ultimately
provide useful information. At least one group\textsuperscript{61} has
used the Quin-2 test to distinguish between MH-
susceptible and control pigs, but human data are
lacking.

**Other Tests**

Other tests such as creatine phosphokinase (CPK)
screening,\textsuperscript{62} platelet ATP depletion,\textsuperscript{63} muscle \textsuperscript{5+}
uptake,\textsuperscript{64} erythrocyte fragility,\textsuperscript{65,66} and exercise
ability\textsuperscript{67} as indicators of susceptibility have been found
to be lacking in predictive correlation or have been
discredited.

**In Summary**

No single noninvasive screening test with a
potential for widespread applicability is sensitive and
specific enough to identify persons at risk for MH.
Therefore, a diagnosis of susceptibility must be made
based upon a family history, elevated serum CPK
levels, and a strong index of suspicion, and must be
confirmed by a muscle contracture test.

**Genetics**

Malignant hyperthermia susceptibility was origi-
nally believed to be transferred within families through
an autosomal dominant gene.\textsuperscript{54} Although the
incidence of MH is greater in men than in women,
it is not believed to be X-chromosome linked because
severity of the condition does not differ between the
sexes. The higher incidence in men is probably the
result of the comparatively greater muscle mass in
men.\textsuperscript{1} Variation in expression and severity of the
condition within families suggests that transmission
of susceptibility may occur through more than one
gene or allele and that there may be modifying factors
present that govern the transmission characteristics,\textsuperscript{45}
resulting in a pattern of genetic transfer that may
range from recessive to dominant.\textsuperscript{52} However, the
work of MacLennan et al\textsuperscript{64} and McCarthy et al\textsuperscript{69}
suggests that it may be possible to isolate the MH
genetic defect to Chromosome 19. Genetic disorders
affecting the musculoskeletal system have also been
identified with MH susceptibility.\textsuperscript{70,71} For clinical and
counseling purposes, first-degree relatives of MH-
susceptible persons should be considered to have a
50% chance of susceptibility, whereas second-degree
relatives should be considered to have a 25% risk.\textsuperscript{54}

In summary, it is generally believed that the pattern
of inheritance of MH susceptibility is autosomal
dominant. However, some evidence suggests that the
susceptibility may be multigenic in origin. Variability
in incidence and severity of MH among subjects,
within subjects at different times, and also among
family members has been noted.\textsuperscript{3}
Pathophysiology

Although the precise intracellular defect responsible for the development of MH has not been defined, it is generally accepted that the primary defect involves Ca++ regulation in the skeletal muscle of susceptible persons. It has been suggested that MH results from an uncontrolled rise in myoplasmic Ca++ levels in response to triggering factors.

Sarcoplasmic Reticulum

Defects in Ca++ storage, Ca++ uptake, and regulation of Ca++ levels at the membrane of the sarcoplasmic reticulum (SR) have been suggested as sources of elevated sarcoplasmic Ca++ concentrations. Enhanced Ca++ release upon exposure to halothane has been demonstrated, suggesting hypersensitivity of Ca++ channels to triggering agents.

Plasma Membrane

Nonspecific sarcolemmal abnormalities have been demonstrated; however, it is uncertain whether these were artifactual. Changes in sarcolemmal permeability to extracellular Ca++ secondary to various triggers producing increased sarcoplasmic Ca++ levels has also been suggested. Defects in Ca++ channel or Ca++-ATPase pump activity at the cell membrane may contribute to elevated myoplasmic Ca++ levels.

Phospholipase A2

Increased activity of the enzyme phospholipase A2 (PLA2) may contribute to MH. PLA2 activation may promote release of fatty acid from membranes stimulating intracellular Ca++ release and inhibiting Ca++ uptake by the SR. Attenuation of halothane- and succinylcholine-induced contractures using PLA2 inhibitors has been demonstrated. Fatty acid production has been found to be higher in MH-susceptible persons compared to controls, and certain fatty acids have been shown to promote Ca++ release from SR.

Mitochondria

MH-induced accumulation of Ca++ by the mitochondria, an energy-requiring process leading to depletion of ATP stores, has been demonstrated in pigs. This results in reduced Ca++ uptake by the SR and in elevated myoplasmic Ca++ levels. Similar results have been found by other researchers. Reduced ATP levels produce ion imbalances due to failure of ATP-driven pumps and muscular rigidity due to the requirement of ATP for actin and myosin separation.

It was originally believed that the observed temperature rise resulted from heat generated through the uncoupling of oxidative phosphorylation. Calculations by Wang et al suggest that heat generated from uncoupling of oxidative phosphorylation would be insufficient to produce the degree of temperature elevation seen in MH over the time period in which it occurs. The temperature rise in MH probably results from heat generated by hydrolysis of high-energy phosphate bonds, by SR-ATPase activity, by Na+-K+ pump activity, by neutralization of hydrogen ions, by anaerobic metabolism, and by free energy liberated but not converted to ATP.

In Summary

It appears that MH occurs as a result of an increase in myoplasmic Ca++ concentration due to a malfunction in Ca++ regulation at the level of the plasma membrane, SR, mitochondria, PLA2, or some combination of these four sites. Ca++-induced interaction between actin and myosin filaments produces contracture and rigidity. Depletion of ATP stores in an effort to restore Ca++ levels leads to increased metabolism, respiratory and metabolic acidosis, and heat production. The acidosis and elevated temperature alter membrane characteristics, which leads to electrolyte imbalances and myoglobinemia. Cardiac dysrhythmias develop secondary to acid-base and electrolyte imbalances. If untreated, the signs and symptoms worsen and death ensues.

In Conclusion

Malignant hyperthermia is a rare but potentially fatal condition precipitated by certain pharmacologic and environmental agents, including agents administered during anaesthesia. While the pathophysiologic defect remains unknown, the available data suggests...
that abnormalities in Ca$^{2+}$ regulation in skeletal muscle cells may be responsible. At present, the only definitive test available to identify MH-susceptible individuals is the caffeine-halothane contracture test. Treatment of an MH reaction consists of administration of dantrolene, systemic cooling, correction of acid-base disturbances, oxygenation, ventilation, and symptomatic relief of cardiac dysrhythmias. Because respiratory care practitioners and anaesthetic technicians may be involved in the care of MH-susceptible patients, greater awareness of the syndrome, early recognition of signs and symptoms of a reaction, and prompt activation of appropriate treatment protocols may prevent disastrous outcomes.

ACKNOWLEDGMENTS

The authors thank Ms Polly Moors for her secretarial assistance in the preparation of the manuscript.

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Systemic Antifungal Agents

Hugh S Mathewson MD

The incidence of fungal infections of the respiratory tract is rising, due mainly to the growing numbers of immunodeficient persons. Immuno-deficiency in the host substantially accounts for the opportunistic pulmonary infections caused by nonvirulent fungi, such as Candida and Mucor, and for many of the disseminated (‘deep’) infections that appear during local epidemics of histoplasmosis, blastomycosis, or coccidioidomycosis. Hard data concerning the prevalence of fungal lung infections are difficult to obtain. Diagnostic pitfalls are numerous, even in epidemics, and the offending organisms may be recoverable only by open lung biopsy. Testing for antibodies is often of little value—immunodeficient patients are likely to be anergic and those with intact immune systems may already have acquired immunity to the more common fungi. The critically ill patient may be contending with an unknown and unidentifiable foe.

Pulmonary mycotic infections were reviewed by Davies (1987), who presented a simple classification based on the nine principal genera of fungi encountered in clinical practice. Four mycoses are endemic to specific geographic areas: histoplasmosis, blastomycosis, coccidioidomycosis, and paracoccidioidomycosis. Three of the genera are totally opportunistic invaders: Aspergillus, Mucor, and Candida. Lastly there are Cryptococcus, more often found as a meningeal infection, and Sporothrix, which usually enters through a skin wound rather than by inhalation.

The drugs recommended for each of these systemic mycotic infections, as set forth in the Medical Letter of Dr. Mathewson are adapted from Reference 3.

Table 1. Drugs for Systemic Fungal Infections

<table>
<thead>
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<th>Drug</th>
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<tbody>
<tr>
<td>Amphotericin B (no dependable drug alternative)</td>
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<tr>
<td>Aspergillosis</td>
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<tr>
<td>Mucormycosis</td>
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<tr>
<td>Sporotrichosis</td>
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<tr>
<td>Amphotericin B or ketoconazole</td>
</tr>
<tr>
<td>Blastomycosis</td>
</tr>
<tr>
<td>Coccidioidomycosis</td>
</tr>
<tr>
<td>Histoplasmosis</td>
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<tr>
<td>Paracoccidioidomycosis</td>
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<tr>
<td>(alternative drug—a sulfonamide)</td>
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<tr>
<td>Amphotericin B with or without fluconazole</td>
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<tr>
<td>Candidiasis</td>
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<tr>
<td>Cryptococcosis (alternative drug—fluconazole)</td>
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<tr>
<td>Flucytosine</td>
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<tr>
<td>Chromoblastomycosis</td>
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<tr>
<td>Ketoconazole or miconazole</td>
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<tr>
<td>Pseudallescheriasis</td>
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Adapted from Reference 3.

June 15, 1990, are shown in the accompanying table. Also listed are infections with two less common genera that respond favorably to special agents: chromoblastomycosis, for which fluconosine is effective, and pseudallescheriasis, which can be treated with either ketoconazole or miconazole. In all the common systemic mycoses amphotericin B is the drug of choice, possibly substituted by or supplemented with ketoconazole or fluconosine.

These compounds, particularly amphotericin B and fluconosine, are markedly toxic. Fungi are eukaryotic cells, similar to mammalian cells, and not prokaryotic like bacteria. Therefore, drugs that destroy fungi are also likely to damage host organs and tissues. Targeting biochemical processes that are completely unique to fungal species is a therapeutic goal yet to be attained.

The list of available systemic antifungal drugs is short: amphotericin B, fluconosine, ketoconazole, miconazole, and fluconazole. Each of these will be reviewed briefly.

Amphotericin B is a polyene macrolide antibiotic produced by the actinomycete, Streptomyces nodosus. It is similar in structure to nystatin, another fungicidal agent that is used only topically. Polyene compounds attach firmly to sterols in the cell membrane, primarily ergosterol in sensitive fungi. This causes membrane disruption and leakage of cytoplasmic constituents. Amphotericin B is
administered in an intravenous infusion under controlled conditions. Fever and chills occur on initial treatment in about 50% of cases, and vomiting in about 20%. Over 80% of persons given the drug develop some degree of renal impairment. Amphotericin B attaches to the liposomes of renal tubular cells, which are rich in cholesterol, causing irreparable epithelial damage. The cellular destruction is dose-dependent, and is often the limiting factor in treatment. Reduction in dosage is recommended when the plasma creatinine value rises above 3.5 mg/dL. The list of other toxic complications is extensive; many of these are life-threatening. Obviously the drug should be reserved for patients in whom the pulmonary mycosis is progressive and will not resolve spontaneously.

Flucytosine is an antimetabolite closely related to the antineoplastic drugs fluorouracil and flouxuridine. It is given orally. The fungal cell converts flucytosine to the cytotoxic fluorouracil, but this occurs to only a limited degree in host cells. However, the therapeutic plasma concentration of flucytosine is limited to 100 mcg/mL; at this point the corresponding fluorouracil level is about 1 mcg/mL, which is destructive to bone marrow cells. Flucytosine is used for treatment of cryptococcosis and candidiasis, but drug resistance commonly develops, restricting its usefulness. Suppression of blood-cell formation heads the list of toxic side actions, which include enterocolitis and hepatomegaly.

