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# CONTENTS

## EDITORIALS

<table>
<thead>
<tr>
<th>Number</th>
<th>Title</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>17</td>
<td>Pulse Oximetry in the Neonatal Intensive Care Unit</td>
<td>by John W Sayer RRT—Cleveland, Ohio</td>
</tr>
<tr>
<td>21</td>
<td>Research-Protocol Review: A New Editorial Service</td>
<td>by Phil Kittredge, Little River, California</td>
</tr>
<tr>
<td>23</td>
<td>How to Write a Concrete Abstract, Or Avoiding the Common Mistakes</td>
<td>of Would-Be Open Forum Participants, Or How To Fit All That Information on One Little Old Page</td>
</tr>
<tr>
<td></td>
<td>How To Write a Concrete Abstract, Or Avoiding the Common Mistakes</td>
<td>by Pat Brougher—Dallas, Texas</td>
</tr>
</tbody>
</table>

## ORIGINAL CONTRIBUTIONS

<table>
<thead>
<tr>
<th>Number</th>
<th>Title</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>25</td>
<td>Pulse Oximetry and Normoxemia in Neonatal Intensive Care</td>
<td>by Tim Blanchette, John Dziodzio, and Kathryn Harris—Portland, Maine</td>
</tr>
<tr>
<td>33</td>
<td>Sputum Induction: A Quick and Sensitive Technique for Diagnosing</td>
<td>Pneumocystis carinii Pneumonia in Immunosuppressed Patients by Cynthia R Godwin, Dennis T Brown, Henry Masur, Vee J Gill, and Frederick P Ognibene—Bethesda, Maryland</td>
</tr>
</tbody>
</table>

## REPORTS

<table>
<thead>
<tr>
<th>Number</th>
<th>Title</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>45</td>
<td>The Second Nagoya Conference: Triggering and Optimizing Mechanical</td>
<td>Ventilatory Assist by Robert M Kacmarek, Yashuhiro Shimada, Akito Ohmura, Jun Takezawa, Akihiro Tokioka, Tomomasa Kimura, and Masaji Nishimura—Boston, Massachusetts; and Nagoya, Kawasaki, Okayama, and Osaka, Japan</td>
</tr>
<tr>
<td>53</td>
<td>PFT Corner #40—A Bronchodilator Response Does Not Asthma Make</td>
<td>by Jonathan Matz and Charles G Irvin—Denver, Colorado</td>
</tr>
</tbody>
</table>

## BLOOD GAS CORNER

<table>
<thead>
<tr>
<th>Number</th>
<th>Title</th>
<th>Authors</th>
</tr>
</thead>
</table>
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(800) 328-4139
TEST YOUR RADIOLOGIC SKILL
63 Radiographic Findings following Pediatric Blunt Trauma 
   by Carol R Schermer and Frederick W Clevenger—Albuquerque, New Mexico

BOOKS, FILMS, TAPES, & SOFTWARE
68 Hyperbaric Medicine Procedures, by Eric P Kindwall MD and Robert W Goldman MD reviewed by John M Graybeal and Garfield B Russell—Hershey, Pennsylvania
68 Health Measurement Scales: A Practical Guide to their Development and Use, by David L Streiner MD and Geoffrey R Norman MD reviewed by James K Stoller—Cleveland, Ohio

CORRECTION
22 Correction to Open Forum Abstract (Respir Care 1990;35:1100)

ABSTRACTS
8 Summaries of Pertinent Articles in Other Journals

CALENDAR OF EVENTS
71 Meeting Dates, Locations, Themes

NOTICES
73 Examination Dates, Notices, Prizes

PRODUCT PROBLEM REPORTING FORM
74 Tear-Out Reporting Form

CALL FOR ABSTRACTS
75 1991 Call for Open Forum Abstracts

NEW PRODUCTS
79 Mechanical Cardiopulmonary Resuscitator
79 Ambulatory ECG-Monitoring System
79 Infant Resuscitator
79 Night Light

INFORMATION FOR AUTHORS
77 Instructions for Authors and Typists

INDEXES
80 Authors in This Issue
80 Advertisers in This Issue
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The effect of nebulizer solution temperature and dilution air humidity on the size and solute concentration of aqueous aerosol droplets was studied. Four combinations of jet-nebulizers with air compressors or oxygen sources and one ultrasonic nebulizer were tested. The temperature to which the nebulizer solution of each system fell during generation was measured. The nebulizers were then kept at set temperatures, generated aerosols collected and either droplet size or solute concentration measured. The droplet solute concentration was found to increase. The droplet size decreased along with the droplet solute concentration increase. The ultrasonic nebulizer also was tested: its high output made the concentration of the solution in the droplets much more stable. However, the proportion of droplets depositing in the tubing and valves changed markedly with aerosol flow-rate. The potential for large changes in droplet solute concentration, droplet size, and output during nebulization should be considered in therapeutic and diagnostic applications of nebulized aerosols.


Guidelines for use of aerosolized drugs in children are inconsistent. In a study of 14 infants, 22 children, and 4 adults, inspired nebulised aerosols were diluted more for large than for small subjects, because of air entrainment which occurred when inspiratory flow exceeded nebuliser flow. Infants under 6 months of age did not entrain air and would receive undiluted aerosols. All other subjects entrained air, which caused up to a 5-fold dilution in inspired aerosol concentration as subject size increased. In subjects who entrained air, the ratio of inspired nebuliser output versus total nebuliser output was relatively constant, and was related to the respiratory pattern. For a given nebuliser solution concentration, infants who do not entrain will inspire more concentrated aerosols than older children. Once entrainment occurs, the mass of drug inspired is largely independent of size. Regimens for nebulised drug delivery in children may require revision.


Previous reports have disclosed a high morbidity and mortality in hospitalized asthmatics, especially those treated in the intensive care unit. Recently, it has been questioned whether the benefits of treating asthmatics in the intensive care unit outweigh the potential hazards. To address this issue, we examined the outcome of status asthmaticus in our medical intensive care unit between January 1, 1978, and December 31, 1987. Eighty episodes of status asthmaticus occurred in 64 patients. In 50 episodes, respiratory failure (PaCO₂ > 50 torr) was present. In half of these episodes, mechanical ventilation was avoided despite severe acidosis and hypercapnia; in the remainder, mechanical ventilation was required as a lifesaving measure. Most patients improved rapidly and required only a short stay in the intensive care unit. There were no deaths and few complications. This was accomplished by close monitoring and repetitive blood gas analysis. We believe that the previous high complication rates and mortality associated with the hospital care of status asthmaticus can be avoided.


In chronic obstructive pulmonary disease (COPD) patients, there is a difference between PaCO₂ and end-tidal partial pressure of CO₂ (PetCO₂). This gradient Pa(ET-CO₂) is due to ventilation/perfusion mismatching and dead-space, and is usually abolished by forced and prolonged expiration. We hypothesized that this gradient might not be canceled by forced expiration in the case of acute respiratory failure (ARF) related to pulmonary embolism (PE). Forty-four adult COPD patients were prospectively entered into this study; they were suspected of having ARF related to PE on the basis of clinical and biological data on admission. Maximum expired partial pressure of CO₂ (PemCO₂) was measured in mechanically ventilated and sedated patients by an interrupt of mechanical support. CO₂ concentration was recorded during the following prolonged and passive expiration. The test was considered valid if an expiratory plateau was obtained. PemCO₂ was measured in triplicate. Simultaneously, PaCO₂ was measured, and the ratio, R = (I - PemCO₂/PaCO₂) × 100, was calculated. Pulmonary angiography was performed on the same day for all patients. Results showed that 17 patients had PE (PE+), and 17 had no PE (PE−). The two groups were comparable regarding mean age, severity of
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underlying chronic respiratory disease, \( P_{a\text{-CO}_2} \), \( P_{a\text{O}_2} \), and hemodynamic data on admission. \( P_{(a\text{-em})CO}_2 \) and R were significantly different in PE+ and PE− patients at 12 ± 6.9 torr compared to 1 ± 2.4 torr and at 28 ± 14.8% compared to 2 ± 6.2% (p < 0.001), respectively. The positive predictive value of the test was 74%, but the negative predictive value was 100% and the specificity was 65%, but sensitivity was 100%. We conclude that this test may be useful to rule out a diagnosis of PE during ARF of COPD patients.


Sequential changes in pulmonary mechanics in response to single dose exogenous surfactant instillation were studied in 15 preterm neonates who had hyaline membrane disease (HMD). The infants were part of a larger double-blind national study. Birthweight ranged from 0.88 to 1.55 kg, and gestational age was between 27 and 32 wk. There were 6 infants in the surfactant group and 9 in the placebo group. Pulmonary mechanics were studied before and at 2, 24, 60, and 96 h after surfactant or sham instillation using a pneumotachometer and an esophageal balloon catheter. The variables studied were dynamic compliance (\( C_{dyn} \)), pulmonary resistance, work of breathing, tidal volume, and minute ventilation. Infants in the surfactant group showed an immediate and significant (p < 0.05) improvement in gas exchange ratio, decreased mean airway pressure (9.7 ± 0.9 to 7.9 ± 0.4 cm H\(_2\)O) and airway resistance (133 ± 6.3 to 92 ± 14.9 cm H\(_2\)O · s · L\(^{-1}\)) (p < 0.05). Changes in \( C_{dyn} \) were noted only at 24 h after surfactant instillation. In the control group, gradual improvement occurred after the initial deterioration. The findings suggest that the immediate improvement in oxygenation after surfactant instillation is the result of factors other than changes in lung compliance, such as improved ventilation/perfusion and better capillary stability with decreased leakage of fluid into alveoli.


Pulse oximetry is widely used in the critical care setting, but few studies have examined its usefulness in clinical decision making. One area where pulse oximetry might be useful is in the titration of fractional inspired \( O_2 \) concentration (\( F_{iO_2} \)) in ventilator-dependent patients. Unfortunately, documented guidelines for this use do not exist, and in a survey of directors of intensive care units, we found that they employed a wide range of target \( O_2 \) saturation (\( S_{PO_2} \)) values. Consequently, we undertook a study to determine if the \( S_{PO_2} \) could be reliably substituted for measurements of arterial \( O_2 \) tension (\( P_{aO_2} \)), when adjusting \( F_{iO_2} \), in ventilator-dependent patients. We examined a number of \( S_{PO_2} \) target values in 54 critically ill patients aiming for a \( P_{aO_2} \) of ≥ 60 torr, while minimizing the risk of \( O_2 \) toxicity. In white patients, we found that a \( S_{PO_2} \) target of 92% was reliable in predicting a satisfactory level of oxygenation. However, in black patients, such a \( S_{PO_2} \) reading was commonly associated with significant hypoxemia (\( P_{aO_2} \) as low as 49 torr), and a higher \( S_{PO_2} \) target, 95%, was required. In addition, inaccurate oximetry readings (ie, < 4% difference between \( S_{PO_2} \) and direct \( S_{aO_2} \) measurements) were more common in black (27%) than in white patients (11%, p < 0.05). In conclusion, a \( S_{PO_2} \) target of 92% was reliable when titrating supplemental \( O_2 \) in white patients receiving mechanical ventilation; however, in black patients, such a \( S_{PO_2} \) reading was commonly associated with significant hypoxemia, and a higher \( S_{PO_2} \) target, 95%, was required to ensure a satisfactory level of oxygenation.


We sought to determine if biofeedback could reduce weaning time for the hard-to-wean patient by improving important weaning factors that are not effectively dealt with by present weaning methods. These include respiratory muscle electromyograph (EMG) efficiency, respiratory drive, and the anxiety of the ventilator-dependent patient. After the patient had received mechanical ventilation for 7 days and the day weaning began (start), the patient was randomly assigned to biofeedback or to the control group. There were 20 patients assigned to each group, with mean ages of 60.2 (biofeedback) and 59.3 (control) years. The patients assigned to the biofeedback group received daily, until extubation or being placed on no resuscitation status (termination), frontalis electromyographic (EMG) relaxation feedback for anxiety reduction and improved respiratory muscle EMG efficiency, tidal volume/diaphragm EMG (\( V_T/DAP \)), and \( V_T \) feedback for increasing \( V_T \) and respiratory drive defined as tidal volume/inspiratory time (\( V_T/T \)). The control group was visited daily to control for attention and reassurance. The results showed a significant (p < 0.01) reduction in mean ventilator days for the biofeedback group of 20.6 ± 8.9 SD compared with 32 ± 17.6 SD mean days for the control group. From start to termination, there was a significant (p < 0.01) increase in baseline \( V_T \) from 295 ± 41 to 415 ± 45 mL and
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a significant (p < 0.02) increase in V_T/DAP, from 0.33 ± 0.09 to 0.94 ± 0.22 L/mV for the biofeedback group but no significant change in these parameters for the control group. The between-group comparison showed a significant (p < 0.05) increase for the biofeedback group in respiratory drive (V_T/T_i) when compared to the control group (199 ± 44 mL/s). These results show that biofeedback can be an important factor in reducing time on mechanical ventilation when used with either T-piece or intermittent mechanical ventilation weaning.


The effects of body position and anesthesia with mechanical ventilation on thoracic dimensions and atelectasis formation were studied by means of computerized tomography in 14 patients. Induction of anesthesia in the supine position reduced the cross-sectional area for both lungs and caused atelectasis formation in dependent lung regions in 4/5 patients. Conventional ventilation with positive end-expiratory pressure (PEEP) increased thoracic dimensions and reduced, but did not eliminate, the atelectatic areas. The vertical diameters of both lungs were smaller in the lateral position as compared to the supine position (16.7 vs 10.4 cm in the left lung and 17.3 vs 12.8 cm in the right lung). The lateral positioning also caused a large reduction of the atelectatic area in the non-dependent lung. Differential ventilation with selective PEEP to the dependent lung eliminated (3/8 patients) or reduced (5/8 patients) dependent lung atelectasis. It can be concluded that lung geometry is altered in the lateral position; the shape of the lung makes the vertical diameter of each lung less in the lateral position, compared to the supine position. The atelectatic areas are mainly located in the dependent lung in the lateral position, and these atelectatic areas could be further reduced by selective PEEP to this lung.


Cot death could be the result of chance coincidence of an adventitious challenge and a baby ill-equipped to meet it. This work was designed to test the hypothesis that there is a subpopulation of babies who lack the appropriate responses to hypoxia and hypercapnia that would enable them to overcome the effects of, for instance, nasal obstruction. The responses of 630 babies to air mixtures that induced significant changes in ventilation in the overwhelming majority, were recorded in a short protocol in which both the addition and withdrawal of hypercapnia and of hypoxia were effected. The results of each test were placed in one of five categories; in 13.6% there was no response to hypoxia, and in 2% the ventilation fell in hypoxia to a significant degree. The study confirms the existence of a subgroup of normal babies with little defense to the respiratory loading of mild upper respiratory tract infections.


The characteristic pattern of breathing for an individual when awake at rest may be due to forebrain influences upon breathing. To examine this hypothesis we have studied the breathing pattern in 18 healthy subjects during relaxed wakefulness (W) and during Stage 4 sleep (S4), when forebrain influences upon breathing are absent or minimal. Inspiratory and expiratory times, respiratory frequency, tidal volume, and ventilation were quantified noninvasively by respiratory inductance plethysmography. The stability of respiratory variables between W and S4 sleep was tested within individuals. The results show that (1) individuals breathe differently from each other when awake and when in S4 sleep; the range between individuals during sleep being as large as it is when awake; (2) differences in breathing pattern between two S4 periods within an individual are relatively small; (3) the characteristic breathing pattern of an individual when awake tends to be maintained in S4 sleep. This persistence of a respiratory ‘personality’ into S4 sleep probably indicates that there are individual differences in respiratory rhythm generation in the absence of any forebrain influences upon breathing.


Thirty-six former preterm infants undergoing inguinal hernia repair were studied. All were ≤ 51 weeks post-conceptual age at the time of operation. Patients were randomly assigned to receive general or spinal anesthesia. Group-1 patients received general inhalational anesthesia with neuromuscular blockade. Group-2 patients received spinal anesthesia using 1% tetracaine 0.4-0.6 mg/kg in conjunction with an equal volume of 10% dextrose and 0.02 mL epinephrine
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1:1000. In the first part of the study, infants randomized to receive spinal anesthesia also received sedation with intramuscular ketamine 1-2 mg/kg prior to placement of the spinal anesthetic (group 2A). The remainder of group-2 patients did not receive sedation (group 2B). Respiratory pattern and heart rate were monitored using an impedance pneumograph for at least 12 h postoperatively. Tracings were analyzed for evidence of apnea, periodic breathing and/or bradycardia by a pulmonologist unaware of the anesthetic technique utilized. None of the patients who received spinal anesthesia without ketamine sedation developed postoperative bradycardia, prolonged apnea, or periodic breathing. Eight of nine infants (89%) who received spinal anesthesia and adjunct intraoperative sedation with ketamine developed prolonged apnea with bradycardia. Two of the eight infants had no prior history of apnea. Five of the 16 patients (31%) who received general anesthesia developed prolonged apnea with bradycardia. Two of these five infants had no prior history of apnea. When infants with no prior history of apnea were analyzed separately, there was no statistically significant increased incidence of apnea in children receiving general vs spinal anesthesia with or without ketamine sedation. Because of the small numbers of patients studied, and the multiple factors that may influence the incidence of postoperative apnea (eg, prior history of neonatal apnea), standard postoperative respiratory monitoring of these high-risk infants is still recommended following all anesthetic techniques.

Previous studies have reported that exercise tolerance improves with transtracheal oxygen delivery. However, patients were not blinded to the delivery technique used, introducing a potential source of bias. The purpose of this study was to compare exercise tolerance during nasal cannula and transtracheal delivery using a randomized double-blinded technique. Subjects (n = 11) performed 12-min walks on the same day while receiving nasal cannula and transtracheal delivery. Nine of 11 subjects walked farther with transtracheal delivery, a significant increase (p < 0.01). Mean increase in walk distance was 95 ± 86 ft. In addition, a trend was seen toward greater improvement in walk distance with greater flows through the catheter (r = 0.58, p < 0.06). Time into the walk when desaturation (SaO2 < 90%) first occurred was not significantly different. We conclude that exercise tolerance improves when oxygen is delivered by transtracheal catheter. This improvement is unrelated to an increase in SaO2. We speculate that the increase in exercise tolerance may be related to other physiologic effects of flow through the catheter.


We employed a questionnaire to survey 334 registered nurses regarding their knowledge, perceptions, and vaccine acceptance for Hepatitis B virus (HBV) infections. The study population was those persons working in areas considered at high risk for contracting HBV. The nurses were asked to provide information regarding blood and body fluid exposures and the reporting of these exposures. The questionnaire was completed by 169 nurses (50.6%). Less than half of the respondents (42%) had been vaccinated. We found that 13 of 14 black nurses, compared to 85 of 154 nonblack nurses, had not been vaccinated (p < 0.01). No other demographic difference between recipients and nonrecipients was noted. Partially or totally incorrect answers regarding transmittal knowledge were given by 108 of 160 (68%) respondents. Reasons for failure to be vaccinated were varied. Fear of side effects of the vaccine, contracting AIDS or hepatitis from the vaccine, or doubt of efficacy of the vaccine were cited by 50 of 88 (58%) respondents as reasons for not being vaccinated. Inability to schedule an appointment or unawareness of the vaccine’s availability were expressed by 23% and 17% of the individuals, respectively. Our data allow us to conclude that to improve vaccination compliance, the vaccine must be made more readily available, possibly through workplace on-site administration. Additionally, a concerted effort to educate our staff regarding HBV with particular emphasis on minorities is needed.
Pulse Oximetry in the Neonatal Intensive Care Unit

"Next to the promulgation of the truth, the best thing I can conceive that a man can do is the public recantation of an error."

Sir Joseph Lister, quoted in The Art of Scientific Investigation

Opinions regarding the use of continuous pulse oximetry in neonatal patients are divergent. There are those who suggest that it is an important tool for clinicians charged with the difficult task of monitoring oxygenation in this often unstable population. These authors have generally agreed that pulse oximetry is sufficiently accurate and reliable to be useful in monitoring oxygenation in the neonate—going so far as to suggest that it is generally better than transcutaneous monitoring of PaO2.

The opposing camp includes those who submit that although the pulse oximeter might be a useful tool under many circumstances, in general it is not sensitive enough to allow the accurate estimation of PaO2 necessary in the neonatal population—especially to avoid hyperoxemia.

This controversy centers around the need for the accurate control of PaO2 unique to the neonatal population.

**Hyperoxemia in the NICU**

The detection and rapid correction of hyperoxemia is vital in the NICU, as in any critical care population. Moreover, the prevention of hyperoxemia is also a weighty concern in the NICU because of its role in the development of retinopathy of prematurity (ROP). ROP has been reported since the first widespread use of oxygen in the treatment of respiratory distress associated with prematurity during the 1940s, and was originally called retrolental fibroplasia. Early reports established that excessive oxygen played an important role in this condition, which can result in partial or complete loss of vision. This led to the long-held belief that through the meticulous control of PaO2, the disease could be prevented (a misconception that may have led to a great deal of unjustified malpractice litigation). Indeed, a reduction in the indiscriminate use of oxygen in the neonatal population led to a significant reduction in the prevalence of ROP. However, in recent years the disease has recurred, and we have come to understand that its etiology is more complex than previously thought. ROP has been reported in more than 60 full-term infants, the majority of whom received no supplemental oxygen, and in 95 preterm infants who received no oxygen therapy.

Indeed, "The exaggerated etiologic importance of oxygen has been recurrently suggested in the literature for years. ROP has been associated with a number of risk factors, but the most prevalent seem to be prematurity, duration of ventilation, and oxygen therapy. In fact, substantial data suggest that hyperoxemia may play an important role in the development of ROP. Respiratory care practitioners must avoid the myopic view that this disease is caused primarily by hyperoxemia. Sadly, a recent neonatal respiratory text has succumbed to this fallacy.

The complete prevention of ROP is not possible at this time. However, the respiratory care practitioner still must play an important role in reducing the patient's risk of acquiring this disease. Oxygen therapy is a factor in ROP—but not the only factor—and strict attention must be paid to the prevention of hyperoxemia and hyperoxemia.

