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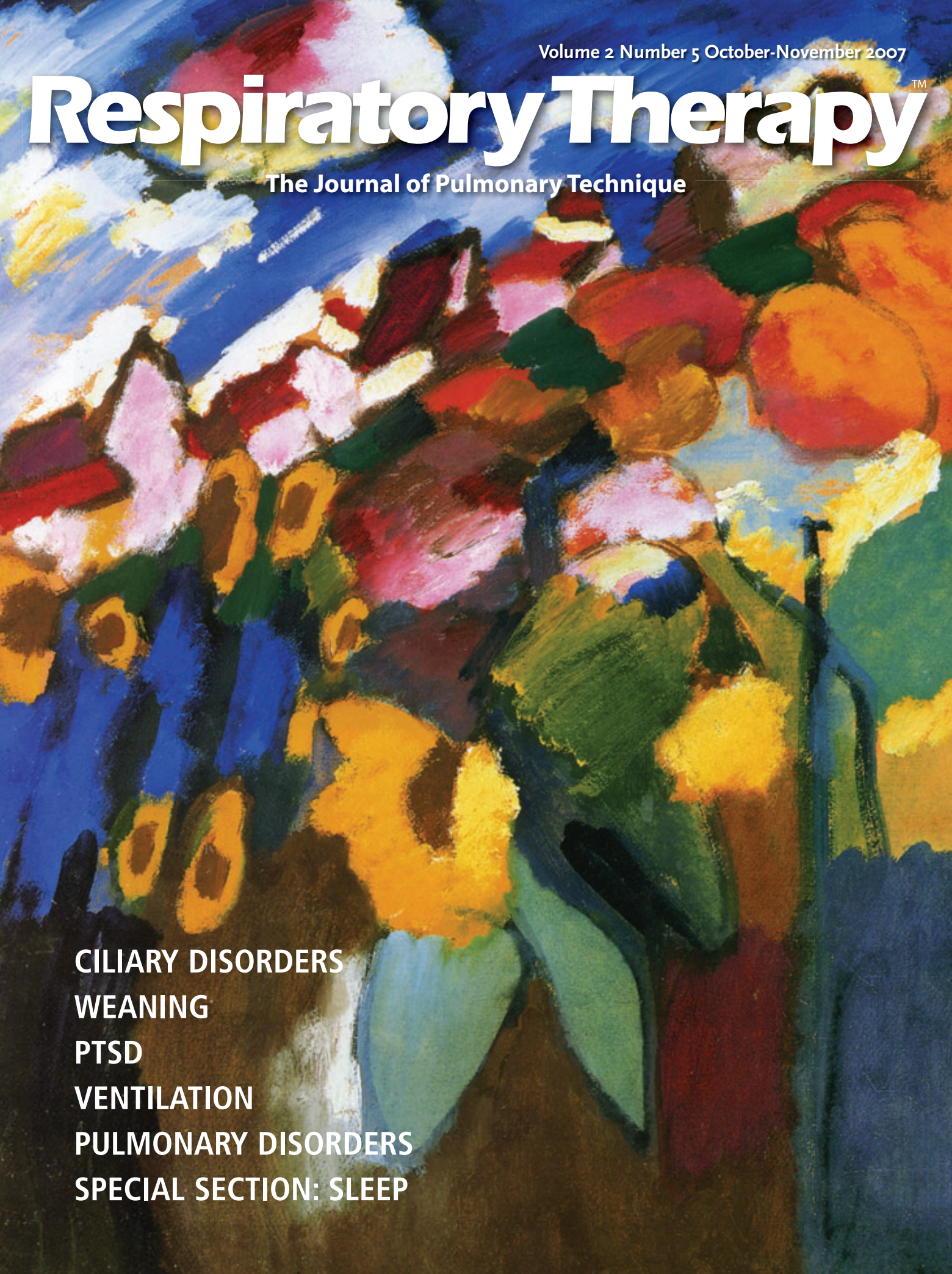
Respiratory Therapy™

The Journal of Pulmonary Technique



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Editorial

Good Better Best?

"In an eight-country comparison, the United States ranked last in years of potential life lost to circulatory diseases, *respiratory diseases* and diabetes and had the second highest death rate from *bronchitis asthma and emphysema*." So reports a recent editorial in The New York Times (World's Best Medical Care?, August 12). How can this be? How can it be explained or excused? If you look at the gamut of papers and topics discussed in this one issue of one journal among the hundreds published every month, you'd be hard put to come up with an answer.

Just for reference, we present papers on underdiagnosed, undertreated conditions, weaning from mechanical ventilation, PTSD requiring mechanical ventilation, chronic fatigue syndrome, and sleep disordered breathing, among other topics. A recent search of clinical trial databases revealed 74 ongoing or pending trials in sleep research alone.

But papers are one thing, praxis is another. Why are we faring so badly? Here's what the Times says: All other major industrialized nations provide universal health coverage with no cost-sharing by patients; the US has 45 million people without health insurance and many more with inadequate coverage. While patients wait a long time for special treatment abroad, this isn't necessarily so for basic treatment. A recent report in Harper's notes that same-day care is more readily available in France. The US, according to the Times, "ranks dead last on almost all measures of equity because we have the greatest disparity in the quality of care given to richer and poorer citizens." While the U.S. ranked first in quality of care among five major industrialized nations, and in preventive care, it scored poorly in coordinating the care of chronically ill patients and in protecting the safety of patients, "which drove our overall quality rating down to last place."

Well, all right, but what do actual patients think? "Americans hold surprisingly negative views of their healthcare system. Only 40% of Americans were satisfied with the nation's healthcare system, placing us 14th out of 17 countries… This may be because Americans face higher out-of-pocket costs, are less apt to have long-term doctors, are less able to see a doctor on the same day when sick, and less apt to get their questions answered or receive clear instructions from a doctor." The Times notes, however, "it is doubtful that many Americans, faced with a life-threatening illness, would rather be treated elsewhere. We tend to think that our very best medical centers are the best in the world. But whether this is a realistic assessment or merely a cultural preference for the home team is difficult to say."

Of course making wholesale changes that would rectify this situation would alter the entire US healthcare landscape, and the consequences of such changes would result, in turn, in other consequences, some of which are bound to be negative – at least depending on one's perspective. Obviously, one reason we have so much new and innovative research (as noted above) is exactly the result of our capital-driven system. Still, if the research isn't applied fairly and equitably, what exactly is the point of it? But that takes us into an entirely other realm of ethical discourse.

Les Plesko, Editor

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News

☐ October-November 2007

LETTER TO THE EDITOR

In his letter criticizing aspects of my article, *High-Frequency Chest Compression: Advanced Therapy for Obstructive Lung Disease* (April-May 2007), Mr Jan Tecklin has failed to read the text carefully. Tecklin states that the studies I cited concerning triangle waveform high-frequency chest compression (HFCC) machines present "…an inaccurate and severely biased perspective on the so-called 'newest HFCC technology'." He goes on to discredit the quality of that research.

If Mr Tecklin had read my conclusion, he would have noticed my statement, "[r]ecent, limited evidence suggests that waveform may be an important component of machine performance and that the triangle waveform may offer other advantages. The actual significance of waveform differences in HFCC machines remains unclear. Further research, some currently in progress, should improve understanding of that aspect of the technology." Clearly, I have made no inaccurate or biased claims.

More disturbingly, Mr. Tecklin asserts that because my co-author (M. Nozzarella) is an executive officer for one of three competing HFCC companies, *Respiratory Therapy* is at fault for not having recognized… "that biased information related to a clear fiduciary conflict of interest by any author(s) does not present useful information." In response, I would refer Mr. Tecklin to the journal's clearly stated "open forum" editorial policy entitled *Truth or Fiction* (February/March 2006). Mr. Tecklin, a consultant for another HFCC device manufacturer, has had his own work published in Respiratory Therapy. In his paper, *High Frequency Chest Wall Oscillation for Individuals With Neuromuscular Weakness* (December/January 2005/2006), his recommendation for therapy mentions by name only his corporation's HFCC device. He then goes on to list "…attractive features" particular to that device.

Respiratory Therapy understands, as stated in its editorial policy, that in the real world, "…there is no such thing as a neutral unbiased outlook." The journal is open to all points of view and believes that its readers are competent to assess information intelligently.

Jane Braverman, PhD
Director: Clinical Programs, RespirTech

REIMBURSEMENT – A RESPONSE

We received a number of questions after running an article on inpatient drug reimbursement. The article, entitled Inpatient Drug Reimbursement —How Can Respiratory Managers Deal With Increasingly Large Drug Budgets? appeared in our April-May issue. (See volume 2 number 2 for more information.) We asked Ken Abreu, the author of the original article, to address these questions.

Some of our private payers do not have provisions for separate reimbursement outside of the DRG. How can our hospital obtain such a provision?

The hospital needs to address this issue directly with the payer. Commonly this is done during contract renegotiation. Typically, a hospital renegotiates payer contracts on a yearly basis. When the hospital's Managed Care Contracting Department goes to the bargaining table, the department generally asks for certain upgrades to its contracts. Since payers want to keep a hospital's business, they usually try to accommodate the hospital if they reasonably can. You may want to seek the addition of such a provision during your hospital's next negotiation with its payers.

Another thing to keep in mind is that, while your payer contracts may not have a drug carve-out or other separate reimbursement provision for a *particular* drug, there may well be a clause in the contract that can help you. Many payer contracts have a new technology clause or other mechanisms that allow the hospital to get special payment terms even before the contracts are renegotiated.

The best way to get more information about what you and your hospital can do, both now and in the future, is to discuss these issues with your Managed Care Contracting Department. They may not know about INOmax because they are accustomed to dealing with the pharmacy, not the Respiratory Therapy

Department, on drug issues. You can help them and yourself by giving them a call.

Please give examples of the common ways that hospitals can get extra drug reimbursement funds.
To obtain fair reimbursement for medically necessary drugs and services, hospitals must apply current contractual provisions or request new ones when they renegotiate their payer contracts. Fair reimbursement for these services can be covered through contractual provisions like drug carve-outs, stop-loss clauses, and cost outlier provisions. Let's look at each one in turn.

Carve-outs are essentially contract clauses that say "When you use a particular (expensive) drug we will pay you an agreed-upon amount of money *in addition to* the DRG or per diem payment." Thus, the drug cost (or a part of it) is "carved out" from the main payment for the patient.

Stop-losses are caps on how much money a hospital spends on a given patient. Once a hospital has spent enough money on the patient to reach the cap, the payer covers the additional costs. Thus, if an expensive drug pushes a patient's costs over the cap, the additional costs are covered.

Cost outlier provisions are similar to stop-losses. If a patient requires services that significantly exceed the typical treatment on which the DRG or per diem is based, the payer will give additional reimbursement. Thus, if a patient takes an expensive drug and the overall treatment costs push the patient past the typical cost range and into outlier status, the hospital may be eligible to receive more money to help defray the additional costs of treatment.

Generally speaking, who in the hospital is the most expert in exploring reimbursement options?
While there are probably several people in the hospital's administration who may be helpful in seeking additional reimbursement, your best bet is to contact the Director of Managed Care Contracting.

Please list those internal and external resources that are available to do the work of seeking reimbursement for high budget items.
The hospital has a number of people who can help. While we already mentioned the Director of Managed Care Contracting, it bears repeating that this person can be extremely helpful. Others are the Charge Master, the Director of Revenue Integrity, and the Director of Planning and Analysis. These people may not be known to you now, but it is a great idea to pick up the telephone, call them and have a conversation. The more they help you with your expensive budget items, the more they help the hospital and the better they do their jobs.

Ken Abreu, MBA, CMR, is Director of National Accounts and Managed Markets, INO Therapeutics. For more information, please contact Ken Abreu at ken.abreu@inotherapy.com.

NEWS

GENE-IUS

A gene associated with a risk of developing childhood onset asthma was identified by scientists from the University of Michigan and their international colleagues. In a genetic study

of more than 2,000 children, the scientists found genetic markers that dramatically increase a child's risk for asthma. Children with markers located on chromosome 17 had higher levels of a new gene called ORMDL3 in their blood, which occurs in higher amounts in children with asthma. The presence of this gene increases the risk of asthma by 60-70% percent. Researchers hoped the discovery would lead to new therapies, since the results point to a specific biological molecular pathway that could be targeted with specific drugs. The discovery of an asthma gene would provide a new set of mechanisms to try and modify and manage childhood asthma. In the study, scientists compared the genetic makeup of 994 patients with childhood onset asthma and 1,243 non-asthmatics and looked at mutations in the nucleotides, as well as how genes were being expressed within human blood cells. The team confirmed its findings by analyzing the genetic makeup of more than 2,000 children from Germany and more than 3,000 subjects from the United Kingdom born in 1958 and monitored until now for the presence of disease.

KILL THE CAT

The presence of a cat could exacerbate allergies even if you're not specifically allergic to cats, according to a study at Imperial College of London. Researchers at 20 locations throughout Europe, were surprised to find that people who were not specifically allergic to cats experienced higher bronchial responsiveness when in the presence of cats. The study indicated that people who are allergic to mold, timothy grass and dust mites are most affected. Researchers said more studies should be done before people take drastic action.

A CURE AT LAST?

The first cousin of the president of Chile, a doctoral candidate at the Hebrew University of Jerusalem, says he has found a cure for allergies. Ido Bachelet has already won a prize for his efforts. His research focused on mechanisms that regulate the function of mast cells, which trigger allergic reactions. When exposed to allergens, these cells release pro-inflammatory substances like histamine. Bachelet identified a receptor protein on mast cells, termed CD300a, which has a prominent negative effect on mast cell activity, virtually stopping the cell from unleashing allergic responses. On the downside, CD300a is widely found throughout the immune system, and simply targeting it could result in undesired, overall immune suppression. In order to overcome this problem, Bachelet designed a small, synthetic, antibody fragment that has the ability of recognizing two targets simultaneously — the receptor CD300a and a mast cell-specific marker. Thus, the antibody targets CD300a only on the surface of mast cells, avoiding suppression of other immune cells. This antibody potently eliminated four different types of allergic diseases in mice. Moreover, when mice suffering from severe chronic asthma received the antibody in nose drops, they completely reverted to normal, healthy mice in less than two months. Bachelet's project, named Receptra, is currently under development and in line for clinical trials.

BE STILL MY HEART

People with reduced lung capacity may have a greater risk of heart attack and stroke because they show evidence of inflammation, according to a New Zealand study. Researchers took measurements of lung capacity and inflammation in 1,000 adults aged between 26 and 32 years. To measure inflammation, they looked at the amount of C-reactive protein. Higher levels of CRP were found in the blood of those with smaller lung capacities. Although increased markers for inflammation have previously been found in the blood of older people with reduced lung function and COPD, the authors said this was the first report of an inverse association between lung function and CRP in young adults. The results showed that this association was not related to smoking, lung disease, or obesity. Nor was atherosclerosis a culprit, since the subjects were as young as 26. Thus, the researchers said their findings indicate that the association between lower lung function and increased inflammation predates the development of either chronic lung disease or clinically significant atherosclerosis.

DRINKING AND BREATHING

Researchers at Virginia Commonwealth University found that in their intensive care unit, 40% of the patients had alcohol-related problems. The researchers examined a year's worth of data from the Nationwide Inpatient Sample, a large, all-payer inpatient database representing approximately 1,000 hospitals, and analyzed all adult patients with one of the six most common diagnoses associated with admission to the intensive-care unit: pneumonia, sepsis, gastrointestinal hemorrhage, asthma, COPD, and respiratory failure. Of the 785,602 patients diagnosed with one of these six medical diagnoses, 65,071 (8.3%) required mechanical ventilation. Those with an AUD (26,577) appeared to have an increased risk of requiring mechanical ventilation, while those who developed alcohol withdrawal (3,967) appeared to need mechanical ventilation for a longer duration. Researchers concluded that excessive alcohol consumption not only causes serious adverse events such as alcohol intoxication and accidents, but also increases the chances of developing life-threatening medical illnesses such as pneumonia, asthma, chronic obstructive lung disease, infections and respiratory failure. Patients with AUDS were said to be at risk for ventilator-associated pneumonia, and possibly acute respiratory distress syndrome. It was urged that patients should be identified for problems with alcohol consumption.