Ketoconazole and miconazole are members of a family of imidazole derivatives, some of which are used topically ( clotrimazole, econazole), and others that are still under experimental study. These compounds inhibit ergosterol synthesis in fungi. Ketoconazole can be taken orally, and is much less toxic than amphotericin B, but it is also less effective; however, it can be given for periods of several months. Miconazole, as stated previously, is effective for pseudallescheriasis, but it must be given intravenously.6

Fluconazole is a triazole derivative recently approved by the FDA. Like the imidazoles it inhibits ergosterol formation, presumably by the same mechanism.12 Its clinical uses are mainly for systemic candidiasis and for cryptococcal meningitis. A recent clinical report stated that fluconazole was effective for control of disseminated candidiasis in AIDS patients who were unable to take amphotericin B.13 Fluconazole is better tolerated than ketoconazole, and is definitely less toxic than amphotericin B or flucytosine. It is very expensive, and its eventual price in treatment of systemic candidiasis is still undefined.12

It is evident that inhibition of the biosynthesis of ergosterol and uracil-antimetabolite substitution are pharmacologic mechanisms that operate with a heavy price to the host. It is unlikely that newer imidazole derivatives will raise the rates of cure of pulmonary mycoses, especially in immunodeficient patients. AIDS victims are particularly difficult. They are often intolerant to amphotericin B or flucytosine, and may have to be treated with second-line drugs that will at best hold the mycotic infection in remission. Relapse rates are very high, because the patients eventually become immunologically defenseless and serve simply as nutrient sources for otherwise innocuous microorganisms.

In patients with some degree of immunocompetency, systemic mycoses must be treated promptly and aggressively. Surgical excision may be the most effective treatment of localized lung infections. Amphotericin B, with its unpleasant subjective side actions and its forbiddingly high incidence of nephrotoxicity, continues to be the agent of choice for disseminated disease.

The AIDS epidemic has stimulated research efforts to develop new antifungal drugs. Currently there are no new polyenes, but some ketoconazole congeners are undergoing trials in immunodeficient patients, notably itraconazole.14 Several ergosterol biosynthesis inhibitors of totally different structure are under study. One of them, terbinafine, an allylamine derivative, is in Phase III clinical trials, and is said to have no effect on cholesterol biosynthesis, suggesting that it may be less nephrotoxic.15

Fungal cells contain unique components, such as chitin and carbohydrate moieties such as glucans and mannans. These may prove to be valuable targets for new antifungal chemicals that have more sparing effects on mammalian cells. Chitin-synthetase inhibitors that disrupt fungal cell-wall synthesis have been discovered. A group of antibiotics called nikkomycins are representative of this type of agent; some of them, including their semisynthetic derivatives, show efficacy in a murine-disseminated candidiasis model.16

The beta-glucan-synthesis inhibitors are another class of antifungal agents under investigation. Cilofungin, a semisynthetic echinocandin B derivative, was reported to be as effective as amphotericin B for disseminated candidiasis in rabbits.17 Papulacandin B17 and aculeacin A18 are other antibiotic compounds identified as beta-glucan synthase inhibitors in Candida albicans.

A rather lengthy list of other antibiotics with antifungal activity could be presented.19 In-vitro studies demonstrate the antibiotic’s capacity to inhibit fungal growth, but the biochemical mechanisms are yet unknown. Improved techniques for studies in mice are bringing more of these promising compounds to experimental trial. It is hoped that new agents will be advanced for clinical study that will improve the prognosis of systemic fungal infections and permit effective therapy without the toxicities associated with the drugs in current use.
REFERENCES


The key published in the September issue of the Journal was incorrect. The correct key is published here. Please be assured that your answer sheet was graded with the correct key.

CRCE through the Journal

For your information, answers to the 50 questions for CRCE through the Journal, which appeared in the July issue of RESPIRATORY CARE, are given below. No scores will be available from the AARC until 1990 CRCE transcripts are released in early 1991. Deadline for submission of Answer Sheets for CRCE credit was August 15, 1990.

The correct answers to questions are

1. e  6. d  11. a  16. a  21. b  26. b  31. a  36. c  41. e  46. c
2. a  7. b  12. c  17. d  22. Omitted  27. d  32. e  37. e  42. b  47. c
3. c  8. a  13. d  18. e  23. d  28. a  33. d  38. c  43. a  48. a
5. c  10. d  15. e  20. a  25. c  30. b  35. a  40. d  45. e  50. b

NOTE: Because of a production error, this test will be scored on the basis of 49 questions. (Question 21 appeared twice, and Question 22 was omitted.)
New Problems in Supply, Reimbursement, and Certification of Medical Necessity for Long-Term Oxygen Therapy: Consensus Conference Report

Introduction

In 1970, Neff and Petty reported that long-term continuous oxygen therapy resulted in increased survival of patients with chronic obstructive lung disease (COLD) and cor pulmonale. Subsequently, the efficacy and clinical benefits of long-term oxygen therapy in COLD patients have been demonstrated by multicenter clinical trials conducted in both the United Kingdom and in North America. Reduced mortality and fewer hospitalizations occurred in patients who received nearly continuous ambulatory oxygen for a shorter period of time in the Nocturnal Oxygen Therapy Trial (NOTT), compared to patients who received only stationary oxygen in the Medical Research Council report. The technology for ambulatory oxygen therapy with portable liquid systems has improved the quality of life for many patients with chronic hypoxemia. Ambulatory oxygen has allowed these patients to carry out activities of daily living, including a return to work for some patients, while substantially improving compliance with therapy.

Two previous oxygen consensus conferences convened in Denver, Colorado, addressed issues of supplying and prescribing oxygen that were primarily related to Medicare policies that were operative at the time. Many of the recommendations of these two consensus conferences were incorporated into Medicare guidelines for coverage of home oxygen therapy. Today the issues and problems have changed to a considerable degree because of new reimbursement legislation introduced in 1987 for implementation in 1989. This is commonly called the “Six-Point Plan” for reimbursement of durable medical equipment (DME). Home oxygen therapy is one of the six components of this plan and represents about 45% of the cost for all DME. Under the Six-Point Plan, home oxygen therapy is reimbursed on a prospective basis, meaning that the cost of all systems are “averaged” and there is no recognized difference between the various types of oxygen delivery systems (liquid versus gaseous). Payment is modulated by the amount of oxygen being used. In each Medicare region a base fee has been calculated for reimbursement of oxygen administered at flowrates of 1-4 liters per minute (L/min). There is provision for a 50% reduction in reimbursement when oxygen flowrates of less than 1 L/min are prescribed and for a 50% increase when flowrates of greater than 4 L/min are prescribed. A small add-on component is allowed for portable oxygen, with no distinction between portable and ambulatory sources.

Recently a new certificate for medical necessity (CMN) (HCFA Form 484) was introduced that required greater direct physician participation in ordering long-term oxygen therapy and documenting need. This form was estimated by the Health Care Financing Administration (HCFA) to require 25 minutes for completion, and only the physician or

The conference was held March 15-16, 1990, in Washington DC, and was co-sponsored by PSI. Center for Health Sciences Education, 1719 E 19th Street, Denver CO 80218; and Creighton University Medical Center, 601 N 30th Street, Omaha NE 68131.

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Reprints: Walter J O'Donohue Jr MD, Professor and Chairman, Department of Medicine, Creighton University Medical Center, 601 N 30th Street, Omaha NE 68131-2197.
a member of the physician’s staff could complete the form. Because Form 484 is long and complex, many primary care physicians have been unwilling and perhaps unable to complete it properly, resulting in an adverse effect on oxygen suppliers’ ability to be reimbursed for services and equipment and a negative impact on patient care. It is in the context of these recent changes in policies and procedures for home oxygen therapy that this Third Consensus Conference on Home Oxygen Therapy was convened in Washington DC.

Reimbursement for Home Oxygen under the Six-Point Plan

A major problem created by the new Six-Point Plan for reimbursement of DME providers relates to the assumption that all oxygen delivery systems are modality neutral, while in fact they are neither clinically nor cost neutral. As stated in the Second Oxygen Consensus Report,5 most patients with chronic lung disease who are candidates for long-term oxygen therapy are neither bedbound nor homebound. For those individuals who are able and desirous of frequent trips outside of the home, a liquid ambulatory oxygen system is the “standard of care.” The higher cost of ambulatory liquid oxygen systems is a disincentive for most DME suppliers to provide this system, even when liquid oxygen is prescribed and is judged to be medically necessary. This cost disadvantage is magnified when patients live in rural areas where mileage and delivery time are substantially increased. In many rural areas in the United States, liquid oxygen is no longer available to patients because the cost of providing this therapy, relative to the allowable reimbursement under the Six-Point Plan, is prohibitive to the supplier. Keying the cost of home oxygen therapy to the flow of oxygen is a major policy mistake because the primary cost of home oxygen therapy is related to the oxygen equipment, delivery, user instruction, and service. Oxygen itself is the least expensive component of home oxygen therapy and should not be the major determinant for reimbursement.

Patients receiving home oxygen therapy often fit into one of three major groups that will usually predict the oxygen delivery system that is medically necessary:

1. **Sedentary, bedbound, or homebound patients (or patients requiring oxygen only during sleep).** These patients usually require an oxygen concentrator with a backup oxygen cylinder for use in the event of a power failure or for the rare occasion when the patient may need to leave the home.

2. **Ambulatory or mobile patients who only occasionally go out of the home for essential medical care or other infrequent visits.** These patients can usually be managed with an oxygen concentrator and supplemental cylinders on a stroller for portability.

3. **Ambulatory patients who leave home several times each week.** These patients should have liquid oxygen systems with lightweight ambulatory units that can be transfilled in the home.

**Recommendation 1:** A new policy for oxygen reimbursement is necessary. It is illogical and detrimental to patient care to base reimbursement of home oxygen therapy on flowrate and ignore the source of oxygen and the variable costs involved in providing oxygen from different devices and in different forms (liquid versus gaseous). Reimbursement should be based on the DME provider’s reasonable costs for supplying these different modalities of oxygen therapy. The geographic discrepancies in reimbursement for oxygen therapy must also be addressed. Rural areas should be considered as high cost areas when liquid oxygen systems are being provided because of the low population density and the greater distances involved in providing service. It should also be recognized by HCFA that portable oxygen and ambulatory oxygen are not synonymous. Portable systems, such as cylinders on strollers, are cumbersome and impede full activity. Ambulatory systems can be carried easily by the patient and are designed to allow and encourage full mobility. Ambulatory systems (eg, liquid ambulatory units) allow daily exercise, which is key to pulmonary rehabilitation.

**Current Disincentives for Development and Use of Oxygen-Conserving Devices**

The lower rates of reimbursement for flowrates of less than 1 L/min (50% reduction in basic reimbursement) now serve as a major disincentive to the development of oxygen-conserving devices. In a recent study of 100 patients using transstracheal oxygen catheters, which is an important new oxygen-conserving technique, the average resting flowrate was less than 1 L/min.6 The cost of therapy with
TRANSTRACHEAL oxygen catheters is clearly not reduced by 50% simply because flowrates of less than 1 L/min can be achieved in a majority of patients. This policy penalizes DME providers who supply oxygen-conserving devices or who have patients using oxygen-conserving techniques because the savings in liquid or gaseous oxygen are never in the magnitude of 50% of the total cost of therapy.

**Recommendation 2:** In redesigning the reimbursement system, the development and use of oxygen-conserving devices and techniques should be encouraged and not discouraged by reimbursement policy. Cost savings that can be achieved through oxygen conservation must be viewed in the context that oxygen is the least expensive component of oxygen therapy. The real benefit from oxygen-conserving technology is improved patient care and better compliance with therapy because of smaller, lightweight ambulatory oxygen units that allow greater mobility and an improved quality of life. The increased costs of new oxygen delivery equipment and technology can be balanced to some degree by the modest reduction in the cost of supplying oxygen, but the cost savings for the DME provider are likely to be small.

**Recommendation 3:** Until reimbursement policy can be changed, it is recommended that all oxygen be reimbursed at the flowrate necessary to achieve correction of hypoxemia using oxygen flow through a nasal cannula as a uniform standard and that the “dual reporting” of flowrate and amount of oxygen delivered be eliminated.

**Short-Term and Long-Term Oxygen Therapy**

Today it must be recognized that with increased pressure for early discharge from the hospital, brought about by the prospective payment system for reimbursement of hospital care, many patients with acute or chronic respiratory disease require home oxygen therapy in order to leave the hospital. Some of these patients are still clinically unstable and may not be receiving optimal therapy at the time of discharge. The need for supplemental oxygen may persist for several weeks, but in a significant number of patients the need for oxygen does not continue for the lifetime of the patient. Once the patient has reached a state of clinical stability and is receiving optimum therapy, which may require a period of 1 to 3 months, the need for long-term (lifetime) oxygen therapy should be reassessed by the measurement of arterial oxygen tension or saturation.