The contemporary rationale for oxygen therapy generally suggests keeping the PaO2 between approximately 50 and 80 torr [6.7-10.6 kPa], although...
no scientific evidence defines the \( P_{aO_2} \) at which the risk for retinal disease increases substantially. \( ^{15,19,20} \)

### The Pulse Oximeter and Neonatal Hyperoxemia

So, why all this talk about ROP in an editorial concerning neonatal pulse oximetry? Because the pressing question is Is the pulse oximeter sufficiently sensitive to be able to reliably detect a \( P_{aO_2} < 50 \) torr \([6.7 \text{ kPa}]\) or \( > 80 \) torr \([10.6 \text{ kPa}]\)? In this issue of the Journal, an important investigation is reported that helps to answer this question.\(^{25}\)

As previously stated, many authors (including me\(^ {17} \)) have expressed concern about the ability of the pulse oximeter to detect hyperoxemia. These concerns have been largely theoretical and based on: (1) the shape of the oxyhemoglobin dissociation curve (ODC) seen in Figure 1, (2) factors that affect the position of the ODC, and (3) the accuracy of the pulse oximeter.

![Figure 1. A representation of the oxygen dissociation curve. As the saturation increases, small changes in the saturation can result in large changes in the \( P_{aO_2} \). The striped horizontal area reflects the range of saturations possible for a given value of 95% ± 4%. The shaded vertical area indicates the range of \( P_{aO_2} \) values associated with this estimated inaccuracy of the pulse oximeter.](image)

Oxygen exists in blood in two forms: dissolved in the plasma and chemically bound to hemoglobin molecules. As the partial pressure of oxygen in plasma increases (or decreases), the chemical bonding of oxygen to hemoglobin changes in the same direction. However, the relationship is not linear. Small changes in the partial pressure can result in large changes in the volume of oxygen bound to hemoglobin (saturation) on the lower (or steeper) portion of the curve. Conversely, the upper (or flatter) portion of the curve shows that big changes in the partial pressure of oxygen are required to effect small changes in the saturation. It is this flat portion of the curve that has been of such concern to practitioners considering the use of the pulse oximeter in neonates.

This concern was succinctly described by Tobin\(^ {24} \) when he discussed the effect of the relative inaccuracy of oximeters on their ability to predict \( P_{aO_2} \). He described the 95% confidence limits of oximeters (in general) as ± 4%. Using this value, he went on to point out that an oximeter reading of 95% could represent a \( P_{aO_2} \) as low as 60 or as high as 160 torr \([8.0 \text{ or } 21.3 \text{ kPa}]\). This is based on the assumption that saturation could be as low as 91%, or as high as 99%. This is the kind of reasoning that has often led clinicians (including me) to question the pulse oximeter’s ability to reliably identify periods of hyperoxemia. This reasoning appeared to be logically sound, but the role of reason based on logic in science is sometimes exaggerated, as clearly pointed out by Francis Bacon in 1605 and quoted in The Art of Scientific Investigation.\(^ {1} \)

"... the present system of logic rather assists in confirming and rendering inveterate the errors founded on vulgar notions, than in searching after truth, and is therefore more hurtful than useful."

Unfortunately, we (those skeptical of neonatal pulse oximetry) had sparse scientifically gathered data to support our assumptions and, as is often the case, we were operating in that gray region between art and science.

I am now happy to admit that I appear to have been in error in not more heartily promoting the widespread use of continuous pulse oximetry in the NICU. Recent investigations (including the one presented by Blanchette et al in this issue of the Journal\(^ {23} \)) have laid to rest concerns that the pulse oximeter is unable to reliably identify hyperoxemia. Hay et al\(^ {6} \) reported that if pulse oximeter readings were kept at \( 92 \pm 3\% \), this always resulted in \( P_{aO_2} \) > 45 torr \([6.0 \text{ kPa}]\) and < 100 torr \([13.3 \text{ kPa}]\). Deckardt and Steward\(^ {7} \) reported that if pulse oximeter readings were kept between 80% and 95%, the transcutaneous \( P_O_2 \) remained between 40 torr and 80 torr \([5.3 \text{ to } 10.6 \text{ kPa}]\) in 94% of patients studied. These findings are similar to the results of Blanchette et al,\(^ {23} \) who are to be commended for the investigation they report here.
It has now been demonstrated to my satisfaction that in fact the pulse oximeters tested are capable, when properly applied, of being enormously helpful in identifying (and thereby leading to the subsequent correction of) periods of occult hyperoxemia. However, the successful use of these instruments requires rigorous training of individuals and proper protocols; it has been my experience that in some NICUs neither of these goals is realized.

The end-user of the pulse oximeter is often uncertain about what is actually being measured and precisely what actions to take in response to changes in pulse oximeter readings. Teaching clinicians when the pulse oximeter readings are likely to be unreliable and how to respond to changes in readings must be an important part of in-service training.

In spite of these recent important findings, it is certain that there will continue to be many times when clinicians will be forced to ponder the difficult question of whether their pulse oximeter readings are to be believed. This is evidenced by the work of Barrington et al., who pointed out that the most popular brand of pulse oximeter was unreliable due to motion artifact between 12% and 29% of the time, depending on the averaging mode used in a population of neonatal patients. Unfortunately, Blanchette et al. failed to adequately discuss motion artifact and its effect on their attempts to gather data or ways in which it might affect the utility of the pulse oximeter.

One other reported limitation of pulse oximeters is their tendency to overestimate true arterial saturation in the presence of profound hypoxemia ($S_{aO_2} < 70\%$). The clinical relevance of this finding within the intensive care environment escapes me. Any patient whose pulse oximeter is reading $< 70\%$ will be aggressively treated for hypoxemia, whether the true saturation is 40% or 60%.

Regardless of the type of monitor used, nothing can replace the bedside clinician, whose diligence and assessment skills must remain the first line of defense in monitoring the critically ill. This is well stated by Severinghaus who said, “I have asked myself whether there may be a dark side to our technological explosion? I began to wonder about the effect on vigilance of devices that do all the measuring and warning and alarming, assuming they are working.”

The adroit application of the pulse oximeter in neonatal monitoring clearly is an enormous advance that may serve to significantly improve the quality of care.

**The Effect of Pulse Oximetry on Morbidity and Mortality**

I have said that continuous pulse oximetry in the NICU may improve the quality of care, but such a statement requires that a definition be established for quality of care as it applies to this form of monitoring. The most fundamental concern is, What is the pulse oximeter’s ability to affect respiratory morbidity and mortality? More specifically, the vital questions are: (1) Do patients continuously monitored with a pulse oximeter have a better overall survival rate than those who are not monitored? (2) Is the duration of mechanical ventilation or oxygen therapy any less in monitored patients than in those not monitored? (3) Does the use of continuous pulse oximetry reduce the incidence of ROP, and, finally, (4) Does the use of pulse oximetry reduce (or increase) the cost of care or the frequency of invasive procedures? In other words, what is the effect of pulse oximetry on quality of care?

Certainly, anecdotal reports abound that describe the benefits of pulse oximetry. Some authors now suggest that pulse oximetry join the continuous electrocardiogram as basic monitoring equipment for all patients in critical care units. This suggestion seems a bit expansive and premature. The issue of whether all mechanically ventilated patients in critical care units should be monitored seems a much more meaningful question. Certainly, this is a widespread practice in many NICUs. Unpublished data from a survey of more than 612 hospitals that I conducted revealed that nearly 80% of all NICUs with more than 20 beds reported monitoring more than 80% of their mechanically ventilated neonates continuously by pulse oximeter.

I have for some time wished that we could randomize patients prospectively to be monitored with a pulse oximeter or to be managed without one. We could then measure the outcome variables I described and find out whether the device really has a measurable impact on care. Unfortunately, pulse oximeter use is so widespread, and so often regarded as a standard of care, it would be difficult to convince clinicians to withhold the pulse oximeter from some patients.

Bancalari et al. studied whether the use of a continuous oxygenation monitor (in this case the transcutaneous $PO_2$ [PtcO2] monitor) could reduce the incidence of ROP. They concluded that continuous...
$P_{\text{CO}_2}$ monitoring may reduce ROP in infants with birthweights $>1000$ g but not in smaller infants. This of course supports the opinion that in the very premature infant, this disease may not be preventable.

However, it now seems unlikely that the pulse oximeter will ever be subjected to this kind of test in the neonatal population. Many universally accepted standards of care (such as continuous bedside electrocardiographic monitoring and continuous monitoring of $F_{\text{O}_2}$) have not been tested as far as I know to determine their overall impact on patient outcome.

Clearly, it is no longer justifiable to limit the availability of pulse oximetry in the neonatal population. A proverb tells us that there is wisdom in the counsel of many. Certainly many people use continuous pulse oximetry in the NICU on most, if not all, of their ventilated patients. Perhaps it is time the rest of us got wise.

**John W Salyer RRT**

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**REFERENCES**


Research-Protocol Review: A New Editorial Service

Over the years, this journal’s editors have tried to help authors—and would-be authors—prepare manuscripts that can pass easily and quickly through review and thus be published without undue delay.

However, in the case of a paper that reports research, even the best prepared manuscript won’t make it if something was wrong with the work behind the paper—that is, the research itself. Many times, an editor has had to send sad news to an author: “Your results are not useful because you did not employ a control group. Therefore we regret we cannot accept your paper.” Or, maybe, “Subjects 4 and 7 did not respond to either bronchodilator. For this kind of study, all the subjects must be responders.” It is always unfortunate when someone has done all that work—planned a research project, carried it out, written it up, submitted it to a journal—and then has learned that something went wrong early in the process, something that spoils it all. It’s particularly unfortunate when the error could have been prevented.

Advance Protocol-Review

This journal now offers researchers a new service meant to avert that calamity. Before you perform the research, you can write up your protocol (which you should do anyway) and send it to our editors for review. As soon as possible, you will receive a message from us that may say, for example, “Your protocol looks OK—go ahead with the research,” or “Your plans have one or more problems; here are our suggestions for changes to overcome them,” or something else, such as that “In our opinion, your research question has already been adequately answered by prior work.”

Another, perhaps simpler way to do this is to write what has been called a “zeroth draft” of the paper that you expect to prepare after you complete the research. A zeroth draft is called that because, coming before the usual first draft (Draft #1) it takes the number zero (Draft #0). The idea of writing a scientific paper prior to the research comes from a 1985 article by Babbs and Tacker. Writing a draft in advance seems strange at first, but we think it’s an excellent plan.

Draft #0 consists of the paper’s title and three of the sections that are found in most research papers. These sections are the Background (usually called the Introduction, but we now prefer the term Background), the Materials and Methods section, and the Results section.

The Background section summarizes what lies behind your research project. It states the research question and tells why you are performing the study.

The Materials and Methods section tells exactly how the research is to be performed, giving all details. If there are to be drawings of test setups or whatever, rough sketches can be included.

The Results section will have some blanks in it, because the actual results—the data—won’t be known when Draft #0 is written. Rough sketches of graphs can be part of Draft #0, showing how the vertical and horizontal axes will be labeled; the internal data will be missing, of course. Tables can be constructed in the same way, showing their titles and the headings of the columns, but with no data in the tables’ bodies.

A Draft #0 is a good way to present your protocol. It also can have other advantages. For one thing, writing a draft before you start the research can force you to visualize every detail of the work in advance, which may make your plans more realistic than they otherwise would be. Another point is that preparing
Draft #0 gets quite a bit of the writing behind you before you collect any data. Afterward, your main job will be to interpret the results and write the Discussion section.

What sort of suggestions might you receive from us? Here are some fictional examples:

- "What criteria will you employ to select the subjects?"
- "Squeeze the resuscitator only at rates of 12/min and 20/min. A rate of 30/min results in a high inspiratory flow with adult resuscitators and is not specified by the ASTM, ISO, AHA, or ECRI."
- "Use the John Doe test lung if possible. It has an RS232 connection at the back, which will connect to a Handy Dandy computer and allow you to store all your data in a file for later review."
- "Please provide a reference to support your use of the Mickey Mouse statistical test. Cite a textbook or article, not your statistical software's handbook."

Do It

Send us your protocol or Draft #0, and we will review it as promptly as possible. Yes, your research will be delayed briefly, but having your plans reviewed in advance can provide you with some peace of mind—and just possibly prevent grief or long delays later. Furthermore, the time taken for this advance review probably will be saved in the final review.

Phil Kittredge
Adjunct Editor

REFERENCES


An error occurred in the Open Forum Abstracts published in the November issue (Respir Care 1990;35:1100). A corrected version appears below. We regret the error.

Non-Bronchoscopic Diagnosis of Pneumocystis carinii Pneumonia: Is it Cost-Efficient?—Michael S Benson BS RRT, Robb Glenny MD, Paul S Kubilis MS, Harborview Medical Center; David J Pierson MD, Harborview Medical Center and University of Washington—Seattle, Washington.
How To Write A Concrete Abstract, or Avoiding the Common Mistakes of Would-Be Open Forum Participants, or How To Fit All that Information on One Little Old Page

Soon it will be Spring and with Spring come thoughts of abstract writing and deadlines and participation in the Open Forum. And, following those thoughts come phone calls to the Journal's Editorial Office from anxious authors and would-be authors. "I just can't fit all this information onto one double-spaced page. I've been trying all week to finish this abstract and . . ." And following the deadlines and the review, come rejection letters and disappointment for some.

Of course, many abstracts are rejected because they report results generated by a study of poor design, a superficial evaluation performed with inadequate tools, or a case report with little or no teaching value. The most beautifully written abstract cannot cure those problems. A study design that cannot possibly answer the research question, an evaluation that gives us no answers about the real-world application of a device, or a case report that does little but establish the presence of poor clinical practice is doomed to rejection—and rightfully so. But, let's assume that the study design, or evaluation, or case report is solid and appropriately executed—what mistakes can you avoid and how do you put all that information on one page?

Remember that the abstract is the only basis that the reviewers have for judging the adequacy of your work. The review process is blind—reviewers don't know you, or your co-authors, or your institution. They see only what is presented in your abstract.

So, let's talk about writing abstracts, and let's see if we can avoid some of the pain.

First, read the 1991 Call for Abstracts printed in each issue of the Journal from January through May. Note the deadlines, schedule the stages of your work, and follow the Call's instructions carefully.

Then, examine the basic structure that the abstract you are writing should assume. The Call lists the essential elements of each abstract type. Read that paragraph with care before you begin to work and to write.

Next, jot down the essentials in outline form to fit that structure. This can be a part of the "zeroth" draft written in preparation for the actual work. Check and double check to assure that there's a place for all of the essential information. When all of the information is in hand (ie, when your work is complete), plug in the 'numbers.'

There's a cliche in management that goes like this: Don't tell me what you've done, tell me what you're gonna' do. But we insist on the antithesis: Don't tell me what you're gonna' do, tell me what you've done.

Now let's talk about some common mistakes and omissions:

— failure to formulate a clear introductory statement that tells the reviewer what the abstract addresses. Make clear the study question, the method or device evaluated, or the importance of the case reported. The reviewer shouldn't have to read the whole abstract to be able to decide what it's about.

— incomplete data—for example, failure to include the number of subjects, number of trials, or statistical analysis and its results.
failure to identify the measuring devices or instruments used to evaluate devices or methods and failure to supply the number of units tested.

— failure to draw a conclusion or conclusions. It may be easier to leave things up in the air, but a conclusion is essential.

— failure to limit statements in the Conclusion to information actually revealed by the study—extrapolation beyond the data, if you will. A common mistake authors make is to suggest that the described method or device is less expensive than some other—but provide no evidence for that conclusion.

If you’ve done all of this careful planning and organization, and included all of the pertinent data but find yourself unable to confine the text to one page, adopt a ‘telegraphic’ style: (1) omit articles (a, an, the); (2) abbreviate often-repeated terms after first introducing them—sputum induction by ultrasonic aerosol becomes USSI; (3) use numerals instead of words—8, not eight; (4) use standard abbreviations without explanation—mL, f, Raw, FRC; and (5) present data in a simple, compact table.

Submit your abstract to in-house review. It works for papers, and it can work for abstracts. This is no time to be thin-skinned. If your best friend and your medical director don’t understand the Conclusion, then chances are the reviewers won’t either.

Use the dictionary or a spelling checker program. ‘Typos’ make reviewers nervous because they suggest lack of care.

Make your abstract ‘look good.’ Several years ago in a well-known medical journal, a reviewer reported that his inspection and analysis of abstracts submitted for possible presentation at a prestigious annual meeting had revealed that regardless of the content or potential value of the abstracts, those submitted in typeset form had a greater likelihood of being accepted than those prepared on a typewriter or dot-matrix printer. Don’t take this observation too seriously (we like to think that our reviewers see beyond mere appearances), but do take the extra few minutes necessary to submit a clean, legible copy. Borrow the use of a good laser printer if you don’t have one or, at least, buy a new ribbon for your typewriter.

Still stymied? Call the Editorial Office to arrange a consultation. Send your abstract in by the early deadline to allow time for feedback from reviewers and consequent revision.

Remember that about 45% of each year’s Open Forum originates with authors who have never presented before—and a good number of those have submitted before and failed to be accepted. The opportunity is yours. Take advantage of it!

Pat Brougher
Editor

REFERENCES*

6. Hess D. In-house manuscript review smooths the path to publication (editorial). Respir Care 1986;31:766-767.

*References 2, 5, & 6 have been reprinted in RESPIRATORY CARE’s Author & Typist’s Kit, which is available on request from the Editorial Office.
Pulse Oximetry and Normoxemia in Neonatal Intensive Care

Tim Blanchette MS RRT, John Dziodzio BA, and Kathryn Harris BA RRT

BACKGROUND: Conflicting results are found in the medical literature concerning the reliability of pulse oximetry in predicting hyperoxemia (P_{O2} > 90 torr [12 kPa]) and hypoxemia (P_{O2} < 45 torr [6.0 kPa]) in neonates. This study was designed to determine whether oximeter saturation limits could be established that would adequately predict normoxemia in our neonatal population. METHODS: Fifty-two infants in our intensive care unit were studied over a wide range of values for temperature, pH, and P_{O2}. Three-hundred fifty-three arterial blood gas samples from umbilical or arterial catheters were drawn and simultaneous pulse oximeter saturation (S_{PO2}) values recorded using Nellcor oximeters. RESULTS: S_{PO2} values ranged from 61% to 100%, and P_{O2} from 15 to 421 torr [2.0 to 56.1 kPa]. Predictive value analyses were applied to determine the reliability of S_{PO2} in detecting the absence of hyperoxemia and hypoxemia. By comparing the sensitivity, specificity, and positive predictive value (PPV) for different limiting values of S_{PO2}, an upper limit of 96% proved to be optimal in preventing hyperoxemia. S_{PO2} values < 90 torr [5.8 kPa] in 239 out of 246 samples, yielding a PPV of 97% and a 2.8% rate of false positives (S_{PO2} > 96% with P_{O2} < 90 torr [12 kPa]). S_{PO2} values > 92% were associated with P_{O2} values > 45 torr [6.0 kPa] in 262 of 273 samples, producing a PPV of 96% with a 4% false-positive rate (S_{PO2} > 92% with P_{O2} < 45 torr [6.0 kPa]). Hypoxemia was avoided in 96% of the samples with the S_{PO2} maintained at > 92%, and hyperoxemia was avoided in 97% of the samples with S_{PO2} maintained at < 96%. DISCUSSION: Although establishing S_{PO2} limits can reliably prevent hyperoxemia and hypoxemia, a considerable number of false negatives do occur in which a P_{O2} of 45-90 torr [6.0-12 kPa] is associated with a S_{PO2} value outside the 92% to 96% range. Also, occasional aberrant S_{PO2} values can occur for unexplained reasons. CONCLUSION: This study demonstrates the ability of pulse oximetry to predict (with reasonable certainty) a clinically acceptable range of P_{O2} values in neonates, under variable conditions. However, the range of saturations to protect against hyperoxemia and hypoxemia must be established for each brand or model of oximeter. (Respir Care 1991;36:25-32.)

Introduction

The pulse oximeter is generally acknowledged to be one of the most important advances in the history of clinical monitoring. The accuracy of pulse oximetry oxygen saturation (S_{PO2}) measurements as compared to arterial oxygen saturation (S_{AO2}) or arterial oxygen tension (P_{AO2}) measurements in newborns has been well researched. However, there is conflicting evidence about pulse oximetry’s reliability in predicting normoxemia and identifying hyperoxemia (P_{AO2} > 90 torr [12 kPa]) and hypoxemia (P_{AO2} < 45 torr [6 kPa]) in neonates.\textsuperscript{1-13} It has been demonstrated that pulse oximeters are not accurate in certain circumstances,\textsuperscript{1,14-18} and that oximeter bias, resulting from differing instrument calibration techniques or intrinsic algorithms, may lead to variations in S_{PO2} values from one instrument to another.\textsuperscript{1,14,19-21}
PULSE OXIMETRY IN NEONATAL INTENSIVE CARE

Abbreviations Used in this Paper

ABG — Arterial blood gas
BPD — Bronchopulmonary dysplasia
\( \text{PaO}_2 \) — Arterial oxygen tension (pressure)
PPV — Positive predictive value
ROC — Receiver operating characteristics
\( \text{SaO}_2 \) — Arterial oxygen saturation
\( \text{SpO}_2 \) — Oxygen saturation measured via pulse oximetry

A Guide to the Use of SI in This Paper

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

\[
(\text{cm H}_2\text{O})(0.09806) = \text{kPa}.
\]

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

An article by Baeckert et al., which demonstrated that \( \text{SpO}_2 \) measurements were poor predictors of hyperoxemia, concerned us because we had become increasingly reliant on pulse oximetry in our neonatal intensive care unit. In this study we sought to determine how well pulse oximetry could predict normoxemia (45 torr [6.0 kPa] \( \leq \text{PaO}_2 \leq 90 \) torr [12 kPa]) and thus enable us to avoid hyperoxemia and hypoxemia in our neonatal intensive care population. We, therefore, compared \( \text{PaO}_2 \) to \( \text{SpO}_2 \) values in our varied neonatal intensive care population to determine if hypoxemia and hyperoxemia could be prevented by establishing \( \text{SpO}_2 \) limits.