A LOT OF WHEEZING!

About 3 of every 10 children and teens admitted to the hospital from the emergency room have asthma, pneumonia, acute bronchitis or another respiratory disorder, according to the Agency for Healthcare Research and Quality. Asthma alone accounted for more than one-third of respiratory admissions - 95,000 cases - through the emergency room in 2004. The AHRQ also found that respiratory conditions are the leading cause of hospital admissions through the emergency room for infants and toddlers, accounting for nearly 40% of such admissions. For children age 5 to 9, respiratory illnesses account for 26%. Among older children age 10 to 14 and teenagers age 15 to 17, injuries become the leading reasons for admission, with respiratory disorders becoming the third and fourth factors leading to admission for these children. About half of the 2.3 million admissions of children to hospitals begin in the ER. The report used statistics from the HCUP Nationwide Inpatient Sample, a database of hospital inpatient stays that is nationally representative of all short-term, non-federal hospitals. The data are drawn from hospitals that comprise 90% of all discharges in the United States and include all patients.

STAY OUT OF TRAFFIC

Scientists in Germany have showed that exposure of pregnant women to fine particulate matter from traffic may reduce their children's birth weight. After the scientists had investigated the

effects of the exposure of adults and children to particulate matter in the past, they are now first focusing on the risks to fetuses. Researchers studied the influence of living conditions and behaviors on the development of the immune system and allergic system in 1,016 mothers and their children born in a one-year period. On the basis of a measuring campaign at 40 locations in the city of Munich, the concentrations of traffic-related atmospheric pollutants during pregnancy, including fine particulate matter (a diameter of less than 2.5 micrometers), could be modeled at the home address of the pregnant women. The model took into account the distance of each home from streets, the population density near the home as well as the fluctuations in the concentration of the air pollutants over time during the pregnancies. Using a detailed questionnaire, the study authors disentangled the influence of air pollutants from other factors known to influence birth weight. The proportion of newborns with a birth weight below 3,000 grams increased with increasing concentrations of fine particulate matter during pregnancy. Researchers noted that the biological mechanisms which could explain the influence of air pollutants on the growth of the fetus are not yet known, since such fine particulate matter consists of hundreds of chemical substances. It is conceivable that a minor fraction of the fine particulate matter reaches the blood through the lungs and influences the placenta or other organs which are responsible for regulating the growth of the fetus.

TEST THEM ALL

The ATS and European Respiratory Society (ERS) published a new statement urging pulmonary function testing for preschool children. The working group hopes its recommendations, which focus on spirometry, tidal breathing measurements, the interrupter technique, forced oscillation, gas washout techniques and bronchial responsiveness tests, will serve as a resource for healthcare professionals and facilitate good laboratory practices by providing guidelines on how to perform the various techniques and how to interpret the measurements. The group envisions that their guidelines will help facilitate multi-center collaboration using these pulmonary function testing techniques.

APPLE A DAY

Children of mothers who eat apples during pregnancy are less likely to develop asthma, according to research at The University of Aberdeen, where 2,000 expectant mothers were questioned about their eating habits, and their children's health evaluated over five years after the kids were born. Researchers found that moms who ate four or more apples a week were half as likely to have an asthmatic child, compared with those who ate no apples or just one. No-one could say why apples produced this benefit, but the fruit has been linked to better lung health when eaten by adults, perhaps because of its antioxidant properties. The project has also linked vitamin consumption in pregnancy to lower levels of asthma. Researchers cautioned that eating apples doesn't guarantee lower asthma rates in children, since other factors may be at play.

TENDERED

Cardinal Health Care has completed its initial tender offer for Viasys Healthcare, with more than 80% of Viasys common stock tendered. The move makes Viasys a majority-owned subsidiary of Cardinal Health. "Now that Viasys is part of Cardinal Health, we are ready to move forward and bring our combined offerings to global customers," said R. Kerry Clark, chief executive officer of Cardinal Health. The tender offer and merger plans are valued at approximately $1.5 billion including the assumption of outstanding debt. Viasys had revenue of $610 million in 2006. The acquisition expands Cardinal Health's clinical and medical product offerings for acute-care customers and the business will be integrated into Cardinal Health's Medical Products Manufacturing segment.

PRODUCTS & PEOPLE

TAKING CARE

Invacare Corporation announced that Joseph S. Lewarski is joining the company as vice president, respiratory products group. Lewarski will be responsible for all of the Company's respiratory and sleep related activities, including new product development, business development, and creation of sales and marketing programs. Lewarski has served in numerous management and leadership roles in both the acute care and alternate healthcare settings. Contact invacare.com.

NO CODING

An advanced blood glucose monitoring system from Nova Biomedical provides a fast, convenient solution for diabetes self-testing. The newly released Nova Max system is built around patented new technology test strips that are so precise they do not require the calibration or coding required of currently available blood glucose monitors to perform a glucose test. This first true "no coding" technology eliminates user coding errors which studies have shown to be on the order of 16% with competitive test strips. Nova Max affords diabetes patients a new level of convenience and increased testing speed for monitoring their blood glucose levels. Test results from Nova Max are reported in just 5 seconds on a 0.3 microliter sample, the smallest sample size of any meter available today. Results are accessed from the meter's 400-test memory encouraging patient adherence to healthcare professional testing regimens. With the introduction of Nova Max, Nova Biomedical now offers a comprehensive product line of advanced technology glucose monitoring systems that also includes StatStrip and StatStrip Xpress professional meters for bedside testing in the hospital. Integrated health systems and individual hospitals with a need for hospital bedside meters and meters for self-testing can look to Nova for a single source. Contact novabiomedical.com.

BREATHTAKING

Dräger Medical announced the release of Innovian Anesthesia 2.0. The new release offers system improvements based on extensive customer feedback and includes expanded capability for IT performance in the perioperative suite. It provides additional support for ease of access, enhanced security functions to promote compliance with HIPAA regulations, and additional notification capabilities that increase visibility to data coming from other systems. Innovian 2.0 is the latest example of Dräger's expertise in developing and supporting IT Solutions at the acute point of care. Innovian Anesthesia 2.0 is the information backbone that integrates near real-time, life-critical information from patient monitoring, medical therapy devices, and other ancillary information systems. It gives clinicians fast access to patient data which can minimize multiple handoffs and duplication of effort that occurs with paper records. Hospitals that standardize on Dräger Medical solutions for IT, patient monitoring, and therapy devices, have a consistent,

harmonized graphical user interface (GUI) across their devices. This simplifies user training and provides a common look and feel. Implementing Innovian Anesthesia across the entire perioperative process impacts charting efficiency by reducing paper records. This allows clinicians to spend more time delivering care while enhancing documentation compliance and expediting reimbursement. Robust data mining capabilities facilitate more aggressive pursuit of quality improvement and cost-containment initiatives. Because Innovian Anesthesia can offer seamless connectivity to medical devices and other XML or HL7 capable hospital software systems, clinicians are able to access vital signs, patient demographics, and lab results quickly and easily. The information is then automatically integrated into the Innovian Anesthesia patient's electronic record in a secure manner, supporting the clinician in their patient care process. Contact draeger.com.

CRITICAL

Medical Graphics Corporation, St Paul, MN, introduces the Express for Critical Care Management. The Express is an Indirect Calorimeter used to optimize nutritional support, monitor patients with metabolic disorders and reduce the number of ventilator days in ICU. The system's Direct Connect preVent Pneumotach allows measurement at the endotracheal tube eliminating the need to correct for bias flow & pressure support. The system can easily be used with a wide variety of ventilators found in the ICU/CCU. Spontaneous breathing patients can be assessed using mouthpiece, mask or face tent. The small, compact design, onboard computer with intuitive software and gasless calibration make the Express a cost effective and simple to use system. Contact (800) 950.5597, medgraphics.com.

PRODUCT REVIEW

CONTINUOUS MONITORING

The information below was a symposium presentation during the 2007 European Society of Anaesthesia Congress in Munich, Germany, and cites independent research showing the potential value of this new noninvasive diagnostic tool.

Masimo, the inventor of Pulse CO-Oximetry and read-through motion and low perfusion pulse oximetry, reported that new independent studies presented at a symposium during the European Society of Anaesthesia in Munich, Germany last week indicated that Masimo's Plethysmograph Variability Index (PVI), a noninvasive measurement that quantifies changes in the plethysmographic waveform derived from pulse oximetry, can provide clinicians with a noninvasive way to monitor functional hemodynamics in their patients.

Clinicians who have evaluated PVI believe this technology will prove to be a valuable clinical tool with significant advantages over currently available indicators of changes in functional hemodynamics that are invasive, operator dependent, often inaccurate, and expensive. PVI displays a numeric representation of the changes to the pleth waveform on the pulse oximeter and allows clinicians to track and trend these changes over time as well as a Diagnostic Plethysmograph that maintains the morphology of the true pleth for clinicians to view.

In his presentation in Munich, Maxime Cannesson, MD from the Claude Bernard University and Louis Pradel Hospital in Lyon, France cited research he coauthored entitled, "Ability of a Novel Algorithm for Noninvasive Automatic Estimation of the Respiratory Variations in the Pulse Oximeter Waveform to Detect Changes in Ventricular Preload," scheduled for publication in the journal Anesthesiology later this year. The research that indicates Masimo's PVI method of quantifying changes in ventricular preload, and therefore the ability of the heart to pump adequate blood to the tissues, correlated well with invasive methods.[1]

Cannesson said the authors had demonstrated in an earlier study that a change in the beat to beat amplitude of the oximeter waveform of greater than 15% predicted whether a patient was able to adapt to fluid restoration with an appropriate physiological response.[2] Cannesson also said that it was his belief that "Pleth Variability Index can automatically and noninvasively detect changes in ventricular preload in mechanically ventilated patients in the operating room. PVI shows great promise for use in perioperative fluid optimization, which will have both clinical and economical impact. Other clinical applications for PVI are in the areas of fluid depletion/restriction, mechanical ventilator settings/adjustments, detection of changes in myocardial contractility."

Other clinicians have concurred with these observations and believe that the ability of Masimo Rainbow SET oximeters to display PVI may provide them with a useful clinical data point in the management of their patients.

Dr Mitchell Goldstein of Loma Linda University Children's Hospital said, "Trending of PVI may be useful in monitoring critical care and surgical patients, both intraoperatively and postoperatively, for appropriate hydration and ventilation status."

Dr Dan Redford of the University of Arizona, University Medical Center added, "PVI may be useful in monitoring surgical patients, for appropriate intravascular volume status. For example, a rising PVI may indicate developing hypovolemia, and a falling PVI post-fluid resuscitation is evidence of an appropriate fluid responsiveness, an important measure in the critical care/OR environment." For more information contact masimo.com.

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1 "Ability of a Novel Algorithm for Noninvasive Automatic Estimation of the Respiratory Variations in the Pulse Oximeter Waveform to Detect Changes in Ventricular Preload," Maxime Cannesson, Bertrand Delannoy, Antoine Morand, Pascal Rosamel, Jean-Jacques Lehot. Anesthesiology 2007 (In Press).

2 "Relation Between Respiratory Variations in Pulse Oximetry Plethysmographic Waveform Amplitude and Arterial Pulse Pressure inVentilated Patients," Maxime Cannesson, Cyril Besard, Pierre G. Durand, Julien Bohe, and Didier Jacques. Critical Care 2005.

SPOTLIGHT ON SPIROMETRY

MOST WITH MICRO

The new generation **MicroLab** from VIASYS has been developed for today's professional. Using Micro Medical's Gold Standard Digital Volume Transducer, it is especially suited for measuring low flow rates in patients with COPD and meets the ATS 2005 Update for performance and accuracy. The MicroLab has a high-resolution color touch screen, large function icons, three pediatric incentives and contextual Help screens. The clinician can use the internal thermal printer or connect directly to an external printer for a full page color report. With over 40,000 Micro Medical MicroLab Spirometers in use worldwide, the MicroLab is truly the most advanced portable Spirometer. With its high definition color touch screen, easy to access Icons, and small size, the **MicroLoop** is designed for today's professional on the go. Employing Micro Medical's precision Gold Standard Digital Volume Transducer, the MicroLoop is especially suited to measuring low flow rates in patients with COPD. The MicroLoop provides real time graphics with pediatric incentives and its help screens provide a built-in operator's manual. It can measure 41 parameters and stores 2000 patient tests. With the included docking station, the MicroLoop will print directly through an external printer to provide the clinician with an 8-1/2" x 11" color report. Contact VIASYS Respiratory Care, viasyshc.com.

EASY ACTION

At ndd Medical Technologies, we want to be recognized by our customers for setting new standards in pulmonary function testing and offering innovative, easy-to-use products and excellent customer support. The EasyOne Frontline is the first and only spirometer to meet the NLHEP criteria for office based spirometry. EasyOne spirometers utilize TrueFlow ultrasonic technology for superior accuracy and reliability. The EasyOne meets 2005 ATS/ERS spirometry standards and runs on AA batteries for true hand-held portability. For real-time pc based testing ndd offers the EasyOne Screen, a great option with pediatric and curve incentives. Look for ndd to introduce exciting new products based on TrueFlow ultrasonic technology in the near future.

INNOVATIVE

Smiths Medical PM, Inc is pleased to introduce a new line of diagnostic spirometry devices with a high degree of technological and functional innovation. The Smiths family of spirometry products and services include diagnostic devices, dedicated software, and telespirometry units and services. The spirometers' safety and quality systems are verified by national and independent organizations. They include the spirobankII and spirobank G handheld multifunction spirometers with graphic display, MiniSpir portable USB spirometer, Spirolab III diagnostic spirometer with oximetry option, and spirotel pocket-sized spirometry device. Smiths spirometers are designed to be patient friendly and easy to operate. Each spirometer uses turbine technology that does not need calibration, and is extremely sensitive to patient airflows. Smiths spirometers provide real time graphical curves, automatic test interpretation, and quality control monitors to evaluate patient performance. An internal temperature sensor provides automatic BTPS (Body conditions: saturated with water vapor at body temperature and ambient pressure) conversion which eliminates the need for manual entry of barometric pressure and/or room temperature. Pulse oximetry is an option on most models, with oxygen evaluation and overnight sleep screening modes standard. Smiths spirometers come standard with the WinspiroPro software and interface cables giving the user a complete pulmonary diagnostic system. For more information, please contact smiths-medical.com, (800) 558-2345.

THE ULTIMATE

Medical Graphics Corporation, St Paul, MN produces and markets the MedGraphics Ultima Series. The Ultima offers configurations for pulmonary, exercise and nutrition testing. The system measures the performance of your patients' respiratory and cardiovascular systems giving you the most complete global indicator of functional capacity. The Ultima interfaces with several external devices, including: cycle ergometer, treadmill, ECG or pulse oximeter. With state-of-the-art analyzers, as well as true breath-by breath analysis, the Ultima sets a new standard in patient testing. Using the patented preVent pneumotach, The Ultima Series offers unsurpassed accuracy, reliability and infection control. Its compact design is adaptable to any testing environment producing accurate results for you and your patients. Backed by over 20 hardware and software patents, MedGraphics leads the way in bringing the latest technology for simplified testing. Contact (800) 950-5597, medgraphics.com.

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Medical Graphics Corporation, St Paul, MN, produces and markets the MedGraphics Elite Series Plethysmograph. Winner of the Medical Design Excellence Award, the Elite offers complete spirometry, diffusing capacity, lung volumes by nitrogen washout and/or plethysmography, and airway resistance in a networked or stand-alone system. The Elite's unique "zero-clearance" door wraps around the system, allowing it to operate in small spaces while accommodating patients of all sizes, from pediatric to adult. The Elite's clear chamber improves patient comfort by providing an open field of vision, and makes testing accessible for all ambulatory and wheelchair-bound patients. Contact (800) 950-5597, medgraphics.com.

SPOTLIGHT ON BLOOD GAS

EXPRESSIVE

Nova Biomedical's Stat Profile Critical Care Xpress provides a comprehensive test menu including blood gases, electrolytes, chemistry, hematology and on-board co-oximetry in a single, compact analyzer. Tests include pH, PCO_2, PO_2, SO_2%, hematocrit, hemoglobin, sodium, potassium, chloride, ionized calcium, ionized magnesium, glucose, BUN, creatinine, lactate, and bilirubin. SmartCheck software frees the operator from performing maintenance. Snap-in reagent packs contain all required liquid and gas calibrators, and on-board controls, eliminating bulky gas tanks, regulators and humidifiers, as well as calibration and cleaning bottles. A sealed waste container stores sample and reagent waste. Integrated, on-board data management software allows data capture, manipulation, and reporting. Contact Nova Biomedical at novabiomedical.com.