Stable patients receiving optimal therapy who have been followed as outpatients and who qualify for home oxygen therapy do not require further recertification. Once the need for long-term oxygen is documented, repeated measurements of arterial blood gases or saturation are no longer indicated or necessary for recertification. In fact, improvements in arterial blood gases may occur in some patients after 3 months as a result of the reparative effects of oxygen therapy, including a reduction in pulmonary hypertension. Cessation of oxygen therapy at this point could be detrimental to the patient and is not medically recommended in patients with chronic cardiopulmonary disease.

**Recommendation 4:** It is recommended that patients with unstable respiratory disease, such as those leaving an acute care hospital, who have not previously qualified for long-term oxygen therapy and who at the time of discharge qualify for home oxygen therapy under currently established criteria be issued a CMN for short-term oxygen therapy. Recertification for long-term oxygen therapy would be necessary in 30-90 days when the patient is judged by the physician to be clinically stable and receiving optimum therapy. Once the need for long-term oxygen therapy is established, repeat measurement of arterial blood gases or saturation is not medically necessary or justifiable for recertification, although these measurements are medically necessary and justifiable for the physician to evaluate the course of the disease and to make adjustments in oxygen flowrates.

**Recommendation 5:** The previous recommendation has a great potential for cost savings and can improve quality of care by eliminating unnecessary oxygen therapy. Some of the cost savings achieved in this area should be applied to correction of the inequities that exist in the current reimbursement system. Consideration should also be given to reimbursement for short-term oxygen therapy at a higher rate because the initial delivery, setup, and educational costs must be amortized over a shorter time period and the maintenance and storage costs will increase.
The Certificate of Medical Necessity—
The Prescription

The HCFA Form 484, which is required for certification of medical necessity, was undergoing revision by HCFA at the time of this consensus conference. All parties recognize that the current form is inadequate and must be revised. A new form has been developed by the National Association of Medical Directors of Respiratory Care (NAMDRC) and has received the approval of both the American College of Chest Physicians (ACCP) and the American Thoracic Society (ATS) Committee on Health Care Policy and Clinical Practice. Hopefully, HCFA will give appropriate consideration to this CMN form because it carries the approval of the major professional and scientific societies that represent pulmonary medicine in the United States today. Because the revised HCFA form was not available at the time of this conference, no specific comments can be made concerning the contents; however, there are several recommendations that can be made concerning the oxygen prescription.

Recommendation 6: It is recommended that the CMN form be a one-page document with the information necessary for certification on one side and instructions for completion on the reverse side. The physician should continue to provide the documentation of medical necessity and prescribe the oxygen delivery system that is medically indicated for the patient, as well as the appropriate oxygen flow rates, which may vary between rest, activities of daily living, and sleep. Many items can be more quickly and efficiently completed by a checklist format. The DME provider should be able to fill in portions of the certificate that do not change the prescription.

Recommendation 7: It is recommended that when home oxygen is prescribed in the hospital, the CMN form should be completed prior to discharge and the DME supplier notified in ample time to provide the equipment and instructions necessary for appropriate therapy. Physicians recognize that other medications cannot be dispensed until the discharge prescription is completed. Each hospital should be responsible for compliance with this policy, and this should become an area for review by the Joint Commission on Accreditation of Healthcare Organizations (JCAHO). The CMN forms must be readily available in all acute-care medical facilities where patients are likely to be discharged with home oxygen therapy.

Recommendation 8: A program must be established to educate primary care physicians who are responsible for ordering home oxygen therapy. The CMN form is now and will continue to be one of the most complex prescriptions that physicians are required to complete. The prescribing physician not only must understand the indications for short-term and long-term oxygen therapy but also must be familiar with the various systems for supply and delivery of oxygen and know which are best for each patient. At the present time, there are striking variations in Medicare carrier policies for documentation, certification, and reimbursement for home oxygen therapy. Education of carrier medical directors, who usually are not specialists in respiratory disease, would help to alleviate most of these discrepancies. Other health care providers who are involved in home oxygen therapy would also benefit from a comprehensive educational program.

Conclusions

The recommendations of this consensus conference can have far-reaching effects in improving the existing program for home oxygen therapy while reducing expenditures for medically unnecessary therapy. Major modifications in reimbursement policies are necessary to cover the cost of liquid oxygen systems for ambulatory patients, with full realization that all oxygen systems are not modality neutral. New policies for short-term oxygen therapy can result in major cost savings, but it must be recognized that DME costs for short-term therapy will be considerably greater and reimbursement must be at a higher level. Once long-term oxygen therapy has been initiated under conditions of clinical stability and optimal therapy, then repeat blood gas measurements are unnecessary for recertification and, in fact, recertification is unnecessary other than to assure that the equipment is being used as ordered and to document changes in the physician’s prescription.

Additional research is still needed to continue to refine oxygen therapy in the United States. Long-term, multicenter clinical trials have clearly identified major clinical benefits from oxygen therapy in patients with COLD. It is reasonable to believe that these findings extend to other patients with chronic
hypoxyemia; however, there may be subgroups of patients with other types of disease for whom the indications for oxygen therapy may vary. In patients with pulmonary hypertension and cor pulmonale, oxygen has a reparative effect, and it is unclear at what point improvement in arterial blood gases reflects disease instability versus the beneficial therapeutic effects of oxygen therapy itself. At the present time, medical knowledge does not allow discontinuation of oxygen therapy based on benefits that are seen after a period of 3 months. Important information is still unavailable concerning the magnitude of use and the true cost of oxygen therapy in the United States.

REFERENCES


Writing Committee:

Thomas L. Petty MD, Co-Chairman
Walter J O'Donohue Jr MD, Co-Chairman
Douglas F Gracey MD
Jerry G Greene MD
David J Pierson MD
Alan L Plummer MD

Conference Participants:

Thomas L. Petty MD
Committee Co-Chairman
Professor of Medicine
PSL Center for Health Sciences Education
1719 East 19th Street
Denver CO 80218

Walter J O'Donohue Jr MD
Committee Co-Chairman
Professor and Chairman of Medicine
Creighton University Medical Center
601 North 30th Street
Omaha NE 68131

Cara Racheheimer
HDIA
1701 Pennsylvania Avenue NW
Suite 470
Washington DC 20006

Charles Baudouin
Puritan-Bennett Corporation
10800 Plummm Road
Lenexa KS 66215-2198

Peter Benjamin
Abbey Foster
PO Box 9100
Fountain Valley CA 92728

Peter Berry
Minnesota Valley Engineering
407 South 7th Street
New Prague MN 56071-0234

Dale E Braddy
Transtracheal Systems
Suite 607
8755 East Orchard Road
Englewood CO 80111

Cheryl A Brown MHA
Director of Government Affairs
American Association for Respiratory Care
Suite 700
1655 North Fort Myer Drive
Arlington VA 22209

Louis W Burgher MD PhD
Pulmonary Medicine
Clarkson Hospital
44th and Dewey
Omaha NE 68105

Kent Christopher MD
Suite 366
1721 East 19th Avenue
Denver CO 80218

Joseph Cottrell MD
University of Pittsburgh
440 Scaife
Pittsburgh PA 15261

Howard Deutch
Lincare
PO Box 42002
St Petersburg FL 33742

Patrick J Dunne RRT
Southwest Medical HC
821 West Wilshire Avenue
Fullerton CA 92632

Douglas R Gracey MD
Professor of Medicine
Vice Chairman for Practice
Department of Internal Medicine
Mayo Medical School
Mayo Clinic
Rochester MN 55905

Jerry G Greene MD
Professor of Medicine
Chief, Pulmonary Service
VA Medical Center
Eln Street and 21st Avenue
Fargo ND 58102

Don Hicks
Glasrock Home Health Care
2480 Mt Wilkinson Parkway
Atlanta GA 30339
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PFT Corner #38—
The Case of the Supranormal FEV₁

Christopher G Green MD and Ronald F Rankin RRT RCPT

A 7-year-old white boy was admitted to our hospital for treatment of status asthmaticus. He had been diagnosed with asthma at 6 months of age and had been receiving oral steroid treatment for 5 years. He had been admitted to the hospital many times previously, and during one admission at age 3 he had been intubated and treated with positive pressure ventilation.

The patient's height was 102 cm (< the 5th percentile for age) and his weight was 26 kg (at the 75th percentile for age). Pulmonary function testing was performed on the 6th day after admission. The results of spirometry before and after bronchodilator are listed in Table 1. Skin testing was performed and the patient responded negatively to eggs, peanuts, milk products, vegetables, various grains, shellfish, fruits, and Aspergillus. The histamine skin test response was 2+.

Table 1. Spirometric Data of a 7-year-old, 102-cm White Boy (before and after Administration of Terbutaline)

<table>
<thead>
<tr>
<th></th>
<th>Predicted</th>
<th>Observed</th>
<th>% Predicted</th>
<th>Post-terbutaline</th>
<th>% Change</th>
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<tr>
<td>FVC (L)</td>
<td>1.12</td>
<td>1.19</td>
<td>106</td>
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<tr>
<td>FEV₁ (L)</td>
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<td>1.04</td>
<td>237</td>
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</tr>
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<td>1.28</td>
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<td>1.76</td>
<td>37</td>
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</tbody>
</table>

*Predictions are from Weng and Levison.

Questions
1. What is your impression of the predicted and % predicted values for this patient?

2. How would you interpret the patient's response to bronchodilator therapy?

Answers and Discussion on Next Page
Answers and Discussion

1. Predicted and % Predicted PFT Values: Based on the reference values of Weng and Levison, the patient's FEV₁ is 237% of predicted, which is questionably high. The FEF₁₅₀ and FEF₂₅₋₇₅ values are also well above predicted values. The FEV₁/FVC is 0.88, which is within the normal range, but the predicted ratio is surprisingly low, 0.39. Because proper system calibration had been performed, these predicted values suggest either a problem with the software that computes the predicted values or a problem with the regression equations used to predict values for this patient.

The software package has been used repeatedly by us and in other centers and is not likely to have been incorrectly programmed. The regression equations developed by Weng and Levison have been used successfully for many years. However, a closer look at the original data collected by Weng and Levison reveals that the height of the shortest patient studied as they developed their regression equations was 111 cm. The height of our patient was only 102 cm. Therefore, these predicted values represent extrapolations from the original data.

In addition to using regression equations appropriately matched for the height, sex, and ethnic background of the patient, we must also pay attention to the range of the independent variable (height, in this case) used in deriving the original regression equations. Weng and Levison studied 139 normal children ranging in height from 111 to 182 cm. Therefore, Weng and Levison's regression equations should not be used outside of this range of heights. The reliability of the predicted values is less at the extremes of height (near 111 cm and 182 cm) because there are fewer datapoints at these extremes.

Other study populations from which predicted PFT values have been derived may not have included such short subjects either. For example, Hsu et al. studied 1,805 normal Mexican-American, white, and black students in Houston, Texas; however, there were only 4 white boys in the height range 111-120 cm and no boys with a height lower than 111 cm. Therefore, Hsu et al.'s data would also have to be extrapolated to provide predicted values for our patient.

Another set of regression equations is needed to avoid extrapolation. Strope and Helms collected longitudinal data on young white and black children. Most of their population were black children, but there were at least 15 white boys with heights below 110 cm. The shortest white boy was 94 cm in height. The authors state that 100 cm to 150 cm is the height range of interest for their data; therefore, the regression equations derived from their data can be confidently applied to our patient.

Figure 1 graphically shows FEV₁ vs height using regression equations derived from the three aforementioned study populations. Note that regression equations from all three populations predict a similar FEV₁ for a patient 120 cm tall; however, the predictions diverge below this height.

Several factors may contribute to this divergence. As we mentioned, Weng and Levison studied no subjects shorter than 111 cm and only 12 who were less than 120 cm in height. They

![Fig. 1. Predicted normal FEV₁ vs height using regression equations from three studies of children: □—Weng and Levison; ●—Hsu et al.; ○—Strope and Helms.](image-url)
used linear regression for estimating FEV₁. This may not be the best approach over a wide range of the independent variable (height) and may make estimates of FEV₁ down to a height of 102 cm very uncertain.