Methods

A total of 52 infants, newborn to 78 days old (mean age 4.6 days), with umbilical or peripheral arterial catheters, were studied. Diagnoses are listed in Table 1. Mean birthweight was 1.67 kg (range 0.550-4.39 kg), and mean gestational age was 31.4 wk (range 24-42 wk). Fetal hemoglobin, 2,3-DPG, and bilirubin levels were not recorded.

The infants were routinely managed in isolettes, and axillary temperatures ranged from 36.2 to 38°C. Oxygen and ventilation requirements in the group ranged from spontaneous room-air breathing to 100% oxygen via mechanical ventilation with peak inspiratory pressure (PIP) > 40 cm H\(_2\)O [3.9 kPa] and respiratory rate (f) > 60.

One Nellcor N-100* and four Nellcor N-200 pulse oximeters were used in this study, and Nellcor N-

25 or I-20 sensors were placed securely on a foot, toe, hand, or thumb. \( \text{SpO}_2 \) values were compared to \( \text{PaO}_2 \) because \( \text{SpO}_2 \) is the accepted oxygenation index in infants.\(^{5,15,22,23}\)

Arterial blood gas (ABG) samples were drawn from an arterial catheter while \( \text{SpO}_2 \) values were being observed. Care was taken to assure that arterial sample site and oximeter sensor site were both either prero or postductal when blood samples were obtained. Infant handling (particularly in stress intolerant infants) or movement may cause a change in \( \text{SpO}_2 \) values. Therefore, \( \text{SpO}_2 \) values before and after ABG sampling were recorded to monitor changes related to handling. If the \( \text{SpO}_2 \) values before and after drawing the ABG sample were within 1% of each other—and the pulse signal was consistent and of sufficient amplitude, and the electrocardiogram (ECG) and pulse-oximeter heart rate correlated—they were included in the study data. Post-ABG \( \text{SpO}_2 \) values were used for data analysis if pre- and post-ABG values differed (eg, if \( \text{SpO}_2 = 95\% \) before drawing ABG and \( \text{SpO}_2 = 94\% \) after drawing ABG, then the 94% \( \text{SpO}_2 \) value was used for data analysis). All N-200 pulse oximeters were run in the Default Mode 1 (5-7 second averaging time) without the use of

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Table 1. Diagnostic Data for the 52 Infants in Whom Pulse Oximetry Saturation and CO-Oximetry Saturation Were Compared

<table>
<thead>
<tr>
<th>Diagnoses</th>
<th>No. of Infants</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory distress syndrome (RDS)</td>
<td>28</td>
</tr>
<tr>
<td>RDS and pulmonary interstitial emphysema</td>
<td>2</td>
</tr>
<tr>
<td>Septicemia</td>
<td>5</td>
</tr>
<tr>
<td>Abdominal surgery</td>
<td>4</td>
</tr>
<tr>
<td>Bronchopulmonary dysplasia</td>
<td>3</td>
</tr>
<tr>
<td>Persistent pulmonary hypertension</td>
<td>2</td>
</tr>
<tr>
<td>Transitic tachypnea</td>
<td>2</td>
</tr>
<tr>
<td>Congenital cardiac malformation</td>
<td>1</td>
</tr>
<tr>
<td>Cardiac surgery</td>
<td>1</td>
</tr>
<tr>
<td>Seizures</td>
<td>1</td>
</tr>
<tr>
<td>Apnea</td>
<td>1</td>
</tr>
<tr>
<td>Meconium aspiration</td>
<td>1</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>1</td>
</tr>
</tbody>
</table>

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

RESPIRATORY CARE • JANUARY '91 Vol 36 No 1
C-Lock. (C-Lock synchronizes pulse-oximeter signals with ECG signals.) All ABG samples were analyzed using an AVL 945 blood gas analyzer that was checked every 4-8 hours with GAS Trak blood gas controls. The median number of samples per patient was 5, with a range of 1 to 21 samples; however, 70 samples were obtained from one patient in order to evaluate how consistently pulse oximetry trended oxygenation in a single patient.

Predictive value calculations (including sensitivity, specificity, positive predictive values, and receiver operating characteristics [ROC] for various $S_pO_2-P_aO_2$ relationships) were analyzed to determine how well $S_pO_2$ values could detect selected $P_aO_2$ ranges.

Results

From the 52 infants studied, 353 paired measurements of $S_pO_2$ and $P_aO_2$ were obtained. $S_pO_2$ values ranged from 61% to 100%, and $P_aO_2$ values ranged from 15 to 421 torr [2.0 to 56.1 kPa]. Mean $P_aO_2$ was 68 torr [9.1 kPa]. In the study group, arterial pH ranged from 6.74 to 7.65 (mean pH 7.34), and arterial CO$_2$ tension ($P_aCO_2$) ranged from 13 to 87 torr [1.7 to 12 kPa] (mean = 40 torr [5.3 kPa]).

The paired data (including $P_aO_2$ < 160 torr [21.3 kPa]) are plotted in Figure 1 referenced to a normal adult $O_2$ dissociation curve. Table 2 summarizes the predictive value of $S_pO_2$ between 89% and 96% in yielding $P_aO_2 \geq 45$ torr [6.0 kPa]. Table 3 summarizes the predictive value of $S_pO_2$ between 92% and 99% in yielding $P_aO_2 \leq 90$ torr [12 kPa].

Table 2. Using $S_pO_2$ To Predict $P_aO_2 \geq 45$ Torr

<table>
<thead>
<tr>
<th>Operating Point $S_pO_2$ (%)</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>Positive Predictive Value (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>89</td>
<td>96.0</td>
<td>44.4</td>
<td>90.5</td>
</tr>
<tr>
<td>90</td>
<td>95.0</td>
<td>51.9</td>
<td>91.6</td>
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<tr>
<td>91</td>
<td>92.0</td>
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</tr>
<tr>
<td>93</td>
<td>79.9</td>
<td>88.9</td>
<td>97.6</td>
</tr>
<tr>
<td>94</td>
<td>71.6</td>
<td>98.2</td>
<td>99.5</td>
</tr>
<tr>
<td>95</td>
<td>60.9</td>
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</tr>
<tr>
<td>96</td>
<td>50.5</td>
<td>100.0</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Table 3. Using $S_pO_2$ To Predict $P_aO_2 \leq 90$ Torr

<table>
<thead>
<tr>
<th>Operating Point $S_pO_2$ (%)</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>Positive Predictive Value (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>92</td>
<td>35.8</td>
<td>100.0</td>
<td>100.0</td>
</tr>
<tr>
<td>93</td>
<td>45.4</td>
<td>98.0</td>
<td>99.3</td>
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<tr>
<td>94</td>
<td>56.0</td>
<td>96.1</td>
<td>98.8</td>
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</tr>
<tr>
<td>96</td>
<td>79.1</td>
<td>86.3</td>
<td>97.2</td>
</tr>
<tr>
<td>97</td>
<td>89.1</td>
<td>74.5</td>
<td>95.4</td>
</tr>
<tr>
<td>98</td>
<td>94.4</td>
<td>51.0</td>
<td>92.0</td>
</tr>
<tr>
<td>99</td>
<td>97.7</td>
<td>19.6</td>
<td>87.8</td>
</tr>
</tbody>
</table>

Discussion

Previous studies have depicted $S_pO_2$ as a poor predictor of hyperoxemia. However our data seem to corroborate studies by Hay et al. and Bucher et al. and demonstrate that pulse oximetry can effectively predict a normoxemic range of $P_aO_2$ values.
Furthermore, it was possible to improve considerably the sensitivity for $S_{pO_2}$ in predicting normoxemia by including $S_{pO_2}$ values of 96%. $S_{pO_2}$ values ≤ 96% were associated with $P_aO_2$ values ≤ 90 torr [12 kPa] in 97% of the cases and decreased the false-negative rate (improving sensitivity) in which a saturation over 96% was associated with a $P_aO_2$ ≤ 90 torr [12 kPa] (Table 3). However, as $S_{pO_2}$ increases, close surveillance is necessary in infants at risk for retinopathy of prematurity (ROP). Because a wider range in $P_aO_2$ will occur as $S_{pO_2}$ increases, the $O_2$ dissociation curve becomes flattened and small changes in $S_{pO_2}$ equal large changes in $P_aO_2$ (Fig. 1). This may be of particular importance in very-low-birthweight infants or those less than 30 weeks gestational age because there may be increased risk of retinopathy if oxygen pressure is permitted to exceed 90-100 torr [12-13.0 kPa] in these infants.5,6,25

Pulse oximetry inaccuracy increases as $S_{aO_2}$ decreases.17,18,26,27 This may be of little clinical importance because therapeutic intervention is most likely indicated whether the $S_{aO_2}$ is 75% or 68%. Motion artifact can present serious problems in monitoring neonates. Besides false alarms, there were many instances (no count was kept) when $S_{pO_2}$ values were inconsistent or changed during blood gas drawing and thus the values were not recorded.

The predictive value model was utilized for statistical analysis because it establishes the value of a test (pulse oximetry) in predicting an outcome (normoxemia). The ROC curve plots the sensitivity and specificity of the entire range of operating points. This aids in the determination of the best operating point (in this case the best $S_{pO_2}$ limit). Optimal sensitivity and specificity lies at the point where false positives and false negatives are both minimized—the point closest to 100% sensitivity and specificity.

In this study, maintaining pulse oximetry saturations between 92% and 96% (Fig. 1) proved effective in limiting the hyperoxic episodes ($P_aO_2 > 90$ torr [12 kPa]) to near 3% and hypoxemic episodes ($P_aO_2 < 45$ torr [6.0 kPa]) to 4%. These false-positive rates lead to occasional high and low $P_aO_2$ values even when $S_{pO_2}$ is maintained in the target range. Even infrequent $P_aO_2$ values > 90 torr or < 45 torr may not be acceptable in certain high risk infants, and pulse oximetry cannot replace arterial blood gas analysis in this population of neonates.
A lower $S_{pO_2}$ limit of 93% would have prevented hypoxemia nearly 98% of the time with similar sensitivity and specificity to the 92% limit (Table 2). Although it may be impractical, further narrowing of the $S_{pO_2}$ limits will increase the positive predictive value of the selected $S_{pO_2}$ ranges for assessing normoxemia (ie, in our group, maintaining an $S_{pO_2}$ of 94% would have avoided hyperoxemia and hypoxemia in 99% of the cases) (Tables 2 & 3).

We compared $S_{pO_2}$-$P_{aO_2}$ relationships at various gestational and postnatal age and pH ranges because of variations in the $O_2$ dissociation curve caused by fetal hemoglobin and changes in pH. $^{28-30}$ When $S_{pO_2}$ was compared to $P_{aO_2}$ in 20 infants of gestational age $\geq$ 32 weeks and in 32 infants of $<32$ weeks, no difference in correlation or shift in the $O_2$ dissociation curve could be identified. The relationship was also unchanged when we compared values from infants $\leq$ 3 days to those $>3$ days of age and $\leq$ 8 days to those $>8$ days of age. When infant pH values were compared to their data pairs, no difference was demonstrated from data obtained at a pH $<7.34$ and those at a pH $\geq 7.34$, or even 7.40 were compared. No difference in the $S_{pO_2}$-$P_{aO_2}$ relationship based on gestational age, postnatal age, or pH could be identified.

As previously noted, we also looked at 70 $S_{pO_2}$-$P_{aO_2}$ pairs in a single neonate to determine whether $S_{pO_2}$ trended $P_{aO_2}$ more closely in this individual than in the general population. Seventy $S_{pO_2}$ values from a 32-wk gestational-age male neonate (Neonate P) during his first 9 days of life were plotted against $P_{aO_2}$ values (Fig. 4) in a similar fashion as in the general intensive care population. A comparison of Neonate P's data with those of the general population demonstrates a similar scatter of $P_{aO_2}$ in relation to $S_{pO_2}$, with the exception that slightly higher $S_{pO_2}$ values were more consistently associated with normoxemia. It appears that individual $S_{pO_2}$-$P_{aO_2}$ relationships may be somewhat skewed upward or downward from infant to infant. This suggests that the saturation limits required to maintain normoxemia may vary from infant to infant.

Establishing $S_{pO_2}$ limits will help avoid hyperoxemia and hypoxemia. However, the problem of a high false-negative rate, where $S_{pO_2}$ values outside the established limits yield normoxemic $P_{aO_2}$ values, and the occasional aberrant $S_{pO_2}$-$P_{aO_2}$ relationships will occur. The shape of the $O_2$ dissociation curve, its complexity, and the complexity of pulse oximetry signal processing make overcoming these problems formidable.

Work by Severinghaus and Naifeh and Nickerson et al. has shown that bias and precision differ from oximeter to oximeter. This is a result of manufacturers' using different empirical calibration curves and standards for determining oxygen saturation in their instruments. Therefore, there is no one range of saturation that will protect against hyperoxemia and hypoxemia. The range must be established for each specific oximeter. For example, using an Ohmeda 3700 pulse oximeter, normoxemia was found to be predictable, but with substantially different $S_{pO_2}$ limits than we found with Nellcor oximeters. $^{10,13}$ It is important to remember that bias and precision do differ from oximeter to oximeter. $^{19,21,31}$ Because we were assured by the manufacturer (personal communication, J Stephani, Nellcor Inc, Hayward CA) that the N-100 and N-200 oximeters react similarly and should produce identical $S_{pO_2}$ values in spite of software differences, we chose to include in our study $S_{pO_2}$ values from both types of oximeters.

Caution should be used when applying these results to bronchopulmonary dysplasia (BPD) patients because BPD patients made up only a small percentage of our population (Table 1). The majority of our patients were in their first week of life (mean postnatal age 4.6 days) at the time of study comparisons. However, other authors have demonstrated satisfactory correlation between pulse oximetry saturation values and CO-oximetry saturation values in the BPD population. $^{9,12}$
Pulse Oximetry in Neonatal Intensive Care

Summary and Conclusion

In summary, infants in the neonatal intensive care setting are susceptible to rapid changes in oxygenation. Intermittent arterial blood gas sampling cannot quickly and accurately identify acute episodes of hypoxemia and hyperoxemia, but simple continuous oxygen monitoring by pulse oximetry can identify such circumstances and allow for quick intervention. Furthermore, maintaining pulse oximetry saturation values within established limits will prevent hypoxemia and hyperoxemia in most cases despite the nuances of the oxyhemoglobin dissociation curve in the neonatal period.

Further studies are necessary to determine the significance of acute increases and decreases in $SpO_2$, how great a saturation change should be of concern, and how long a change should be tolerated before intervention is begun. Pulse oximetry monitoring cannot be expected to identify all hypoxemic and hyperoxic episodes in neonatal intensive care. It is recommended that different brands (or makes) of pulse oximeters not be used interchangeably in the neonatal intensive care setting because $SpO_2$ readings from different brands correlate differently with $SaO_2$ and $PaO_2$. Industry standardization of calibration algorithms would help to assure better agreement from one oximeter to the next. However, it is our opinion that $SpO_2$ limits (specific to pulse oximeter brand) can reasonably be used to maintain $PaO_2$ within a specified range and to assure normoxemia in the newborn population.

ACKNOWLEDGMENTS

The authors thank Nancy Scarborough for her help in the typing of this manuscript.

PRODUCT SOURCES

Pulse Oximeters:
Nellcor N-100, Nellcor Inc, Hayward CA
Nellcor N-200, Nellcor Inc, Hayward CA

Sensors:
N-25 Neonatal Sensor, Nellcor Inc, Hayward CA
I-20 Infant Digit Sensor, Nellcor Inc, Hayward CA

Blood Gas Analyzer and Controls:
AVL 945 Blood Gas Analyzer, AVL Scientific Inc, Graz, Austria
GAS Trak Blood Gas Controls, Curtin Matheson Scientific Inc, Houston TX

REFERENCES


APPENDIX

Definitions

Sensitivity is conventionally defined* as the ability of a test to single out people who have disease. In the context of this study, the ‘disease’ is normoxemia (i.e., $P_{aO_2} \geq 45$ torr [6.0 kPa] and $\leq 90$ torr [12 kPa]) and sensitivity refers to the ability of pulse oximetry ($S_{PO_2}$ within specified limits) to detect normoxemia.

Specificity is conventionally defined* as the ability of a test to classify people who do not have disease as negative. In the context of this study, those without disease are those with a $P_{aO_2} < 45$ torr [6.0 kPa] or $> 90$ torr [12 kPa], and specificity refers to the ability of pulse oximetry ($S_{PO_2}$ outside specified limits) to detect hypoxemia or hyperoxemia.

Positive predictive value is conventionally defined* as the frequency with which a positive test actually signifies disease. In the context of this study, positive predictive value refers to the frequency with which pulse oximetry oxygen saturation ($S_{PO_2}$) within specified limits accurately reflects normoxemia (45 torr [6.0 kPa] $\leq P_{aO_2} \leq 90$ torr [12 kPa]).

True positive is a positive test ($S_{PO_2}$ within the specified range) that accurately reflects the presence of disease (normoxemia).

False positive is a positive test ($S_{PO_2}$ within the specified range) that fails to accurately reflect the absence of disease (hyperoxemia or hypoxemia).

True negative is a negative test ($S_{PO_2}$ outside the specified range) that accurately reflects the absence of disease (hyperoxemia or hypoxemia).

False negative is a negative test ($S_{PO_2}$ outside the specified range) that fails to accurately reflect the presence of disease (normoxemia).


Predictive Value Calculations

| Normoxemia ($45$ torr [6.0 kPa] $\leq P_{aCO_2} \leq 90$ torr [12 kPa]) |
|---|---|---|
| Yes | True Positive (TP) | False Positive (FP) |
| No | False Negative (FN) | True Negative (TN) |

Sensitivity = TP/(TP + FN).
Specificity = TN/(TN + FP).
Positive Predictive Value = TP/(TP + FP).

Example

(353 cases included in the study)

$P_{aO_2} \leq 90$ torr [12 kPa]

<table>
<thead>
<tr>
<th>Yes</th>
<th>True Positives (TP = 198)</th>
<th>False Positives (FP = 4)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No</td>
<td>False Negatives (FN = 104)</td>
<td>True Negatives (TN = 47)</td>
</tr>
</tbody>
</table>

Sensitivity = 198/(198 + 104) = 0.6556.
Specificity = 47/(47 + 4) = 0.9216.
Positive Predictive Value = 198/(198 + 4) = 0.9802.
Sputum Induction: A Quick and Sensitive Technique for Diagnosing *Pneumocystis carinii* Pneumonia in Immunosuppressed Patients

Cynthia R Godwin BA RRT, Dennis T Brown CRTT, Henry Masur MD, Vec J Gill PhD, and Frederick P Ognibene MD

The incidence of *Pneumocystis carinii* pneumonia has increased primarily because of the increase in the number of persons with human immunodeficiency virus (HIV) infection. Consequently, there has been an impetus to develop faster and more economical diagnostic techniques. Earlier reports have indicated that pneumocystis pneumonia can be diagnosed from recovery of the pneumocystis organism from sputum. We sought to determine the sensitivity of sputum induction supervised by respiratory care practitioners using ultrasonic nebulization as a noninvasive diagnostic procedure in patients with suspected pneumocystis pneumonia.

**METHODS & MATERIALS:** From January 1987 through December 1989, all patients at our institution suspected of having pneumocystis pneumonia underwent sputum induction with 3% sodium chloride administered via ultrasonic nebulizer.

**RESULTS:** 429 sputum induction procedures were performed on 272 patients (198 HIV-positive, 74 HIV-negative). In patients who had multiple inductions, each procedure was considered a separate patient episode. Eighty-eight sputum specimens were positive for *P carinii*, 328 specimens were negative, and sputum could not be obtained 13 times. One hundred and fifty-four of the 328 sputum-negative patient episodes were followed by bronchoscopy that yielded sputum positive for *P carinii* in 16 patients. The 174 sputum-negative patients who were not followed by bronchoscopy were followed clinically for a minimum of 3 months. Of those 174 patients, 161 (128 HIV-positive and 33 HIV-negative) improved without treatment and 13 were treated empirically and improved. The 13 patients who were unable to produce sputum were followed by bronchoscopy with 5 positive and 8 negative specimens resulting. If one considers the true positives to be comprised of those who were positive by sputum induction, those negative by sputum induction but positive by bronchoscopy, and those negative by sputum induction but positive by clinical presentation who improved with treatment, and if one considers the false negatives to be those who were negative by sputum induction but positive by bronchoscopy or negative by sputum induction but positive by clinical presentation who improved with treatment, then sensitivity was 83% in HIV-positive patients and 69% in HIV-negative patients. **CONCLUSIONS:** Sputum induction via ultrasonic nebulization is a sensitive, noninvasive technique that can be effectively administered by respiratory therapists. Although we did not directly study cost, time, and morbidity, the technique may contribute to a reduction in those factors in establishing the diagnosis of *P carinii* pneumonia. (Respir Care 1991;36:33-39.)

Ms Godwin is Chief and Mr Brown is Supervisor, Critical Care Therapy Section; Dr Masur is Chief; and Dr Ognibene is Senior Investigator and Head, Pediatric Section—Critical Care Medicine Department, Warren G Magnuson Clinical Center, National Institutes of Health, Bethesda, Maryland. Dr Gill is Chief, Microbiology Laboratory, Department of Clinical Pathology at the Clinical Center.

This study was completed at the National Institutes of Health, Warren G Magnuson Clinical Center, Bethesda, Maryland. Ms Godwin presented some of the data in preliminary form at the Respiratory Care Open Forum during the 1987 AARC Annual Meeting in Las Vegas, Nevada.

Reprints: Cynthia R Godwin BA RRT, Critical Care Medicine Department, Warren G Magnuson Clinical Center, National Institutes of Health, Building 10, Room 10D48, Bethesda MD 20892.
Introduction

The frequency of Pneumocystis carinii pneumonia has greatly increased in the United States during the last decade, due in large part to the growing number of people with the acquired immunodeficiency syndrome (AIDS). As a result, there has been an urgent need to develop faster and more economical diagnostic techniques. Bronchoscopy, with bronchoalveolar lavage and transbronchial biopsy, has become the standard method of making the diagnosis of *P. carinii* pneumonia. Although highly sensitive and accurate in yielding the diagnosis, bronchoscopy is invasive and associated morbidity and occasional mortality have been reported.