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The Roche Diagnostics cobas b 221 blood gas system helps

clinicians maximize uptime while providing significant convenience and control. The configurable menu has options for blood gas, electrolytes, and metabolites. With the only FDA 501(k) clearance for testing pleural fluid pH, as well as patient trend data, automated acid-base mapping, and 42-day onboard load-and-go smart reagents, the cobas b 221 system enhances operational efficiency and simplifies regulatory compliance. (Cobas and cobas b are trademarks of Roche.) Contact roche-diagnostics.com.

A REAL GEM

Instrumentation Laboratories announces its GEM Premier 4000, the revolutionary analyzer (BG, electrolytes, metabolites, Glu, Lac, Hct, tHb, O_2Hb, COHb, HHb, MetHb, sO_2, BUN,, Creat, Total Bili, HCO3-) with integrated CO-Oximetry that quickly provides consistent, accurate, lab-quality results throughout your hospital. Easy-to-use touch-screen displays make it simple to select and customize parameters. Self-contained cartridges incorporate all components for patient testing and are maintenance-free. iQM automates quality control and continuously detects, corrects and documents to assure quality results and compliance, 24/7, regardless of operator or testing location. GEMweb® Plus software enables remote access to any networked analyzer for real-time status updates and supervision of remote locations. (BUN, Creat, Total Bili and HCO3 are in development).

IT'S TRUE

The addition of the lactate assay to IRMA TRUpoint Blood Analysis System further enhances its value for rapid, accurate point-of-care testing in emergency and critical care situations. The portable IRMA TRUpoint measures blood gases, hematocrit, electrolytes and other chemistries easily in the OR, NICU, ICU, or wherever your patient may be, so you can make treatment decisions without delay. IRMA TRUpoint utilizes single-use cartridges that are available in a variety of analyte configurations. Additional features include automatic electronic quality control, data management and an on-board printer. For more information, visit itcmed.com, (800) 631-5945.

FAST AND ACCURATE

The AVOXimeter 1000E and 4000 from ITC Medical are portable, easy to use analyzers that provide rapid, accurate assessment of a patient's true oxygenation status at the bedside. Testing is performed in single use disposable cuvettes with no storage requirements. No sample preparation is required; simply inject whole blood sample into the cuvette, and insert into the analyzer. Results are displayed and recorded in less than 10 seconds, improving efficiencies and allowing for timely diagnosis and intervention. The AVOXimeter 4000 measures oxyhemoglobin, carboxyhemoglobin, methemoglobin, total hemoglobin and oxygen content and is used in the respiratory department, ICU, OR, NICU, and ED. The AVOXimeter1000E is specifically designed for the cardiac cath lab and measures oxyhemoglobin, oxygen content and total hemoglobin concentration. Contact itcmed.com, (800) 631-5945.

SOLUTIONS

Radiometer is a leading provider of medical solutions for blood gas and acute care testing. Our instruments are used in the laboratory as well as the clinical areas of the hospital, including emergency rooms, intensive care units, surgical departments and respiratory departments. Our mission is to make testing simpler and better by expediting turnaround time, streamlining workflow and reducing the risk of errors. To help achieve that, our solutions simplify and automate key testing processes, leaving more time and confidence to focus on what matters most: patient care. Contact radiometeramerica.com.

QUALITY

RNA Medical is a leading developer of Quality Control materials for the hospital laboratory with a wide variety of products to improve patient care. For daily QC, calibration verification and linearity, RNA offers aqueous, dye-based, and bovine blood-based products for use with blood gas, critical blood analyte and CO-Oximetry instrumentation. In addition to QC products. RNA recently introduced heparinized glass capillary collection tubes with a puncture-resistant Mylar covering. Safe-Wrap Blood Collection Tubes reduce the risks associated with broken glass while at the same time offer the preferred collection material for capillary blood gas samples. Contact rnamedical.com.

FAST MOVES

Siemens announces the release of software Version 2.0 for the Rapidlab 1200 Series Blood Gas System. This software introduces many new features that help to increase system uptime and improve laboratory workflow. Custom Panels: Up to six fully customizable panels are available for easy selection of frequently-run profiles. Quality Control and Maintenance Charts: QC performance and maintenance are monitored and tracked to facilitate regulatory compliance and ensure maximum system uptime. On-line Troubleshooting Guidance: On-line program helps to pinpoint and resolve fluidic difficulties quickly. Calibration Sequences: The calibration sequences have been optimized with the ability to interrupt the calibration to run a patient sample. The Rapidlab 1200 Version 2.0 software adds many new capabilities to meet the demands of today's laboratory. Contact Siemens.com.

QUICKER

RAPIDComm, from Siemens, is an innovative, next-generation blood gas data management and connectivity software solution. RAPIDComm's flexibility, real-time data collection and control of remote and local analyzers, provides easy and efficient management of blood gas data in a secured environment. RapidComm provides the ability to control multiple functions from a single, automatic interface, helping to streamline your administration and management of all your blood gas data. RapidComm is designed to fit the needs of the entire hospital, providing flexible QC management, remote monitoring and control of all blood gas systems and IT controlled passwords. The RAPIDComm software solution can be fully integrated into healthcare institutions through multiple and widely accepted communication interfaces. Contact siemens.com.

EXECUTIVE PROFILE

Covidien

Brent Boucher
Brent Boucher is the VP of Global Marketing for Covidien's Respiratory and Monitoring Solutions global business unit.

Describe your product(s) and its unique features.
The Respiratory and Monitoring Solutions business unit of Covidien, formerly Tyco Healthcare, is located in Boulder, Colorado and is part of a $5.7 billion segment of the company. Our systems are used to monitor, diagnose and treat respiratory disease and sleep disorders, and they provide life sustaining ventilation and oxygen support for patients. We have several different categories of products, namely Ventilation and Airway Management, Monitoring, and Homecare. The Ventilation and Airway Management products are crucial to Intensive Care Units (ICU) and their patients. Seriously ill patients in the ICU have ventilators do the work of breathing for them when they are unable to do so because of their medical condition. The market leading Puritan Bennett 840 is the flagship ventilator product for Covidien. The sophisticated and versatile device uses innovative software that allows the user to access a range of customizable options and accessories. This ventilator brings care to a variety of patients, from premature infants to adults. The Portable Ventilator Line from Puritan Bennett is designed for patients on long-term or permanent breathing support. Our Airway Management products include endotracheal tubes, made by Mallinckrodt; Shiley tracheostomy tubes; and other critical devices for establishing and maintaining an open airway to facilitate breathing including ventilation filters, CO_2 detectors and resuscitation bags. Covidien also provides healthcare professionals with a wide range of products for managing the patient's airway in anesthesia, intensive care and emergency medicine. Covidien's Pulse Oximetry/Capnography/Temperature Monitoring and Management products make up the Monitoring division of this business unit.

As the industry leader in pulse oximetry, Covidien supplies far more medical device original equipment manufacturers (OEMs) with their pulse oximetry solutions than any other company. Through the Nellcor brand, Covidien offers bedside and handheld pulse oximeters and the broadest selection of pulse oximetry sensors. Continual monitoring of oxygen in the bloodstream and body temperature during serious illness or surgery is critical to ensuring patient stability and safety. The pulse oximeter connects to a sensor that is attached to the patient, usually on the finger, toe, ear or forehead. Originally introduced by Nellcor to improve patient safety during anesthesia, pulse oximeters are now used in virtually every area of care to assess patients' respiratory status and to alert caregivers early in cases of respiratory distress. Nellcor pulse oximetry, featuring the innovative OxiMax monitoring technology, is also offered to customers through other manufacturers of patient monitoring systems. These systems rely on Nellcor's accuracy and reliability, and are compatible with Nellcor's adhesive and specialty sensor line.

Also part of Covidien's patient monitoring products are portable capnography monitors for tracking ventilatory status and a remote respiratory monitoring system to improve patient safety on the general care floor. Maintaining normal body temperature for surgery patients can make a critical difference in their recovery time and minimize post-surgical complications. To help with this goal, Covidien provides a highly effective convective air warming system that blows warmed air into soft, durable warming blankets placed on patients before, during and after surgery. Monitoring body temperature requires precision and responsiveness. Covidien's well-respected and comprehensive Mon-a-therm line of temperature probes and sensors aids clinicians in accurately monitoring temperature in infants through adults.

The third division of products is Homecare, which is comprised of Oxygen Therapy, Sleep Therapy and Diagnostic products. In the past "supplemental oxygen" meant carting around a bulky, heavy oxygen cylinder or being confined to a home oxygen system that runs on electricity and limits mobility. As a leader in oxygen therapy for over 20 years, Covidien offers these patients a far better option with its portable liquid oxygen systems. The portable units are so small and lightweight that patients can tote them via a belt pack or shoulder strap, leaving their hands free. The units refill in about a minute from a home reservoir, and allow long-term oxygen patients to be out and about for extended periods. Covidien's products help patients with obstructive sleep apnea or other respiratory ailments, such as chronic obstructive pulmonary disease, live full, productive lives at home, at the office and on the road. Building on the success of the Companion line, the latest HELiOS technology enables portables that are even smaller and lighter, yet provide a long-lasting oxygen supply. Covidien also produces oxygen conservers—devices that attach to the home reservoir to provide oxygen to the patient while at home. The leading systems for diagnosing sleep-breathing disorders are offered along with a full line of sleep therapy and portable oxygen therapy systems.

What sets your product apart from others in the field?
Our company enhances patient care by providing doctors, nurses and patients the products to improve patient outcomes at home and in the hospital. We look forward to bringing that same spirit of innovation to a next generation of respiratory and monitoring solutions, improving patient care and safety.

Discuss the educational services you offer for use of your product.
Our internal employee teams provide first-rate customer service and technical support complemented by a wide selection of clinical education resources. Free, accredited online continuing education courses for registered nurses and respiratory therapists are offered through our Center for Clinical Excellence website: ccexcellence.org.

Discuss the role of critical care providers in developing and upgrading your product.
Healthcare professionals are essential partners in our mission to develop medical solutions that improve patients' lives. We believe our integral partnership with doctors, nurses and other healthcare professionals is the key to delivering best-in-class products and solutions.

The Primary Ciliary Disorders: Underdiagnosed and Undertreated

Jane Braverman, PhD; Barbara Stewart, MD

"Primary ciliary dyskinesia is a prototypical example of a condition in which poorly functioning cilia contribute to the retaining of secretions and recurrent infections that, in turn, lead to bronchiectasis… Enhancing the removal of bronchial secretions in bronchiectasis has been shown to be useful… Traditional chest clapping or cupping has largely been replaced by the use of inflatable vests… applied to the chest."

Alan F. Baker, Bronchiectasis. N Engl J Med 2002; 346 (18): 1383-1393

Medicine is replete with examples of diseases hidden in plain sight. A broad array of inherited abnormalities of ciliary structure and function fall into this category. The primary ciliary dyskinesias (PCD), disorders which were previously little-known and poorly understood, are rapidly gaining recognition as an important cause of chronic, refractory childhood ear, nose, throat and respiratory infections and of progressively deteriorating pulmonary health.[1,44] Increasingly, unexplained bronchiectasis is now attributed to untreated PCD.[2,31,32]

In order to make a correct diagnosis, physicians must be aware of the disorder, maintain a reasonable level of suspicion for its occurrence, recognize its symptoms and have access to appropriate diagnostic tools. An explosion of research in molecular genetics, macromolecular analysis and imaging have expanded knowledge of the structure, biochemistry and physiology of both healthy and abnormal cilia. In addition, there is a body of longitudinal data to demonstrate the health consequences of ciliary dysfunction.[9,17,25,26,42] Awareness of the disorder has led alert physicians to consider PCD in the differential diagnosis of persistent, frequent sino-respiratory symptoms in infants and young children.[5,10,14,15,17] Sophisticated diagnostic tools, most notably tracheal biopsies evaluated with transmission electron microscopy and genetic tests, have permitted definitive diagnoses.[19,25,28,37] A definitive diagnosis includes evidence of specific ultrastructural defects and a compatible genetic phenotype.[13,43] Following the hugely successful "centers of excellence" model pioneered by cystic fibrosis (CF) physicians, PCD testing resource centers are growing in number.[4,5] Their experience suggests that PCD prevalence figures, currently estimated at one case per 15,000 individuals, may represent only the tip of an iceberg. Some authorities suspect that as many as one person in 1,500 may be affected.[5] Unsurprisingly, cases of PCD cluster around medical specialist centers that have the knowledge and facilities to diagnose the condition.

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History

In 1901, a German physician, A.Oeri, described the first case of advanced lung disease, or bronchiectasis, associated with the anatomical variation known as *sitis inversus*, in which the thoracic and abdominal organs are positioned in mirror image to the typical arrangement.[1,42] Three years later, Dr A.K Siewert reported on a patient who also exhibited chronic sinusitis. In 1935, as more cases were seen, Dr. A. Kartagener described a series of four patients who presented with male infertility in addition to the previously described triad, *sitis inversus*, severe, refractory sinusitis and bronchiectasis. Subsequently, this clinical pattern was recognized as a distinct disease entity, eponymously called "Kartagener's Syndrome" (KS). Although *sitis inversus* appeared to be rare, Kartagener's description raised awareness of the condition, resulting in the recognition of quite a number of new cases. Currently, KS is classified as a subtype of a spectrum of inherited disorders characterized by dysmotility (primary ciliary dyskinesia, or PCD) or immotility of the cilia (immotile cilia syndrome, or ICS). Interestingly, only 50% of persons with KS also have dysmotile cilia.[4]

Etiology and clinical manifestations

PCD is the most common variant of a constellation of recessive heritable disorders characterized by defects in the structure and function of all ciliated cells throughout the body. The assortment and severity of clinical manifestations vary greatly depending upon the nature of the abnormality.[4,6,20,25,28,37]

Respiratory complications associated with inadequate secretion clearance in upper and lower airways are the major source of pathology in this population.[23,25,37] Although other important mechanisms - alveolar clearance and cough - remain intact, stasis of airway secretions is manifested as chronic sinusitis, otitis media, nasal polyposis and recurrent respiratory infection. Over time, following a pattern similar to that seen in cystic fibrosis (CF), the accumulation of mucus in PCD lungs promotes bacterial colonization, setting in motion a vicious cycle of atelectasis, impaired gas exchange, increasingly antibiotic-resistant exacerbations, mucus plugging and ultimately irreversible lung damage.[26] Other clinical features may include male infertility, hearing loss, visual deficits, congenital heart disease, renal dysfunction and, less commonly, central nervous system involvement including hydrocephalus and joint pathology.[4,7,18,37]

The consequences of untreated PCD are cumulative and devastating.[29, 31, 32] Chronic illnesses, frequent hospitalizations, decreased quality of life, and progressive pulmonary decline are the norm. Individuals with PCD are frequently colonized with antibiotic-resistant organisms including those considered to be normal oral flora that have now colonized the airway because of inability to clear organisms from them effectively. Consequently, patients experience an array of febrile illnesses. A large proportion develops bronchiectasis. Increasingly, those with advanced disease are referred for lung transplantation.[25]

Importance of early diagnosis

"Early recognition and treatment of respiratory infection, as well as chest physical therapy and postural drainage, have done more than anything else to reduce the morbidity and mortality of immotile cilia syndrome." [42]

Recent evidence has shown that neonatal respiratory distress is a common clinical presentation of PCD.[5,10,14,15] Full-term neonates who develop respiratory distress or persistent hypoxemia or who have *sitis inversus* or an affected sibling should be screened for the condition.[14,15] Increasingly, PCD is diagnosed retrospectively in older children whose histories show transient respiratory distress during the newborn period and have subsequently developed persistent cough or chronic otitis media.[5,36,37] Numerous studies demonstrate a correlation between late diagnosis and poorer outcomes:

- In a longitudinal study of 24 PCD patients (12 adults and 12 children 7-18 years of age) with mild to moderate pulmonary impairment who adhered to standard protocol of twice daily physiotherapy and antibiotics as needed, lung function at baseline was significantly lower for those who started therapy as adults. (forced vital capacity FVC 70% vs 85% predicted; forced expiratory volume in one second (FEV_1) 59% vs 72% predicted). Bronchiectasis was found in 2/12 children and 6/12 adults. Most patients remained stable on the protocol. Results suggest that 1) lung damage begins early in life and will progress without treatment; 2) lung function can be maintained with aggressive physiotherapy and prompt treatment of infections and 3) early diagnosis and treatment have a strong impact on outcomes.[9]

- Several reports have shown extensive atlectasis and pneumonia in neonates diagnosed subsequently with PCD. Evidence suggests that ciliary dysfunction is critical for effective clearance of fetal lung fluid; infants with PCD fail to rapidly and fully transition to air breathing. Undiagnosed infants are at high risk for progressive pulmonary disability and possible respiratory failure. [4,10,14,15]

- In 47 children aged 1-15 with recurrent or chronic refractory respiratory infections undergoing nasal biopsies evaluated with transmission electro microscopy (TEM), 13 were diagnosed with PCD. Earlier diagnosis and treatment may have modified their lung damage.[17]

Mechanisms of disease

"Since patients with PCD are unable to clear secretions …by mucociliary transport, they are totally dependent on ancillary clearance mechanisms…"[35]

The importance of normally beating cilia to maintain healthy airways is well-recognized in both CF and PCD.[3,16,25,26] Although effective ciliary function is innately absent in PCD and is merely disabled by an excess burden of thick secretions in CF, both conditions result in serious chronic respiratory disease.[26,31,33] While each condition has unique features, the pathogenesis of respiratory tract disease – prevalence of lung infection, contributing pathogens, longitudinal declines in pulmonary function and development of bronchiectasis- is similar. However, there are also considerable differences between PCD and CF. Persons with PCD are more likely to spike fevers and be more frequently diagnosed with pneumonia than those with CF. In contrast to infants and toddlers with CF, those with PCD have a higher prevalence of high spiking fevers and draining ears.[5,43] Despite a delayed clinical course, PCD and CF patients have similar end-stage disease patterns and, as an alternative to death from respiratory failure, are faced with lung transplantation as a final option.[25,26]

A number of factors may explain differences in the natural history of PCD versus CF:

Variable cough efficacy

Cough is the default mechanism for airway clearance in patients with impaired MCC[3,6,24]

- In PCD airway surface liquid content is assumed to be normal, thus permitting more effective cough clearance.

- In CF, owing to a defect in chloride ion transport, the volume of airway surface liquid is markedly reduced. Secretions are correspondingly denser, making cough clearance more difficult.

Variable patterns of particle deposition and clearance

Lung health depends upon effective defense mechanisms to clear inhaled debris from the airways. [3, 16, 24] Larger particles are cleared from the upper airways by triggering coughing and sneezing. Smaller particles are entrapped in mucus lining the lower airways and are removed by the unidirectional "escalator" effect of the mucociliary clearance (MCC) system. Finally, alveolar macrophages and other scavenger cells ingest very small particles not captured by the MCC. The longer foreign substances including bacteria and viruses reside in the airways, the greater likelihood of inflammation and infection. Studies comparing particle clearance in CF and PCD versus healthy subjects shows that:

- Compared to healthy controls, both subjects with PCD and CF demonstrated significantly prolonged retention of radio-labeled particles but had different distribution patterns.[33]

  1) CF subjects showed an increased retention in smaller airways at 24 hours - consistent with typical patterns of CF lung pathology

  2) PCD subjects showed increased retention in larger airways at 24 hours -consistent with typical patterns of PCD lung pathology

  3) CF subjects showed clearance rate delay in smaller lung regions

  4) PCD patients showed clearance rate delay in larger airways

  5) An inverse relationship emerged between lung function and regional deposition of particles in CF and PCD subjects versus normal controls

- Compared with healthy non-smoking subjects, PCD subjects showed significant delay in clearance of experimentally deposited microparticles.[30]

  1) In healthy non-smoking subjects 49±9 % of particles were cleared with a mean half time of 3.0±1.6 hours.

  2) In PCD patients, particle clearance was retarded and prolonged; 42±12% of particles were cleared with a mean half time of 16.8±8.6 hours

Clinical Implications

MCC function and therefore particle clearance is impaired in both CF and PCD. Differences in regional particle deposition and clearance rates may explain differences in patterns of lung disease and progression.[9,30,33,38,39] In CF patients, whose secretions are very thick and tenacious, alveolar and cough clearance mechanisms are considerably impeded in the small, lower lung airways; prolonged regional particle retention may contribute to the predominately lower lobe pathology characteristic in CF.[24,26] In contrast, in PCD the physical qualities of airway secretions are normal, permitting alveolar and cough effects to move secretions from the tiniest passages even in the absence of ciliary movement.[3,26] The prolonged retention of particles in the larger, upper airways in PCD result in prolonged regional exposure to bacteria and viruses and may account for the greater prevalence of upper respiratory illness in pre-bronchiectasis patients and the correspondingly slower rate of lung deterioration.[30,33]

Treatment

"Unfortunately, no specific therapeutic modalities are available to correct the ciliary dysfunction [in PCD]... Consequently, treatment focuses on facilitating the clearance of retained mucus secretions from the respiratory tract."[10]

PCD treatment is closely modeled on CF care paradigms.[26] The therapeutic goals are to control symptoms, maintain lung health and prevent or delay the onset of bronchiectasis. Accordingly, care plans include prompt intervention for acute exacerbations with administration of antibiotics to suppress microbial load, management of underlying conditions, reduction of excessive inflammatory response and, most importantly, promotion of effective secretion clearance.[4,12,26,34,43] Aggressive daily airway clearance therapy (ACT) is the "cornerstone of treatment" for both CF and PCD, but the higher prevalence of early morbidity in PCD demands an even more rigorous treatment plan.[34,42,43] Because ACT benefits are diminished if treatment is deferred until the development of irreversible lung disease, therapy should be begun at the time of diagnosis.[15,42]

High-frequency chest compression therapy: Standard of Care

"High frequency chest compression (HFCC) …technology has proven to be the most effective way to remove mucus from the lungs of patients with CF and many other lung diseases."[41]

High-frequency chest compression (HFCC) therapy is recognized as a *standard of care* ACT for patients with ineffective MCC. Its importance as a treatment modality is based upon a significant body of peer-reviewed research and more than a decade of clinical experience.[8] Since its introduction in 1988, HFCC has gained rapid acceptance and widespread use. Currently, it is used by more than 70% of American CF patients.

HFCC is the most logical choice of therapy for patients with PCD and CF because it accelerates the rate of secretion clearance from both peripheral and central airways. By reducing exposure to excess mucus and to inhaled particulate matter, inflammation, infection and bacterial colonization are less likely to occur.

Several studies show increased rates of tracheal mucus clearance with HFCC:

- In 9 anesthetized dogs receiving HFCC at frequencies of 3 to 17 Hz, tracheal mucus clearance rate (TMCR) was determined by direct observation of the rate of displacement of a charcoal particle spot by means of a fiberoptic bronchoscope. Baseline TMCR during spontaneous breathing averaged 8.2 ±5.6 mm/min. The TMCR during 2 min of HFCC was increased at 5, 8, 11, 13, 15, and 17 Hz. The enhancement of clearance was most pronounced in the range of 11 to 15 Hz, reaching a peak value of 340% of control at 13 Hz.[21]

- A comparison of TMCR in anesthetized dogs during spontaneous breathing (SB) showed that high frequency chest compression (HFCC) enhances tracheal mucus clearance when compared with spontaneous breathing, *whereas high-frequency oscillation (HFO) at the mouth does not*. Rate of displacement of a charcoal marker in the lower trachea was observed by fiberoptic bronchoscope. Mean TMCR with HFCC was 240% of control ($p = < 0.001$) and 76% of control with HFO/AO (NS).[22]

- To investigate whether increases in TMCR in dogs during high frequency chest wall compression (HFCC) is due, in part, to the expiratory bias in peak flow rate (VE/VI greater than 1) that occurs during HFCC, TMCR in 8 anaesthetized, spontaneously breathing dogs was studied by comparing several randomized maneuvers designed to assess that effect. TMCR determined by direct bronchoscopic visualization of charcoal particle transport showed clearance rates during HFCC at 240% of rates obtained during spontaneous breathing ($p = < 0.001$), and that rates were influenced by expiratory flow-rate bias.[24]

HFCC promotes secretion clearance in peripheral as well as central airways:

- To investigate the effect of high frequency chest compression (HFCC) on clearance of secretions from peripheral lung regions, 5 anesthetized, spontaneously breathing dogs received 30 minute treatments of HFCC at 13 Hz & cuff pressures at 50-60 cm H_2O. Correlations between peripherial mucus clearance indices (PMCI) and tracheal mucus clearance rates (TMCR) in two outer peripheral regions located under the cuff were statistically significant (13.0 $\pm$ 2.6; p = < 0.05); lower-middle outer peripheral region (9.1 $\pm$ 3.0; p = < 0.05). Overall, HFCC enhanced both central and peripheral mucus clearance.[11]

Summary

The inherited dyskinetic ciliary disorders, including PCD, are an important cause of chronic respiratory illness and declining pulmonary health. In PCD, secretion retention is a direct cause of pulmonary deterioration. To prevent a growing burden of debilitating, costly respiratory illness, appropriate diagnostic screening and care plans are urgently needed. Consistent, effective removal of airway secretions is a critical component of every PCD treatment regimen. HFCC is an established *"standard of care"* therapy used widely for patients with absent or defective airway clearance mechanisms. It is the only ACT shown 1) to move secretions from smaller to progressively larger airways and; 2) to significantly increase tracheal mucus flow above rates achieved during spontaneous breathing. HFCC contributes to reduced morbidity, better survival and dramatically improved quality of life. PCD is a complex, multi-system disorder with serious complications arising from delayed clearance of particle-laden respiratory tract secretions. HFCC therapy provides a practical, effective, evidence-based intervention.

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Recommendations for Weaning from Mechanical Ventilation

Jeff Borrink

Prolonged mechanical ventilation is associated with significant morbidity and mortality. Therefore, patients should be weaned as soon as possible following the initiation of mechanical ventilation. However, there is uncertainty and disagreement about the best methods for conducting this process. In April 2005, an International Consensus Conference sponsored by five scientific societies was held in Budapest, Hungary to provide recommendations regarding the management of weaning patients from mechanical ventilation. This was the sixth such International Consensus Conference on Intensive Care Medicine. At this conference, an 11-member international jury answered several pre-defined questions in regards to weaning from mechanical ventilation and made some recommendations that were recently published in an article by J-M Boles et al, in the European Respiratory Journal.[1] The following is a synopsis of some key recommendations.

In their consensus statement, the jury stated that patients requiring mechanical ventilation should be categorized into three groups based on the difficulty and duration of the weaning process. The three groups include simple weaning, difficult weaning, and prolonged weaning. The simple weaning group, which represents 69% of weaning patients, includes patients who successfully pass the initial spontaneous breathing trial (SBT) and are successfully extubated on the first attempt. The difficult weaning group includes patients who require up to 3 SBTs, or as long as 7 days from the first SBT to achieve successful weaning. The prolonged weaning group includes patients who require > 3 SBTs or > 7 days from the first SBT to achieve successful weaning.[1]

Weaning should be considered as early as possible in the hospital course. A thorough and systematic search for potentially reversible pathologies should be done for all patients who do not fulfill simple weaning as previously defined.[1] Reversible pathology should be repeatedly and aggressively sought in all patients that fall into groups 2 and 3. Once the underlying pathology has been improved or reversed, the patients should be assessed for readiness to wean. An initial assessment of the likelihood of a successful SBT is appropriate in order to avoid trials in patients with a high probability of failure.[1] Criteria for readiness to begin weaning should be systematically evaluated each day to allow prompt initiation of weaning as soon as the patient is ready.[2] This will shorten the weaning process and minimize time on mechanical ventilation.[3] The initial assessment of the readiness for discontinuation of mechanical ventilation support often involves calculation of the rapid shallow breathing index (RSBI). In general, patients should be considered for an RSBI calculation and subsequent SBT earlier rather than later, since physicians frequently underestimate the ability of patients to be successfully weaned.[1] A value of <100-105 predicts a successful SBT, however if other clinical criteria are met for readiness to wean, the RSBI should not preclude an SBT attempt. For many patients, discontinuation of sedation is a critical step that can be achieved by either daily interruption of sedation or continuous titration of sedation to a level that allows the patient to be adequately responsive.[4]

An SBT is the major diagnostic test to determine whether patients can be successfully extubated.[1] An SBT should be considered as soon as possible once the patient meets criteria from the initial assessment, as the majority of patients can be successfully weaned on the first attempt.[1] Multiple studies have examined the methodology for performing an SBT. There appears to be no difference in either the percentage of patients who pass the SBT or the percentage of patients successfully extubated when a T-tube trial is compared with the use of low levels of pressure support (PS), such as 7 cmH_2O[5] or 8 cmH_2O[6] in adults or 10 cmH_2O[7] in pediatric patients, or the use of CPAP.[8]

The initial SBT should last 30 min and consists of either T-tube breathing or low levels of pressure support (PS) (5–8 cmH_2O in adults; 10 cmH_2O in pediatric patients), with or without 5 cmH_2O positive end expiratory pressure (PEEP). CPAP may be effective in preventing hypoxic respiratory failure in patients after major surgery; otherwise there is no clear advantage over other modes of mechanical ventilation used during weaning.

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Synchronized intermittent mandatory ventilation (SIMV) should be avoided as a weaning modality. Weaning protocols are most valuable in hospitals in which physicians otherwise do not adhere to standardized weaning guidelines. PS or assist–control (AC) ventilation modes should be favored in patients failing an initial trial/trials.[1]

Noninvasive ventilation techniques should be considered in selected patients to shorten the duration of intubation but should not be routinely used as a tool for extubation failure. There is promise that for some subgroups (hypercapnic respiratory insufficiency, especially in COPD patients) NIV may be helpful in expediting the weaning process.[1]

Servo-controlled ventilation may be an additional tool for reducing time on mechanical ventilation. Rapid adaptation of the ventilatory support to the changing situations of a patient is one of the major factors determining the length of the weaning process. Automatic ventilatory modes provide a tool to achieve optimal ventilatory support and an individual level of PS with the aim of rapid extubation. One such mode, adaptive support ventilation (ASV) has been integrated into conventional intensive care ventilators and is available on the market.[1] The numbers of studies are too limited to make firm recommendations just yet, therefore, further studies comparing ASV with other weaning techniques are necessary to gain insights of the utility of this mode.[1]

Future studies should also define the minimal criteria required for assessment of readiness for weaning (in order to allow earlier weaning), the need for a screening test such as the RSBI prior to the SBT, identification of patients who pass an SBT but fail extubation, the role of CPAP/PEEP in the COPD patient undergoing SBT, the required duration of the SBT in patients who fail the initial trial, and the specific aspects of weaning protocols which improve weaning outcome. When initial attempts at spontaneous breathing fail to achieve the goal of liberation from mechanical ventilation, clinicians must choose appropriate mode(s) of ventilatory support which: 1) maintain a favorable balance between respiratory system capacity and load; 2) attempt to avoid diaphragm muscle atrophy; and 3) aid in the weaning process.[1]

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Risk Factors for Post-Traumatic Stress Disorder Symptoms Following Critical Illness Requiring Mechanical Ventilation: A Prospective Cohort Study

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Abstract

Background: Posttraumatic stress disorder (PTSD) has been identified in a significant portion of intensive care unit (ICU) survivors. We sought to identify factors associated with PTSD symptoms in patients following critical illness requiring mechanical ventilation.

Methods: Forty-three patients who were mechanically ventilated in the medical and coronary ICUs of a university-based medical center were prospectively followed during their ICU admission for delirium with the Confusion Assessment Method for the ICU (CAM-ICU). Additionally, demographic data were obtained and severity of illness was measured with the APACHE II score. Six months after discharge, patients were screened for PTSD symptoms using the Post-Traumatic Stress Syndrome 10-Questions Inventory (PTSS-10). Multiple linear regression was used to assess the association of potential risk factors with PTSS-10 scores.