Furthermore, Weng and Levison did not separate their subjects by race and sex. Black subjects are known to have a lower FEV₁ for a given height. Weng and Levison do not state whether their reference population was racially mixed. If blacks were included, this would tend to lower the predicted FEV₁ for a given height.

In contrast, Table 2 shows predicted values for our patient from the regression equations of Strope and Helms, who studied white boys in the height range of interest. FEV₁/FVC is not predicted by Strope and Helms, and the 95% tolerance limits for their data are very wide. Forced expiratory flows at 25%, 50%, and 75% of vital capacity are less than predicted, whereas FVC and FEV₁ are greater than predicted but within the 95% confidence interval. Thus, our patient's predicted values from Strope and Helms are consistent with the clinical presentation, whereas the values generated using the equations of Weng and Levison are not.

We emphasize the importance of applying appropriate regression equa-

2. The Bronchodilator Response: Despite questions about the predicted values for this patient, the response to bronchodilators can still be helpful. This response is usually expressed as a relative change (% change) rather than an absolute change (change in liters). The patient's response to bronchodilators includes an 11% increase in FVC, a 12% increase in FEV₁, and a 37% increase in FEF₂₅₋₇₅%. Snider et al1 state that the response to bronchodilators "should be interpreted according to the recognized variability in the laboratory in which the test is being performed." They do not comment on how this variability should be defined. Lacking an intra-

laboratory definition of variability of the three tests (FVC, FEV₁, and

FEF₂₅₋₇₅%) at least two should show significant improvement (defined as an improvement of 15% or more) after bronchodilator to indicate overall reversibility.4 Using these criteria, our patient did not show a significant improvement overall since only one of three tests increased more than 15% after bronchodilator.

Lorber et al5 defined a significant change after bronchodilator as an improvement greater than that seen in 95% of a control group chosen to define the inherent variability of the test rather than a response to medication. They found a 7.7% improvement in FVC and a 10.7% improvement in FEV₁ to be significant. They did not study FEF₂₅₋₇₅%. Using these criteria, our patient had a significant improvement after bronchodilator. This approach considers only the Type I error, which is analogous to a false-positive conclusion. A Type I error occurs if the null hypothesis is rejected when it is actually true. In this context, the null hypothesis is that there is no difference between the pre-bronchodilator and post-bronchodilator measurements. Rejecting the null hypothesis is equivalent to stating that the pre-bronchodilator and post-bronchodilator measurements are significantly different. There is always the chance that such a statement is false. Such a false presumption is a Type I error. In Lorber et al’s work, the chance of a Type I error was set at 5%. This means that a change greater than the level defined by Lorber et al will occur by chance only 5% of the time. Therefore, 95% of the time a change in FVC greater than 7.7% will be a real change, and only 5% of the time will such a change occur by chance.

Table 2. Spirometric Data of a 7-year-old, 102-cm White Boy (using Predicted Values from Strope and Helms3)

<table>
<thead>
<tr>
<th></th>
<th>Predicted</th>
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<td>142</td>
</tr>
<tr>
<td>FEV₁ (L)</td>
<td>0.82</td>
<td>± 0.41</td>
<td>1.04</td>
<td>126</td>
</tr>
<tr>
<td>FEV₁/FVC</td>
<td>—</td>
<td>—</td>
<td>0.87</td>
<td>—</td>
</tr>
<tr>
<td>FEF₅₀ (L/s)</td>
<td>2.62</td>
<td>± 1.34</td>
<td>2.69</td>
<td>102</td>
</tr>
<tr>
<td>FEF₂₅₋₇₅ (L/s)</td>
<td>1.76</td>
<td>± 1.21</td>
<td>1.35</td>
<td>77</td>
</tr>
<tr>
<td>FEF₂₅₋₇₅ (L/s)</td>
<td>1.02</td>
<td>± 0.98</td>
<td>0.68</td>
<td>67</td>
</tr>
</tbody>
</table>

The use of linear regression for estimating FEV₁ may not be the best approach over a wide range of the independent variable (height).
Table 3. Reproducibility of Spirometric Data: Comparison of Individual FVC Maneuvers Performed by a 7-year-old White Boy

<table>
<thead>
<tr>
<th>Trial Number</th>
<th>FVC (L)</th>
<th>FEV1 (L)</th>
<th>FEV1/FVC</th>
<th>FEFmax (L/s)</th>
<th>FEF25-75% (L/s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1.08</td>
<td>0.99</td>
<td>0.91</td>
<td>1.75</td>
<td>1.16</td>
</tr>
<tr>
<td>2</td>
<td>1.10</td>
<td>1.04</td>
<td>0.94</td>
<td>2.39</td>
<td>1.33</td>
</tr>
<tr>
<td>3</td>
<td>1.17</td>
<td>1.04</td>
<td>0.89</td>
<td>2.59</td>
<td>1.23</td>
</tr>
<tr>
<td>4</td>
<td>1.13</td>
<td>1.04</td>
<td>0.92</td>
<td>2.40</td>
<td>1.27</td>
</tr>
<tr>
<td>5</td>
<td>1.19</td>
<td>0.96</td>
<td>0.80</td>
<td>2.28</td>
<td>0.95</td>
</tr>
<tr>
<td>6</td>
<td>1.09</td>
<td>1.00</td>
<td>0.91</td>
<td>2.11</td>
<td>1.27</td>
</tr>
<tr>
<td>Mean</td>
<td>1.127</td>
<td>1.011</td>
<td>0.95</td>
<td>2.253</td>
<td>1.202</td>
</tr>
<tr>
<td>SD</td>
<td>0.045</td>
<td>0.0337</td>
<td>0.4930</td>
<td>0.293</td>
<td>0.135</td>
</tr>
<tr>
<td>Δ*</td>
<td>7.5%</td>
<td>6.2%</td>
<td>10.3%</td>
<td>24.3%</td>
<td>21.1%</td>
</tr>
</tbody>
</table>

*Percent change that would be considered by Nickerson et al. to be a significant bronchodilator response.

where n is the number of FVC trials per subject, SD is the standard deviation, and \( \bar{x} \) is the mean of the measured values of several trials, and \( t_{0.05, n - 1} \) and \( t_{0.10, n - 1} \) are t values with \( n - 1 \) degrees of freedom.

Spirometric data from individual FVC maneuvers are listed in Table 3. Using Nickerson's formula, a significant change in FVC is 7.5%, a significant change in FEV1 is 6.2%, and a significant change in FEF25-75% is 21.1%. Using these criteria, our patient had a significant improvement after bronchodilator treatment. This approach considers both Type I and Type II errors.

A Type II error is analogous to a false-negative conclusion, and occurs if the null hypothesis is accepted when it actually is false. This means the pre-bronchodilator and post-bronchodilator measurements are not accepted as being significantly different when indeed they are different. The chance of a Type II error was considered by Nickerson and set at 10%. Using Nickerson's approach, a change in FVC of < 7.5% is considered to be insignificant and that conclusion will be wrong only 10% of the time (a Type II error). A change in FVC of more than 7.5% is considered to be significant, and that conclusion will be wrong only 5% of the time (a Type I error). Using this formula also allows the calculation of variability for each subject. Determining individual variability is calculation-intensive and requires careful assessment of each FVC maneuver. With the use of digital computers, this approach could easily be implemented, but to the best of our knowledge it has not been.

There is no consensus on how best to assess the response to bronchodilators. The approaches discussed above may yield disparate results. What does represent a significant response to bronchodilator treatment? Should one test be evaluated or some combination of tests? How large a change is significant? These are all questions that remain to be answered. Readers interested in this subject are referred to Miller, Scaccial, and Gasl and Ries for further discussion of this issue.

REFERENCES

Advanced bronchodilator therapy at home or in the hospital... rapidly relieves reversible bronchospasm and maintains significant lung function improvement for up to 6 hours in some patients... minimal cardiac stimulation in most patients at the recommended dosage* of 2.5 mg tid or qid.

*Potency expressed as albuterol.
*In individual patients, any beta-2-adrenergic agonist should be used with caution in patients with cardiovascular disorders.

Please see next page for Brief Summary of Prescribing Information.
**BRIEF SUMMARY**

**Ventolin® (albuterol sulfate, USP)**

**Solution for Inhalation, 0.5%**

*Potency expressed as albuterol.*

The following is a brief summary only. Before prescribing, see complete prescribing information in Ventolin® Solution for inhalation product listing.

**CONTRAINDICATIONS:** Ventolin® Solution for Inhalation is contraindicated in patients with a history of hypersensitivity to any of the components.

**WARNINGS:** As with other beta-adrenergic agonists, Ventolin® Solution for Inhalation can produce paradoxical bronchospasm that can be life threatening. If this occurs, the preparation should be discontinued immediately and alternative therapy instituted.

**Patients:** Paradoxical bronchospasm has been reported rarely in association with the use of other sympathomimetic drugs and the home use of nebulized solutions. It is therefore essential that the physician instruct the patient in the need for further evaluation if his asthma becomes worse. In individual patients, any beta-adrenergic agonist including albuterol solution for inhalation, may have a clinically significant cardiac effect.

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

**PRECAUTIONS**

**General** As with all sympathomimetic agents, this product should be used with caution in patients with cardiovascular disease, especially coronary insufficiency, cardiac arrhythmias, and hypertension, as patients with constrictive pericarditis, hyperthyroidism, or diabetes mellitus, and in patients who are unusually responsive to sympathomimetic agents. Large doses of intravenous albuterol have been reported to aggravate pre-existing tachycardia and arrhythmias. Additionally, beta-adrenergic agents, including albuterol, given intravenously may cause a decrease in serum potassium, possibly through extracellular shifting. The decrease is usually transient, not requiring supplementation. The relevance of these observations to the use of Ventolin® Solution for Inhalation is unknown.

**Information for Patients:** The action of ventolin Solution for Inhalation may last up to six hours, and therefore it should not be used more frequently than recommended. Do not increase the dose or frequency of medication without medical consultation. If symptoms persist worst medical consultation should be sought promptly, while taking Ventolin Solution for Inhalation, other anticholinergic medications should not be used unless prescribed.

See directions for Patient Information for the section of the package insert.

**Drug Interactions** Often sympathomimetic aerosol bronchodilators should not be used concomitantly with anticholinergics.

Albuterol should be administered with extreme caution to patients being treated with monoamine oxidase inhibitors or tricyclic antidepressants because the sudden withdrawal of the vasoconstrictive effects of these drugs may precipitate serious reactions. These reactions are more common in elderly patients and in those who have been dependent on the drugs for a prolonged period of time.

**Animal Data** Mutagenicity, Impairment of Fertility, Abnormalities. Other adverse effects in rats given the maximum human inhalation dose in another study. This effect was blocked by the co-administration of propranolol. The relevance of these findings to humans is not known. A 18-month study in mice with a limited study of hamsters revealed no evidence of tumorigenicity. Studies with albuterol revealed no evidence of mutagenicity. Reproduction studies in rats revealed no evidence of impaired fertility.

**Pregnancy** Teratogenic Effects. Pregnancy Category C. Albuterol has been shown to be teratogenic in mice when given subcutaneously in doses corresponding to the maximum human inhalation dose, respectively, shown in Table 4. These findings are not relevant to humans. Pregnancy Exposure and Labor. Albuterol should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

**Lactation** Nursing Mothers. The uses of albuterol sulfate in nursing mothers are not known. Ethanol administered to pregnant rats has been shown to impair testicular, brain, and heart development. However, the significance of these findings in human infants has not been established.

**HOW SUPPLIED:** Ventolin® Solution for Inhalation. 0.5% is supplied in bottles of 20 ml (NDC 0173-0235-58) with accompanying calibrated droppers in boxes of one.

**INDICATIONS:** Advanced bronchodilator therapy at home or in the hospital. Effectively controls acute and chronic symptoms of reversible bronchospasm in patients with asthma, COPD, or cystic fibrosis. Usual dosage of 2.5 mg tid or qid for patients 12 years of age and older. 40-dose (20 ml) bottle is ideal for home use. Sulfite-free formulation minimizes potential for preservative-induced reactions.