Earlier published reports suggesting that *P. carinii* could be recovered in sputum in up to 55% of patients with human immunodeficiency virus (HIV) infection and documented pneumocystis disease, led us to evaluate the accuracy of sputum induction as a noninvasive diagnostic procedure in patients in our institution. Our premise was that if the diagnosis of *P. carinii* pneumonia could be established in an induced sputum specimen with a reasonable degree of accuracy, then many patients could avoid the discomfort and risks of bronchoscopy or open-lung biopsy procedures. In addition, the sputum-induction technique might provide a more rapid and less costly method of making the diagnosis for the many institutions that have been overwhelmed with escalating numbers of patients requiring workup for *P. carinii* pneumonia.

Methods & Materials

Patient Population

All patients at risk for developing *P. carinii* pneumonia who had clinical signs and symptoms of pneumonia were considered for sputum induction. The patients were under treatment by either the National Cancer Institute, the National Institute of Allergy and Infectious Diseases, or the Critical Care Medicine Department of the Warren G Magnuson Clinical Center, National Institutes of Health, between January, 1987 and December, 1989. HIV status was assessed by standard ELISA and Western Blot tests* using conventional methods.

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

Sputum-Induction Techniques and Sputum Processing

All sputum inductions were done by the same group of critical care therapists at the Clinical Center, according to a standardized technique.

Therapists followed the Clinical Center's universal precautions policy, which included the wearing of a mask and gloves during the sputum-induction procedure and the handling of the specimen afterwards. All items used were single-use items (with the exception of the ultrasonic nebulizer) and were disposed of immediately after the procedure. The ultrasonic nebulizer couplant cup was decontaminated with a disinfectant spray.

A 3% sodium chloride solution was obtained by mixing 5 10-mL vials of 10% sodium chloride with the 150 mL of 0.9% sodium chloride present in a prefilled reservoir designed for use with an ultrasonic nebulizer. Approximately 50 mL of the resulting 3% sodium chloride solution were poured into a cup, and patients were instructed to gargle and to rinse their mouths several times immediately prior to sputum induction in order to reduce the quantity of oral contaminants (vegetable particles, yeast, and other debris). In our laboratory's experience, the presence of some common organisms, such as *Candida* species, can make it difficult for the microbiologist to identify *P. carinii* with certainty, especially when the pneumocystis organism is present in scant amounts. The gargling was omitted in children who were too young to cooperate.

A DeVilbiss Ultra-Neb 100 ultrasonic nebulizer was used for all sputum inductions. Nebulization time ranged from 10 to 45 minutes (with an average time of 20 minutes) for an acceptable specimen to be obtained. Most patients preferred to hold the aerosol mask in front of their faces rather than to have it strapped in place. Small children usually sat on a parent's or a therapist's lap with that person directing the mist from the aerosol tubing toward the child's face. Patients were instructed to produce a deep-cough specimen if possible, and the induction procedure continued until the therapist was satisfied that an 'optimal' specimen had been obtained or until the procedure had taken 45 minutes without results. An optimal specimen was one that resulted from a maximal patient effort and appeared to be a deep-cough specimen. Patients were instructed to expelorate all sputum into a sterile specimen cup.
Patients who were too young to follow directions for expectorating their sputum inhaled the aerosolized 3% sodium chloride solution, but they were then oropharyngeally or nasotracheally suctioned by the therapist when cough or auscultation indicated the presence of secretions. During suctioning procedures, patients were continuously monitored by electrocardiogram and pulse oximeter. Children did not drink or eat for the 4 hours prior to the induction procedure to limit the risk of aspiration if suctioning was required to obtain satisfactory specimens.

All specimens judged to result from maximal patient effort were processed (even those that may have appeared to be saliva). The therapist diluted the specimen with an equal volume of sterile water to diminish possible hypertonic effects of the saline solution on the pneumocystis organisms. The duration of nebulization, patient effort, specimen appearance, and any variance from standard technique (e.g., the need to suction a small child) for each sputum-induction procedure were documented. Specimens were taken immediately to the clinical microbiology laboratory for processing or were refrigerated overnight before being processed by the laboratory the next morning. All specimens were processed and read within 24 hours of being obtained.

Sputum was stained by the standard toluidine-blue-O12 and human monoclonal immunofluorescent techniques.13 A sputum sample positive for *P carinii* is considered diagnostic for pneumocystis pneumonia8 and thus can be considered a true positive. Our protocol required that one characteristic cluster of cysts be seen by one method for the diagnosis of pneumocystis pneumonia to be made.

**Results**

During the 3-year period of the study, 429 sputum-induction procedures were performed on 272 patients considered at risk for pneumocystis pneumonia. In patients who underwent multiple induction procedures, most procedures performed represented separate patient episodes and were counted as such. Patients had evidence of pulmonary dysfunction manifested by either symptoms, hypoxemia, or chest roentgenographic abnormalities, or they were being evaluated by a protocol assessing subclinical pulmonary disease resulting from pneumocystis or cytomegalovirus (CMV) infection. The majority (73%) of patients had HIV infection. Seventy-four patients (27%) who were negative for HIV antibodies also underwent sputum induction. The HIV-negative patients were at risk for *P carinii* pneumonia because of immunosuppressive disorders or therapies. Patients ranged in age from 8 months to 65 years. Eighty-six patients underwent multiple sputum inductions during the course of the study for the same or serial episodes of pneumocystis infection.

**HIV-Infected Patients**

**All Patients:** One hundred ninety-eight HIV-infected patients underwent sputum induction for 342 episodes of clinical pulmonary disease (Fig. 1). The diagnosis of pneumocystis pneumonia was made from 76 (22%) sputum specimens from 65 patients. (Eighteen of the patients with 76 positive-sputum specimens underwent bronchoscopy that also demonstrated *P carinii.*) Two hundred fifty-six specimens in 123 patients were

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![Fig. 1. The results of sputum induction and bronchoscopy to establish the diagnosis of *P carinii* pneumonia in HIV-infected patients with signs and symptoms of pneumonia. (PC = *P carinii.*)](image-url)
negative for *P. carinii*, and 10 patients (3%) were unable to produce sputum after 45 minutes of the procedure. Of the 256 sputum-negative patient episodes, 118 were subsequently followed (within 2 days) by bronchoscopy, and only 10 of 118 episodes (8.5%) were diagnosed as pneumocystis pneumonia. The other 108 specimens were negative for *P. carinii* on bronchoscopy, bronchoalveolar lavage, or biopsy, thus confirming the negative-sputum results. Of the 10 patients unable to produce sputum, all underwent bronchoscopy, which yielded 4 additional cases of pneumocystis pneumonia. The 138 sputum-negative, HIV-positive patients who were not followed with bronchoscopy were followed clinically for a minimum of 3 months. One hundred and twenty-eight of those 138 improved without treatment. The remaining 10 (9 of whom were small children) were treated empirically and improved. Of the 86 HIV-infected patients who were able to produce sputum, 76 were diagnosed with pneumocystis pneumonia by the sputum-induction technique. Overall sensitivity of the procedure was 88.3% (76 of 86), and specificity was 100%. One hundred and eighty-four of 194 sputum-induction procedures provided an accurate result when bronchoscopy was used as the confirmatory procedure.

**Pediatric Patients:** Seventeen of the 198 HIV-infected patients who underwent this procedure were less than 15 years of age. Data from some of these patients have been presented elsewhere. The children ranged in age from 8 months to 15 years, with a mean age of 5.6 years. The same method was followed for adults and children. Ten of the children were 5 years of age or younger, and all but one of these younger children required oropharyngeal or nasotracheal suctioning in order to obtain an optimal specimen. No adverse reactions resulted from the suctioning procedure.

The 17 children underwent sputum induction for 28 episodes of clinical pulmonary disease. The diagnosis of pneumocystis pneumonia was made in 8 episodes, and 19 episodes were negative for pneumocystis. One child was unable to produce a specimen after 45 minutes of the procedure. Of the 19 specimens negative for *P. carinii*, 2 were confirmed negative by bronchoscopy, and the remaining 17 were considered negative due either to other documented infectious processes or clinical follow-up. The child who was unable to produce sputum underwent bronchoscopy, which was also negative.

**HIV-Negative Patients**

**All Patients.** Seventy-four patients without HIV infection underwent sputum induction for 87 episodes of possible pneumocystis pneumonia (Fig. 2). The diagnosis of *P. carinii* was made in 12 sputum specimens from 10 patients. Seventy-two specimens from 62 patients were negative for *P. carinii*, and 3 patients were unable to produce sputum after 45 minutes of the procedure. Of the 72 sputum-negative patient episodes, 36 were subsequently followed by bronchoscopy that yielded 6 additional cases of *P. carinii*. Of the three patients unable to produce sputum, all underwent bronchoscopy, and one was positive for *P. carinii*. In those patients uninfected by HIV, who were able to produce sputum, 12 of

![Diagram](attachment:image.png)

**Fig. 2.** The results of sputum induction and bronchoscopy to establish the diagnosis of *P. carinii* pneumonia in HIV-negative patients with signs and symptoms of pneumonia. (PC = *P. carinii*.)
18 episodes of pneumocystis pneumonia, or 66.7%, were diagnosed by the induced-sputum technique. Thus, sensitivity of the procedure in these patients was 66.7%, and specificity was 100%. Forty-two of the 48 patient episodes in which sputum was obtained yielded the correct diagnosis when bronchoscopy was used as the confirmatory procedure.

Pediatric Patients. Eight of the 74 patients without HIV infection who underwent this procedure were children. The children ranged in age from 3 to 9 years and had underlying diagnoses of aplastic anemia (n = 2), acute lymphoblastic leukemia (n = 2), and other malignancies (n = 4). Only the 3-year-old required nasotracheal suctioning to obtain a specimen after saline nebulization. The suctioning process was uneventful. The 8 children underwent 12 sputum inductions resulting in 3 specimens positive for *P carinii* and 9 negative specimens. Of the 9 specimens negative for pneumocystis, 3 were confirmed negative by bronchoscopy. Patients producing the remaining 6 specimens were either treated empirically for pneumocystis pneumonia or were treated definitively for another bacterial pathogen documented by the procedure.

Adverse Effects of the Sputum-Induction Technique

All 272 patients tolerated ultrasonic nebulization. No episodes of bronchospasm were induced by the procedure, and only two patients who had histories of bronchospasm were pretreated with bronchodilators. No patients developed laryngospasm, progressive hypoxemia, or hoarseness during or after the procedure. Nausea and rare instances of vomiting (in two adult patients) were the only adverse effects of the procedure, and the effects did not persist after the procedure was completed.

Only 10 of the 272 patients (all children) required either oral or nasotracheal suctioning after the induction procedure, and none of them experienced airway trauma or bleeding as a consequence of the ultrasonic nebulization and suctioning. Like adults, children did not receive sedative agents or other medications for the procedure.

Discussion

In this study, application of the sputum-induction technique provided specimens that allowed the diagnosis of *P carinii* pneumonia to be made with a high rate of sensitivity in both adult and pediatric patients (83% in HIV-infected patients and 69% in HIV-negative patients). These results are quite different from previous studies,7-9 which reported sensitivities of induced specimens ranging from 2-55%. One study15 reported that specimens obtained by sputum induction were less sensitive than nasotracheal suctioning and lavage specimens for the diagnosis of *P carinii*. In that study, 10% sodium chloride solution was administered by handheld nebulizer, and ultrasonic nebulization was not utilized to obtain specimens. Although many hospitals may not own ultrasonic nebulizers because they are not as commonly used as they once were, we speculate that ultrasonic nebulization may be a major factor for our success in obtaining diagnostic specimens.

The sputum-induction technique described in our study appears to be a consistent means of noninvasively obtaining optimal specimens, even though a number of therapists perform the procedure. During the 3-year course of this study, approximately 20 critical care therapists administered the sputum-induction procedure following a standard protocol. Accuracy of diagnosis from sputum was related to whether the patient actually had *P carinii* rather than variance in technique among therapists. Previously, other studies have demonstrated the potential of the induced-sputum technique to produce a high degree of sensitivity and specificity,16-20 but these studies have not been addressed specifically to the respiratory care practitioner's performing the procedure.

The noninvasive, high-yield nature of our sputum-induction procedure has expedited the diagnosis of *P carinii* in many patients in our institution and often has allowed them to avoid discomfort and risks involved in bronchoalveolar lavage or nasotracheal suctioning. Although bronchoscopy with bronchoalveolar lavage and transbronchial biopsy continues to have a higher overall yield for *P carinii* than does the sputum-induction technique, sputum induction, due to its high sensitivity, has replaced bronchoscopy in our institution as the initial procedure for those patients suspected of having *P carinii*. If a negative sputum is obtained, then bronchoscopy is required in order to make the diagnosis of *P carinii*.

With the advent of monthly prophylaxis with aerosolized pentamidine to prevent pneumocystis pneumonia in HIV-infected patients, it is quite
possible that the sensitivity of the induced-sputum technique will decrease, perhaps because disease develops in association with fewer organisms, because the disease is confined to upper lobes, or because pentamidine may alter organism morphology. During the last 3 months of our study, when pentamidine prophylaxis had become common, four patients with negative sputum specimens were found to be positive for P carinii by bronchoscopy (three had been receiving aerosolized pentamidine prophylaxis). This is in contrast to a total of 20 false-negative sputums in HIV-infected patients for the entire 36-month period of the study. A recent abstract that retrospectively examined the effect of prophylaxis for pneumocystis pneumonia on the diagnosis of P carinii by induced sputum during the past 2 years, concluded that induced sputum from HIV-infected patients receiving prophylaxis had a lower sensitivity for organism detection than sputum from patients not treated prophylactically (71.4% versus 96% in HIV-infected patients, p < 0.05).21

Conclusions

The data demonstrate that sputum specimens obtained by our induction technique made the diagnosis of P carinii with a 83% sensitivity in immunosuppressed patients with HIV disease. In other immunosuppressed patients, the sensitivity fell to 69%. This noninvasive technique has proven to be quick and safe in both adult and pediatric patients. Although we did not directly study cost, reductions in time and morbidity may lead to reduction in the cost of establishing the diagnosis of P carinii pneumonia.

ACKNOWLEDGMENTS

We thank the team of Critical Care Therapists, the Microbiology Service, and Frida Stock and Nancy Nelson for their participation in this study.

PRODUCT SOURCES

Ultrasonic Nebulizer:
Ultra-Neb 100, DeVilbiss Health Care Inc, Somerset PA

Disinfectant:
Sporicidin spray, Sporicidin International, Rockville MD

Saline Solutions:
10-mL vials of 10% sodium chloride, Dey Laboratories, Napa CA
0.9% sodium chloride, #145 AquaPak, Respiratory Care Inc, Arlington Heights IL

Sputum Stains:
toluidine-blue O, Aldrich Chemical Co Inc, Milwaukee WI
human monoclonal immunofluorescent stain:
(1) an in-house direct fluorescent antibody stain (IFA-NIH) National Institutes of Health, Bethesda MD
(2) Mab immunofluorescent stain, Pneumocystis carinii Immunofluorescent Test Kit, Genetic Systems Corp, Seattle WA

HIV-Detection Tests:
ELISA, DuPont, Wilmington DE
Western Blot, Bio-Tech, Rockville MD

REFERENCES


Bench Evaluation of the Novametrix Neonatal C/D Endotracheal Tube Adaptor for Use during High-Frequency Oscillatory Ventilation

Kaye R Weber BA RRT, Sherry E Courtney MD, John F Hopson CPFT RRT

BACKGROUND: High-frequency ventilation (HFV) is being used for the treatment of newborn respiratory distress in many intensive care nurseries. Some therapies important to the care of infants receiving HFV, such as endotracheal-tube (ETT) suctioning, have not been thoroughly studied. Suctioning frequently results in the clinical deterioration of the infant and is probably due to the loss of lung volume and oxygen delivery that occurs when the ventilator is disconnected. Although some methods of closed airway suctioning have been used successfully with conventional ventilation, their use with HFV has not been reported. EVALUATION METHOD: We conducted a bench evaluation of the Novametrix Neonatal C/D suction adaptor (CD) in conjunction with the SensorMedics 3100 High Frequency Oscillatory Ventilator. We determined the differences in % CO₂ between CD and standard (STD) connectors for three ETT sizes (2.5-, 3.0-, and 3.5-mm at 20-second time intervals). Test-lung CO₂ concentrations were sampled and recorded by a clinical capnometer coupled to a recording oscilloscope. Significance of differences was tested by analysis of variance for repeated measures. Resistances to airflow (R) offered by CD and STD, with ETT of the three sizes were compared. RESULTS: The differences in % CO₂ were not significant; however, increased resistance was seen with CD. In addition, we were able to show that the presence of the suction catheter in the lumen of the ETT causes only a small pressure rise in the test lung. CONCLUSIONS: The use of the Novametrix Neonatal C/D suction adapter does not affect the performance of the SensorMedics 3100 and thus may be advantageous in the care of the neonate managed on this ventilator. However, the increase in resistances seen with the CD especially when coupled to the 2.5-mm ETT may be clinically important, particularly in the spontaneously breathing patient. (Respir Care 1991;36:40-44.)

Introduction

High frequency ventilation (HFV) is a relatively new technique currently either in use or under investigation in intensive care nurseries. ¹²

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A version of this paper was presented by Ms Weber at the RESPIRATORY CARE Open Forum during the 1989 AARC Annual Meeting in Anaheim, California.

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In our nursery, when patients on the high-frequency ventilator commonly used (SensorMedics 3100 High Frequency Oscillatory Ventilator, or SM*) require suctioning to maintain endotracheal-tube (ETT) patency, they must be removed from the ventilator to allow passage of the catheter. During this procedure, infants may experience some degree of clinical decompensation as evidenced by changes in transcutaneous partial pressure of oxygen (PteO₂), carbon dioxide (PteCO₂), and pulse oximetry oxygen saturation (SpO₂). The degree of decompensation is highly variable and is probably related to the loss of mean airway pressure, functional residual capacity (FRC), and FIO₂.¹³⁴ Investigators have shown that when ETT suctioning can be accomplished without

*Suppliers are identified in the Product Sources section at the end of the text.
total interruption of ventilation and oxygenation, the degree of decompensation is significantly less.\(^5\)\(^-\)\(^9\) The Novametrix Neonatal C/D suction adaptor (CD) has been used successfully during conventional mechanical ventilation,\(^8\)\(^,\)\(^9\) but its use with HFV has not been reported. Any alteration in the patient circuit, including the ETT, can produce changes in ventilator performance and subsequently in patient condition, and thus must be carefully evaluated prior to use in patients.\(^10\)\(^-\)\(^12\) Therefore, we compared CD to the standard ETT connector (STD) for CO\(_2\) elimination and resistance to airflow using a test lung ventilated by the SM. Additionally, we tested CD using a simulated suction procedure to verify that the presence of a catheter in the lumen of the ETT would not cause an inadvertent increase in distal test-lung pressure.

**Description of Devices**

CD is a low dead-space, flush-design ETT connector (Fig. 1). On two sides are zipper-like openings provided for the insertion of the suction catheter. When the catheter is withdrawn, the ‘zipper’ is closed and the ventilator circuit is again a closed system. SM is a true oscillator by virtue of its active expiratory phase. The driver is a linear piston that provides variable inspiratory times (30-70%), and displaces a maximum of 500 mL/stroke, unloaded.

The patient circuit is designed to offer very low impedance and is constructed primarily of acrylic resin.

**Methods of Evaluation**

One of each neonatal size (2.5-, 3.0-, and 3.5-mm) Murphy-eye ETT was cut to 9.0-cm length and combined alternately with CD and STD connectors. A 1.2-L glass bottle was used as the test lung. The neck stopper contained a 5-Fr umbilical artery catheter, which terminated 10 cm inside the bottle, for the introduction of CO\(_2\); a short catheter for CO\(_2\) sampling, which extended through the stopper, and the ETT (Fig. 2). An airtight seal was verified by pressurizing the test lung and observing a stable, elevated water-column pressure. The sampling of % CO\(_2\) in the test lung was accomplished with a clinical capnometer, and its analog output was displayed on a digital storage oscilloscope (DSO). The DSO was calibrated to 1% CO\(_2\)/volt allowing us to read the concentration of CO\(_2\) in the jar directly from the DSO. The sampling line from the capnometer was attached to the sampling port of the test lung and 5% CO\(_2\) was supplied from a cylinder via the CO\(_2\) introduction catheter. A 3-way stopcock was placed in the gas line to allow us to quickly close the test lung to the cylinder gas.

![Fig. 1. Novametrix Neonatal C/D Suction Adaptor. (A) Airway lumen. (B) Solid plastic body. (C) Zipper-like suction ports. (D) Endotracheal tube connection.](image)

![Fig. 2. Experimental setup for CO\(_2\) washout. DSO = digital storage oscilloscope. HFOV = high-frequency oscillatory ventilator.](image)
SM was preset to standardized settings: mean airway pressure 10 cm H₂O [0.9 kPa], root-mean-square pressure 10 cm H₂O [0.9 kPa] (corresponding to a pressure amplitude of 32-34 cm H₂O [2.9-3.1 kPa]), flow 10 L/min, frequency 15 Hz, and percent inspiratory time 33. An ETT was inserted into the stopper, and the test lung was flushed with the 5% CO₂ mixture. Once the concentration of CO₂ in the test lung reached 4.8%, the stopcock was closed, and the ventilator was connected. The curve of CO₂ elimination was traced by the oscilloscope. The test was repeated three times for each ETT size using the CD and STD connectors. Percent CO₂ was read at each second of each trial beginning with 4.5% CO₂ as Time 0 (zero) and ending when % CO₂ was less than 0.1. The time required for complete washout of the test lung varied inversely with ETT size. All trials were done by a single investigator (KRW) to ensure reliability. The mean CO₂ elimination curve for each ETT-connector combination is shown in Figure 3.

These data were recorded on a computer spreadsheet for later analysis. The mean and standard deviation of % CO₂ were calculated for the three trials. The mean values of % CO₂ for CD and STD at 20-second intervals were compared for each ETT size by analysis of variance for repeated measures.

The CD and STD for each tube size were then tested for resistance to airflow (R). The respective ETT connector was attached to a U-tube water manometer and to 6 L/min gas flow. The back pressure created by the ETT connector was recorded, and R (in cm H₂O · s · L⁻¹) was calculated.