Results: At follow-up, 6 (14%) patients had high levels of PTSD symptoms. On multivariable analysis, women had higher PTSS-10 scores than men by 7.36 points (95% CI, 1.62-13.11; p=.02). Also, high levels of PTSD symptoms were less likely to occur in older patients, with symptoms declining after age 50 (p=0.04). Finally, though causation cannot be assumed, the total dose of lorazepam received during the ICU stay was associated with PTSD symptoms; for every 10 mg increase in cumulative lorazepam dose, PTSS-10 score increased by 0.39 (95% CI, 0.17 to 0.61; p=.04). No significant relationship was noted between severity of illness and PTSD symptoms or duration of delirium and PTSD symptoms.

Conclusions: High levels of PTSD symptoms occurred in 14% of patients 6 months following critical illness necessitating mechanical ventilation, and these symptoms were most likely to occur in female patients and those receiving high doses of lorazepam. High levels of PTSD symptoms were less likely to occur in older patients.

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Introduction

The life-sustaining therapies employed in the intensive care unit (ICU) commonly result in pain and anxiety as reported by survivors of critical illness.[1,2] In addition, the acute illnesses that threaten each patient's life create formidable stress. These experiences may result in long-term morbidity in survivors of critical illness, including depression, anxiety, and other psychological diseases.[3] One such psychological outcome, posttraumatic stress disorder (PTSD), has been identified in a significant portion of ICU survivors.[4] Early identification of patients who are at high risk for the development of PTSD after critical illness may facilitate the implementation of strategies focused on preventing this untoward outcome.

The current literature offers little in the way of identification of patients at high-risk for PTSD after critical illness. While female gender has long been recognized as a risk factor for the development of PTSD,[5,6] the significance of gender on the development of PTSD after critical illness remains unclear. One recent study determined that ICU patients subjected to a daily interruption of sedatives developed fewer symptoms of PTSD.[7] Also, recent work has shown that ICU patients with delusional memories of their ICU stay are more likely to develop PTSD than those with factual memories.[8] Critical illness is frequently complicated by delirium,[9] and delusions are a common

Table 1. Baseline characteristics and ICU outcomes for patients evaluated at 6 months and those not tested

Characteristics	6-month Follow-up (n=43)	Not Tested (n=136)	*p* value*
Age, median years [IQR]	52 [39-65]	55 [42-68]	0.39
Female, % (n/total)	53 (23/43)	49 (66/136)	0.60
Black, % (n/total)	16 (7/43)	24 (33/136)	0.40
Charlson Comorbidity Index, median [IQR]	3 [2-5]	3 [1-5]	0.34
APACHE II, median [IQR]	25 [20-31]	25 [18-31]	0.63
ICU admission diagnosis,† % (n/total)			
Sepsis and/or ARDS	42 (18/43)	49 (66/136)	0.49
Pneumonia	26 (11/43)	15 (21/136)	0.17
MI/CHF	9 (4/43)	9 (12/136)	1.00
Hepatic or renal failure	12 (5/43)	1 (1/136)	0.003
COPD	2 (1/43)	10 (14/136)	0.12
Gastrointestinal bleeding	2 (1/43)	10 (14/136)	0.12
Malignancy	5 (2/43)	2 (3/136)	0.59
Drug overdose	5 (2/43)	7 (9/136)	1.00
Other	21 (9/43)	34 (46/136)	0.13
ICU length of stay, median days [IQR]	10 [5-13]	7 [5-10]	0.08
Days on mechanical ventilation, median [IQR]	5 [3-12]	6 [3-9]	0.61
Duration of coma, median days [IQR]	1 [0-3]	1 [0-2]	0.16
Duration of delirium, median days [IQR]	2 [1-3]	2 [1-3]	0.39

APACHE II = Acute Physiology and Chronic Health Evaluation II; ICU = intensive care unit; ARDS = acute respiratory distress syndrome; MI = myocardial infarction; CHF = congestive heart failure; COPD = chronic obstructive pulmonary disease.

*p values were obtained using Wilcoxon rank sum tests for all variables except female and black/white, for which Fisher's exact tests were used.

†Primary and secondary admission diagnoses are included, resulting in some patients being listed twice—e.g., as having both sepsis and COPD.

component of delirium suggesting delirium may be associated with the development of PTSD. However, no previous studies of PTSD after critical illness have incorporated formal delirium evaluations. Therefore, this pilot investigation was conducted to identify factors associated with the development of PTSD symptoms in patients after critical illness. Specifically, we hypothesized that ICU delirium is a risk factor for the development of PTSD symptoms following critical illness and mechanical ventilation.

Materials and Methods

Subjects: All patients requiring mechanical ventilation admitted to the medical and coronary care ICUs of the 631-bed Vanderbilt University Medical Center between February 21, 2001, and May 3, 2001 were prospectively evaluated for enrollment. Those with neurologic disease impairing cognitive function (eg, stroke, Parkinson's disease, etc) or mental retardation were excluded, as were non-English speakers and those with sensory deficits limiting their ability to communicate with examiners. Although no history of PTSD was identified at enrollment, it is possible that some study patients had pre-existing PTSD that was not reported as patients were not prospectively assessed for symptoms of PTSD prior to enrollment due to the non-elective nature of their ICU admissions. The study was approved by the Vanderbilt University Institutional Review Board, and informed consent was obtained from the patients or their surrogates before study enrollment. Consent was also obtained from all patients at the 6-month follow-up visit. While no outcomes data from this manuscript have been previously reported, other data from this cohort have been published.[9-12]

Procedures: Baseline data included demographics, ICU admission diagnoses, and data needed to calculate the Acute Physiology and Chronic Health Evaluation II (APACHE II)[13] score and the Charlson Comorbidity Index, as calculated per Deyo et al.[14] While in the ICU, patients were evaluated daily for delirium with the Confusion Assessment Method for the ICU

Table 2. Factors associated with PTSD symptoms at 6-month follow-up

Factor	Univariate Analysis*		Multivariable Analysis†	
	rho	*p* value	B (95% CI)	*p* value
Age (years)	-0.297	.05	Nonlinear effect‡	.04
APACHE II	0.039	.80	0.02 (-0.32, 0.37)	.90
Duration of delirium (days)	0.030	.84	0.91 (-0.82, 2.63)	.31
Total lorazepam dose (10 mg)	0.300	.05	0.39 (0.17, 0.61)	.001
Gender, median PTSS-10 score [IQR]		.06§	7.36 (1.62, 13.11)	.02
Female	22 [16-35]			
Male	17 [12-27]			

CI = confidence interval; APACHE II = Acute Physiology and Chronic Health Evaluation II; IQR = interquartile range.

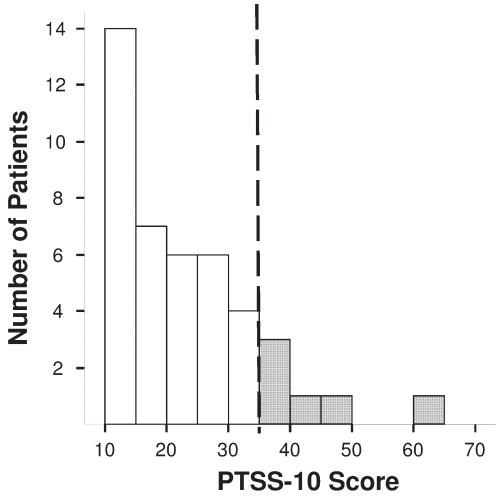
*Spearman's correlation coefficients (*rho*) unless otherwise noted

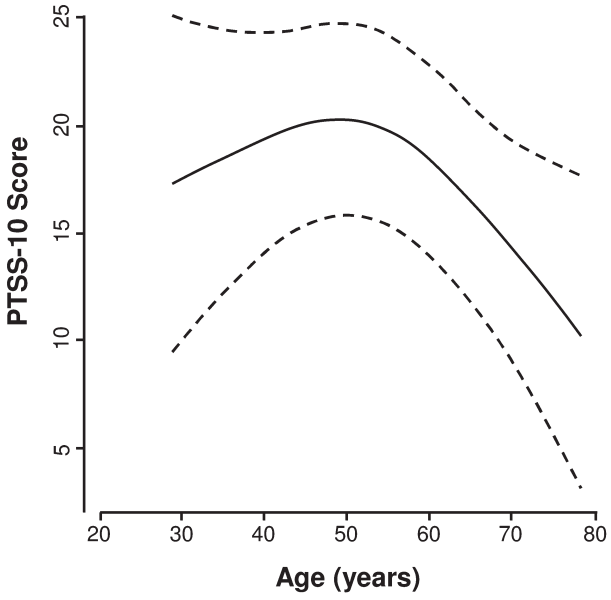
†Multiple linear regression with B representing regression coefficients

‡See Figure 2

§Wilcoxon rank sum tests were used

(CAMICU).[9,15] The CAM-ICU had a high sensitivity (93%-100%), specificity (89%-100%), and interrater reliability (Î, 0.96; 95% CI, 0.92-0.99) when evaluated against a reference standard rater in 2 cohorts of medical ICU patients. Each dose of sedative (lorazepam, midazolam, and propofol) and analgesic (fentanyl and morphine) medication received was recorded daily throughout the ICU stay. Follow-up testing was conducted 6 months after hospital discharge; this interval was arbitrarily defined a priori. Patients were screened for PTSD symptoms using the modified Post-Traumatic Stress Syndrome 10-Questions Inventory (PTSS-10).[16] This 2-part questionnaire assesses for memories of traumatic experiences during the ICU stay: nightmares, panic, pain, and suffocation (Part A). It then measures the intensity of 10 PTSD symptoms presently experienced (i.e., at or around the time of evaluation) by the patient (Part B), including sleep disturbance, nightmares, depression, hyperalertness, emotional numbing, irritability, labile mood, guilt, avoidance of activities prompting recall of the traumatizing event, and muscular tension; each symptom is rated from 1 (never) to 7 (always). Total scores >35 on Part B predict the diagnosis of PTSD by the criteria outlined in the Diagnostic and Statistical Manual of Mental Disorders, Third Edition (DSM-III).[16] The PTSS-10 has a high sensitivity (77%) and specificity (97.5%) and has been validated for use in ICU patients, with a reliability coefficient (Crohnbach's alpha) of 0.914 in this patient population.[4,16] Quality of life was assessed using the Short Form Health Survey-12 (SF-12),[17] and a





comprehensive neuropsychological battery was performed.[11] The PTSS-10, SF-12, and neuropsychological battery were conducted in person by a neuropsychologist (SMG) or a clinical psychologist (JCJ).

Terminology: Although a PTSS-10 score >35 predicts the diagnosis of PTSD,[16] this screening instrument cannot make a formal diagnosis of PTSD. As formal psychiatric evaluations were not done, results are reported in terms of PTSD symptoms rather than a diagnosis of PTSD.

Statistical Analysis: Baseline characteristics are presented using median and interquartile range (IQR) for continuous variables and proportions for categorical variables. Patients evaluated at 6-month follow-up were compared with patients not tested at 6 months using Wilcoxon rank sum tests for continuous variables and Fisher's Exact tests for categorical variables. Spearman's rank correlations were employed to evaluate the correlations between PTSS-10 score and duration of delirium (defined as the total days of delirium measured in the ICU), age in years, APACHE II score, cumulative dose of sedative drug (defined as the total amount of drug received during the ICU stay separately for lorazepam, midazolam, morphine, fentanyl, and propofol), total days in the ICU, total days of mechanical ventilation, the presence of memories of traumatic ICU experiences (PTSS-10, Part A), quality of life as measured by SF-12 scores, and composite neuropsychological test scores. A Wilcoxon rank sum test was used to compare PTSS-10 scores among men and women.

To assess the independent association of each factor with PTSD symptoms, multiple linear regression was employed with PTSS-10 score as the outcome variable. Although a threshold value of 35 on the PTSS-10 has been recommended in order to maximize sensitivity and specificity, across the spectrum of possible scores (10-70) higher PTSS-10 scores are associated with a higher likelihood of diagnosing PTSD[16] making the PTSS-10 score a suitable continuous outcome variable. A priori, we chose to include age in years,[18] gender,[5] APACHE II score, sedative exposure,[7] and days of delirium[8] in the regression model because we suspected these factors to be associated with PTSD based on existing literature and clinical suspicion. To assess the association between sedative exposure and PTSD symptoms, cumulative lorazepam dose was chosen based on the Spearman's correlation analysis; cumulative lorazepam dose was most correlated with PTSS-10 scores compared with cumulative fentanyl and propofol doses. Total days in the ICU and days of mechanical ventilation were not included in the model because of their possible correlation with cumulative sedative drug dose. No variables were removed from the model. Nonlinear associations between each continuous variable and PTSS-10 score were assessed by including nonlinear cubic splines in the regression model. Nonlinearity of the effect of age was included in the regression model because significant nonlinearity was detected in its association with the outcome. In order to correct for possible overfitting of the regression model, penalized maximum likelihood estimation was used to allow shrinkage for nonlinear effect of age. Residuals of the multiple linear regression model were examined by graphically plotting residuals against predicted values, plotting normal Q-Q plots, and using the Shapiro-Wilk test. Additionally, bootstrap model validation was used to assess the robustness of the regression model for its predictability for future data. R-software version 2.11,[19] SAS version 9.0 (SAS Institutes; Cary, NC), and SPSS version 14 (SPSS Inc.; Chicago, IL) were used for data analysis, and a two-sided 5% significance level was used for all statistical inferences.

Results

Of 555 mechanically ventilated ICU patients admitted during the study period, 275 (49.5%) patients were enrolled in the study. A total of 280 patients were excluded: 86 had stroke or other primary neurologic disorder, 13 were deaf or unable to understand English, 44 died prior enrollment, 69 were extubated prior to enrollment, 27 had been previously enrolled, and consent was not obtained for 41 patients.[12] After enrollment, 96 patients died prior to hospital discharge. Of the remaining 179 patients, 23 (13%) patients died within 6 months of discharge, 27 (15%) were too ill to participate in follow-up evaluation or declined further participation, and 86 (48%) patients were lost to follow-up. Therefore, a total of 43 (24%) patients were evaluated 6 months after hospital discharge (Table 1). There were no significant differences in baseline demographics or outcome measures between the patients tested at 6-month follow-up and those not tested (e.g., due to death or illness, or lost to follow-up), except that hepatic and renal failure were more common in those tested (p=.003).

At 6-month follow-up, 6 (14%) of 43 patients scored >35 on the PTSS-10 (Figure 1), i.e., reported high levels of symptoms consistent with PTSD. These patients reported frequent feelings of guilt (83%), mood swings (67%), and sleep disturbances (67%) while muscular tension was the symptom experienced least often, with 16% of patients reporting frequent muscular tension. The majority of patients with PTSD symptoms at 6 months reported memories of panic (67%) and suffocation (50%) during the ICU stay while memories of nightmares (20%) and severe pain (20%) were less common. Spearman's rank correlation coefficients (rho) between PTSS-10 score and cumulative doses of sedative drugs were 0.30 for lorazepam (p=.05), -0.22 for midazolam (p=.16), 0.09 for fentanyl (p=.56), 0.07 for morphine (p=.66), and -0.16 for propofol (p=.30). Thus, cumulative lorazepam dose is included in the multivariable model.

Results of the multivariable analysis are shown in Table 2. Women had higher PTSS- 10 scores than men by 7.36 points (95% confidence interval [CI], 1.62-13.11; p=.02). PTSD symptoms were less likely to occur in older patients, with symptoms declining after age 50 (p=.04) (Figure 2). The total dose of lorazepam received during the ICU stay was associated with PTSD symptoms; for every 10 mg increase in lorazepam dose, PTSS-10 score increased by 0.39 (95% CI, 0.17 to 0.61; p=.04). The bootstrap model validation showed that optimism of the model indicating degree of overfitting is 2.3%, suggesting an excellent robustness of the regression model.