**Usual Dosage:**

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

- **Nursing Mothers:** It is not known whether this drug is excreted in human milk. Because of the potential for serious adverse reactions in neonates, breastfeeding should be interrupted while the drug is taken.

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

**ADVERSE REACTIONS:** The results of clinical trials with Ventolin® Solution for Inhalation as adjunctive therapy in children and adults showed the following side effects that were considered probably or possibly drug-related:

- **Central Nervous System:** Agitation (2%), excitation (1%), insomnia (3%), nervousness (6%), tremors (1%), headache (1%), palpitations (1%), anxiety (1%), restlessness (1%), and seizures (2%).

- **Cardiovascular System:** Supraventricular tachycardia (3%), hypertension (1%), tachycardia (1%), angina pectoris (1%), and myocardial infarction (1%).

- **Gastrointestinal System:** Nausea (1.5%), vomiting (1.5%), diarrhea (1.5%), and abdominal pain (1.5%).

- **Respiratory System:** Generalized bronchospasm (1.5%), cough (1.5%), bronchitis (1.5%), rhinitis (1.5%), laryngitis (1.5%), and sinusitis (0.5%).

**OVERDOSAGE:** Information concerning possible overdose and its treatment appears in the full prescribing information.

**OSAGE AND ADMINISTRATION:** The usual dosage for adults and children 12 years and older is 2.5 mg of albuterol administered three to four times daily by inhalation. More frequent administration or higher doses are not recommended. To administer 2.5 mg of albuterol, 0.5% Solution for Inhalation with 7.5 ml of sterile normal saline solution. The flow rate is regulated to shut the particular nebulizer so that Ventolin® Solution for Inhalation be delivered over a period of about five to 10 minutes. The use of Ventolin Solution for Inhalation can be combated as medically indicated to control recurring bouts of bronchospasm. During the same illness, patients may be benefited from regular use of the solution for maintenance. If a previously effective dosage regimen fails to provide the usual relief of symptoms, should be increased immediately as this is often a sign of seriously worsening asthma that would require readmission of therapy.

**HOW SUPPLIED:** Ventolin® Solution for Inhalation. 0.5% is supplied in bottles of 20 ml (NDC 0173-0235-58) with accompanying calibrated droppers in boxes of one.

Allen & Hanburys

DURHAM, N.C. 27709

882-417

May 1980
Blood Gas Corner #27—
Nonventilatory Cause of Hypercapnia during Weaning

Robert S Campbell RRT, Richard D Branson RRT, and James M Hurst MD

An 82-year-old white man presented to the emergency room with sudden onset of abdominal pain and weakness. Vital signs deteriorated rapidly and the patient was taken to the operating room for repair of a ruptured abdominal aortic aneurysm. His postoperative course was complicated by adult respiratory distress syndrome (positive end-expiratory pressure [PEEP] of 15 cm H₂O [1.5 kPa] was required) and sepsis induced by ischemic bowel. Bowel resection was performed and antibiotic therapy initiated.

On Postoperative Day (POD) 4, ventilator settings were intermittent mandatory ventilation (IMV) 6 breaths/min, tidal volume (VT) 1.0 L, PEEP 8 cm H₂O [0.8 kPa], and inspired oxygen concentration (FiO₂) 0.45. Analysis of arterial blood revealed pH 7.42, PaCO₂ 39 torr [5.2 kPa], PaO₂ 87 torr [11.6 kPa], BE 1.2 mEq/L [1.2 mmol/L], HCO₃⁻ 26 mEq/L [26 mmol/L] and SaO₂ 97%. Indirect calorimetry revealed carbon dioxide production (VCO₂) 0.238 L/min, oxygen consumption (VO₂) 0.318 L/min, respiratory quotient (RQ) 0.75, and energy expenditure (EE) 2122 kilocalories (kcal)/day. Total parenteral nutrition (TPN) was instituted delivering 2100 kcal/day using a 15% dextrose base (D15) with 42.5 g protein/bottle, and daily lipids.

Ventilatory support was slowly decreased to IMV 2, VT 1.0 L, PEEP 5 cm H₂O [0.5 kPa], FiO₂ 0.40; analysis of arterial blood revealed pH 7.37, PaCO₂ 35 torr [4.7 kPa], PaO₂ 85 torr [11.3 kPa], BE -4.7 mEq/L [-4.7 mmol/L], HCO₃⁻ 20 mEq/L [20 mmol/L], SaO₂ 95%. On POD 9 an enteral feeding tube was placed, and tube feeding was initiated at 20 mL/hour using a 55% carbohydrate (CHO)-31% lipid solution. The rate was increased to a target of 1900 kcal/day based on EE measurement of 1860 kcal/day. Parenteral nutrition was to be tapered gradually and discontinued as the enteral rate reached the target, which took approximately 22 hours.

On the morning of POD 10, it was noted that the patient had become increasingly tachyphneic (f = 46) and diaphoretic; analysis of arterial blood revealed pH 7.31, PaCO₂ 65 torr [8.7 kPa], PaO₂ 87 torr [11.6 kPa], BE 5.1 mEq/L [5.1 mmol/L], HCO₃⁻ 32 mEq/L [32 mmol/L], and SaO₂ 95%. Indirect calorimetry revealed VCO₂ 0.290 L/min, VO₂ 0.260 L/min, RQ 1.12, and EE 1900 kcal/day.

Study Questions
1. How do you interpret the results of the arterial blood analysis and indirect calorimetry performed on POD 4?
2. Why do you think daily lipids were given as part of this patient’s parenteral nutrition?
3. How do you interpret the results of the arterial blood analysis and indirect calorimetry performed on POD 10?

Answers and Discussion on Next Page
Answers

1. First Interpretation. Arterial blood analysis performed on POD 4 reveals normal ventilation and acid-base balance with corrected hypoxemia. Indirect calorimetry reveals normal $V_{CO_2}$, elevated $V_O_2$, low RQ, and a resting caloric requirement of 2100 kcal/day. Elevation of the patient’s $V_O_2$ is most likely due to sepsis, and the decreased RQ reflects catabolism from his malnourished state.

2. Rationale for Lipid Administration. Our standard ICU practice specifies that lipids (500 kcal) be administered daily as part of parenteral nutrition in patients with respiratory failure and no contraindications to lipid administration. In this case, lipids accounted for 23% of total caloric intake. Delivery of a mixed source of nonprotein calories in the form of lipids and CHO helps to reduce $V_CO_2$, compared to providing all nonprotein calories by CHO.

3. Second Interpretation. Arterial blood analysis performed on POD 10 reveals a noncompensated respiratory acidosis with corrected hypoxemia. This patient’s high $P_ACO_2$ is probably due either to an increase in dead-space-to-tidal-volume ratio ($V_D/V_T$) or an increase in $V_CO_2$. Indirect calorimetry at this time showed an increased $V_CO_2$ and a normal $V_O_2$, resulting in an RQ of 1.12. An RQ greater than 1.0 suggests that lipogenesis is taking place. Lipogenesis occurs when CHO calories in excess of daily requirements are delivered and converted to fat, a process that markedly increases $V_CO_2$.

Discussion

Patients with normal pulmonary function respond to increased $V_CO_2$ by increasing minute ventilation. However, patients with limited ventilatory reserve will often respond to increased $V_CO_2$ with signs of respiratory distress, respiratory muscle failure, and failure to wean from mechanical ventilation.

Askanazi and co-workers described a case in which excess CHO administration (2.25 x resting EE) precipitated respiratory distress. A decrease in CHO delivery to 0.75 x resting EE resulted in improved ventilatory status. Other investigators have reported hypercapnia and exacerbation of respiratory failure associated with increased $V_CO_2$ from excessive CHO intake in patients with normal and altered lung function.

More recently, Jannace and colleagues reported the case of a patient experiencing hypercapnia and repeated weaning failure due to excessive caloric intake. Their patient received 2900 kcal/day, given in 18.5 hours, leaving 5.5 hours without nutritional support. This cyclic method of TPN delivery was done because abnormal results had been obtained from liver function studies. The cyclic TPN schedule caused a 51% increase in $V_CO_2$ during the TPN administration period with resultant hypercapnia and respiratory distress. The same number of TPN calories (2900 kcal/day), delivered continuously, stabilized $V_CO_2$ and the patient then tolerated periods of unassisted ventilation.

Our patient required the addition of 20 cm H2O [2.0 kPa] of pressure support ventilation (PSV) to assist his respiratory efforts and normalize $P_ACO_2$. The results of the indirect calorimetry performed on POD 10 prompted further clinical investigation. This patient had inadvertently continued to receive TPN at a rate of 2100 kcal/day as his enteral feeding was being increased to 1900 kcal/day. This resulted in a 48% increase in caloric intake causing excessive $V_CO_2$ and respiratory distress. TPN delivery was discontinued, and the patient’s respiratory status improved. He was quickly weaned successfully to minimal ventilator settings of PSV 10 cm H2O [1.0 kPa], CPAP 5 cm H2O [0.5 kPa], and $F_{1O_2}$ 0.35; and arterial blood analysis revealed pH 7.41, $P_{ACO_2}$ 39 torr [5.2 kPa], $P_AO_2$ 84 torr [11.2 kPa], BE 0.1 mEq/L [0.1 mmol/L], $HCO_3^-$ 25 mEq/L [25 mmol/L], and $S_O_2$, 97%. The patient’s condition progressively improved and he was extubated 3 days following discontinuation of TPN.

REFERENCES

Exertional Dyspnea in a Patient with Chronic Urinary Tract Infection

Dominic P Coppolo RRT and James I Couser MD

A 69-year-old woman was referred to our hospital for evaluation of exertional dyspnea. Minor foot surgery had been performed under local anesthesia 3 weeks earlier. She noticed during her recuperation that she could not climb one flight of stairs without feeling short of breath. She denied fever, cough, chest pain, or orthopnea.

She did not have a history of occupational exposure to organic dusts, chemicals, or asbestos nor did she have a history of rheumatic disease. She did have a history of hypertension, chronic bladder infections, and osteoarthritis. She also had a 50-pack-year history of cigarette smoking but had not smoked for 18 years. Her prescribed medications included nitrofurantoin (which she had been taking for 2 years), ibuprofen, and clonidine.

Physical examination revealed that her temperature was 37.6 °C, blood pressure 130/82 torr, pulse 82 beats/min, and respiratory rate 16 breaths/min. Auscultation revealed fine crackles in both lung bases. Analysis of arterial blood drawn while the patient was at rest and breathing room air revealed: pH 7.38, PaCO₂ 34 torr [4.5 kPa], and PaO₂ 60 torr [8.0 kPa]. Oxygen saturation (SpO₂) measured by pulse oximetry was 91% while the patient was at rest and 73% after the patient had walked 100 yards. Complete blood count was normal with no eosinophilia. Erythrocyte sedimentation rate was 64 mm/h. Rheumatoid factor was negative. Pulmonary function testing showed that the forced vital capacity (FVC) was 65% of predicted, total lung capacity (TLC) 69% of predicted, residual volume (RV) 77% of predicted, and diffusion capacity (DLCO-sb) 59% of predicted. The chest radiograph is shown in Figure 1.

Fig. 1. Initial chest radiograph of a 69-year-old woman complaining of exertional dyspnea.

Questions

Radiographic Findings: What are the abnormalities on this chest radiograph?
Diagnosis: What is the probable diagnosis? What further actions or tests will be necessary to make the diagnosis?
Treatment: What treatment is indicated?

Answers and Discussion on Next Page

Mr Coppolo is Chief, Respiratory Care Services, The Mary Imogene Bassett Hospital; Dr Couser is Attending Physician, Pulmonary Medicine, The Mary Imogene Bassett Hospital, and Assistant Professor of Clinical Medicine, College of Physicians and Surgeons, Columbia University—Cooperstown, New York.
Answers

Radiographic Findings: The chest radiograph shows increased diffuse interstitial markings, with honeycombing in both lungs. Findings are greater on the right than the left. There is associated bilateral pleural thickening.

Diagnosis: The probable diagnosis is drug-induced interstitial pulmonary fibrosis from the use of nitrofurantoin—'Macrobidantin Lung.' Open lung biopsy was performed and confirmed this diagnosis.