To test the effect of the presence of a suction catheter in the lumen of the tube on test lung volume, the mean pressure in the test lung was monitored during a simulated suctioning procedure using CD, a 2.5-mm ETT, a 6 Fr suction catheter, and the test lung. Because of the small lumen of the 2.5-mm ETT, data derived from this combination should represent 'worst case' for this phenomenon. Mean pressure and amplitude changes in the test lung were measured during ventilator operation. A pressure transducer was connected to the short catheter, which terminated just inside the test lung (Fig. 2). The signal from this transducer was conditioned and traced on a chart recorder. Measurements were made with (1) the suction port closed, (2) the suction port open, (3) with a catheter in the lumen of the ETT, and finally (4) with the catheter withdrawn from the lumen and

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Fig. 3. (A) Graph of mean % CO₂ vs time for 2.5-mm; (B) 3.0-mm; (C) 3.5-mm ETT. Square symbols represent the datapoints corresponding to the trials using the Novametrix Neonatal C/D adaptor and the triangular symbols those for the standard adaptor.
the suction port closed. The operator left the catheter in the lumen for 1 minute to verify that any pressure change caused by its presence was stable.

Results

No significant differences were seen in % CO₂ between CD and STD for any ETT size at any time period. R was higher for CD for all ETT sizes (Table 1).

Table 1. Resistance Values for the Novametrix Neonatal C/D Endotracheal Tube Adaptor (CD) and a Standard Adaptor (STD), with Endotracheal Tubes of Three Sizes and Gas Flow at 6 L/min

<table>
<thead>
<tr>
<th>Tube Size mm ID</th>
<th>Resistance of CD cm H₂O • s • L⁻¹</th>
<th>Resistance of STD cm H₂O • s • L⁻¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.5</td>
<td>72 [6.5]</td>
<td>46 [4.2]</td>
</tr>
<tr>
<td>3.0</td>
<td>30 [2.7]</td>
<td>21 [1.9]</td>
</tr>
<tr>
<td>3.5</td>
<td>17 [1.5]</td>
<td>12 [1.1]</td>
</tr>
</tbody>
</table>

During the simulated suctioning procedure, an almost complete loss of oscillation amplitude occurred, particularly during the time that the catheter was inside the lumen of the tube. When the port was opened, mean pressure in the test lung fell from 10 cm H₂O to 4 cm H₂O [0.9 to 0.4 kPa]. The catheter was inserted in the ETT tube, and mean pressure rose to 5 cm H₂O [0.5 kPa]. Regardless of the time interval for suctioning (up to 1 minute), no further rise in pressure occurred in the test lung.

Discussion

Because the design of the mechanical circuit, including the ETT, affects the efficiency of HFV, the addition of adaptors to any part of such a circuit must be thoroughly evaluated to ensure that their inclusion will not adversely affect ventilator function and, therefore, the patient's response.

The results of our evaluation of CD show that it does not alter the rate of CO₂ elimination from the test lung when compared to STD. Thus, ventilator performance is unaffected by the use of CD. Anecdotally, we have observed no change in patient condition when CD is used in conjunction with SM. Moreover, during suctioning we have seen little or no decompensation of the infants. Following closure of the suction port, it is possible that some recruitment maneuver such as sustained inflation will be required to re-establish lung volume. With SM, however, we have not found this maneuver to be necessary, even following complete lung volume disconnection as when the patients are suctioned using STD.

The higher R of CD was particularly marked with the 2.5-mm ETT. This may be of clinical importance especially in the spontaneously breathing patient. Resistance-related work of breathing may seriously impair the patient's response to therapy when the 2.5-mm CD is used.

We were not able to demonstrate a large increase in distal test-lung pressure during a simulated suctioning procedure using CD. It is likely, therefore, that only a small increase in lung volume will occur with the suction catheter in the ETT when the CD is used. Because in our experiment, test-lung pressure did not approach the therapeutic level of 10 cm H₂O [0.9 kPa], it is unlikely that a dangerous increase in pressure could occur during the maneuver. Although ventilation is interrupted as evidenced by complete loss of amplitude with the suction catheter in place, the test lung received essentially low-level continuous positive pressure, ensuring fresh gas delivery and maintenance of some test-lung volume during the procedure.

ETT suctioning has been implicated as causal in a variety of complications that can occur during the neonatal period. Hypoxemia is believed to be one of the first deleterious physiologic events that occurs during suctioning, and may contribute to the incidence of intraventricular hemorrhage. Some authors have shown that sedation improves the patient response to suctioning, whereas others have evaluated various methods of closed airway suction to minimize the harmful effects of the procedure. Guntupalli et al showed that during high-frequency jet ventilation adult patients do not exhibit significant hypoxemia during suctioning because ventilation and fresh gas flow are maintained throughout, via the distal jet lumen. However, the SM delivers fresh gas and ventilation via the proximal lumen of a standard, single-lumen ETT, and patients must be disconnected from the ventilator to allow suctioning. Because the suction catheter is inserted between the fresh gas source and the patient, the risk of overinflation during suctioning is minimal.

In conclusion, the use of CD may be a useful adjunct to suctioning patients on SM by assuring fresh
gas flow and maintaining lung volume. We recommend that infants on whom this device is used be monitored closely to ensure that acceptable levels of oxygenation and ventilation are maintained.

We have shown that the use of CD does not affect the efficiency of SM and that serious increases in test-lung pressure do not occur during a prolonged suctioning procedure. CD has been shown to be of value in reducing hypoxemia during suctioning of infants on conventional ventilation; further studies are needed to document the patient response to the use of CD with SM in neonates.

ACKNOWLEDGMENTS

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PRODUCT SOURCES

SensorMedics 3100 High Frequency Oscillatory Ventilator
SensorMedics Corp, Anaheim CA

Novametrix Neonatal C/D Suction Adaptor
Novametrix Medical Systems Inc, Wallingford CT

Murphy Eye Endotracheal Tubes
National Catheter Corp, Argyle NY

Umbilical Artery Catheter
CR Bard Inc, Bellerica MA

TMM 2000 Capnometer
Traverse Medical Monitors, Saline MI

Gould Model 1604 Digital Storage Oscilloscope
Gould Electronics, Cleveland OH

Gould P-50 Pressure Transducer
Gould Electronics, Cleveland OH

Gould Transducer Amplifier
Gould Electronics, Cleveland OH

Gould Chart Recorder
Gould Electronics, Cleveland OH

5% CO₂ Calibrating Gas
Corning Medical, Corning NY

U-Tube Water Manometer
Dwyer Instruments, Michigan City IN

REFERENCES


The Second Nagoya Conference: Triggering and Optimizing Mechanical Ventilatory Assist

Robert M Kacmarek PhD RRT, Yasuhiro Shimada MD, Akito Ohmura MD, Jun Takezawa MD, Akihiro Tokioka MD, Tomomasa Kimura MD, and Masaji Nishimura MD

Introduction

The purpose of the second Nagoya Respiratory Intensive Care Conference held in Nagoya, Japan, March 3, 1990, was to bring together researchers and clinicians with a stated interest in the work of breathing during assisted, or patient-initiated, ventilation (ie, ventilatory assist). Whereas the 1988 Nagoya Conference had focused on systems design and patient-ventilator interactions during pressure support ventilation, the presentations summarized in this report focus on aspects of work of breathing and ventilatory muscle function, with the unifying theme of the Conference being triggering and optimizing mechanical ventilatory assist. The specific questions were: What is the optimal method of triggering ventilator systems? and What parameter best assesses the most appropriate level of ventilatory assist?

We have organized this report to reflect the individual comments of each presenter, including discussions that occurred following individual presentations, and conclude with the results of a general discussion on the optimal method of triggering and assessing ventilatory assist.

Dr Kacmarek’s Presentation

This initial presentation focused on methods used to assess work of breathing and patient effort during ventilatory assist. Discussion also centered on the work imposed by the newest generation of mechanical ventilators during spontaneous ventilation.

Work of breathing (WOB), regardless of circumstances under which it is assessed, must relate the volume (V) change of the lung to the pressure (P) differential required to establish that volume change.

\[ WOB = (P)V \]

The most accurate estimate of work associated with moving the lung during spontaneous breathing requires an assessment of transpulmonary pressure changes (intrapleural minus intrapulmonary) with intrapleural pressure changes being reflected by intraesophageal pressure changes. However, only ventilator-circuit pressure changes are normally available at the bedside. Although ventilator-circuit pressure changes grossly underestimate actual intrapleural pressure changes, they do reflect the effort required during spontaneous ventilation. That is, a ventilator-circuit pressure drop of 5 cm H₂O during a spontaneous inspiration may, in fact, reflect an intrapleural pressure change of 15-20 cm H₂O or...
During assisted ventilation, the primary resistor within the respiratory system is the artificial airway. The effect of the artificial airway on patient work, or effort, is dependent upon the diameter and length of the airway and its actual contour. Of the airways commonly utilized, the nasal endotracheal tube appears to impose the greatest inspiratory load because of its length and the likelihood of its lumen’s being decreased by secretions and kinking within the nasal pharynx. As a result, pressure differences of 5 to 10 cm H2O are frequently noted across nasal endotracheal tubes during spontaneous breathing.

Mechanical ventilators have also imposed increases in work of breathing. However, recent data suggest that in spite of variation in the imposed WOB associated with present-day ventilators, overall imposed WOB is decreased over previous generations of ventilators, if they are functioning optimally and appropriately set. The addition of CPAP to ventilator systems increases the imposed work primarily as a result of the resistance offered by PEEP and exhalation valves. Flow triggering, as used in the Puritan-Bennett 7200a, appears to impose less WOB than the commonly used pressure-triggering mechanisms.

**Dr Ohmura’s Presentation**

Dr Ohmura presented data on the effects of chest-wall vibrations (100 Hz) applied at the second and third parasternal intercostal spaces on functional residual capacity (FRC) and tidal volume (VT) after upper abdominal surgery (UAS) and in healthy volunteers (HV). Triggering of the inspiratory chest-wall vibration was accomplished via a hot-wire flowmeter connected to the mouthpiece through which subjects breathed. In both groups, FRC increased (604 ± 429 mL HV and 199 ± 142 mL UAS) as did VT (66 ± 36% HV and 21 ± 13% UAS). Respiratory inductance plethysmography was used to identify chest-wall contribution to the increased volumes. It was noted, however, that chest-wall movement remained essentially constant before and after stimulation. As a result, the increased volumes were attributed to improved function of the diaphragm, which was confirmed with fluoroscopy.

It was concluded that stimulation of the tonic vibratory reflexes of the chest wall could not account for the changes noted, and that unknown reflex mechanisms that stimulate diaphragm activity play a significant role in increasing FRC and VT during chest-wall vibration. It was speculated that because lung volumes are commonly decreased as a result of atelectasis and upper abdominal surgery, and because diaphragm dysfunction is also common in these settings, the use of this chest-wall vibration technique in the postoperative period may improve ventilatory efficiency and decrease overall work of breathing.

**Dr Tokioka’s Presentation**

Auto-PEEP during weaning and the effect of pressure support ventilation (PSV) on the level of auto-PEEP were discussed by Dr Tokioka. Auto-PEEP is defined as the maintenance of a positive end-expiratory pressure in the alveoli as a result of insufficient expiratory time or airflow limitation, and may be present during either mechanical ventilation or spontaneous breathing. Auto-PEEP can be measured during controlled mechanical ventilation by occluding the airway at end-expiration. However, assessment during spontaneous breathing is best accomplished by the use of respiratory inductive plethysmography, simultaneous assessment of esophageal pressure, airway opening pressure and inspiratory flow, or by end-expiratory occlusion, followed by two to three spontaneous attempts at inspiration with relaxed exhalation. The end-expiratory plateau established following occlusion during spontaneous breathing is equal to the auto-PEEP present. However, the end-expiratory-plateau technique can only be used if relaxation of ventilatory muscles is assured during the expiratory phase.

The presence of auto-PEEP during spontaneous breathing markedly increases WOB because it increases the pressure differential that the patient must establish to decrease pressure in the ventilator circuit and initiate flow. If no auto-PEEP is present and ventilator sensitivity is set at -1 cm H2O, a pressure gradient of about 1 cm H2O is necessary to trigger flow. However, if the alveolar end-expiratory pressure is +5 cm H2O (auto-PEEP), a pressure gradient ≥ 6 cm H2O is required to trigger flow; thus, WOB is markedly increased.
In a series of 13 patients being weaned from mechanical ventilation, Dr Tokioka identified auto-PEEP in 5 patients (1 to 3 cm H2O). The application of PSV at 10 cm H2O decreased the auto-PEEP level to 0 cm H2O in four patients, and to 1 cm H2O in the remaining patients (Fig. 1). The elimination of auto-PEEP lessened the pressure gradient required to trigger the ventilator and thus decreased patient effort. The mechanism responsible for this reduction appears to be a change in ventilatory pattern. Pressure support ventilation increases the tidal volume while decreasing respiratory rate; therefore, expiratory time is lengthened, allowing alveoli to empty more completely and decreasing the likelihood of the development of auto-PEEP.

**Dr Takezawa’s Presentation**

Dr Takezawa’s presentation focused on the benefit of triggering and monitoring patient-initiated ventilation by changes in pleural rather than airway pressure. He argued that pleural pressure changes precede airway pressure changes, particularly in patients with long time constants, and that the ideal ventilatory assist, or sensitivity, mechanism would minimize actual pleural pressure fluctuations. Dr Takezawa presented data comparing pressure support triggered by pleural pressure changes (pleural pressure support ventilation, PPSV) to standard PSV and T-piece breathing in a mechanical lung model. Work of breathing and trigger delay time were determined under four conditions: normal compliance and resistance; decreased compliance; increased resistance; and increased resistance with decreased compliance. In all experimental settings, WOB was greatest during T-piece breathing, followed by PSV, and was least with PPSV. (Fig. 2). Because PPSV uses pleural pressure as a target pressure and attempts to minimize pleural pressure swings, it results in decreased WOB, particularly if resistance to gas flow is increased. However, the trigger delay time with PPSV did vary considerably with experimental setting. When compliance was reduced (0.04 L/cm H2O), a greater delay occurred with PPSV (200 ms) than with PSV (150 ms) regardless of resistance. However, when compliance was increased (0.12 L/cm H2O), the opposite relationship developed. The delay with PPSV was again 200 ms, but with PSV it increased to 350-400 ms. In addition, because PPSV attempts to maintain a constant pleural pressure, pressure assist continues into the initial aspect of expiration; that is, an end-inspiratory pressure spike develops as exhalation begins. This was also noted with PSV in the setting of high airway resistance.

**Dr Kimura’s Presentation**

Methods of titrating PSV to unload ventilatory muscles but still allow active patient involvement in ventilation vary considerably. Approaches presently used include: algorithms based on endotracheal tube size, calculations based on airway resistance, evaluation of accessory muscle function, evaluation of ventilatory pattern, and the actual calculation of work of breathing. All of these may provide appropriate levels of PSV, but frequently over- or underestimation of needed PSV levels occurs, or the WOB measurements needed are technically difficult to perform at the bedside. Dr Kimura presented data
on the selection of PSV levels based on the monitoring of transdiaphragmatic pressure (Pdi) levels. Using a single nasogastric tube with both esophageal and gastric water-filled catheters, Dr. Kimura evaluated Pdi in six mechanically ventilated patients during randomized 20-minute trials of 0 (spontaneous breathing) 5, 10, 15, and 20 cm H2O PSV (Fig. 3). He defined optimal PSV as the level that minimizes Pdi but maintains the normal negative inspiratory and positive expiratory esophageal pressure changes. He reasoned that if esophageal pressure becomes positive during inspiration, PSV is excessive and actually assumes most of the WOB. Because normal Pdi is about 10 cm H2O, and unloading of ventilatory muscles is the desired outcome, he defined the optimal Pdi during PSV as 4 cm H2O. In this series of patients, optimal PSV varied from 13 to 26 cm H2O, producing VT that varied from 6 to 19 mL/kg. He also noted that a minimal respiratory rate did not necessarily relate to the optimal PSV level.
Using least-squares analysis for determining the relationship between $P_{di}$ and PSV, the $P_{di}$ measured during a T-piece trial, and the optimal $P_{di}$ of 4 cm H$_2$O, Dr Kimura derived a formula for determining the optimal PSV level:

$$A = \frac{B - 4}{0.8},$$

where $A$ is the optimal PSV level, $B$ the $P_{di}$ on a T-piece trial, and 0.8 the slope of the $P_{di}$-PSV regression line determined by the least-squares method.

Dr Nishimura's Presentation

The relationship between patient WOB and percent of ventilatory support provided by SIMV in adults has been described by Marini et al. They noted in a group of critically ill adults requiring ventilatory support that patient work during spontaneous breaths and mechanical breaths increased as percent of support provided by SIMV decreased. Their study raised questions about the practice of maintaining critically ill patients at the lowest SIMV rate compatible with adequate gas exchange without considering the effect of the low SIMV rate on total patient effort.

Dr Nishimura presented data from three pediatric patients indicating the same phenomenon potentially exists in this population. Each of his patients was acutely ill and required ventilatory support. All had a nasogastric tube in place that allowed the measurement of gastric and esophageal pressures, and a pneumotachograph and pressure transducer affixed to the airway for flow measurement. Ventilatory support levels, using pressure-limited ventilation, were randomly applied at 100%, 75%, 50%, 25%, and 0% (CPAP) IMV. The index of patient effort used by Dr Nishimura was the product of inspiratory pressure and time (PTP). In addition, surface diaphragmatic electromyogram (EMG) sensors were placed at the seventh and eighth intercostal space. In all infants, during 75% or less ventilatory support, the esophageal pressure was negative during both mechanical and spontaneous inspirations. The total summed PTP for all breaths remained constant as percent ventilatory support decreased from 75 to 0, as well as the individual breath-by-breath PTP, for both spontaneous and mechanical breaths (Fig. 4). In this group of pediatric patients, IMV did not demonstrate a gradual increase in effort as rate decreased, but resulted in a consistent and sustained increase in effort as percent support decreased.

Discussion

The capabilities of the newest generation of mechanical ventilators are greatly improved over those of previous generations. Each can provide multiple ventilatory modes and gas delivery patterns and monitoring of the ventilator system and patient. However, in spite of these improvements, mechanisms for triggering gas flow and assessing the adequacy of ventilatory assistance have progressed little since the introduction of the first mechanisms for assisted ventilation, the IPPB devices. All modern ventilators, with the exception of the Puritan-Bennett 7200a, incorporate pressure-triggering mechanisms. Most units sense pressure on the inspiratory or expiratory limb internal to the ventilator. Few of today's ventilators sense pressure at the attachment to the artificial airway.
The flow-by triggering mechanism of the 7200a appears to be an improvement over standard pressure triggers. With the 7200a, triggering occurs when a patient diverts a bias gas flow from the expiratory flow sensor. Thus, anything that inhibits gas movement through the circuit (eg, water in the tubing) improves sensitivity, making triggering easier. This is the opposite of what occurs with a pressure trigger; however, self-cycling does occur with flow-by to a greater extent than with pressure triggering. In spite of the improvement in triggering afforded by flow-by, the sensor is still physically remote from the site of patient-generated effort.

Ideally, ventilators should be triggered by the motor impulses to the ventilatory muscles. This would, at least theoretically, eliminate much of the delay between the patient initiation of inspiration and the ventilator's delivery of gas and would ensure that only minimal patient effort would be required. However, at present this is not feasible and probably would require an unnecessary invasion of the patient.

However, options are available that could move triggering closer to the origins of patient muscular effort. A first step toward improved triggering would be triggering based on tracheal pressure changes. Because endotracheal tubes with proximal pressure-sensing ports are available, this option is easy to exercise. As noted already, pressure differences of 5 to 10 cm H2O do occur across endotracheal tubes. Triggering at the tracheal level would reduce work by eliminating the effect of the resistance of the artificial airway on triggering effort. This would be a marked improvement over present systems, but is not the ultimate approach because the effect of patient compliance and airways resistance on effort are not considered. Triggering based on diaphragmatic EMG activity, esophageal pressure, or transdiaphragmatic pressure would take into account the effects of compliance and airway resistance. Any of these approaches would minimize the effort required of patients with decreased compliance, increased airways resistance, or auto-PEEP. Although each of these techniques is associated with technical problems and patient invasion, the use of esophageal pressure change seems the most promising, as demonstrated by Dr Takezawa.

The problems in establishing a better triggering mechanism seem small when compared to defining the ultimate variable to determine the optimal level of ventilatory assistance. Investigators attempt to minimize ventilatory muscle effort during assisted ventilation by monitoring airway pressure changes, ventilatory patterns, and cardiopulmonary stress. These approaches are helpful in establishing ventilatory assistance levels, but are all crude assessments of patient effect. Oxygen consumption, work of breathing, pressure-time products, esophageal pressure and transdiaphragmatic pressure changes, and ventilatory muscle EMG activity provide a better assessment of patient effect. However, all are technically complex and some are invasive. The information presented by Dr Kimura regarding the use of Pdi seems promising. Although nasogastric catheters are invasive, the development of nasogastric catheters with water-filled double pressure monitoring ports (esophageal and gastric) makes monitoring of Pdi easier than historic approaches. However, much research is required before recommendations can be made.

Ventilatory assist techniques presently available are capable of meeting patient demands. Machines are responsive and delivery patterns almost infinitely variable. However, research related to ventilatory assistance must develop a broader scope, and future directions need to focus more on methods to optimize trigger efficiency and level of assistance as discussed in this conference. Much work is still needed.

REFERENCES

5. Kacmarek RM. The role of pressure support ventilation in reducing work of breathing. Respir Care 1988;33:99-120.
7. Gibney RTN, Wilson RS, Pontoppidan H. Comparison of work of breathing on high gas flow and demand valve
10. Samodelov LF, Falke KJ. Total inspiratory work with modern demand valve devices compared to continuous flow CPAP. Intensive Care Med 1988;14:632-639.
27. Fiastro JF, Habib MP, Quan ST. Pressure support compensates for inspiratory work due to endotracheal tubes and demand continuous positive airway pressure. Chest 1988;93:499-505.
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A 20-year-old woman was seen in the outpatient clinic with the complaint of "worsening asthma and allergies." Four years earlier she had first developed symptoms characterized by chest tightness and wheezing while participating in sports. Within the past 2 years she had experienced more frequent symptoms, worse from September to May. During the past winter she visited the emergency room twice for wheezing and shortness of breath, and subsequently was given short courses of oral steroids. Recent history also revealed that the patient was having frequent early morning cough or was awakening at night with symptoms. She was self-administering two dogs and two cats. Pulmonary function testing was performed before (medication withdrawn for 48 hours) and after bronchodilator (Table I and Fig. 1).