No significant correlation between PTSD symptoms and duration of delirium or APACHE II scores was demonstrated (Table 2). Additionally, PTSD symptoms were not significantly correlated with duration of mechanical ventilation (Spearman's rho, 0.034; p=.83) or with duration of ICU stay (Spearman's rho, 0.10; p=.51). Thus, the observed association between cumulative lorazepam dose and PTSD symptoms does not seem to be confounded by duration of ICU stay nor mechanical ventilation. As expected, the presence of memories of traumatic ICU experiences (PTSS-10, Part A) was positively correlated with PTSD symptoms (PTSS-10, Part B) (Spearman's rho, 0.366; p=.02). Additionally, there was a significant inverse correlation between PTSD symptoms and quality of life as measured by SF-12 scores (Spearman's rho, -0.565; p<0.0001). There was no correlation noted between PTSD symptoms and composite neuropsychological test scores (Spearman's rho, -0.079; p=.63).

Discussion

In this investigation, high levels of PTSD symptoms after critical illness requiring mechanical ventilation were most likely to occur in female patients and in patients treated with high doses of lorazepam, while PTSD symptoms were less likely to occur in older patients. Understanding these risk factors may facilitate preventive strategies and direct screening for symptoms of PTSD after critical illness. In this study, 14% of patients evaluated 6 months after discharge reported high levels of symptoms consistent with PTSD. This coincides with the existing literature which reports a prevalence of 10%-30%.[4,7,16,18,20-25] Despite occurring frequently, PTSD goes unrecognized in many patients. The current study confirms previous work showing that high levels of PTSD symptoms are associated with impaired quality of life,[4] underscoring the importance of diagnosing and treating this disorder in survivors of critical illness.

In this study, women were significantly more likely than men to have high levels of PTSD symptoms after critical illness. The association between PTSD and female gender has been reported previously,[5] but few studies have evaluated the significance of gender on the development of PTSD after critical illness. Several studies have demonstrated that women are more vulnerable to PTSD, even after controlling for differences in the type of trauma,[5,6] and a higher incidence of preexisting anxiety and/or depression disorders is postulated to play some role in the difference in PTSD rates between the sexes.[6]

This study reveals a significant relationship between age and PTSD symptoms, with older patients being less likely to experience high levels of PTSD symptoms after critical illness. A nonlinear relationship between age and PTSD symptoms was observed, but caution is appropriate in interpreting this finding due to the small number of younger patients studied and the results of previous research. For example, Scragg et al evaluated 80 ICU patients for symptoms of PTSD and reported that scores on the screening instrument were inversely correlated with age (p=.05).[18] Rattray et al similarly found that symptoms of anxiety (p=.04) and avoidance (p=.01) were inversely correlated with age 12 months after discharge in 80 ICU survivors.[23] In the current study, older patients were significantly less likely to have high levels of PTSD symptoms than middle-aged patients. Several possible explanations for this relationship exist. Although each patient studied was mechanically ventilated, older patients are less likely to receive aggressive interventions that may predispose to the development of PTSD.[26] Additionally, older patients may be less likely to view critical illness as a traumatic event since they may have multiple comorbidities and a history of hospitalization.

We hypothesized that patients who experienced longer periods of delirium would be more likely to develop high levels of PTSD symptoms after critical illness, but the data do not support this hypothesis. Jones et al has demonstrated that the recall of delusions rather than factual memories of the ICU experience is associated with the development of PTSD symptoms.[8] Their study assessed 45 patients after ICU discharge and revealed that patients with delusional memories and no recall of factual events in the ICU were more likely to develop PTSD symptoms than those patients with factual memories (p<.0001). These data[8] suggest that periods of delirium, with associated delusions, may predispose to PTSD while periods of alertness, which allow for the consolidation of factual memories, may protect patients from developing PTSD-related symptoms after discharge. However, the association between days of delirium and PTSD symptoms in this study was not statistically significant (p=.31).

Cumulative lorazepam dose correlated with PTSD symptoms. While Nelson et al studied 24 ARDS survivors and noted that days of sedation correlated with symptoms of both PTSD (p=.006) and depression (p=.007),[27] the current investigation is the first to report an association between sedative dose and PTSD symptoms. However, it cannot be concluded from these analyses that lorazepam causes PTSD. The possibility exists that ICU patients who demonstrate symptoms of anxiety during their ICU stay are likely to receive higher sedative doses than those patients who are not anxious. Therefore, high lorazepam doses may identify those ICU patients with acute stress disorder, a known risk factor for PTSD.[28]

While the administration of lorazepam may lead to more PTSD symptoms or, alternatively, may identify anxious ICU patients, the daily interruption of sedatives may facilitate periods of alertness and reduce the risk of PTSD. Kress et al. evaluated 32 patients who were randomized to the daily interruption of sedatives or standard sedation to determine the long-term psychological effects of this intervention.[7] The 13 patients who had been treated with daily interruption of sedation had better Impact of Events scores (11.2 vs. 27.3, p=.02) and a lower incidence of PTSD (0% vs. 32%, p=.06). Further study of the effect of the daily interruption of sedation on the development of PTSD is needed.

Limitations of the current study warrant comment. Because the PTSS-10 does not make a formal diagnosis of PTSD, the results of this study may not be generalizable to the clinical syndrome of PTSD. Also, the PTSS-10 does not assess for delusional

memories. Data on the frequency of delusional memories, such as that provided by the ICUM tool,[29] would have allowed for more in-depth analysis regarding the relationship between delusional memories, delirium, and PTSD symptoms. There were a significant number of patients lost to follow-up. Analysis suggests that baseline and outcome characteristics were similar between those patients lost to follow-up and those evaluated at 6 months (Table 1), but this does not rule out the possibility of selection bias. In fact, "avoidance of activities prompting recall of traumatizing events" is a symptom of PTSD, and patients experiencing this symptom may have been less likely to return for follow-up testing. Thus, this study may underestimate the prevalence of PTSD after critical illness. Also, the findings regarding risk factors may differ if all survivors were evaluated. It was not systematically determined whether any patient sought psychiatric care prior to 6-month follow-up, and follow-up was limited to a single visit. Therefore, it is possible that some patients experienced PTSD symptoms prior to follow-up, but psychiatric treatment resulted in the resolution of such symptoms prior to testing at 6 months. Also, no evidence exists to define the ideal follow-up interval after which to screen for PTSD symptoms. Therefore, it is possible that screening after a shorter interval would have identified a higher number of patients with PTSD symptoms. Because of the non-elective nature of critical illness, it could not be prospectively confirmed that patients did not have PTSD prior to ICU admission. This diagnosis was not reported by family members and was not recorded in the medical record for the patients in this study. Finally, no data was collected regarding corticosteroid and beta blocker administration, two possible confounders.[30,31] This planned pilot investigation was limited by a small sample size, and a larger study to confirm these findings is warranted.

Conclusion

In conclusion, this study shows that high levels of PTSD symptoms occurred in 1 out of every 7 patients 6 months following critical illness and mechanical ventilation. High levels of PTSD symptoms were most likely to occur in females and less likely to occur in older patients. Additionally, lorazepam dose in the ICU was associated with PTSD symptoms at follow-up, though causation cannot be assumed. A significant minority of patients who survive critical illness will develop symptoms of PTSD; screening for these symptoms and warning all patients about the possibility of experiencing such symptoms is prudent. Knowledge of the risk factors demonstrated in this study may facilitate identification of PTSD after critical illness. However, it is unclear what component(s) of ICU experience (eg, the critical illness itself or treatments rendered, etc) may contribute to the development of PTSD. The current data cannot help to answer this question, and this is an important area to be addressed by future studies. Also, additional studies are needed before firm conclusions can be made regarding the relationship between ICU delirium and the development of PTSD after critical illness.

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Adaptive Pressure Control Ventilation: One Size Fits All?

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As advances are made in the science of mechanical ventilation, clinicians are faced with a plethora of mode choices when initiating mechanical ventilation of a patient. There have always been arguments for and against the different modes that are available. One certain fact is that a patient's lung condition is dynamic and therefore parameters of ventilation may require modification in order to keep the lungs safe and provide comfort when appropriate. In a recent article by Branson and Chatburn,[1] volume control pressure control and adaptive pressure control were reviewed while visiting the question, "Should Adaptive Pressure Control Modes Be Utilized for Virtually All Patients Receiving Mechanical Ventilation?" With respect to Chatburn's classification of ventilation modes,[2] volume control, pressure control, and adaptive pressure control are all breath types that can be delivered through continuous mandatory ventilation (CMV), intermittent mandatory ventilation (IMV), and continuous spontaneous ventilation (CSV).

Volume controlled breaths are described as breaths with a set point that limits flow or volume. During volume control, peak inspiratory pressure is varied dependent on lung mechanics and patient interaction with the breath.

Pressure control delivered breaths use a constant pressure as a set point. The flow rate is variable in order to meet the pressure set point. Tidal volume and flow varies with changes in lung mechanics and patient effort.

Adaptive pressure control breaths use some advantages of both previous breath types. This breath is delivered using a pressure set point during the inspiratory phase while "adapting" pressure between breaths to maintain a target tidal volume. If the breath cannot be delivered within the specified pressure limits an alarm will be activated. As such, the targeted tidal volume may not be reached if, for example, the patient's respiratory system compliance is too low and the pressure limit is set too low. It is important to realize that tidal volume may be higher or lower than expected on a breath by breath basis as the ventilator needs time to adjust/adapt pressure over the course of several breaths to maintain the target Vt.

As pointed out by Branson and Chatburn, no randomized controlled trials using large numbers of patients have been done to study adaptive pressure control. There have been several smaller trials that have suggested some advantages of adaptive pressure control. Piotrowski et al[3] found that adaptive pressure control ventilation compared to volume control, had a lower incidence of intraventricular hemorrhage grade II and higher in neonates and that the duration of ventilation was shorter along with a lower incidence of hypotension in infants who weighed less than 1kg. Alvarez et al[4] found a lower peak airway pressure as well as a slight improvement in carbon dioxide elimination using adaptive pressure control as compared to volume control ventilation. Peak pressures were also lower compared to volume control as noted by Guldager et al.[5] Kocis et al[6] also note the lower peak airway pressures using adaptive pressure control compared to volume control in infants post congenital heart disease surgeries. Considering all of the above studies in support of using adaptive pressure control ventilation, it is shown that peak airway pressures are consistently lower than with volume control ventilation. These studies were unable, however, to show any reduction in the duration of mechanical ventilation, reduction of complications, improvement in patient-ventilator asynchrony, or improvements in rate of survival.

There are several potential advantages to using adaptive pressure control ventilation although those advantages could be seen as a disadvantage in some perspectives. A targeted set tidal volume is achieved using the lowest possible peak airway pressures. Pressures are titrated on a breath to breath basis to the target tidal volume without clinician intervention. Some clinicians note that the "peak" pressure is lower than in volume control mode, but this is misleading as the lower peak pressure to achieve the same tidal volume is due to the resistive effects of a fixed flow pattern vs. a variable flow pattern. The plateau pressure will be the same if the tidal volume and PEEP targets

The authors are with Hamilton Medical, which provided this article.

are the same regardless of mode (assuming there's no differences in potential autopeep).

Indeed, as the flow is variable, patients can generate whatever tidal volume they want if they're capable, so the volume target is really a minimum tidal volume guarantee. Therefore, the inspired tidal volume may be much higher than the targeted tidal volume if the inspiratory effort generated by an active patient is great. Some clinicians may consider this harmful. (This is subject to much debate.) Another potential advantage to the variable tidal volume that could be produced with adaptive pressure control ventilation is a possible increase in surfactant production, which can improve lung mechanics and gas exchange as evidenced in studies done by Arnold et al.[7,8] (However, as a counterpoint, this assumes that the Vt can vary during adaptive pressure control to mimic the normal variation in spontaneous breathing pattern, which is the opposite of what the adaptive pressure algorithm is trying to do.)

When considering better patient-ventilator synchrony, proponents of adaptive pressure control point out that both tidal volume and flow can vary to meet patient demand while guaranteeing a minimum tidal volume. Pressure control does not guarantee a tidal volume minimum. During volume control, both tidal volume and flowrate are fixed values and do not change with patient demand. For this reason, if patient demand is increased, work of breathing (WOB) may also be increased. The disadvantage with adaptive control ventilation lies within human decision making about where to set the target tidal volume. If the patient's demand is not met because of an inappropriate target tidal volume setting or inappropriate pressure limit setting, then better patient-ventilator synchrony cannot be guaranteed. Ventilator-induced lung injury could be caused or exacerbated if tidal volumes are not reduced (indexed to ideal body weight) in ALI/ARDS.

Less clinician intervention and automated weaning are also identified by Branson and Chatburn as potential advantages of adaptive pressure control ventilation. Studies have suggested that there are fewer alarms and interventions needed from staff when using adaptive pressure control ventilation as compared to conventional modes. As a patient is able to maintain the target tidal volume or exceeds it, the pressure is decreased during adaptive pressure control, which helps to automate the weaning process. When considering weaning, the process can only be automated when the work of breathing is transferred appropriately from the ventilator to the patient as the patient's ability to breathe spontaneously improves. Also, it is important to point out that adaptive pressure control does not distinguish between improving lung mechanics and an increase in demand and drive due to such things as anxiety, fever or other factors that may cause that increase. In that situation the ventilator could decrease support to the patient and exacerbate the condition.

Branson and Chatburn state the only clear advantages of adaptive pressure control seem to be "more stable gas exchange than conventional pressure-controlled ventilation, better patient-ventilator synchrony than conventional VCV, and probably less human time spent at the bedside making sure the ventilator is meeting the patient's needs."[1] Branson and Chatburn also state, "Modes that use this strategy are a step above tactical control (ie, that require the operator to select and adjust all ventilator output set points), they are still a step below intelligent control (ie, those that allow the ventilator to mimic human decision making)."[1]

Adaptive support ventilation (ASV) is an intelligent control ventilation system. It delivers breaths in the same fashion that adaptive pressure control delivers breaths. However, human decision is not required for setting the target tidal volume, as the Vt is adapted to the patients lung mechanics. ASV employs the Otis Least Work equation to find the optimal tidal volume target and rate for each individual patient. The ASV algorithm takes in to consideration the patient's pulmonary mechanics and expiratory time constants in order to target an optimal tidal volume and rate to meet a target minute ventilation. The pressure, target tidal volume, target rate and I:E ratio all adapt to changes in the patient's lung mechanics and respiratory drive. The tidal volume and rate are also a function of the minute ventilation which is input by the clinician and is expressed as percent of normal minute ventilation for a patient of a particular ideal body weight. With ASV, the tidal volume is no longer an arbitrary clinician selected set-point. Lung protective strategies are incorporated into ASV based on pressure limitations that are clinician set, as well as by other limitations on tidal volume and rate which are dynamically modified as changes occur in the patient's respiratory system. ASV represents the dawn of the intelligent ventilation era and may be adaptive pressure control ventilation's answer to the "one size does not fit all" theory.

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Chronic Productive Cough in School Children: Prevalence and Associations With Asthma and Environmental Tobacco Smoke Exposure

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Abstract

Background: The relationships between chronic productive cough (CPC), environmental tobacco smoke (ETS) exposure, and asthma are not clearly established in children. Therefore, we wished to determine the prevalence of CPC and examine the relationships between CPC, ETS exposure, and asthma in young teenagers.

Methods: We performed a cross sectional survey of 2,397 Seattle middle school students, 11-15 years old, using written and video respiratory-symptom questionnaires. We defined CPC as daily cough productive of phlegm for at least 3 months out of the year; current asthma as *yes* to "Have you had wheezing or whistling in your chest in the past 12 months?" and *yes in the past year* to any of the four video wheezing/asthma video scenarios; and ETS exposure as exposed to tobacco smoke at least several hours each day. We used multilogistic regression to examine relationships between CPC, asthma, and ETS exposure and included in the model the potentially confounding variables race, gender, and allergic rhinitis.

Results: The prevalence of CPC was 7.2%. Forty-seven percent (82/173) of children with CPC met criteria for current asthma, while only 10% (214/2224) of those without CPC had current asthma. Current asthma had the strongest associated with CPC, odds ratio (OR) 6.4 [95% CI 4.5–9.0], and ETS was independently associated with both CPC, OR 2.7 [1.8–4.1] and asthma, OR 2.7 [1.5–4.7].

Conclusion: In a population of young teenagers, CPC was strongly associated with report of current asthma symptoms and also with ETS exposure. This suggests that asthma and ETS exposure may contribute to CPC in children. However, this study was not designed to determine whether asthma was the actual cause of CPC in this population of children.