Treatment: The appropriate course of action is to discontinue the drug, administer oxygen to treat hypoxemia, and consider prescribing systemic corticosteroids.

Discussion

Nitrofurantoin (Macrobidantin) is one of more than 40 pharmacologic agents that have been associated with some form of pulmonary toxicity.\(^1\) It is a widely prescribed antibiotic used for the prophylactic treatment of recurrent urinary tract infections. In 1962 the pulmonary toxicity of nitrofurantoin was first reported,\(^2\) and more than 500 cases of adverse pulmonary reactions have been reported since then.\(^3\) Although the incidence of pleuropulmonary reactions to nitrofurantoin is unknown, Hailey et al reported 237 cases occurring in an estimated 44 million courses of therapy.\(^3\)

Pulmonary reactions to nitrofurantoin occur in two distinct patterns, acute and chronic. In the more common acute syndrome, patients present with fever, dyspnea, chills, chest pain, nonproductive cough, and leukocytosis. Pulmonary function tests characteristically show a restrictive ventilatory defect with a reduced Dl,CO. The chest radiograph in the acute phase of this toxicity can be normal, but more often demonstrates bilateral lower-lobe interstitial infiltrates, sometimes accompanied by pleural effusions.\(^3\),\(^4\)

This case represents the chronic syndrome of nitrofurantoin pulmonary toxicity. In the chronic syndrome, symptoms may be more insidious and may include dyspnea and nonproductive cough, however fever and blood eosinophilia are not seen. Pulmonary function tests show a restrictive pattern. Bibasilar interstitial infiltrates are the most common roentgenographic findings.\(^3\),\(^4\)

The differential diagnosis of diffuse interstitial infiltrates encompasses a large number of diverse entities including drug reactions. Drugs that cause chronic pneumonitis or fibrosis as seen in this case include nitrofurantoin, amiodarone, penicillamine, gold salts, tocinamide, and cytotoxic agents such as bleomycin and cyclophosphamide.\(^1\) Drug-induced pulmonary disease is often suggested by the history.

Diagnostic studies performed in the evaluation of patients with interstitial infiltrates include serologic tests for hypersensitivity pneumonitis, lung scanning with gallium-67 citrate, and bronchoalveolar lavage. Although these studies may provide clues to diagnosis, histologic confirmation is usually required.\(^5\) Transbronchial biopsy is performed when a disorder is suspected for which histologic diagnosis can be made on the basis of analysis of small bits of tissue (eg, sarcoidosis, lymphangitic carcinoma, opportunistic infection).

Diagnosis in this case was made by open-lung biopsy. Histopathologic findings may be similar in the acute and chronic forms of the disease. These include interstitial and alveolar inflammation with a variable amount of eosinophilia. Fibrosis is generally more prominent in the chronic syndrome.\(^3\)

Treatment for either form of this disease includes the immediate discontinuation of the drug. Patients with the acute syndrome usually recover completely. Complete or partial resolution of symptoms and radiographic abnormalities may occur in patients with chronic nitrofurantoin toxicity when the drug is stopped. Mortality with the chronic form of the syndrome may be as high as 10%.\(^3\),\(^4\)

In our patient, nitrofurantoin was discontinued, home oxygen was prescribed, and systemic corticosteroids were administered. Although there are little data on the efficacy of using corticosteroids to treat chronic nitrofurantoin toxicity, their use has hastened recovery in some patients.\(^3\),\(^6\),\(^10\) We chose to use corticosteroids due to the severity of radiologic changes and physiologic impairment in our patient.

During the 6 months subsequent to the open-lung biopsy and initiation of therapy, symptomatic and physiologic improvement occurred: Steroids were gradually tapered off; FVC and TLC increased to 103% and 81% of predicted, respectively; S\(_{\text{PO}}\) improved to 96% while the patient was at rest and breathing room air, and to 91% after the patient had walked 100 yards; home oxygen was discontinued; and improvement was seen on radiograph (Fig. 2).
Fig. 2. Chest radiograph of a 69-year-old woman 6 months after diagnosis of 'Macrodantin Lung' had been confirmed by open-lung biopsy and appropriate treatment initiated.

REFERENCES


Instructions for Authors

In addition to case reports strictly involving pulmonary-medicine radiography, case reports involving adult, pediatric, and neonatal critical-care radiography can be submitted to the 'Test Your Radiologic Skill' corner. However, all case reports should relate somehow to respiratory care. Illustrative radiographs may be of routine chest exams or of other less-common exams, such as digital subtraction or computerized axial tomography.

Given the inconsistency among respiratory care practitioners in identifying and reporting breath sounds,1 we believe that it is appropriate to review this new idea in breath-sounds training material. RALE is a computer-assisted instruction (CAI) package for teaching chest auscultation and lung sounds. The authors, from the Departments of Pediatrics and Electrical Engineering of the University of Manitoba, have digitally recorded a wide variety of normal and abnormal lung sounds that can be played on a personal computer (PC) in an interactive instructional mode.

The package consists of an audio-output PC interface card, high-quality headphones, a bound manual, and software diskettes that are not copy-protected. Utilization requires an IBM PC, AT, PS/2 Model 30, or a 100%-compatible PC with an XT-type slot. 5 megabytes on a hard disk, and an EGA or VGA display (preferably color). We evaluated the package using an IBM PS/2 Model 30-286 with a color VGA display.

The startup screen looks like the table of contents of a book, which quickly made us feel comfortable with the software. It provides chapter headings and subheadings or you can turn right to the desired page. The presentation is intuitive and provides an excellent example for others interested in writing CAI.

The first chapter, "How To Use This Program," covers basic navigational rules. The use of directional keys is obvious and a small menu is always available at the bottom of the screen with the other appropriate commands. At any time, pressing "C" returns you to the table of contents page where you may select another chapter. Context-sensitive help screens explain features and options.

An excellent tutorial is provided in the second chapter on the physics of acoustics as it relates to breath sounds. Frequency, amplitude, harmonics, noise, and pitch perception are discussed with both audible and visual demonstrations.

The third chapter explains how lung sounds are generated and how they are changed (attenuated) as they pass through lung tissue and chest wall. The best feature, generally missing from audiotape instructional material, is the simultaneous display of airflow, sound wave, and sonogram, presented synchronously with the actual lung sounds. The audible and visual information together clarifies the concepts presented on each page more adequately than does a simple audiotape. The compressed sound wave display shows the relative loudness of each portion of the tracing. The sonogram shows the frequency and sound intensity in a pattern similar to a "voice print," with constituent frequencies and their intensity displayed using high-resolution color graphics.

The sonogram, with its frequency-component display, provides much more information than does a simple sound wave, such as those found in an earlier paper by Wilkins and Dexter in this journal. For example, it is difficult to visually differentiate monophonic and polyphonic wheezes from a sound wave, whereas the sonogram distinctly shows the multiple components.1

The user may freely flip back and forth between pages, enabling easy comparison of subtle differences in sounds. The sound wave display may be expanded by zooming in to expand an area of interest—helpful when rapid respiratory rate or a few crackles are present.

Two additional tools are available as needed—the ability to show a 100-ms portion of the waveform expanded as it would be seen on an oscilloscope and a spectrum analyzer by Fourier transform. The oscilloscope view is simply a 100-ms expansion of the sound wave similar to the zoom-in function, except that it does not use a variable time base. The spectrum analyzer displays frequency against amplitude. Sounds that have only a few frequency components (such as wheezes) have distinct peaks, whereas other noises are composed of many intermixed frequencies. (The powerspectrum display converts time domain to frequency domain.)

Normal sounds are included from simultaneous recordings over the trachea and chest wall, and you can readily see the attenuation of sound as it passes through the soft tissue in the chest. Several examples of breath sounds from babies are provided with the electrocardiogram also displayed, so that the cardiac sounds can be readily distinguished from breath sounds. The names of adventitious lung sounds conform with ATS recommended terminology.1 Wheezes are continuous musical sounds, whereas crackles are noncontinuous sounds of short duration. The combined audiovisual presentation graphically reinforces the differences. The spectral display of monophonic wheezes also makes them easy to distinguish from lower-pitched rhonchi. Examples of stridor, grunting, squeaks, and friction rubs are also included.

The digitized sound reproduction is of such high quality that you can actually hear typical background noises from the room in which the recordings were made. In several examples of newborn and pediatric breath sounds, other children can be distinctly heard in the background.

The fourth chapter presents four case studies: asthma, wheezy infants, pneu-
TAKE THE CHALLENGE...

PULL THE PLUG ON YOUR VENTILATOR!

TAKE THE CHALLENGE...

Will your present ventilator stand up to the ADULT STAR CHALLENGE? Try the first in a series of head-on challenges and find out why you should contact Infrasonics today!

Challenge #1...with your ventilator operating on wall air and oxygen, disconnect the electrical cord. If it stopped operating, it failed the challenge.

The same test performed on ADULT STAR will continue operating for up to 30 minutes. ADULT STAR's battery operation eliminates electrical failure, effects of brown outs and insulates the internal microcomputers from electrical surges and current fluctuation.

Adult Star® Ventilator "Plus Much More"
Call 1-800-STARVENT now for more information and be watching for Challenge #2 coming soon.

Infrasonics, Inc.
A Public Company
9944 Barnes Canyon Road, San Diego, CA 92121 U.S.A.
619 / 450-9898, 1-800-STARVENT
FAX: 619 / 450-4372
Patent Pending

Visit AARC Booth 1120 in New Orleans
mona, and interstitial fibrosis. Most of the examples in this chapter are recorded from two sites at the same time. Helpful text comments are included that explain the probable sources of the sounds.

The final chapter is a quiz that randomly presents a group of additional breath sounds and provides checkoff boxes to record answers. The sounds can be repeatedly replayed, and the answers are provided when the participant requests them.

Additions to this package that we believe would further expand its utility for educators would include more case studies, more quiz questions, and the ability to store quiz results for a number of students.

We highly recommend this package as an excellent, inexpensive educational tool for all respiratory therapy and medical schools. Reach for your purchase-order requisition book or credit card before the authors realize that they could get three times as much for this package. It is the best CAI program in pulmonary medicine that we have ever seen.

Steve Nelson MS RRT
Paul Enright MD
Mayo Clinic
Rochester, Minnesota

REFERENCES
1. Wilkins RL, Dexter JR. Comparing RCPs to physicians for the description of lung sounds: Are we accurate and can we communicate? Respir Care 1990;35:969-976.
2. Wilkins RL, Dexter JR, Smith MP, Marshak AB. Lung-sound terminology used by respiratory care practitioners. Respir Care 1989;34:36-41.


The death of master puppeteer Jim Henson shocked many Americans who found themselves wondering how a previously healthy man could die from bacterial pneumonia in this age of miracle antibiotics and technology. Of course, medical personnel know that pneumonia remains a major health problem in the U.S.—it accounts for nearly 10% of hospital admissions, is the fifth leading cause of death, and is the most common cause of death from infection. Physicians, nurses, and respiratory care practitioners realize that they must always keep informed about respiratory infections.

This book conveniently bridges the gap between the limited information found in general medicine textbooks and the overwhelming information found in some encyclopedic infectious disease textbooks. The 49 contributors are experts, many of whom are well-known contributors to the aforementioned infectious disease texts. [The list of contributors understates remarkably the expertise of the authors!] Thirty-eight chapters are organized into five sections. The first section discusses the pathogenesis of infection with chapters on host defenses, bacterial colonization, and pulmonary clearance mechanisms. The next section reviews noninvasive and invasive diagnostic techniques (eg, the sputum smear, immunofluorescent stains, genetic probes, bronchoalveolar lavage, and lung biopsy techniques). The third section contains 12 chapters that address common clinical settings (eg, infections in the upper-respiratory tract in infants, in the elderly, in the immunocompromised, and in patients with AIDS). The 17 chapters in the fourth section cover specific infectious agents either grouped or individually (eg, gram-positive bacteria, gram-negative bacteria, tuberculosis, and Pneumocystis carinii). The final section considers the specifics of therapy, such as supportive measures, pharmacokinetics of antibiotics in respiratory secretions, and rational use of new antibiotics.