Table 1. Pulmonary Function Data Pre- (Baseline) and Post-Bronchodilator Treatment*

<table>
<thead>
<tr>
<th></th>
<th>Predicted</th>
<th>Baseline</th>
<th>% Predicted</th>
<th>Post Bronchodilator</th>
<th>% Change</th>
</tr>
</thead>
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<tr>
<td><strong>Lung Volume</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TLC (L)</td>
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<td>5.73</td>
<td>106</td>
<td>5.71</td>
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<tr>
<td>TGV (L)</td>
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<td>2.94</td>
<td>101</td>
<td>2.92</td>
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<tr>
<td>RV (L)</td>
<td>1.56</td>
<td>1.70</td>
<td>109</td>
<td>1.71</td>
<td>1</td>
</tr>
<tr>
<td><strong>Forced Expiratory Volume</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FVC (L)</td>
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<td>4.57</td>
<td>111</td>
<td>4.59</td>
<td>0</td>
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<tr>
<td>FEV₁ (L)</td>
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<td>3.73</td>
<td>100</td>
<td>3.83</td>
<td>3</td>
</tr>
<tr>
<td>FEV₁/FVC (%)</td>
<td>90</td>
<td>81</td>
<td>90</td>
<td>83</td>
<td>2</td>
</tr>
<tr>
<td><strong>Additional</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SGaw (cm H₂O⁻¹ · s⁻¹)</td>
<td>0.24</td>
<td>0.27</td>
<td>111</td>
<td>0.26</td>
<td>-4</td>
</tr>
<tr>
<td>[kPa⁻¹ · s⁻¹]</td>
<td>2.4</td>
<td>2.7</td>
<td>111</td>
<td>2.6</td>
<td>-4</td>
</tr>
<tr>
<td>D_LCO (mL · min⁻¹ · torr⁻¹)</td>
<td>32.53</td>
<td>40.70</td>
<td>125</td>
<td>244.0</td>
<td>305.3</td>
</tr>
</tbody>
</table>

*Bronchodilator used was albuterol.

Questions

1. What is your interpretation of the baseline PFT results?
2. In your opinion does this patient have asthma?
3. What follow-up testing would you recommend?

Answers and Discussion on Next Page
Answers

1. Interpretation of Baseline PFTs: The baseline PFT results (Table 1 and Fig. 1) reveal normal pulmonary function.

2. Diagnosis: Though the patient’s history seems to strongly suggest asthma, her normal pulmonary function and lack of reversibility with an inhaled bronchodilator does not support this diagnosis.

3. Additional Testing: Aside from reversibility and variability, asthma is also characterized by variable airflow limitation and hyperresponsiveness to external ‘triggers.’ Of note, this patient arrived for her testing after having been on vacation and had not yet returned to her home, which was laden with allergic triggers. This may have accounted for her lack of airflow limitation when tested. Therefore, to determine the presence or absence of bronchial hyperreactivity, a methacholine provocation test was ordered. The indication for this challenge was a known history and the need to confirm and evaluate the severity of her asthma.

Baseline spirometric measurements were performed; then placebo (saline) and increasing concentrations of methacholine were administered, with forced expiratory maneuvers after each dose. The results of the challenge are shown in Figure 2. The PC20 (provocative concentration that causes a 20% decrease in FEV1) was between 0.15 and 0.31 mg/mL (at 0.25 mg/mL), indicating moderate to severe airways hyperresponsiveness consistent with a diagnosis of asthma. Normal subjects will not respond to concentrations of methacholine until exposed to 8 mg/mL or more.

Discussion

Asthma is a syndrome that, unlike other lung disorders, is diagnosed based on physiologic abnormalities; in fact, asthma is the only lung disorder for which pulmonary function testing alone is diagnostic. As such, asthma is characterized by (1) reversibility of airflow limitation by bronchodilator therapy, (2) variability of airflow limitation, and (3) hyperresponsiveness of the airways to provocation. It is this latter feature that, as we will see, may be the most important. Variability of airflow rates, and reversibility to acute administrations of bronchodilator is often not evident during a single set of tests. In this case, medical history was crucial in the eventual correct diagnosis of this case. Despite normal initial pulmonary function, the 4-year history of wheezing and the added information of likely allergic triggers suggested that this patient might indeed have asthma.

It has been repeatedly demonstrated that pulmonary function, peak expiratory flow rate (PEFR) in particular, varies widely in asthmatics. Studies have further shown that the amplitude of variation in PEFR correlates with severity of disease. The patient in this PFT corner had been tested at 1530 (3:30 PM), which is when pulmonary function might be expected to be at its best. Because readings during the daytime may be completely normal, deteriorated function may not be observed during normal working hours or at the time of visit to the doctor’s office.

Our patient was sent home with a peak flowmeter to keep a diary of peak flow rates measured four times a day (Fig. 3). She showed consistent and important reductions in peak flow in the morning. This phenomenon is referred to as ‘morning dipping.’ On the basis of PFTs alone, the professional staff may be misled into believing that a patient’s symptoms have no real organic basis.

In the current case, our patient was quite sensitive to inhaled methacholine. Bronchial hyperresponsiveness is a cardinal feature of asthma, and often cannot be demonstrated by simple spirometry. Further, unlike COPD, in asthma the degree of airflow limitation does not correlate to the degree of hyperresponsiveness. Therefore, measurement of baseline pulmonary function is crucial.
function is inadequate for diagnosing asthma. Hence, airways challenge with some sort of external trigger is employed. Histamine, methacholine, or specific antigens (eg, ragweed) may be used as provoking agents; however, methacholine is most often used. The degree of sensitivity to methacholine does correlate to the severity of asthma and need for treatment, and is a reliable prognostic indicator. Patients found to be most sensitive to methacholine are much more likely to require more aggressive therapy and frequent follow-up. The PC_{20} is therefore a reasonable index of the presence of 'twitchy' airways and can be used to assess asthma severity. On the basis of this finding and the nocturnal symptoms exhibited, we judged that our patient had quite severe asthma and was in need of more aggressive management. A regular regimen of inhaled bronchodilator and steroid therapy was prescribed in place of 'as needed,' or prn, therapy; the patient went through a patient education program to learn more about asthma management; and the patient moved out of her home into an environment free of cat and dog dander.

In conclusion, while it is important to recognize that asthma is often characterized by variability and reversibility in air flowrates, the most important physiologic feature of the asthmatic patient may be bronchial hyperresponsiveness. Even when initial PFT values are normal and reversibility is absent, if a patient's history strongly suggests asthma, the measurement of circadian variations in pulmonary function and/or bronchoprovocation testing (such as methacholine challenge testing) may reveal the presence of asthma, and as such is paramount to correctly diagnose asthma.

REFERENCES

1. Irvin CG. Airways challenge. Respir Care 1989;34:455-469.
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Blood Gas Corner #28—
Oxygenation of the Carbon Monoxide Poisoned Patient

John M Graybeal CRTT, Garfield B Russell MD, and John Basile RRT

A 52-year-old man, with a history of ischemic heart disease, arrived in the emergency department comatose after having been found unconscious in his parked car, with its motor running. An endotracheal tube had been placed by the paramedics, and assisted ventilation with 100% oxygen had been initiated. Vital signs on ER arrival, an estimated 30 minutes after removing the patient from the car, were sinus tachycardia 132 beats/min, respiratory rate 15 breaths/min with assisted ventilation, and blood pressure 126/67. Oxygen saturation ($\text{SpO}_2$) measured by pulse oximeter was 100%. Arterial blood analysis revealed pH 7.14, $P_{\text{CO}_2}$ 24 torr [3.2 kPa], $P_{\text{O}_2}$ 511 torr [70.0 kPa], and $HCO_3^-$ 7.9 mEq/L [7.9 mmol/L]. The carboxyhemoglobin (COHb) level was 41.9%. Approximately 4 hours after exposure to the carbon monoxide the patient remained unconscious, apneic, and unresponsive to verbal commands but responsive to painful stimuli. During this time, the patient was mechanically ventilated with 100% oxygen.

Study Questions

1. How do you interpret the results of pulse oximetry in this patient?
2. How do you interpret the results of the arterial blood gas and pH analysis?
3. Does the clinical severity of the carbon monoxide poisoning and prognosis correlate with the COHb level?
4. What further therapeutic action is indicated?
5. What are the difficulties and techniques associated with the sampling and analysis of arterial blood of patients in a hyperbaric chamber?

Answers and Discussion on Next Page
mental pollution on a prolonged low-level basis.

2. Arterial Blood Gas and pH Interpretation. Arterial blood gas values from patients poisoned by CO must be carefully evaluated. Specifically, (1) the calculated $S_AO_2$ will be inaccurate, (2) the $P_AO_2$ will not reflect adequacy of oxygen for cellular respiration, and (3) a metabolic acidosis may be present.

An uncoupling of the relationship between the partial pressure of oxygen dissolved in the plasma and the oxygen bound to hemoglobin results in the inaccuracies seen in the $S_AO_2$ calculated from blood gas values. In cases of suspected CO poisoning, the $S_AO_2$ should be measured using a multiple-wavelength spectrophotometer, such as the Instrumentation Laboratories IL-282 (Lexington MA). Usually 4 or 5 wavelengths are necessary to distinguish between COHb and HbO2, which have similar absorption coefficients at the wavelengths (660 and 940 nm) used in most two-wavelength devices.

Despite apparently normal or elevated partial pressures of oxygen in the arterial sample, intracellular hypoxia may exist. Carbon monoxide’s affinity for hemoglobin is 240 times greater than is oxygen’s, causing displacement of oxygen from the hemoglobin molecule. Also the oxyhemoglobin dissociation curve is shifted to the left, decreasing the $P_{so}$. The decreased oxygen-carrying capacity and the diminished oxygen release to the tissues cause the supply of oxygen to be inadequate for intracellular respiration.

Carbon monoxide also exhibits toxicity by direct interference with intracellular respiration. Myoglobin, reduced cytochrome c-oxidase, cytochrome a3, reduced cytochromes of the P450 type, and tryptophan dioxygenase all bind with carbon monoxide sufficiently to interfere with function. Inadequate cellular respiration results in metabolic acidosis, which is considered a poor prognostic sign and has been reported to occur in up to 25% of cases of CO poisoning. Aside from other metabolic abnormalities, such as extracellular shifting of potassium, it is believed that there is a positive correlation between metabolic acidosis severity and poor clinical outcome. This patient initially had severe metabolic acidosis (with a $HCO_3^{-}$ of 7.9 mEq/L [7.9 mmol/L] and pH of 7.14).

3. Correlation between COHb Level and Clinical Status and Prognosis. The severity of CO poisoning, clinical status, and prognosis are not consistently reflected by the COHb level. This is because COHb levels do not reflect tissue levels of CO. The duration of exposure, along with the COHb level at the time of the exposure, may better correlate with the clinical status and prognosis. If a significant amount of time elapses between exposure and blood sample analysis, washout of CO, which has a circulating half-life (COHb t½) of 5.3 hours, can begin to occur.

A high level of dissolved CO is required for cellular respiration to be blocked; at times a high COHb level may be associated with a small amount of dissolved CO in plasma and thus low toxicity. The difference between COHb levels in survivors and nonsurvivors is not significant. However, some fairly clear relationships have been established. Cerebral edema, seizures, and coma are frequently related to COHb levels above 50%. Survival is unusual when COHb levels are above 70%. Myocardial tolerance to COHb is significantly lowered in patient’s with pre-existing cardiac disease. In primate experiments, COHb levels as low as 9.3% resulted in significant lowering of the ventricular fibrillation threshold. A quick test to assess intracellular dysfunction is not available, but metabolic acidosis in the presence of normal or elevated $P_AO_2$ is suggestive of severe CO poisoning.

4. Further Therapeutic Action. Immediate administration of 100% oxygen is always indicated in a case...
of CO poisoning. In the past, other therapies have been tried, including hypothermia and exchange blood transfusions, but these therapies are no longer utilized.

Hyperbaric oxygen (HBO) therapy is indicated for CO-poisoned patients who are unconscious, acidotic, or pregnant, or who have neurologic or cardiac symptoms. COHb above 25% is considered, by some, to be an indication for HBO.

Two separate, potentially beneficial aspects of HBO therapy are elevated environmental pressure and increased partial pressure of oxygen. The combination of these two entities allows administration of oxygen ($F_{O_2} = 1.00$) at pressures greater than normal atmospheric pressure (760 torr [101 kPa]). Inspired $P_{O_2} > 2,000$ torr [266 kPa] are easily attained (Fig. 1).

![Fig. 1. The partial pressure exerted by oxygen increases in direct proportion to the increase in atmospheric pressure for different concentrations of delivered oxygen. Compressed air at 6 atmospheres (ATA), with an oxygen concentration of 0.21, can exert a partial pressure of 958 torr of oxygen, which is greater than the partial pressure of pure oxygen at normobaric conditions. (Refer to box for SI conversions.)](image)

HBO was originally considered beneficial to CO-poisoned patients because it markedly shortens the COHb $t_{1/2}$. During normobaric air breathing the COHb $t_{1/2}$ is approximately 5.3 hours. During normobaric 100% oxygen breathing, the COHb $t_{1/2}$ is reduced to 1.3 hours, and with HBO treatment at 3 atmospheres (ATA) (1 ATA = 760 torr) it is further reduced to 0.38 hours. True benefits of HBO are believed to also include relief of tissue hypoxia by saturation of the plasma with dissolved oxygen (Fig. 2) despite high COHb levels, mass action removal of the CO by the increased partial pressure of circulating oxygen, and improved intracellular respiration with shortened time of CO attachment to cytochromes. Lipid peroxidation and free radical formation may also be reduced. In the case we report, the elevated dissolved oxygen concentration of the plasma may provide extra protection for the patient's myocardium, which is at special risk in light of his history of ischemic heart disease. Associated cerebral edema and increased intracranial pressure may also be reduced.

Central-nervous-system disorders associated with neurologic and neuropsychiatric deterioration can occur in up to 11% of victims as sequelae to CO poisoning. This may range from diffuse demyelination to mood changes and increased irritability. Because HBO helps prevent this significant secondary deterioration, many argue that all CO-poisoned patients should be treated with HBO.

This patient was transferred to a regional center with hyperbaric chamber facilities. The patient was noted to be as described but with electrocardiographic evidence of left axis deviation and ST-segment depression. Hyperbaric therapy at 2.8 ATA was initiated. During therapeutic compression, short 5-min intervals of air breathing were interspersed with 100% oxygen therapy. During the initial air break, arterial blood analysis revealed pH 7.40, $P_{ACO_2} = 37$ torr, $P_{O_2} = 128$ torr, $HCO_3^- = 22.6$ mEq/L [22.6 mmol/L]. The initial treatment session lasted for 3 hours. ECG abnormalities resolved. Upon return to the ICU (approximately 9 hours after exposure to CO) arterial blood analysis revealed pH 7.37, $P_{ACO_2} = 41$ torr [5.5 kPa], $P_{O_2} = 255$ torr [34.0 kPa], and COHb 0% during mechanical ventilation with 100% oxygen. The patient's mental status was somewhat improved, as exhibited by frequent openings of the eyes and spontaneous respiratory efforts. A 3-hour treatment was repeated 18 hours later. The patient's condition improved gradually. He was extubated 72 hours after admission. Neurologically, he was noted to have periods of confusion and memory loss, which continued to become less frequent and of shorter duration up to discharge.

5. Arterial Blood Analysis during HBO Therapy. There are several techniques used to evaluate blood gas
values in the hyperbaric setting. Within a multipurpose chamber there is often room to set up and maintain a dedicated blood gas analyzer. Only modified blood gas analyzers are capable of working within the hyperbaric environment—all analyzer parts that may contain trapped gas must be vented to minimize the risk of implosion during chamber compression. Under hyperbaric conditions some analyzers are unable to electronically display correct $P_{A\text{O}_2}$ values, which may be in excess of 1,000 torr (133 kPa), due to an inadequate number of digits in the display window.

Blood gas analyzers used under HBO conditions must be calibrated appropriately. Supranormal calibration standards and control samples for validation of instrument accuracy are required. This is also true for a measuring system operated under normobaric conditions but used to analyze blood samples obtained in the hyperbaric chamber.

Blood gases obtained in the hyperbaric environment can be measured at normobaric conditions, but not without creating specific problems. If the sample is not measured rapidly, frothing of the sample can occur. The dissolved gases come out of solution, forming bubbles. Studies have shown that if the sample is rapidly decompressed and analyzed within 3 minutes, the resulting $P_{A\text{O}_2}$ value is within the same % error as that of a sample measured under normobaric conditions. Arterial blood gas analysis performed 3 minutes or more after obtaining the sample have variable results. Careful handling of the sample is important, because shaking or jarring may speed the formation of bubbles within the sample. Blood gas samples obtained from this patient during compression were analyzed outside the chamber.

The $P_{A\text{O}_2}$ can be predicted. The arterial-alveolar oxygen tension ratio ($P_{A\text{O}_2}/P_{A\text{O}_2}$) has been shown to be stable for the same patient, despite the presence or absence of abnormal gas exchange, through the range of atmospheric pressures from 1 to 3.1 ATA. If pre-HBO arterial blood gas values are known, the $P_{A\text{O}_2}/P_{A\text{O}_2}$ can be easily calculated. The predicted $P_{A\text{O}_2}$ during compression with 100% oxygen can be calculated as follows:

$$\text{Predicted } P_{A\text{O}_2} = \left( \frac{P_{A\text{O}_2}}{P_{A\text{O}_2}} \right) \left( \frac{[760 \text{ Pata} - 47]}{P_{A\text{CO}_2}} \right).$$

The Pata is the ambient pressure in ATA, and $P_{A\text{O}_2}$ and $P_{A\text{CO}_2}$ are measured in torr. While this may be very helpful for estimating the $P_{A\text{O}_2}$ expected under hyperbaric conditions, it does not provide any information about the $P_{A\text{CO}_2}$. Therefore, whenever ventilatory adequacy is uncertain, direct analysis of the arterial blood must be done.

Transcutaneous $P_{O_2}$ can also be measured in the HBO environment. These monitors must also be altered, by adding a fourth digit to the display screen, to enable the display of supranormal $P_{O_2}$ during HBO therapy. Supranormal calibration gases must also be provided for validation of the instrument's accuracy.

**Discussion**

Carbon monoxide poisoning is the most commonly reported poisoning in the United States each year. Carbon monoxide poisoning results in an increase in the affinity of hemoglobin for oxygen and a consequent blockage of oxygen transport to the tissues. It shifts the oxyhemoglobin dissociation curve to the left, facilitating pulmonary oxygen loading but impairing oxygen release at the tissue level. Intracellular respiration is disrupted. In acute poisoning, myocardial hemorrhage and necrosis can result. Initial cerebral edema may develop into diffuse demyelination. HBO therapy offers a method of reducing the circulating half-life of COHb, while simultaneously increasing the dissolved oxygen concentration in plasma and shortening the attachment time of CO to intracellular enzyme systems.

HBO therapy may decrease morbidity, particularly the secondary neurologic deterioration that occurs 7-14 days after poisoning, and the immediate cardiac ischemia. However, HBO therapy has not been studied in a controlled manner, so there is some variation in recommendations.^20,21^ Treatment in a monoplace hyperbaric chamber usually consists of 2.5 ATA (100% oxygen) exposure for 90 minutes. For residual or recurring symptoms, treatment may be repeated in 6 to 12 hours. In severe cases, treatment two or three times a day for several days may be considered. In a multipurpose chamber, compression to 3 ATA is usually recommended, with 23-to-25-min periods of 100% oxygen breathing interspersed with 5-min periods of air breathing to decrease the risk of neurologic oxygen toxicity and convulsions. After 1 hour, pressure is decreased to 2 ATA and oxygen breathing continued at 45-minute intervals until there is improvement or clinical judgment indicates that no improvement is occurring.

Patients can be monitored during HBO therapy. Chambers can be equipped to have full hemodynamic monitoring, with passage of hemodynamic pressure tubing through chamber-wall conduits and patient ventilators mounted inside the chamber. Arterial blood gas analysis poses specific problems, as discussed, but can be accurately performed if appropriate equipment modifications are made and calibration and sampling routines are followed.

**REFERENCES**

3. Barker SJ. Tremper KK. The effect of carbon monoxide inhalation on
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Radiographic Findings
following Pediatric Blunt Trauma

Carol R Schermer BA and Frederick W Clevenger MD

A 5.5-month-old girl was brought to the trauma center after a motor vehicle accident. She and her grandmother had been restrained with the same shoulder harness and lap belt and both were reportedly thrown forward at the time of the head-on collision. Neither the grandmother nor the driver of the car sustained clinically important injuries, despite the fact that the impact of the collision had caused a 2-foot indentation in the front bumper of the automobile. The grandmother and driver were fully ambulatory when the paramedics arrived at the scene of the accident and were not transported to a hospital for evaluation.

At the scene of the accident, the child was moving all extremities; however, her skin was cool and she was somewhat cyanotic. Her blood pressure was 85/60, heart rate 150-220, and respiratory rate 35 (normal for her age). She 'pinked up' quickly after 40% oxygen by face mask was initiated. The lungs were clear with good breath sounds heard bilaterally. The abdomen was soft and did not appear to be tender to palpation. Because of the mechanism of injury and the uncertainty on the part of the paramedics regarding the status of the child, she was transported (immobilized on a long spine board) to the trauma center.

Upon arrival at the trauma center, an arterial blood gas sample was obtained, and analysis revealed pH 7.37, P_{aCO_2} 35 torr [4.7 kPa], P_{aO_2} 87 torr [12 kPa], HCO_3 20.2 mEq/L [20.2 mmol/L], and {aO_2} 96.7%. The patient's cardiac exam was unremarkable except for tachycardia. Because of the mechanism of injury, the child underwent radiographic evaluation. The cervical-spine films were normal. The chest radiograph is shown in Figure 1.