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Background

Asthma is a recognized cause of persistent cough in both adults[1,2] and children,[3] but cough productive of sputum for more than three months out of the year, referred to as chronic productive cough (CPC), is not considered common in children with asthma. The NHLBI guidelines do not discuss productive cough as a separate sign,[4] and little is known about the prevalence of CPC and its causes in children.

Chronic productive cough is a hallmark of the rare conditions cystic fibrosis, ciliary dysmotility, and bronchiectasis, but it is possible that asthma and ETS exposure lead to CPC as well. However, the relationships between asthma, ETS exposure, and CPC in children have not been delineated. Peat et al followed a cohort of school children for six years and found that the majority of those with asthma also had at some time a productive cough lasting two or more weeks, but this duration of cough was too short to be termed chronic.[3] In addition, while ETS exposure has been linked to asthma,[5-8] its association with CPC, especially in children, is less clear. Lewis et al found that ETS exposure was associated with asthma symptoms but not with CPC in Alaskan native teenagers.[8] However, Janson et al surveyed young adults and identified both asthma and ETS exposure as risk factors for CPC.[9]

The prevalence of CPC in a large population of children has not been well established, in part due to variations in the definition of CPC. The American Thoracic Society (ATS) defines chronic bronchitis as "cough productive of sputum for at least 3 months of the year for at least 2 years,"[10] and this has become the standard for adults. However, these criteria have not been used consistently in studies of chronic cough in children. Amaral-Marques et al did use criteria that were similar to the ATS definition, and they found the prevalence of CPC in Portuguese children to be 4.9%.[11] However, they did not account for asthma or ETS exposure. Establishing the prevalence of CPC and the relationships between CPC, asthma, and ETS in children could lead to earlier diagnosis and treatment of asthma and a better understanding of the causes of CPC.

Table 1: Demographics of middle-school children with CPC*

Characteristic	Children with CPC (n = 173)	Children without CPC (n = 2224)
Prevalence in population	7.2% [3.3–11.1]	--
Age (years): median (range)	13 (12–15)	13 (11–16)
% Female	63% [56–70]	50% [48–52]
Race (%)		
Caucasian	29% [22–36]	31% [29–33]
African American	27% [20–34]	20% [18–21]
Asian	23% [17–29]	32% [30–34]
Native American	4% [1–7]	2% [1–3]
Other	17% [11–23]	15% [13–16]

* See text for the definition of chronic productive cough (CPC). Values in brackets are 95% confidence intervals.

In 2003 we participated in Phase III of the International Study of Allergies and Asthma in Childhood (ISAAC) as part of an effort to determine the prevalence of asthma symptoms in children throughout the world.[12,13] Seattle middle-school students completed written and video respiratory-symptom surveys. We added questions on ETS exposure and CPC in order to determine the prevalence of CPC and examine the relationships between CPC, asthma, and ETS exposure. Some of the results of this study have been published in abstract form.[14]

Methods

Subjects: In June 2003, students from the six middle schools in Seattle, Washington that participated in the ISAAC Phase III study were asked to complete written and video respiratory-symptom questionnaires. The Seattle School Board and The University of Washington Human Subjects Committee approved the protocol and waived written informed consent. We provided detailed written information to parents as well as verbal and written information to the students, and gave them ample time to refuse participation. We targeted children in the 7th and 8th grades, but 6th grade students were also eligible. Investigators oversaw completion of the questionnaires during typical class periods. Eighty-six percent (2397/2797) of the eligible students completed the questionnaires. School absenteeism accounted for the vast majority of students who did not complete surveys.

Study questionnaire and administration: The written survey contained core ISAAC questions on asthma, allergic rhinitis, and eczema, and we added questions on CPC and tobacco smoke exposure. In addition to the written questionnaire, students viewed the international version of the ISAAC video, which has one cough and four wheezing scenarios depicting children with signs of asthma.

Definitions: CPC required positive responses to both of the written questions, "Have you had a daily cough as often as 3 months out of the year?" and "Do you bring up phlegm, sputum, or mucous from your lungs as often as 3 months out of the year?" The personnel assisting with the study asked students if they knew what was meant by "sputum/phlegm," and if there was any confusion then they provided explanations. We did not use responses to the cough video question because this scenario showed a child with a non-productive hacking cough.

ETS exposure: The ETS question "How much time do you think that you spend around tobacco smoke?" had three possible responses – never or very little, occasionally, and several hours a day. Students were categorized has having ETS exposure if they answered "several hours a day."

Current asthma required a positive response to the written question, "Have you had wheezing or whistling in your chest in the past 12 months?" and a "yes in the past year" to any of the four video wheezing/asthma video scenarios. We did not use responses from the cough video scenario as part of the diagnostic criteria for asthma because we felt that this scenario was not representative enough of asthma. The current asthma group included both patients with and without a physician diagnosis of asthma.

No asthma required an answer of no to wheezing in the past year, no to a physician diagnosis of asthma, and no in the past year to all four of the video wheezing scenarios.

Possible asthma students who did not fit into either the current asthma or no asthma groups. These students had some positive responses to asthma questions but did not meet our specific criteria for current asthma.

Allergic rhinitis is a common cause of post nasal drip and cough with a high prevalence in patients with asthma. Thus, we wished to identify students who might have allergic rhinitis. We classified students as having allergic rhinitis if they answered yes to either of the two ISAAC questions, "Have you ever had a problem with sneezing, or a runny, or blocked, or stuffy nose when you did not have a cold or flu, that was accompanied by itchy-watery eyes?" or "Have you ever had hay fever?" These ISAAC questions have been validated and have a high specificity for atopy confirmed by skin testing.[15]

Outcomes and statistical analysis: The primary objectives were to establish the prevalence of CPC in this population and

Table 2: Adjusted multivariate associations of asthma and ETS exposure with CPC

Condition	Students with CPC N = 173	Students without CPC N = 2224	Odds Ratio [95% CI]
Current Asthma	82 (47%)*	214 (10%)	5.2 [3.6–7.5]
ETS exposure	43 (25%)	180 (8%)	2.9 [1.4–9.4]
Allergic Rhinitis	103 (60%)	549 (25%)	2.6 [1.9–3.8]
Female gender	108 (62%)	1108 (50%)	1.5 [1.0–2.1]

*The numbers in parentheses are the percent of students in each group who have the condition; i.e., 82/173 (47%) of children with CPC had current asthma.

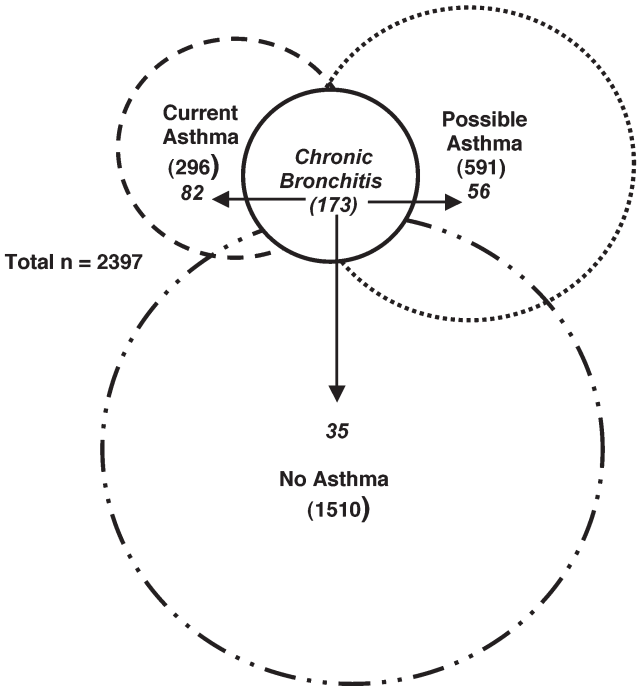

Figure 1
The relationship between CPC and asthma. This Venn diagram depicts the relationship between chronic productive cough (CPC) and asthma. See text for definitions of the current, possible, and no asthma groups. While the criteria for these groups make them mutually exclusive, they are shown as overlapping to indicate that in reality there is crossover between the groups. The numbers in parentheses are the number of children with the condition, e.g. current asthma. The proportions of children in each group with CPC (28%, 9.5%, and 2.3% for the current asthma, possible asthma, and no asthma groups, respectively), were statistically significantly different, $p < 0.001$ for each comparison.

to determine the associations of CPC with current asthma symptoms and ETS exposure. We also examined the relationship between ETS exposure and asthma. Demographic data were characterized using descriptive statistics, and differences between groups were analyzed with chi square. Using SPSS 11.5, we performed univariate analysis followed by multivariable logistic regression to assess independent associations between current asthma, ETS exposure and CPC. We included the potential confounding variables of allergic rhinitis, gender, and race in our model. We explored potential effect modification by adding the following multiplicative interaction terms to each model: gender × ETS exposure, gender × current asthma, ETS exposure × current asthma, race × ETS exposure, and race × current asthma. We did not include any multiplicative terms in our final regression model because we found no evidence of effect modification. We expressed these relationships as odds ratios (OR) with their respective 95% confidence intervals [95% CI].

Results

The demographics of the students are denoted in Table 1. The median age of the students was 13 years, and most of them were Caucasian, African American, or Asian. The prevalence of CPC was 7.2% (173/2397), and the prevalence rates of current asthma and ETS exposure were 12.4% and 9.3%, respectively. Of those students with CPC, 34% stated their sputum was white or clear, 47% reported it was yellow, and 15% claimed that it was green. Compared to the total group, a higher proportion of students reporting CPC were girls (63% vs. 50%, $p = 0.024$). Similarly, a higher proportion of children with current asthma were girls (59% vs. 52%, $p = 0.024$). Five percent of the students claimed to have smoked at least one cigarette in the past month. However, only 30% of the students actually answered this question, a response rate too low to accurately assess the effects of active smoking. Report of allergic rhinitis was also much more common in children with CPC (Table 2).

Current asthma was strongly associated with CPC (OR by univariate analysis 6.4 [4.5–9.0]). The association remained strong after accounting for interactions of gender, ETS exposure, and allergic rhinitis in a multilogistic regression model (Table 2). Children with CPC were five times more likely to have current asthma than those without CPC. Nearly half of the children with CPC (82/173) had current asthma compared to only 10% (214/2224) of those without CPC (Table 2). There were 296 children with current asthma, 1510 that met criteria for no asthma, and 591 children with possible asthma. Of the 173 children with CPC, 138 had current asthma or possible asthma, while only 35 met criteria for no asthma (Figure 1). Twenty-eight percent (82/296) of the current asthma group reported CPC compared to 9.5% (56/591) of the children with possible asthma and only 2.3% (35/1510) of the no asthma group (Figure 1).

Environmental tobacco smoke exposure was associated with CPC by univariate analysis (OR 2.7 [1.8–4.1]), and this association remained similar in the multilogistic regression analysis (Table 2). Environmental tobacco smoke exposure was also associated with current asthma, OR 2.7 [1.5–4.7]. Nineteen percent of the children (43/223) with ETS exposure had CPC compared to 6% (130/2174) of those without ETS exposure, $p < 0.01$. Twenty-three percent (52/223) of the children with ETS exposure had current asthma, while only 8% of those without current asthma reported ETS exposure, $p < 0.01$. More children with both asthma and CPC reported ETS exposure than did the children with asthma but no CPC; 33% (27/82) vs. 12% (25/214), $p < 0.01$.

Discussion

In this survey-based study of almost 3,000 middle-school students, the prevalence of CPC, defined as a daily cough productive of phlegm for at least three months out of the year, was 7.2%. Report of current asthma symptoms was strongly associated with CPC, even after correcting for allergic rhinitis, and almost half of the children with CPC met criteria for current asthma. Environmental tobacco smoke exposure was also independently associated with CPC. These findings suggest that CPC can be a manifestation of asthma and that asthma should be considered in the differential diagnosis of children who present with a CPC.

It is important to establish what is meant by CPC, often referred to as chronic bronchitis. In the 1950s, the British Medical Research Council defined chronic bronchitis as "cough productive of sputum for at least 3 months of the year for at least 2 years without an identifiable cause,[16] and the ATS adopted this definition in 1962.[10] While many investigations of CPC in young adults and children have employed similar definitions,[11,17-20] others have not.[8,9] Consequently, this makes it difficult to compare the prevalence and causes of CPC across studies. In addition, while chronic bronchitis is a term that is inherently linked to CPC, it has many connotations. Taussig et al noted that only 55% of pediatricians and 74% of family practitioners surveyed considered CPC lasting at least 3 months of the year important in diagnosing chronic bronchitis,[21] and Bobadilla et al found that only a minority of patients with physician-diagnosed chronic bronchitis actually met ATS criteria.[22] Thus, it is more precise to use the descriptive term CPC in lieu of the label 'chronic bronchitis'. The ATS definition was established primarily for adults, and there are causes of CPC in adults that are much less common in children, including active cigarette smoking and chronic obstructive pulmonary disease. Nevertheless, by adopting standard criteria for CPC, it will be possible to compare results across studies as well as age groups.

Our study is one of the few to establish the prevalence of CPC in a large population of children using an adaptation of the ATS criteria. Our criteria only differed from the ATS criteria in that we required cough productive for sputum over one year rather than in two consecutive years. We found the prevalence of CPC to be 7.2%, which is higher than the 4.9% prevalence noted by Amaral-Marques et al in 4,148 Portuguese school-aged children.[11] Their prevalence may have been lower because they required productive cough in two consecutive years. As with our study, they observed that a higher proportion (62%) of the young teenagers with CPC were girls. One possible explanation for this female predominance is that asthma and CPC are closely linked, and, as noted in our study as well as others,[23,24] more teenagers with asthma are girls. Girls did not report significantly more ETS exposure, so it is unlikely that ETS exposure was a factor in the female predominance of CPC.

Investigators have surveyed young adult populations to determine the relationships between smoking, CPC, and asthma, but there are few data in children. Cerevi et al identified active cigarette smoking to be the primary risk factor for CPC in young adults.[18] However, almost 20% of their subjects with CPC had asthma and approximately 30% were non-smokers. Compared to the active smokers, the non-smokers were younger and were more likely to be female and to have asthma. Janson et al in a survey of 18,277 young adults noted a positive, albeit weak, association of CPC with ETS exposure and a stronger association with asthma.[9] However, Lewis et al noted that ETS exposure was a risk factor for asthma but not for CPC in Alaska native teenagers.[8] Environmental tobacco smoke exposure has been linked to asthma exacerbations,[5-7] but the association of ETS exposure with persistent asthma symptoms is less well established. While our study was not designed to determine the causes of CPC, our results suggest that asthma and ETS exposure independently increase the risk of having CPC. Furthermore, the fact that 28% of the children with current asthma reported CPC indicates that CPC may be a more frequent complaint in patients with asthma than previously recognized.

There are rare conditions, eg cystic fibrosis, that frequently present with CPC, but the most common causes of CPC have not been determined on a population level. However, the causes of CPC have been studied in select pediatric populations. Seear et al determined the causes of CPC in a group of children specifically referred for evaluation of that complaint.[20] They found that of 81 children presenting with "a productive or rattly cough, with or without wheezing, on most days for 3 consecutive months or more," 14 had probable asthma and 33 had other conditions that explained their cough. However, there were 34 children in whom there were no clear etiologies, and they were labeled as having chronic bronchitis. Of note, eight of these children (24%) were Native American. The authors postulated that lower respiratory tract infections early in life resulted in lung damage/inflammation and a propensity towards chronic cough. Native Americans appear to be prone to CPC. Lewis et al found that 30% of Alaskan native teenagers reported CPC, many of whom did not meet criteria for current asthma symptoms.[8] This population has an unusually high prevalence of bronchiectasis, presumably due to a predilection to damage from lower respiratory tract infections.[25] In our study, only 4% of the students claimed Native American heritage, and it is unlikely that bronchiectasis accounted for many of the cases of CPC. Marchant et al evaluated 108 children referred to a pediatric respiratory practice for assessment of cough of > 3 weeks duration.[26] The mean age was 2.6 years and 89% had wet cough. They found the most common diagnosis to be protracted bacterial bronchitis, based on a positive culture of bronchoalveolar lavage fluid and response to antibiotic treatment. Fewer than 5% had asthma as the primary diagnosis. The studies by Seear et al and Marchant et al suggest that children referred to a respiratory clinic for evaluation of cough often have diagnoses other than asthma. However, it is likely that many of the patients with asthma that have CPC are not referred to specialists, and these studies were not designed to assess the frequency of CPC in children with asthma.