In general, each chapter is written to stand alone, which leads to some repetition that I found helpful. The authors have presented fairly the pros and cons of their approach. There is liberal use of tables and pertinent references; however, some tables are simply too crowded and lack explanatory legends, there is a paucity of figures, several chest radiographs are reproduced poorly, and there is questionable use of black and white photomicrographs to show stained sputum and lung histopathology. These shortcomings suggest an overly tight budget, and typographical errors indicate suboptimal copyediting. It is hoped these distractions will be corrected in future editions.

Busy clinicians, pulmonary and infectious disease trainees, nurses, and respiratory care practitioners will find this book very useful when working with respiratory infections. A sad fact of life is that medical journals and books have been afflicted by marked inflation—the cost of some books has doubled since their last edition! At first glance, this medium-size text seems fairly expensive, but consider the following: the cost is less than half that of an infectious disease tome and roughly equal to an annual subscription for a subspecialty journal. The convenience of learning quickly and honestly how (and why) the experts would approach a respiratory infection is worth the cost. Pulmonary physicians will want to purchase this text, while most nurses and respiratory care practitioners will insist that it be available in the hospital library.

John W Shigeoka MD
Chief, Pulmonary Section
Veterans Administration Medical Center
Salt Lake City, Utah
NOW, THERE'S MORE THAN HOPE FOR INFANTS WITH RESPIRATORY DISTRESS SYNDROME
New, protein-free synthetic lung surfactant that's as easy to use as it is effective

Exosurf® NEONATAL
(Colfosceril Palmitate, Cetyl Alcohol, Tyloxapol) For Intratracheal Suspension

At last, there's more than hope for infants with respiratory distress syndrome (RDS). Clinical trials have shown that protein-free synthetic EXOSURF Neonatal dramatically reduced neonatal morbidity and mortality. In addition to being effective in both prophylactic and rescue use, EXOSURF Neonatal was well tolerated.

Widely studied
To date, in excess of 2,600 premature infants have received EXOSURF Neonatal in controlled clinical trials involving more than 4,400 infants in North America. In addition, 10,000 infants in more than 400 hospitals have received EXOSURF Neonatal under a treatment IND.
Effective in infants at risk of developing RDS

A single, prophylactic dose of EXOSURF Neonatal given immediately following birth reduced death from RDS by 50% and one-year mortality by 33% in neonates weighing 700 to 1100 grams. Two additional prophylactic doses of EXOSURF Neonatal reduced one-year mortality by an additional 30%. EXOSURF Neonatal reduced the severity of RDS and the incidence of lung rupture in these premature infants.

Effective in infants with RDS

In infants weighing 700 to 1350 grams, EXOSURF Neonatal rescue treatment initiated within 24 hours of birth, reduced death from RDS by 66% and one-year mortality by 44%. Survival to day 28 without bronchopulmonary dysplasia was increased significantly. Pneumothorax, pulmonary interstitial emphysema, and overall pulmonary air leaks were significantly reduced. Similarly beneficial effects were also observed in infants with RDS weighing >1350 grams, and chronic lung disease was significantly reduced.

Impressive safety profile

In individual controlled clinical trials, adverse events were comparable to those of placebo, with the exception of apnea. Infants receiving EXOSURF Neonatal required less ventilatory support, possibly contributing to an increased incidence of apnea. In both placebo and treated infants, apnea proved to be a marker for reduced pulmonary air leak and improved survival.

In the treatment IND experience of over 10,000 infants, the reported incidence of pulmonary bleeding was 4%. It appears to be related to improvements in pulmonary function in infants whose ductus arteriosus remains patent. This condition may be prevented by early and aggressive diagnosis and treatment (unless contraindicated) of patent ductus arteriosus during the first two days of life (while the ductus arteriosus is often clinically silent). Additionally, a low incidence (3/1,000) of mucous plugging of the endotracheal tube was observed.

Please see full prescribing information on last pages of this advertisement.
Easy to store

- EXOSURF Neonatal may be stored at room temperature (15° to 30°C [59° to 86°F]).
- Reconstituted suspension may be maintained refrigerated or at room temperature (2° to 30°C [36° to 86°F]) for up to 12 hours.

Easy to use

- Key items needed for EXOSURF Neonatal administration are supplied in one carton: one 10 mL vial of EXOSURF Neonatal, one 10 mL vial of Sterile Water for Injection, and five endotracheal tube adapters (2.5 mm, 3.0 mm, 3.5 mm, 4.0 mm, and 4.5 mm).

Easy to administer

- Each EXOSURF Neonatal dose is administered in two 2.5 mL/kg half-doses.
- EXOSURF Neonatal is administered via a sideport on a special endotracheal tube adapter (supplied with EXOSURF Neonatal) without interrupting mechanical ventilation.

Easy on the infant

- To assist the distribution of EXOSURF Neonatal in the lungs, the infant is simply turned from midline position to the right after the first half-dose and from midline position to the left after the second half-dose.

A complimentary videotape on reconstitution and administration of EXOSURF Neonatal is available from your Burroughs Wellcome Co. representative.

Please see full prescribing information on last pages of this advertisement. Call your Burroughs Wellcome Co. professional representative for further information.
**EXOSURF**

**Colfosceril Palmitate, Cetyl Alcohol, Tyloxapol**

**Neonatal For Intratracheal Surfactant**

**Descripción**: Exosurf neonatal for Intratracheal Surfactant is a protein-free synthetic long surfactant stored under vacuum as a sterile lyophilized powder. It can be reconstituted with sterile water for injection prior to administration. When reconstituted, it contains 10 mg/mL of colfosceril palmitate and 5 mg/mL of cetyl alcohol.

**Clinical results**: In these six controlled clinical studies, infants in the Exosurf neonatal group showed significant improvements in disease characteristics and parameters such as lung compliance and oxygenation. In studies comparing Exosurf with placebo, neonates who received Exosurf neonatal demonstrated significantly improved lung compliance and oxygenation compared to the placebo group.

**indications and usage**: Exosurf neonatal is indicated for:
1. Prophylactic treatment of infants with birth weights of less than 1500 grams who are at risk of developing RDS (see PRECAUTIONS).
2. Prophylactic treatment of infants with birth weights greater than 1500 grams who have evidence of pulmonary maturity.
3. Rescue treatment of infants who have developed RDS.

**Contraindications**: There are no known contraindications to treatment with Exosurf neonatal.

**Warnings**: Intratracheal Administration Only: Exosurf neonatal should be administered only by intratracheal (see DOSAGE AND ADMINISTRATION).

**General**: The use of Exosurf neonatal requires expert clinical care by experienced neonatologists and other clinicians who are acclimated to neonatal intubation and ventilatory management. Adequate personnel, facilities, equipment, and medications must be available for resuscitation.

**Acute effects**: Exosurf neonatal can rapidly affect oxygenation and lung compliance. Lung function: If chest expansion improves substantially after dosing, peak ventilator inspiratory pressures should be reduced immediately, without waiting for confirmation of respiratory improvement by blood gas analysis.

**Pulmonary Hemorrhage**: In the single study conducted in infants weighing <500 grams at birth, the incidence of pulmonary hemorrhage (10% vs 31% in the placebo group) was significantly reduced in the Exosurf neonatal group. None of the five studies involving infants with birth weights >1020 grams showed a significant increase in pulmonary hemorrhage in the Exosurf neonatal group; in a cross-study analysis of these five studies, pulmonary hemorrhage was reported for (14% (14/102) of infants in the placebo group and 3% (7/111) of infants in the Exosurf neonatal group. Prolonged lung hemorrhage occurred only in the Exosurf neonatal group and in the placebo group. In one infant, mortality was (13%) in the placebo group and 3.7% in the Exosurf neonatal group. Pulmonary hemorrhage in both Exosurf neonatal and placebo groups was more frequent in infants who were younger, smaller, more premature, and a patent ductus arteriosus. Pulmonary hemorrhage typically occurred in the first 2 days of life in both treatment groups.

**Acute toxicity**: Infants whose ventilation becomes maximally impaired during or shortly after dosing may have mucous plugging of chest radiographic views, which is usually clinically silent. Other potentially protective measures include frequent radiographic examinations of the chest to monitor for signs of air leak.

**Precautions arch**: In the neonatal period, infants are more susceptible to lung injury and death from air leak. Thus, early diagnosis and intervention are crucial. For infants who are not intubated or ventilated, the effects of Exosurf neonatal on the neonatal lung are not known. Therefore, the safety and efficacy of these additional doses are not available in placebo-controlled trials. Carcinogenesis, Mutagenesis, Impairment of Fertility: Exosurf neonatal at concentrations up to 10,000 µg/mL was not mutagenic in the Ames Salmonella assay.

**Adverse reactions**: Premature birth is associated with a high incidence of morbidity and mortality. Despite significant reductions in overall mortality and morbidity, Exosurf neonatal did not provide any additional benefit to infants who received Exosurf neonatal developed severe complications and/or survived with permanent handicaps or died.

**Clinical studies**: In controlled clinical studies evaluating the safety and efficacy of Exosurf neonatal, numerous safety assessments were made. The results of these assessments are presented in the Clinical Studies section. A number of adverse events were significantly reduced in the Exosurf neonatal group, particularly various forms of pulmonary air leak and use of ventilator treatments (see CLINICAL PHARMACOLOGY: Clinical Results). Tables 3 and 4 summarize the results of the infant survival evaluation using the endpoint clinical score.
<table>
<thead>
<tr>
<th>Treatment Group</th>
<th>Number of Infants</th>
<th>Placebo (Av)</th>
<th>EXOSURF (Av)</th>
<th>Placebo (N=126)</th>
<th>EXOSURF (N=125)</th>
<th>Placebo (N=121)</th>
<th>EXOSURF (N=121)</th>
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<tr>
<td>Interventricular Hemorrhage (VHI)</td>
<td>Overall</td>
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<td>187</td>
<td>172</td>
<td>183</td>
<td>171</td>
<td>182</td>
</tr>
<tr>
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<td>2</td>
<td>6</td>
<td>4</td>
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<td>5</td>
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<tr>
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<td>16</td>
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<td>4</td>
<td>9</td>
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<td>12</td>
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<td>10</td>
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<tr>
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<td>6</td>
<td>5</td>
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<td>6</td>
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<tr>
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<td>Congenital Pneumonia</td>
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<td>2</td>
<td>3</td>
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<td>3</td>
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<tr>
<td></td>
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<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
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</table>

* All parameters were examined with a "t"-test. * Analysis of variance using placebo as the reference group. * Analysis of variance using placebo as the reference group.

<table>
<thead>
<tr>
<th>Safety Assessment</th>
<th>500 to 750 g</th>
<th>750 to 1250 g</th>
<th>1250 g to 1500 g</th>
<th>1500 g to 2000 g</th>
<th>2000 g to 2500 g</th>
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<tbody>
<tr>
<td>Incidence of Interventricular Hemorrhage</td>
<td>Overall</td>
<td>178</td>
<td>178</td>
<td>173</td>
<td>171</td>
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<table>
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<tr>
<th>Events During Dosing in the Open, Uncontrolled Study</th>
<th>EXOSURF</th>
<th>Placebo</th>
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<tbody>
<tr>
<td>Rebleed of Extremity</td>
<td>21</td>
<td>35</td>
</tr>
<tr>
<td>Drop-in O2 Saturation (&lt; 90%)</td>
<td>6</td>
<td>5</td>
</tr>
<tr>
<td>Drop in O2 Saturation (&lt; 85%)</td>
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<td>5</td>
</tr>
<tr>
<td>Increase in ICP &gt; 10 mm Hg</td>
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<td>5</td>
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<tr>
<td>Increase in ICP &gt; 20 mm Hg</td>
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<td>5</td>
</tr>
<tr>
<td>Increase in ICP &gt; 30 mm Hg</td>
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<td>4</td>
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<tr>
<td>Increase in ICP &gt; 40 mm Hg</td>
<td>1</td>
<td>3</td>
</tr>
</tbody>
</table>

* * Analysis of covariance using placebo as the reference group. * Analysis of covariance using placebo as the reference group.