Questions

Radiographic Finding: What radiographic abnormality is present and what pathologic processes could explain this finding?

Diagnostic Confirmation: What further action is required to confirm the diagnosis?

Corrective Action: What corrective action is indicated?

Answers and Discussion on Next Page
Answers

Radiographic Finding: The chest radiograph in Figure 1 demonstrates a density in the lower right hemithorax and a contralateral mediastinal shift. The differential diagnosis of this finding includes right lower lobe atelectasis or consolidation; fluid in the base of the right lung; a mass lesion within the diaphragm, pleura, or lower lung field; right phrenic nerve palsy resulting in diaphragmatic elevation; a pericardial cyst; a subphrenic abscess; or herniation of the liver through a tear in the diaphragm. The curved and distinct nature of the density strongly suggested herniation of the liver. Because the child’s past medical history was unremarkable, acute blunt abdominal trauma was suspected to be the most likely etiology of the elevated hemidiaphragm.

Diagnostic Confirmation: A thoracic CT scan confirmed the suspected pathology. The child was taken to the operating room where the defect shown in Figure 2 (an acute posterolateral tear in the right hemidiaphragm with herniation of the right lobe of the liver into the chest) was identified through an abdominal incision.

Corrective Action: Primary repair was achieved using running monofilament suture. Recovery was uneventful, with extubation 12 hours postoperatively, and no signs of respiratory distress. The patient was fed by mouth on the second postoperative day and discharged 3 days later. A normal chest radiograph taken just prior to discharge is shown in Figure 3.

Fig. 3. Anteroposterior (AP) chest radiograph of a 5.5-month-old girl injured in a motor vehicle accident, obtained just prior to hospital discharge (subsequent to surgical repair of a traumatic diaphragmatic hernia). Note the normal position of the right hemidiaphragm.

Discussion

Traumatic diaphragmatic hernia (TDH) is most frequently caused by motor vehicle accidents. Although diaphragmatic rupture most commonly occurs on the left side, right-sided TDH has been described in an average of 5% of reported cases. Diaphragmatic rupture is seen in 3-6% of patients surviving the initial traumatic event. With right-sided TDH, the liver most commonly herniates, whereas with left-sided TDH the stomach most commonly herniates.

Traumatic hernias may occur anywhere. The posterolateral region of the left hemidiaphragm is frequently ruptured because of presumed embryologic weakness. Other factors thought to contribute to blunt rupture of the diaphragm are compressing forces, which lead to increased abdominothoracic pressure gradients, and shearing forces, which can occur during chest compression.

Patient presentation ranges from tachypnea (with grunting respiration) and hypoxemia, to shoulder or
abdominal pain associated with vomiting, hypotension, and shock. Although the majority of patients present with symptoms, a high index of suspicion is necessary to diagnose TDH because of the non-specificity of the symptoms. On occasion, asymptomatic patients are diagnosed with TDH solely on the basis of an abnormal chest radiograph.

Isolated diaphragmatic injuries are rare. Thus, the physical findings of TDH are generally those of associated injuries. Mortality (ranging from 0 to 50%) is proportional to the number and severity of the associated injuries. Right-sided TDH is associated more frequently with major-organ-system injuries that result in death than is left-sided TDH. Presumably, the force required to cause rupture on the right side is greater than that required on the left side, and thus injuries associated with right-sided TDH are worse than those associated with left-sided TDH.

On occasion, the diagnosis of TDH is delayed and patients may present months to years after the initial appearance of the defect. This phenomenon is seen most frequently in children and in cases of right-sided TDH. The herniation worsens with time due to the pressure differential between the chest and abdominal cavities. Positive pressure ventilation, frequently employed in the management of the multiple-injury patient, arrests herniation; however, when it is discontinued—if TDH is present—the abdominal organs may migrate into the chest. Thus, appreciation of the presence and danger of TDH may be delayed until a patient is weaned from mechanical ventilation. The sequelae of delayed diagnosis range from failure to gain weight and grow (in children), to shortness of breath and fatigue, to abdominal or chest pain resulting from obstructed abdominal contents. Visceral herniation through the defect may also present as an emergency situation with the clinical features of severe respiratory distress, peritonitis, cardiac tamponade, cardiac arrest, or strangulation and necrosis of abdominal organs.

Diagnosis of TDH starts with a high index of suspicion. Pericostal injury, fracture of the pelvis or lumbar spine (reflecting major torso compression), dyspnea, lower chest or upper abdominal pain (particularly if referred to the shoulder), and dullness or tympany heard upon percussion of the lower chest are clinical constellations that arouse suspicion. The pathognomonic physical sign is auscultation of bowel sounds in the chest. Although the chest radiograph may be normal, an abnormal but nonspecific chest radiograph is seen in the majority of TDH cases and provides the first clue that herniation is present. Radiographic findings suggestive of TDH include elevated diaphragm, pleural effusion, pneumothorax, mass lesion above the diaphragm, intestinal shadows above the diaphragm, plate-like atelectasis above an indistinct diaphragm, and mediastinal shift.

If the chest radiograph arouses suspicion of TDH, subsequent diagnostic investigation may be indicated and may include nasogastric tube placement with subsequent identification of TDH on follow-up plain films, barium contrast studies of the upper and lower gastrointestinal tract, computed tomography, ultrasound, liver-spleen scans, fluoroscopy of the diaphragm, thoracoscopy, and induced pneumoperitoneum. Barium studies are more likely to be useful if left-sided rather than right-sided herniation is suspected. The definitive means to diagnose TDH is thoracoscopy—it is 100% accurate and, if positive, repair of the defect is facilitated because the patient is already in the operating room. Although some authors advocate the use of induced pneumoperitoneum for diagnosis of TDH, a negative result does not rule out the possibility of a tear since the omentum or viscera may plug the defect.

Laparotomy is indicated in acute cases because of the high incidence of associated injuries that require operative intervention. However, if the diagnosis is delayed adhesions usually form between the abdominal viscera and the lung or pericardium, and, as a result, thoracotomy becomes necessary to repair the defect.

Because of the sequelae associated with nonfatal diaphragmatic hernia, it is critical to look for occult TDH in the absence of signs and symptoms—particularly if the chest radiograph arouses suspicion. Diagnosis of TDH is often suggested by a chest radiograph that reveals an elevated hemidiaphragm or loops of bowel in the thoracic cavity. Rapid surgical repair of TDH will in all cases decrease the morbidity associated with delayed repair and in some cases be life-saving.

REFERENCES

How You Can Help Patients Stop Smoking

The National Heart, Lung, and Blood Institute has made available "How You Can Help Patients Stop Smoking: Opportunities for Respiratory Care Practitioners." This guide was developed in collaboration with the AARC and provides guidance on talking to patients about smoking. Plus, it tells you how to integrate a smoking intervention program into a respiratory care department. Includes strategies for community outreach and information on smoking intervention techniques and tools.

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Hyperbaric Medicine Procedures, by Eric P Kindwall MD and Robert W Goldmann MD. Softcover, 158 pages. Available by mail order (no telephone orders accepted) from St Luke’s Medical Center, Hyperbaric Department, 2900 W Oklahoma Ave, Milwaukee WI 53215. 1988. $32.00.

Hyperbaric Medicine Procedures is, in this sixth edition, bound in softcover—a format that should prove more durable than has the loose-leaf format used for previous editions. The authors’ preface clearly points out that this book, with its limited references, is intended as a guide only. During our review of this manual we have attempted to keep this in mind.

The most useful and detailed section (94 of 158 pages) is devoted to the disease categories for which hyperbaric oxygen therapy (HBO) has become an accepted treatment. A brief summary of each condition, the expected response to therapy, and a brief reference list for each condition is included. Several practical recommendations (such as appropriate scheduling and sequencing of therapy) are also included. For example, in the section on HBO treatment of skin grafts, the authors recommend that surgery for these patients should be scheduled on Monday or Tuesday, so that HBO therapy, which is recommended for the first 3 postoperative days, can be completed during the normal work week.

A brief review of procedures applicable to HBO therapy (such as obtaining vascular access, myringotomy, thoracotomy tube insertion, mechanical ventilation, and methods of drug administration as used by the authors) and the associated complications is also included and could prove quite useful, particularly for inexperienced personnel. This section is useful, but is lacking in some minor but important details. For example, the inability of mechanical ventilators to deliver preset tidal volumes during HBO is briefly mentioned. The suggestion is made that a “calibrated Wright Respirometer” be used to check the patient’s exhaled tidal volume during compression. This is clearly a sound clinical recommendation, but nowhere do the authors describe the method for recalibration of the Wright Respirometer for use during compression, when gas density is increased.

Drs Kindwall and Goldmann have succeeded in writing this manual in a manner that is both easy to read and to understand; however, some components would benefit from careful editing and review. Some tables and charts (for example, Minimal Recompression, Oxygen Breathing Method for Treatment of Decompression Sickness, on Page 51, and Necessary Perfusion Pressure for Healing Wounds of the Foot, Page 120) are difficult to interpret because headings are not aligned with columns of information. In general, this does not detract from the value of the manual but does at times interrupt the flow of the text. Material on several topics is unnecessarily splintered into different sections. Cross-referencing of the text would guide readers to specific information they might be looking for. For example, information on oxygen toxicity is found in three different areas of the text. Hunting for these sections greatly increases the time required to cover the relevant material. In the Table of Contents, sections are clearly numbered, but the corresponding divisions in the text are not. Numbered sections with subdivisions indicated by letter would make information retrieval more efficient. The addition of descriptive headings and captions to tables and illustrations would be helpful.

Some sections need improvement. The section on anesthesia is badly dated. The whole concept of sedation of patients is not adequately addressed. Many potentially useful drugs such as midazolam, propofol, and short-acting narcotics (such as alfentanil, sufentanil, and fentanyl) are not mentioned.

To get the maximum benefit from this manual, the reader needs some background knowledge in HBO. Basic principles of HBO are not covered with sufficient depth for the newcomer, which is consistent with the authors’ clear statement that they did not intend to write a complete textbook. Hyperbaric Medicine Procedures does have its limitations (not the least of which is its $32.00 price), but it is a manual that may be helpful as a guide for personnel at hyperbaric treatment facilities, especially when they are presented with a patient whose condition they do not treat regularly. It will also provide a simple introduction and useful guide to the clinical utilization of HBO, for respiratory therapists, nurses, and physicians.

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In respiratory care, as in medicine overall, the efficacy of treatment is determined by measuring responses to therapy and resulting outcomes. While measuring in respiratory care focuses on many ‘hard’ outcomes, such as flowrates, tidal volumes, and pressures,
the respiratory care investigator is also frequently challenged to measure 'soft' outcomes, like dyspnea, quality of life, and subjective response to bronchodilation. These soft outcomes are often the most clinically important and compelling outcomes in a study, but because soft outcomes are widely perceived to be difficult to measure reliably, they are often ignored.

The book Health Measurement Scales: A Practical Guide to their Development and Use makes an important contribution by recognizing that soft clinical outcomes are important. The book provides the respiratory care investigator with a practical method for evaluating available measurement scales and guidelines for creating and validating new instruments when published precedents are not available.

The stated goals of the book are "to introduce researchers in health sciences to the concepts of measuring outcomes" that are not usually studied, examples of which are "arthritic pain, return to function in post-MI patients, speech difficulties of aphasic stroke patients, or clinical competence of junior medical students." The focus is on outcomes of interest to health care researchers (eg, "subjective states, attitudes, response to illness") rather than on soft outcomes more traditionally considered by psychologists (eg, personality types).

The respiratory care reader to whom this book will appeal is interested in (1) evaluating an available health scale to measure a soft respiratory outcome, like dyspnea, or (2) creating a respiratory health measurement scale when one does not already exist.

The book has 13 chapters; following an introductory chapter that contains references, Chapter 2 summarizes techniques for researching the literature to find available scales and criteria for critically reviewing these instruments.

The next five chapters review techniques for developing health measurement scales or troubleshooting the instruments: Devising the Items (Chapter 3), Scaling Responses (Chapter 4), Selecting the Items (5), Biases in Responding (Chapter 6), and From Items to Scales (Chapter 7). Remaining chapters discuss methods to validate health measurement scales: Reliability (Chapter 8), Generalizability Theory (Chapter 9), Validity (Chapter 10), Measuring Change (Chapter 11), Latent-Trait Theory (Chapter 12), and Methods of Administering the Scale (Chapter 13). Two appendices detail additional readings and provide a list of sources containing a broad array of available health measurement scales, some of which will interest the respiratory care investigator.

Chapters describing underlying concepts (eg, Devising and Selecting Items, Biases) are very readable, whereas excursions into theory (eg, chapters on Generalizability and Latent-Trait Theory) are expectedly more formidable and dense to read. These chapters are formidable despite the avowed goal of avoiding complex mathematics for the sake of readability. Tables and figures are used amply throughout the text.

The book will be useful to the respiratory care investigator, because the topic is not traditionally included in a respiratory care curriculum or in the training of most respiratory care investigators. Those readers looking for a primer of health measurements scales will find the practice-based chapters (eg, Devising the Items, Selecting the Items. Searching the Literature) most helpful and can refer selectively to the more theory-based chapters. A useful reference function is also served by the appendices. This is a unique book that will appeal to respiratory care investigators seeking guidance in measuring relevant but often-ignored clinical outcomes.

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January 26 in Ruidoso, New Mexico. The NMSRC presents the 2nd Annual Winterfest '91 at the Swiss Chalet Inn. Topics focus on current trends in cardiopulmonary care. Contact Schuyler Michael RRT at (505) 841-1580 or the NMSRC, PO Box 35417, Station D, Albuquerque NM 87176-5417.

January 30-31 in Salt Lake City, Utah. The USRC presents its Annual Meeting at the Airport Hilton Hotel and Conference Center. Theme is "Where Is Technology Taking Us?" Topics include BiPAP, HBO, neonatal ECMO, IVOX, computerized management, pulse oximetry, and medical ethics. Special features include a license update and a lecture by Allan Morris MD concerning the study of computerized protocols for treatment of hypoxemia in ARDS, "ECCO, R vs Traditional Therapy." Social events include the annual Sputum Bowl competition and the 2nd Annual "RespOlympics." Join us for a little winter skiing and a great Annual Meeting. Contact Valerie Thomas RRT, Respiratory Therapy Education, Weber State College, Ogden UT 84408-3904. (801) 626-6835.

February 4-6 in Breckenridge, Colorado. The CSRC presents its first annual Neonatal/Pediatric Review Seminar at Breckenridge Resort. The topics cover the examination matrix for the Perinatal/Pediatric Specialty Examination in March 1991 and provide insights into current perinatal/pediatric modalities. The schedule allows time to enjoy some of Colorado's finest skiing. Contact Dan Van Hise RRT, University Hospital, Respiratory Care Dept, Box C-271, 4200 East 9th Ave, Denver CO 80262. (303) 270-8701.


April 3-5 in Baton Rouge, Louisiana. The LSRC presents the 21st Annual Educational Meeting and Exhibition, "Rx for Preservation of the Respiratory Care Profession," at Embassy Suites on 1-10. Contact Jim Lanoha at (504) 381-6542.

April 10-12 in Bismarck, North Dakota. A New Decade of Strength in Respiratory Care is the theme for NDSRC's annual convention. Topics include nutrition studies, adult and neonatal critical care issues, and management strategies. For more information, call (701) 224-7870.

May 1-3 in Rapid City, South Dakota. Rapid City Regional Hospital hosts the annual convention of the SDSRC. Featured speakers including WJ O'Donohue Jr MD, Robert Kacmarek PhD RRT, and Anthony Talbert MD discuss neonatal, pediatric, and adult critical care issues. Contact Terry Anderson RRT, Respiratory Care Department, 1-800-232-9287.

May 28-31 in Jekyll Island, Georgia. The Georgia/South Carolina Region VI presents its 15th Annual Conference and Assembly at the Holiday Inn, Jekyll Island. Contact Mike Payne RRT, 730 South Pleasantsburg Dr, Suite 525, Greenville SC 29607. (803) 879-0130.

OTHER MEETINGS

January 15-16 in Tampa, Florida. This Joint Commission for the Accreditation of Health Care Organizations seminar is developed specifically for the Home Medical Equipment (HME) supplier and provides the only authoritative coverage of Joint Commission standards for HME and clinical respiratory services. The plenary and workshop sessions are taught by Joint Commission HME surveyors who are Registered Respiratory Therapists and experts in providing home medical equipment services within independent organizations. The seminar is also scheduled for February 21-22 in New York, New York; August 15-16 in San Francisco, California; and November 12-13 in Atlanta, Georgia. For a brochure, contact Customer Service Center of the Joint Commission at (708) 916-5800.

February 2-3 in Shreveport, Louisiana. Respiratory Review Workshops presents an Entry Level Certification Examination review to prepare candidates for the March exam. Instructor is Gary Persing BS RRT, Director of Clinical Education, Respiratory Therapy Program, Tulsa Junior College, Tulsa, Oklahoma; Mr Persing takes the exam each time it is given. The 2-day workshop provides 16 CRCE credits. Other workshops scheduled in February include: Tulsa OK—February 9-10; San Jose CA—February 16-17; and San Antonio TX—February 22-24. (918) 455-0503.

February 10-13 in Sun Valley, Idaho. St Helena Hospital of Deer Park, California, and the American College of Chest Physicians (ACCP) cosponsor a program on
NEw and updated
individual independent
study packages

NEW - Microbiology for Respiratory Care: A Review of Microbial Growth and Cross-Contamination. The CDC estimates that 5% to 15% of patients contract nosocomial infections and about 20,000 die each year. This study package provides you with an overview of some important aspects of microbiology in respiratory care. There are four main sections: classification and characteristics of microbes, requirements for microbial growth, cross-contamination, and prevention of disease transmission. Item CS17 — $10

NEW - Arterial Blood Gas Interpretation. Describes a systematic method that allows you to correctly classify the acid-base dysfunction and to relate the diagnosis concisely and coherently to other members of the health care team. Item PE10 — $10

UPDATED - Ventilation/Perfusion Relationships in Health and Disease. Ventilation/perfusion abnormalities account for the major share of the severe hypoxemia observed in COPD, with alveolar hypoventilation representing an additional contributing factor. Because of the relatively high incidence of COPD in the patient population treated by respiratory care practitioners it is important that you have an understanding of ventilation/perfusion concepts. Item CS21 — $10

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CALENDAR

“Cardiopulmonary Wellness and Rehabilitation.” Contact Darlene Buczak at the ACCP, (708) 698-2200.


March 10-13 in Denver, Colorado. The National Jewish Center for Immunology and Respiratory Medicine, in conjunction with the American College of Chest Physicians, presents the 3rd International Conference on Pulmonary Rehabilitation and Home Mechanical Ventilation, with concurrent workshops on home ventilator care and pulmonary rehabilitation, at the Denver Hyatt. Contact Adele Gelfand, Conference Coordinator, (303) 398-1359.

March 11-20 in Stanford, California. The Sleep Medicine and Technology Training and Education Center—Stanford holds one of many year-round courses with an emphasis on respiration during sleep. Selected audio tapes are also available. (415) 493-0131.

March 15 in New York, New York. The Joint Commission for the Accreditation of Health Care Organizations offers a Quality Assurance in Home Care seminar for providers and/or suppliers of home medical equipment, infusion therapy, and home health, personal care, and support services. Teachers are Joint Commission home-care surveyors with expertise in quality assurance. The seminar offers hands-on experience in quality assurance program development, implementation, and evaluation. Other seminars are scheduled for May 1 in Los Angeles, California; May 22 in Orlando, Florida; and September 27 in Princeton, New Jersey. For a brochure, contact the Joint Commission Customer Service Center at (708) 916-5800.

April 11-12 in Cape Cod, Massachusetts. The New England Association of Allied Health Educators holds the 8th Annual Meeting at the Hyannis Sheraton at picturesque Cape Cod. Conference program topics include Leadership and Communication and Designing Clinical Objectives. Contact Darcy Blitz or Julie Ann Mangini, South Central Community College, 60 Sargent Drive, New Haven CT 06511. (203) 789-6970.

August 25-September 1, Caribbean Cruise. Cruise the Western Caribbean aboard the SS Sea Breeze while earning 8 CRCE credits. Topic is “Aid for AIDS.” $895 prepaid includes airfare, cruise, transfers, food, and entertainment. Friends and family welcome. Call or write Dream Cruises, 10882 LaDonna Ave, Garden Grove CA 92640. 1-800-462-3628.

RESPIRATORY CARE • JANUARY '91 Vol 36 No 1
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.

AARC SUMMER FORUM
The Westin, Vail, Colorado, July 11-13, 1991

AARC ANNUAL CONVENTION SITES & DATES
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
1991 Examination and Fee Schedule:

<table>
<thead>
<tr>
<th>CRTT Examination</th>
<th>RPFT Examination</th>
</tr>
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<tr>
<td>Applications Accepted Beginning: November 1, 1990</td>
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<tr>
<td>EXAMINATION DATE: JULY 20, 1991</td>
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<td></td>
</tr>
<tr>
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CPFT Examination

EXAMINATION DATE: JUNE 1, 1991
Applications Accepted Beginning: December 1, 1990
Application Deadline: April 1, 1991

RRT Examination

EXAMINATION DATE: JUNE 1, 1991
Applications Accepted Beginning: December 1, 1990
Application Deadline: February 1, 1991
EXAMINATION DATE: DECEMBER 7, 1991
Applications Accepted Beginning: June 1, 1991
Application Deadline: August 1, 1991

Perinatal/Pediatric Respiratory Care
Specialty Examination

EXAMINATION DATE: MARCH 9, 1991
Applications Accepted Beginning: July 1, 1990
Application Deadline: November 1, 1990
Application Fee: $150

Fee Schedule

- Entry Level CRTT—new applicant: $75.00
- Entry Level CRTT—reapplicant: $50.00
- RRT Written and Clinical Simulation—new applicant: $175.00
- Written Registry Only new applicant: $75.00
- Written Registry Only reapplicant: $50.00
- Clinical Simulation Only new and 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:
  - 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

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*In Maryland, call collect (301) 881-0256 between 9:00 AM and 4:30 PM
1991 Call for Abstracts

The American Association for Respiratory Care and its science journal, Respiratory Care, invite submission of brief abstracts related to any aspect of cardiorespiratory care. The abstracts will be reviewed, and selected authors will be invited to present papers at the Open Forum during the AARC Annual Meeting in Atlanta, Georgia, December 7-10, 1991. Accepted abstracts will be published in the November 1991 issue of Respiratory Care. Membership in the AARC is not necessary for participation.