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AARC & AFFILIATES

October 19 in Long Island, New York. The Southeastern Chapter of the New York State Society for Respiratory Care presents its 32nd Annual Symposium, "Into the Future," at the Marriott Hotel, Uniondale NY. Speakers include John Back MD—Alternate Forms of Positive Pressure Ventilation; Michael McPeck RRT—A Glimpse of the Soviet Health Care System; Gary Pettett MD—New Advances in Transport of the Ventilated Infant; Daniel Draper MS—Techniques in Diagnosing Sleep Disorders; Mark McCauley MS RRT—Pressure Control-Inverse Ratio Ventilation; Karen Larson BS RRT—Entrepreneurial Management in the Decade Ahead and Respiratory Care Services and the Joint Commission; and John Rowite MD—Pulmonary Rehabilitation: What's New and What's on the Horizon. Contact Ken Axton RRT at (516) 444-3180.

October 30-November 1 in Sturbridge, Massachusetts. The Massachusetts Society for Respiratory Care presents its 13th Annual Meeting at the Sheraton Sturbridge Resort and Conference Center. Topics include cardiopulmonary diagnostics, mechanical ventilation, lung transplantation, research, COPD, sleep apnea, surfactant replacement therapy, ARDS, cystic fibrosis, noninvasive monitoring, respiratory muscle function, AIDS, pressure control ventilation, asthma, home care, and a legislative update. Social events include a masquerade party, golf tournament, walkathon, awards banquet, and vendor reception. Contact Susan Harding RPFT RRT, 1072 High St, Bridgewater MA 02324, (503) 295-0880.

November 2 in Jackson, Mississippi. The MSRC presents a mini-seminar on the ABCs of neonatal/pediatric critical care. This meeting will be at the Holiday Inn-Medical Center. For more information, contact Donna Lindsey CFFT RRT, Northeast MS Community College, Cunningham Blvd, Booneville MS 38829. (601) 728-7751, ext 387.

November 2 in West Point, New York. The Hudson Valley Chapter of the New York State Society for Respiratory Care presents its annual educational seminar at The Hotel Thayer, U.S. Military Academy, West Point NY. The topic for the one-day event is "Ventilator Trends in the 1990s." Speakers include Robert Kacmarek PhD RRT and Norma Braun MD. Tours of the U.S. Military Academy, the scenic Hudson River and fall foliage, and a buffet lunch complement the event. Contact Mike Aiello RRT, Box 150, Glenham NY 12527. (914) 795-5340.

November 9 in Reno, Nevada. The Nevada Society for Respiratory Care and the American Lung Association of Nevada present the 9th Annual Respiratory Health Care Conference at the Washoe Medical Center. The topic of the Conference is "Respiratory Illness Across the Lifespan." Contact Barbara Rottstein at (702) 829-5864.

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

February 21-22 in Kansas City, Missouri. The Kansas and Missouri Respiratory Care Societies, in conjunction with the Respiratory Care Managers Association (KS/MO), sponsors the 3rd Annual Midwest Respiratory Care Symposium at Baptist Medical Center, 6601 Rockhill Rd, Kansas City MO 64131. Contact Clarissa Craig, Program Director, Respiratory Therapy Education, Johnson County Community College, 12345 College at Quivira, Overland Park KS 66210-1299. (913) 469-8500, ext 3939.

OTHER MEETINGS

October 26-28 in Savannah, Georgia. The Armstrong State College Respiratory Therapy faculty sponsors an intensive Registry Preparation Course. Call Coastal Georgia Center for Continuing Education by Oct 16 for details at (912) 927-5322.

November 2 in U.S.A. The AARC and the Annenberg Center for Health Sciences-Eisenhower Medical Center present the 3rd Video Teleconference, "New Approaches to Asthma." Broadcast times are Eastern 10:30 to 11:30 AM, Central 9:30 to 10:30 AM, Mountain 8:30 to 9:30 AM, and Pacific 7:30 to 8:30 AM. Contact the AARC at (214) 243-2272.

November 2 in Atlanta, Georgia. St. Joseph's Hospital of Atlanta presents its 7th Annual Respiratory Care Seminar. Contact Rena Cobb RRT RCP, Education Coordinator, Respiratory Care, St. Joseph's Hospital of Atlanta, 5665 Peachtree Dunwoody Rd, Atlanta GA 30342. (404) 851-7176.

April 14-21 Mexican Riviera Cruise. "Each One, Teach One" is the theme for this year's spring cruise for 8 education units (CEU). Cruise only $775 for prepaid, double occupancy. Call (800) 462-3628; or write Dream Cruises, 10882 La Dona Ave, Garden Grove CA 92640.

October 24-29, 1993 in Israel. The XIV World Congress of the International Association of Asthmaology hosts INTERASMA 93—XIV World Congress of Asthmaology in Israel. Contact President Israel Glazer MD RH, Bognashov 89, Tel-Aviv 63297, Israel. (03) 282925.
Notices

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

ARCF Literary Award

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

AARC ANNUAL CONVENTION SITES & DATES

1990—New Orleans, Louisiana, December 8-11
1991—Atlanta, Georgia, December 7-10
1992—San Antonio, Texas, December 12-15
1993—Nashville, Tennessee, December 11-14
1994—Las Vegas, Nevada, December 12-15
1995—Orlando, Florida, December 2-5

THE NATIONAL BOARD FOR RESPIRATORY CARE

1990 Examination and Fee Schedule

**CRTT Examination**

EXAMINATION DATE: NOVEMBER 10, 1990
Applications Accepted Beginning: July 1, 1990
Application Deadline: September 1, 1990

**RRT Examination**

EXAMINATION DATE: DECEMBER 1, 1990
Applications Accepted Beginning: June 1, 1990
Application Deadline: August 1, 1990

**RPFT Examination**

EXAMINATION DATE: DECEMBER 1, 1990
Applications Accepted Beginning: July 1, 1990
Application Deadline: September 1, 1990

**Fee Schedule**

Entry Level CRTT—new applicant: $75.00
Entry Level CRTT—reapplicant: $50.00

Combined RRT Written and Clinical Simulation: $175.00
Written Registry Only—new applicant: $75.00
Written Registry Only—reapplicant: $50.00
Clinical Simulation Only—new or reapplicant: $100.00
Entry Level CPFT—new applicant: $100.00
Entry Level CPFT—reapplicant: $80.00
Advanced RPFT—new applicant: $150.00
Advanced RPFT—reapplicant: $130.00
CRTT Recredentialing: $25.00
RRT Recredentialing: $25.00
Written Registry Examination: $25.00
Clinical Simulation Examination: $65.00
CPFT Recredentialing: $25.00
RPFT Recredentialing: $90.00

Membership Renewal
CRTT/RRT/CPFT/RPFT: $12.00
Membership Renewal
Combination of CRTT/RRT and CPFT/RPFT: $18.00

8310 Nieman Road • Lenexa, Kansas 66215 • (913) 599-4200
Instructions for Authors and Typists

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

General Requirements

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

Publication Categories

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• Number pages in upper right corner and leave margins of 1½” or more on all four sides of the page.
• For research articles, follow format of Model Manuscript, Respir Care 1984;29:182 (Feb 1984).
• Meticulously follow instructions for typing references.

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

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

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

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1013
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- For the reference list, obtain author names, article and book titles, dates, volume and page numbers from the original cited articles and books, not from secondary sources such as other articles' reference lists, which often are inaccurate.
- Type references in medical-journal style. Examples appear at the end of these Instructions. Abbreviate journal names as in Index Medicus. A list of many journal-name abbreviations was published in Respir Care 1988;33:1050 (Nov 1988).
- DOUBLE-SPACE the lines of references.
- List ALL authors' names. Do not use "et al," to substitute for names.
- Identify abstracts, editorials, and letters as such. See examples.


Examples of How To Type References

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6. Are references typed in requested style?
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PULSE OXIMETER. The 2000 pulse oximeter and the SureSAT line of reusable reflectant sensors are designed to perform monitoring under difficult conditions. According to the manufacturer, the signal processing system of the 2000 minimizes interference by ambient light, motion, and other noise, by filtering the signal before it is amplified; and the reflectant sensors, which can be attached to the forehead, facilitate monitoring in high motion environments and in patients with low peripheral perfusion. Sentinel Monitoring Inc, Dept RC, 6530 Corporate Dr, Indianapolis IN 46278. (317) 328-4530.

POWDER-FREE LATEX GLOVES. According to the manufacturer, Safeskin powder-free latex gloves (examination, sterile surgical, and procedure) are hypoallergenic, durable, cost-effective, and meet or exceed all FDA 510K requirements and ASTM standards. The manufacturer states that their powder-free gloves protect the wearer’s hands from the dryness associated with powder; the patients from postoperative complications such as adhesions, granulomas, and inflammations caused by powder entering open wounds during surgical procedures; respiratory and neonatal patients from risks associated with the presence of powder in a sterile air environment; and laboratory procedures and bar-coding equipment from adverse effects. The sterile surgical gloves are available in sizes 5½ thru 9, and the examination and sterile procedure gloves (singles and pairs) are available in extra-small, small, medium, and large sizes. For further information and a free sample, contact Safeskin Corporation, Dept RC, 12520 High Bluff Dr, Suite 218, San Diego CA 92130. (800) 456-8379.

DUST MITE TEST AND TREATMENT PRODUCTS. According to the manufacturer, the easy-to-administer Acarex dust mite test enables consumers to determine the level and location of dust mite infestation in their homes, and the EPA-approved, non-toxic Acarosan treatment dust (active ingredient Benzyl Benzoate) effectively kills mites and larvae and encapsulates the waste upon contact—creating a dust-mite-free environment that lasts approximately six months. Acarex is sold in four packets ($10.00) and ten packets ($15.00); Acarosan is sold in a standard-sized, three-bag kit (one bag per room) and retail for about $75.00. Both test and treatment products are manufactured by Werner & Mertz GmbH, Division of Environmental Medicine, Mainz, West Germany; sold by Fisons Pharmaceuticals, 755 Jefferson Rd, Rochester NY 14623; and available through allergy specialists and local pharmacies. For additional information, call the Allergy Information Center and Hotline at (800) 727-5400.

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Authors in This Issue

Barnes, Thomas A .......................................................... 960
Branson, Richard D .......................................................... 952, 1001
Campbell, Robert S .......................................................... 952, 1001
Coppolo, Dominic P .......................................................... 1003
Couser, James I .............................................................. 1003
Dexter, James R .............................................................. 969
Enright, Paul ................................................................. 1006
Green, Christopher G ...................................................... 997
Hall, Richard ................................................................ 977
Hess, Dean .................................................................. 945
Hurst, James M .............................................................. 952, 1001
Johannigman, Jay A ......................................................... 952
Kacmarek, Robert M .......................................................... 945
Larach, Marilyn G ............................................................. 949
Mathewson, Hugh S .......................................................... 987
McGarry, William P III .................................................... 960
Nelson, Steve ................................................................ 1006
O’Donohue, Walter J ........................................................ 990
Petty, Thomas L ................................................................ 990
Rankin, Ronald F .............................................................. 997
Shigecoka, John W ............................................................ 1008
Vakharia, Narendra .......................................................... 977
Wilkins, Robert L ............................................................. 969

Advertisers in This Issue

Allen & Hanburys ............................................................. 1000a
Burroughs Wellcome Co .................................................. 1008a, 1009, 1010
CNS ................................................................................. Cover 3
DHD Medical Products .................................................... Cover 4
HealthScan Products .......................................................... 941
Impact Medical Corp. .......................................................... 996
Infrasonics Inc ................................................................. 1007
Lifecare ........................................................................ 932
Medical Equipment Designs .............................................. 935
Netleor ........................................................................ 942
Ohmeda ........................................................................ 944
PPG Sara ...................................................................... 939, 943
Puritan-Bennett ................................................................. Cover 2, 929
Quinton Instrument Co ..................................................... 930
Siemens Life Support Systems ........................................... 937

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York Hospital, York PA ....................................................... 936
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81 82 83 84 85 86 87 88 89 90 91 92 93 94 95 96 97 98 99 100

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81 82 83 84 85 86 87 88 89 90 91 92 93 94 95 96 97 98 99 100

101 102 103 104 105 106 107 108 109 110 111 112 113 114 115 116 117 118 119 120

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