Specifications

An abstract may report (1) an original study, (2) the evaluation of a method or device, or (3) a case or case series. Topics may be aspects of adult acute care, continuing care/rehabilitation, perinatology/pediatrics, cardiopulmonary technology, health occupations education, or management of personnel and health-care delivery. The abstract may have been presented previously at a local or regional—but not national—meeting and should not have been published previously in a national journal.

The abstract will be the only evidence by which the reviewers will decide whether the author should be invited to present a paper at the Open Forum. Therefore, the abstract must provide all important data, findings, and conclusions. Give specific information. Do not write such general statements as “Results will be presented” or “Significance will be discussed.”

Essential Content Elements

An original study abstract must include (1) Introduction: statement of research problem, question, or hypothesis; (2) Method: description of research design and conduct in sufficient detail to permit judgment of validity; (3) Results: statement of research findings with quantitative data and statistical analysis; (4) Conclusions: interpretation of the meaning of the results.

A method/device evaluation abstract must include (1) Introduction: identification of the method or device and its intended function; (2) Method: description of the evaluation in sufficient detail to permit judgment of its objectivity and validity; (3) Results: findings of the evaluation; (4) Experience: summary of the author’s practical experience or a notation of lack of experience; (5) Conclusions: interpretation of the evaluation and experience. Cost comparisons should be included where possible and appropriate.

A case report abstract must report a case that is uncommon or of exceptional teaching/learning value and must include: (1) case summary and (2) significance of case. Content should reflect results of literature review. The author(s) should have been actively involved in the case and a case-managing physician must be a co-author or must approve the report.

Abstract Format and Typing Instructions

An optical scanner will be used to process abstracts. First line of abstract should be the title. Title should explain content. Type or electronically print the abstract double-spaced on plain white bond paper, on one page only (copier bond is excellent). Do not underline or boldface and insert only one letter space between sentences. Provide a 1-inch margin top and bottom, a 1/2-inch left margin, and an approximate 1/2-inch ragged-right margin. Text may be submitted on diskette but must be accompanied by a hard copy.

No identification of authors or institutions is to appear on the abstract sheet or in the abstract itself. Make the abstract all one paragraph. Data may be submitted in table form provided the table width is limited to 60 letter spaces (ie, letters or numbers plus necessary blank spaces = 60). No figures or illustrations are to be attached to the abstract.

Type all information required to complete the author information form on the other side of this page. A photocopy of good quality may be used.

Standard abbreviations may be employed without explanation. A new or infrequently used abbreviation should be preceded by the spelled-out term the first time it is used. Any recurring phrase or expression may be abbreviated if it is first explained.

Check the abstract for (1) errors in spelling, grammar, facts and figures; (2) clarity of language; (3) conformance to these specifications. An abstract not prepared as requested may not be reviewed.

Questions about abstract preparation may be telephoned to the editorial staff of Respiratory Care at (214) 243-2272.

Deadlines

The mandatory Final Deadline is June 5 (postmark). Authors will be notified of acceptance or rejection by letter only—to be mailed by August 15.

Authors may choose to submit abstracts early. Abstracts received by March 20 will be reviewed and the authors notified by April 26. Rejected abstracts will be accompanied by a written critique that should in many cases enable authors to revise their abstracts and resubmit them by the final deadline (June 5).

Mailing Instructions

Mail (Do not fax!) 1 copy of the abstract, 1 author information sheet, and a stamped, self-addressed postcard (for notice of receipt) to:

Respiratory Care
11030 Ables Lane
Dallas TX 75229
1991 OPEN FORUM
AUTHOR INFORMATION SHEET

Please type:

Abstract title

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Mail, with abstract and stamped self-addressed postcard, to RESPIRATORY CARE Open Forum
11030 Ables Lane, Dallas TX 75229
Instructions for Authors and Typists

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

General Requirements

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

Publication Categories

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

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

Editorial Consultation and Author’s & Typist’s Kit

To discuss a writing project, write to RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229 or call 214/243-2272.
Authors are urged to obtain the RESPIRATORY CARE Author’s & Typist’s Kit. The Kit provides authors with specific guidance about writing a research paper, writing a case report, converting to and from SI units, and in-house manuscript review. Typists can use the Kit’s Model Manuscript, a list of journal name abbreviations, and a copy of these Instructions. The Kit is free from the Journal office.

Preparing the Manuscript

General Concerns—Typist

• Double-space ALL lines, including those in references, figure legends, and tables. Do not justify right margins.
• Number pages in upper right corner and leave margins of 1/4” or more on all four sides of the page.
• For research articles, follow format of Model Manuscript, Respir Care 1984;29:182 (Feb 1984).
• Meticulously follow instructions for typing references.

General Concerns—Author:

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

Manuscript Structure

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


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

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

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

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

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

Structure: Important Details

Title Page: List title of paper, all authors’ full names, degrees, credential letters, professional positions, and affiliations. List correspondence address, telephone number, and reprint address if desired. Name sources of grants or other support. Identify any author’s consulting or commercial relationships that pertain to the paper’s topic.
Abstract Page: Number this Page 1. List paper's title but omit authors' names. Abstract should be 200 words or less and must be informative, briefly specifying main points of paper, such as methods, results, and conclusions drawn.

Statistical Analysis: In research articles, identify statistical tests and chosen level of significance in the Methods section. In Results section, report actual P values.

Figures (Illustrations): All photographs, diagrams, & graphs must be numbered as Figure 1, Figure 2, etc., according to the order in which each is first mentioned in the text. Photographs must be glossy prints 5 × 7 to 8 × 10 inches and should be black & white unless color is essential. Letters and numerals must be neat and large enough to remain legible if figure is reduced in size for publication. Final figures must be of professional quality, but 'rough' sketches may accompany the submitted manuscript, with final figures to be prepared after review. Identify each figure on back with a stick-on label showing figure number and arrow indicating top; omit author's name. Cover label with clear tape so ink will not smudge other prints. Supply three sets of unmouted figures. If figure has been published before, include copyright-holder's written permission to use it.

Figure Legends: List figure legends on a separate page, not on figures. If a figure has been published before, list the source in the legend.

Tables: Type each table on a separate page. Avoid more than 8 columns across. Continue a deep table on following pages. Give each table a number and descriptive title, placed above the table. Double-space ALL lines in tables, including column headings and footnotes.

Drugs: Brand names may be given, but always also show generic names.

Units of Measurement: In addition to conventional units of measure, show SI values and units in brackets after conventional expressions, e.g., "PEEP, 10 cm H₂O [0.981 kPa]." For conversion to SI, see RESPIRATORY CARE 1988;33:861-873 (Oct 1988).

Commercial Products: If three or fewer commercial products are named in the text, list the manufacturer's name and location in parentheses the first time each is mentioned. If four or more products are named, do not list manufacturers in the text; instead, name the products and manufacturers in a Products Sources list at the end of the text. Provide model numbers when available.

Abbreviations: Use an abbreviation only if the term occurs several times in the paper. Write out the full term the first time it appears, followed by the abbreviation in parentheses. Thereafter, employ the abbreviation alone. Never use an abbreviation without defining it. Do not create new abbreviations unless absolutely necessary.

References:
- Use references to support statements of fact, indicate sources of information, or guide readers to further pertinent literature.
- Cite only published works—or works accepted for publication. When listing an accepted but still unpublished work, designate the accepting journal's name, followed by "(in press)."
- In the text, cite references by superscript numerals (half space above text), not in parentheses. The first reference cited in the text is number 1, the next is number 2, etc.
- In the reference list, place the cited works in numerical order.
- 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, editorial, and letters as such. See examples.

News releases about new products and services will be considered for publication in this section. There is no charge for these listings. Send descriptive release and glossy black and white photographs to RESPIRATORY CARE Journal, New Products and Services Dept, 11030 Ables Lane, Dallas TX 75229.

MECHANICAL CARDIOPULMONARY RESUSCITATOR. The Thumper cardiopulmonary resuscitator is designed to sustain the patient from the emergency scene to the hospital with completely automatic, synchronized external cardiac compression and intermittent time-cycled, positive-pressure ventilation in accordance with American Heart Association Guidelines. According to the manufacturer, the resuscitator allows more definitive therapy to begin at the scene, reduces the number of rescue workers surrounding the patient, and decreases confusion on the scene and in the hospital. The newest version of the Thumper, Model 1005, features clear measurement of chest compression and continuously displayed ventilation pressure. The fully portable unit is powered by compressed oxygen and weighs less than 25 lb. Michigan Instruments Inc, Dept RC, 6300 28th St, Grand Rapids MI 49546. (616) 942-9721.

AMBULATORY ECG-MONITORING SYSTEM. The Custo Jet System, consisting of the Custo Port recorder, interface unit, and matrix printer, provides ambulatory ECG monitoring and analysis. The recorder provides 24 hours of full disclosure ambulatory monitoring in real-time format and is designed to be lightweight and portable enough so that patients can comfortably perform typical daily tasks during monitoring. Solid-state technology eliminates the need for cassette tapes or reel-to-reel devices. The recorder operates on a single C battery. Static RAM technology enables storage of the complete test for up to 14 days in the recorder. According to the manufacturer, no loss of data occurs if the battery loses power or is removed; the recorder provides complete detailed analysis of ST-segment changes with QRS complex averaging for artifact elimination; and serious abnormalities are stored independently in a two-channel strip format. After monitoring is completed, the recorder is inserted into the interface unit and acquired data are transmitted to the matrix printer, which produces a report. The report provides a narrative and hourly overview, detailed data accumulated during monitoring, exact date and time of stored events, and ST-segment analysis. Siemens-Burdick Inc, Dept RC, 915 N Plum Grove Rd, Suite C, Schaumburg IL 60173. (708) 517-7000.

INFANT RESUSCITATOR. The new Fisher and Paykel RD1000 infant resuscitator is designed to provide an alternative to manual bag resuscitation and to give the respiratory care practitioner precise control of airway pressure for increased emergency resuscitation safety. The RD1000 has built-in safety features including a pressure-relief valve and a manometer gauge that monitors airway pressure; disposable components are used with the RD1000 to reduce the risk of contamination. The adjustable PEEP valve at the patient end of the breathing circuit is designed to allow for easy fingertip control. Baxter Healthcare Corp, Pharmaseal Div, Dept RC, 27200 North Tourney Rd, Valencia CA 91355-8900. (714) 686-8900.

RESPIRATORY CARE ● JANUARY '91 Vol 36 No 1
# Authors in This Issue

<table>
<thead>
<tr>
<th>Author</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basile, John</td>
<td>57</td>
</tr>
<tr>
<td>Blanchette, Tim</td>
<td>25</td>
</tr>
<tr>
<td>Brougher, Pat</td>
<td>23</td>
</tr>
<tr>
<td>Brown, Dennis T</td>
<td>33</td>
</tr>
<tr>
<td>Clevenger, Frederick W</td>
<td>63</td>
</tr>
<tr>
<td>Courtney, Sherry E</td>
<td>40</td>
</tr>
<tr>
<td>Dziodzio, John</td>
<td>25</td>
</tr>
<tr>
<td>Gill, Vee J</td>
<td>33</td>
</tr>
<tr>
<td>Godwin, Cynthia R</td>
<td>33</td>
</tr>
<tr>
<td>Graybeal, John M</td>
<td>57, 68</td>
</tr>
<tr>
<td>Harris, Kathryn</td>
<td>25</td>
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<tr>
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<td>40</td>
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</tr>
<tr>
<td>Kacmarek, Robert M</td>
<td>45</td>
</tr>
<tr>
<td>Kimura, Tomomasa</td>
<td>45</td>
</tr>
<tr>
<td>Kittredge, Phil</td>
<td>21</td>
</tr>
<tr>
<td>Masur, Henry</td>
<td>33</td>
</tr>
<tr>
<td>Matz, Jonathan</td>
<td>53</td>
</tr>
<tr>
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<td>45</td>
</tr>
<tr>
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<td>33</td>
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<td>Ohmura, Akito</td>
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<td>57, 68</td>
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<td>17</td>
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<td>Schermer, Carol R</td>
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<tr>
<td>Takezawa, Jun</td>
<td>45</td>
</tr>
<tr>
<td>Tokioka, Akihiro</td>
<td>45</td>
</tr>
<tr>
<td>Weber, Kaye R</td>
<td>40</td>
</tr>
</tbody>
</table>

# Advertisers in This Issue

<table>
<thead>
<tr>
<th>Advertiser</th>
<th>Pages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bird Products Corp</td>
<td>4</td>
</tr>
<tr>
<td>Ciba-Corning Diagnostics</td>
<td>14, 15</td>
</tr>
<tr>
<td>HealthScan</td>
<td>11</td>
</tr>
<tr>
<td>Monaghan Medical</td>
<td>6, 7</td>
</tr>
<tr>
<td>Puritan-Bennett Corp</td>
<td>Cover 2, 1</td>
</tr>
<tr>
<td>Quinton Instrument Co</td>
<td>13</td>
</tr>
<tr>
<td>Schering Corp</td>
<td>Cover 3, Cover 4</td>
</tr>
<tr>
<td>Sherwood Medical</td>
<td>2</td>
</tr>
<tr>
<td>Siemens Life Support Systems</td>
<td>9</td>
</tr>
</tbody>
</table>

# Employment Opportunities

- Underwood Memorial Hospital, Woodbury NJ 72
- Star Med, Tampa FL 69
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Check the boxes below for information from the AARC
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RESPIRATORY CARE

December Information Service Expires April 30, 1991

Name ____________________________ Ph.# ____________________________

Institution ____________________________

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Please Circle No More Than 15 Items

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1. Type of Inhale/Practice
☐ Hosp. 500 or more beds
☐ Hosp. 300 to 500 beds
☐ Hosp. 200 to 300 beds
☐ Hosp. 100 to 200 beds
☐ Hosp. 100 or less beds
☐ Clinic/Group Practice
☐ Independent RT Provider
☐ Industry (Mfg/Sales)

II. Department
☐ Respiratory Ther.
☐ Cardiopulmonary
☐ Anesthesia Service
☐ Emergency Dept.

III. Specialty
☐ Clinical Practice
☐ Paramed Pediatr
☐ Critical Care
☐ Critical Research
☐ Pulmonary Func Lab
☐ Home Care/Rehab
☐ Education
☐ Management

IV. Position
☐ Dept. Head
☐ Chief Therapist
☐ Supervisor
☐ Staff Technician
☐ Staff Therapist
☐ Educator
☐ Medical Director
☐ Anesthesiologist
☐ Other MD
☐ Nurse

V. Are you a member of the AARC?
☐ Yes
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☐ Other MD
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81 AARC
Membership Info

82 RESPIRATORY CARE
Subscription Info

100 Bird Products Corp
Infant Pediatric Ventilator

104 Ciba-Corning Diagnostics
Data Management System

115 HealthScan Products
Peak Flowmeter

112 Monaghan Medical
AeroVent

108 Puritan-Bennett Corp
7200ae Ventilator

128 Puritan-Bennett Corp
7200ae Ventilator

103 Quinton Instruments Co
CP Exercise System

123 Schering Corp
Metered Dose Inhaler

131 Sherwood Medical

126 Siemens Life Support System
Servo 900C Ventilator
Use these cards to request information from the AARC or advertisers in this issue.
PROVENTIL®
brand of albuterol sulfate

Solution for Inhalation 0.5% *  
Solution for Inhalation 0.083% *  
(Potency expressed as albuterol)

DESCRIPTION
PROVENTIL® brand of albuterol sulfate Soluion for Inhalation, is a selectively beta-2-agonist bronchodilator (see CLINICAL PHARMAcOLOGY section below). Albuterol sulfate has the chemical formula of ([l]-Butylamino) methyl-4-hydroxy-n-ylene oxide-diol sulfate (2:1). It is a white crystalline powder, soluble in water and slowly soluble in ethanol.

PROVENTIL® Solution for Inhalation is available in two concentrations. The 0.5% solution is in concentrated form. Dilute 0.5 ml of the solution in 3 ml with normal saline solution prior to administration. The 0.083% solution requires no dilution prior to use.

Each ml of PROVENTIL® Solution for Inhalation (0.5%) contains 5 mg of albuterol (as 6.8 mg of albuterol sulfate) in an aqueous solution containing benzyl alcohol, sodium chloride, and sulfuric acid to adjust the pH to between 3 and 5. PROVENTIL® Solution for Inhalation (0.083%) contains no sulfuric acid. It is supplied in 3 ml bottles for units-size dispensing.

PROVENTIL® Solution for Inhalation is clear, colorless to light yellow solution.

CLINICAL PHARMACOLOGY
The prime action of beta-adrenergic drugs is to stimulate adenyl cyclase, the enzyme which catalyzes the formation of cyclic-3',5'-adenosine monophosphate (cyclic AMP) from adenosine monophosphate (AMP). The cyclic AMP thus formed mediates the cellular responses. In vivo studies and in vitro pharmacologic studies have demonstrated that albuterol has a preferential effect on beta-adrenergic receptors compared with isoproterenol. While it is recognized that beta-agonists may produce other non-beta-adrenergic effects, current evidence suggests that the dose required to reach 10-20% of the beta-receptors in the human heart may be beta-receptors. The functional action of these receptors, however, is not yet established.

Albuterol has been shown in most controlled clinical trials to have more effect on the respiratory tract, in the form of bronchodilation, than on the heart. Albuterol may produce less cardiovascular effects compared with the other beta agonists, while producing fewer cardiovascular effects. Controlled clinical studies and other clinical experience have shown that inhaled albuterol, like other beta-adrenergic agonists, can produce a significant cardiac-mediated effect in some patients, as measured by pulse, blood pressure, symptoms, and in ECG changes.

Albuterol is longer acting than isoprenaline in most patients by any route of administration because it is not substrate for the cellular uptake processes for catecholamines or for catechol-O-methyl transferase.

Studies in asthmatic patients have shown that less than 20% of a single albuterol dose was absorbed following either theophylline or an uncontrolled route; the remaining amount was recovered from the urine and a proportion excreted in the faeces. Most of the absorbed dose was recovered in the urine 24 hours after drug administration. Following a 3.0 mg dose of inhaled albuterol, the maximum albuterol plasma level at 0.5 hour was 2.1 ng/ml (range 1.4 to 3.7 ng/ml). There was a significant dose-related increase in FEV1 and peak flow rate (PFR) and it has been demonstrated that oral administration of 4 mg albuterol via nebulizer is effective.

Inhalation of the solution is painless to the larynx.

Animal studies show that albuterol does not pass the blood-brain barrier. Recent studies in laboratory animals (minipigs, rhesus monkeys and dogs) recorded the occurrence of cardiac arrhythmias and sudden death (with histologic evidence of myocardial necrosis) when beta-agonists and methylnxanthines were administered concurrently. The significance of these findings when applied to humans is currently unknown.

In clinical trials, most patients experienced an onset of improvement in pulmonary function within 5 minutes as determined by FEV1, FEV2, measurements also showed that the maximum average improvement in pulmonary function usually occurred approximately 1 hour following inhalation of 2.5 mg of albuterol by compressed aerosol inhaler and remained elevated for at least 2 hours. Clinically significant improvements in pulmonary function (defined as maintenance of a 15% or more increase in FEV1, over baseline values) continued for 3 to 4 hours in most patients and in some patients continued up to 6 hours.

In repetitive dose studies, continued effectiveness was demonstrated throughout the three-month period of treatment in some patients.

INDICATIONS AND USAGE
PROVENTIL® Solution for Inhalation is indicated for the relief of bronchospasm in patients with reversible obstructive airway disease and acute attacks of bronchospasm.

CONTRAINdications
PROVENTIL® Solution for Inhalation is contraindicated in patients with hypersensitivity to one or more of its components.

WARNINGS
As with other inhaled beta-adrenergic agonists, PROVENTIL Solution for Inhalation can produce paradoxical bronchospasm, which can be life threatening. If an attack occurs, the preparation should be discontinued immediately and all other available treatment should be used.

Fatalities have been reported in association with excessive use of inhaled sympathomimetic drugs and with the home use of sympathomimetic medications. It is, therefore, essential that the physician restrict the patient in the need for further evaluation if a paradoxical bronchospasm attack occurs. If a paradoxical bronchospasm attack occurs, the patient may have a clinically significant cardiac effect.

Inhaled beta-agonist preparations may cause serious adverse reactions, especially in asthma patients with cardiovascular disease, including angina pectoris, myocardial infarction, and sudden death. Patients with cardiovascular disease are at increased risk for adverse reactions and should be monitored closely. Because of the potential for such reactions, PROVENTIL Solution for Inhalation should be used with caution in patients with cardiovascular disease.

Information For Patients: The admonition to PROVENTIL Solution for Inhalation is that the patient be instructed not to use the inhaler too frequently or for too long periods of time. It is not recommended to take the inhaler more frequently than the label indicates.

Use of an alternative beta-agonist should be considered for patients who experience an adverse reaction to PROVENTIL Solution for Inhalation.

OVERDOSAGE
Overdosage of PROVENTIL Solution for Inhalation may cause symptoms of toxicity such as headache, nervousness, tremor, dizziness, palpitations, tachycardia, and chest pain.

In case of overdose, the patient should be observed for four to six hours after dosing and given symptomatic and supportive treatment. In case of severe symptoms, consultation with a physician should be sought.

PROVENTIL® Solution for Inhalation is contraindicated in patients with hypersensitivity to one or more of its components. If a paradoxical bronchospasm attack occurs, the patient may have a clinically significant cardiac effect.

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Proventil
(albuterol) Solution
for Inhalation
0.063% and 0.5%

WHEN A METERED-DOSE INHALER IS NOT ENOUGH

Hospital therapy that's also comfortable in the home

*potency expressed as albuterol

For Prescribing Information, please see following pages.