Allergic rhinitis is a common cause of post nasal drip and chronic cough, and we found that report of allergic rhinitis was associated with CPC. However, using a multilogistic regression model that included allergic rhinitis as a co-variate, we found that current asthma had the strongest independent association with CPC. Nevertheless, we cannot rule out the possibility that allergic rhinitis was the cause of CPC in some of the children who also reported current asthma symptoms.

There were limitations to our study. This was a cross-sectional study, and we did not follow the children longitudinally. The results were based on self-reports, and we did not use physical examinations or tests to confirm the diagnosis of asthma or

identify other potential causes of cough. Therefore, the children that reported CPC and also met criteria for asthma and/or ETS exposure may have had other causes for their cough, including allergic rhinitis, chronic sinusitis, or the rarer diseases cystic fibrosis and bronchiectasis. Asthma is unlikely to be the cause of CPC in patients with purulent sputum, and only 15% of the students in our study reported green sputum. Thus, it is important to evaluate children who have cough productive of purulent sputum for other conditions even if they have asthma. The questions used to define CPC, while standard, have not been validated in children. Young teenagers may have difficulty recalling their symptoms over a year's time and understanding what is meant by sputum or phlegm production. The study personnel that administered the surveys to the students were available to explain the questions to the students, so we believe that most of the students were capable of answering the questions. Nevertheless, the results of our study should be interpreted with caution due to the lack of physician-confirmation of CPC, asthma, and other respiratory conditions in the respondents. Our criteria for current asthma, which we have used previously,[13] were designed to have a fairly high specificity at the risk of decreased sensitivity[27] and likely resulted in the misclassification of some of the subjects. The prevalence of ETS exposure in our patient population was lower than that reported in other studies,[28,29] possibly due to the reliance on self report and the requirement of being around cigarette smoke for at least several hours each day. However, the prevalence of ETS exposure in the students reporting chronic productive cough (25%) was similar to the prevalence of ETS exposure in homes reported by both Soliman et al and Sexton et al.[28,29] Finally, we could not assess active cigarette smoking as too few students responded to that question. Only 5% of the students in our study reported having smoked at least one cigarette within the past month compared to 9.6% of 3379 8th-graders who responded to this question on the 2004 Washington State Healthy Youth Survey.[30] Therefore, some of the effects attributed to ETS exposure may have been due to active smoking.

Asthma is a common cause of cough but not necessarily of CPC. We found an association of self-reported CPC and asthma symptoms. However, this does not prove that the CPC was due to asthma nor does it suggest that children with asthma and CPC do not require evaluation for other causes of CPC, especially in those with purulent sputum. However, the strong association between asthma symptoms and CPC identified in our study suggests that CPC may be more common in children with asthma than previously thought.

Conclusion

The prevalence of CPC in young teenagers, based on self-report of cough productive of phlegm for at least 3 months out of the year, is approximately seven percent. Children with CPC and/or ETS exposure are more likely to report asthma symptoms. We found that CPC was more common in children with current asthma symptoms and/or ETS exposure than in children without those conditions. Furthermore, current asthma and ETS exposure were strongly and independently associated with CPC. In this limited epidemiological study children reporting CPC had an increased risk of asthma symptoms. However, asthma is unlikely to be the cause of chronic cough productive of purulent sputum, and patients with purulent sputum should be evaluated for other conditions even if they have asthma. Further studies are needed to determine the frequency of CPC in children who have confirmed asthma and to establish whether children presenting with CPC truly have asthma as the cause of their CPC vs. other underlying diseases.

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Flow Trigger vs Pressure Trigger

Tim France RRT

One of the settings on a ventilator that impacts patient/ventilator synchrony and work of breathing is the type of triggering mechanism that is used. Ventilators manufactured prior to the 1990s traditionally only offered pressure triggering systems. Around 1993 when ventilators became more computer controlled, flow triggering systems were implemented.

Pressure trigger (PT) requires a drop in the circuit pressure to initiate a breath, usually set between -0.5 and -2 cmH_2O.[1] The patient's diaphragm drops causing negative intrathoracic pressure. (Remember Boyle's gas law: as the muscles of inspiration expand the thoracic volume the pressure drops.) The ventilator senses this negative pressure drop and once the pressure drop equals the trigger setting, a spontaneous or mandatory breath is delivered. The higher the trigger setting, the less sensitive the ventilator. Although modern ventilator pressure trigger systems impose significantly less imposed work of breathing than older systems, one should consider that the patient has to create the pressure drop across the ventilator circuit to the pressure sensor point and as such one can't avoid some imposed work along with the lag time until flow reaches the patient.

Flow trigger looks at the amount of inspiratory flow the patient inhales and once the flow trigger setting is reached, a breath is delivered. The ventilator flows a known base flow through the circuit during the expiratory phase and compares the base flow leaving the ventilator to the flow returning to the ventilator. If the patient's inspiratory flow exceeds the trigger setting, the ventilator will trigger as it 'knows' that the difference between the sent and returned flow through the circuit has exceeded the flow trigger setting. Flow trigger has been shown to be more sensitive than pressure trigger.[2]

For example the Hamilton Galileo always has twice the flow trigger setting circling thru the circuit (base flow during the expiratory phase). Just like pressure trigger, setting higher levels of flow trigger makes the ventilator less sensitive. For example, it is more difficult to inhale 8 lpm vs 3 lpm. In a patient with poor inspiratory muscle strength, at high levels of FT they may only breathe off the flow by in the circuit and not initiate a spontaneous breath. Relatively speaking, however, an increase in the flow trigger setting from 3 lpm to 6 lpm is much less additional work to trigger than would be increasing the pressure trigger setting from -3cm to -6cm. Just like PT, at low levels of FT, autocycling can occur, as the ventilator can't distinguish flow not returning due to a leak vs the patient receiving the flow. Flow trigger on a ventilator has another advantage in that FT can compensate for leaks during mask ventilation by increasing the FT setting. During autocycling, if there is a 2 lpm leak and you have an FT setting of 3 lpm, turning up the FT setting to 5 lpm should stop the autocycling. Basically you are tricking the vent into thinking the trigger setting is 5 lpm. The trigger is still 3 lpm, but the extra 2 lpm is compensating for the leak.

Flow trigger has also been associated with autocycling even at higher levels. In hyperdynamic patients there are documented situations where the patient's heart rate has cycled the ventilator due to intrathoracic pressure changes.[3] This can also occur with balloon pumps and beds with built in percussion/vibration features. In clinical practice, FT has become the default triggering method. However, just like pressure triggering, setting inappropriate levels can cause autocycling or failure to cycle.

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Metaphorical Medicine: Using Metaphors to Enhance Communication with Patients Who Have Pulmonary Disease

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Although the importance of enhancing communication with patients has been emphasized and is the subject of recent editorial reminders, little formal attention has been given to the way pulmonary physicians speak with patients regarding the underlying nature of their chest illnesses or the rationale, risks, or benefits of treatment. In many forums, including business, education, politics, and medicine, using metaphors has been advocated as a way to enhance teaching and communication and as a means to achieve better insight into institutional character. Derived from the Greek root "metapheiren" meaning "willing to transfer" and defined as a "figure of speech in which a word or phrase literally denoting one kind of object or idea is used in place of another to suggest a likeness or analogy between them", metaphors can facilitate communication because they render new concepts in familiar terms.

On the basis of our impression that using metaphors to explain pulmonary illness can enhance communication with patients, we conducted the current study with two goals in mind: 1) To describe the frequency with which pulmonary and critical care physicians use metaphors in explaining diagnosis or treatment to their patients, and 2) to begin to assemble a "catalog" of metaphors for a variety of pulmonary issues that may be useful to colleagues seeking new communicative strategies.

Methods

To ascertain whether physicians in our department use metaphors and to record their metaphors, we administered a survey to all 22 members of the Department of Pulmonary and Critical Care Medicine at the Cleveland Clinic Foundation, Cleveland, Ohio (11 staff physicians, 11 fellows). Using a brief questionnaire instrument, we asked colleagues whether they ever use metaphors or similes in communicating with patients regarding diagnosis or therapy. In the case of an affirmative answer, the respondent was asked to indicate for which types of pulmonary illness the metaphor or simile is used. Finally, using an open-ended format, respondents were asked to describe the metaphor or simile they use.

The study was conducted with approval of the Cleveland Clinic Foundation Institutional Review Board.

Metaphors were classified according to the well-accepted "contemporary theory" pioneered by Lakoff and Johnson. As discussed and applied by Johnson, Honeck, and Ortony, the contemporary theory holds that metaphors define and reflect deeply entrenched cultural understandings. In contrast to the traditional theory of metaphors, in which metaphors are regarded as mere figures of speech that compare words to ornament their presentation rather than to enhance their meaning (for example, "my love is a rose" expresses a relationship that is both beautiful and thorny but does not suggest that love is a plant), the contemporary theory understands metaphors as conceptual entities that are based in the shared experiences of a given group of people and that structure the language that these individuals speak. According to the contemporary theory, a metaphor associates two mental concepts or "domains," the first of which is understood and experienced in terms of the second. For example, in the conceptual metaphor ARGUMENT IS WAR (capital letters indicate that the metaphors are conceptual), the first domain, ARGUMENT and its attendant attributes, is understood and experienced in terms of the second domain and attributes, WAR. WAR ARGUMENT emerges from the association as a kind of verbal battle, a conceptualization that is then articulated in such familiar expression as "you attacked my point" and "she defended her thesis." Seen this way, metaphors construct meaning rather than merely embellishing it, often within everyday language. This feature distinguishes true metaphors from analogies, which simply expand common concepts through comparison. The analysis that follows recognizes this distinction between analogies and metaphors.

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Results

Responses were elicited from all 22 members of the department, including 11 staff physicians (9 men, 2 women; mean age, 42.5 years [range, 34 to 51 years]; mean years in practice, 10.1 [range 3 to 18 years]) and 11 fellows (4 third-year, 3 second-year, and 4 first-year). Ten of the 11 staff physicians expressed the belief that metaphors enhance communication with patients, and 8 provided examples of metaphors from their practices. One staff physician felt that using metaphoric language could be confusing to the patient. All 11 fellows thought that the use of metaphors enhanced communication with patients, but only 4 could provide examples of metaphors they used.

Altogether, a total of 19 metaphors were offered, addressing 8 separate topics in pulmonary disease: chronic obstructive pulmonary disease, α_1 - antitrypsin deficiency, emphysema, airflow obstruction, asthma, interstitial lung disease, restrictive chest disease, pleural effusion, obstructive sleep apnea, lung cancer treatment, transtracheal oxygen therapy, bronchoscopy, and hemostasis. The percentage of physicians who used metaphors when discussing specific topics varied from 55% for emphysema and chronic obstructive pulmonary disease to 0% for pulmonary vascular disease and pulmonary hypertension.

Classification of the types of images used in these 19 metaphors showed four basic types:

1. Container images (that is, lungs or aspects of them as a balloon, paper bag, white coat, balloon in a balloon, coffee cup in a coffee cup, and collapsed straw);
2. Natural images (that is, lung or aspects of them as a small child, Swiss cheese, a mosquito bite, an upside down tree, killing/roping a best);
3. Mechanical images (that is, lungs or aspects of them as monkey bars, a plumbing system, clogged drainpipe, compressed rubber tube, trash compactor, band around the chest, puncture tie, collapsed straw, rope around a beast, piercing an ear, and a clogged freeway), and
4. Somatic images (that is, lungs or aspects of them as twitching back muscle, cut and scar on arm, and pierced ear).

In addition, the 19 metaphors used by physicians in this series vary from more limited, prosaic figures of speech that may be best categorized as analogies (for example, the bronchi are to the trachea as branches are to a tree trunk) and similes (for example, "the lung is like a jungle gym") to the more imaginative, poetic figures that are true metaphors because they construct fresh meaning by relating two domains with unexpected and new connections (for example, the windpipe is like a small child in that the slightest insult can cause it to spasm). While most of the 19 figures assembled here are best characterized as analogies or similes, the spectrum is broad and reflects the continuum that physicians in this series have found useful as they communicate with patients.

Discussion

The results of this survey show that for almost all pulmonary and critical care physicians at the Cleveland Clinic Foundation, metaphors were considered helpful and were actually used in communicating with patients about diagnostic or treatment issues. Our simple survey generated a total of 19 different metaphors from 12 physicians who offered examples from their experiences. The figures of speech assembled cover a spectrum from analogies and similes, in which the lung is likened to other objects with common features, and true metaphors, in which new meanings are constructed by imaginative and even poetic associations (for example, the windpipe as a small child). This range defies simple classification and establishes the continuum of figures of speech that physicians in this series used in talking with patients.

In this study, metaphors were valued by physicians at all levels of experience, from new fellows to the most senior practitioners in the group. The use of metaphors varied by topic. For example, more than half of physicians surveyed used metaphors in their discussions of emphysema and chronic obstructive pulmonary disease with patients. However, for other topics, such as pulmonary hypertension and vascular disease, metaphors were never used. We speculate that physicians did not use metaphors because they could not identify a suitable analogy rather than because of bias against the potential value or harm of these linguistic devices. Our observations also suggest that certain medical content areas are amenable to metaphoric communication, while others are less so.

Physicians' frequent use of metaphors in this survey bespeaks the widespread endorsement of metaphors as communication aids in our department. Although not formally studied here, physicians' rationales for using metaphors reflect two goals: 1) to transform a complex, clinical concept into a simple explanation for lay persons, and 2) by enhancing the comprehensibility of clinical explanations, to accelerate communication. Participating physicians commonly felt that patients achieved understanding faster when metaphors were used, thereby helping communication by allowing more time for follow-up questions and interaction.

While the current report is, to our knowledge, the first full description of metaphor use in pulmonary and critical care medicine, earlier reports have described the value of metaphors in other medical contexts. For example, Olweny presented an inventory of metaphors used by oncologists in speaking with patients about the diagnosis and treatment of cancer. As an example, Olweny likened the bone marrow to a factory whose three main products are the three blood components: erythrocytes, leukocytes, and platelets. Also, in explaining the role of receptors in treating breast cancer, Olweny likened the breast cancer to a house with many open windows and doors - the receptors. The more windows and doors that are open (that is, the greater the receptor positivity), the better the chance that closing them with receptor antagonists (for example, tamoxifen) will combat the cancer. In another analysis of metaphors in the clinical setting, Mabeck and Olesen reported the importance of metaphors to patients in articulating their understanding from medical encounters. Specifically, in interviewing eight patients after medical visits about somatic symptoms, these investigators reported that patients uniformly used metaphors to "narrate their understanding of medical encounters."

Physicians' practice of using metaphors in this survey is not unique; metaphors have been widely used in other professional settings. For example, metaphors have been successfully used by business consultants to enhance organizational analysis, by educators to strengthen and test analytic skills, and by politicians to enhance the clarity of their messages.

The use of metaphors in medical and other settings is consistent with recent research showing that metaphors are conceptual rather than linguistic entities; they verbalize the shared experiences of a given social group in all language. Because metaphors create meaning, primarily by means of everyday language, they have a normative role in language and are value-laden. In addition, metaphors are grounded in a cultural context, and their meaning is determined by that context. For example, in western cultures, the metaphors "HAPPY IS UP" and "SAD IS DOWN" conceptualize the direction "up" as a positive emotional state and "down" as a negative one, and then express these states in such statements as "I'm feeling up (or down) today." However, in cultures such as the Hmong of Cambodia, where "up" is conceptualized negatively and "down" as positively, the meaning of the aforementioned western metaphors would be radically different.

Our classification of the medical metaphors into four distinct image types helps to clarify the role and value of metaphors in medical communication. In particular, the common use of specific image types suggests that the pulmonary and critical care physicians surveyed constitute a culture whose members share certain values. Within this culture, the high number of natural images (five) suggests that these physicians sought explanations that are familiar and comfortable to the patient rather than threatening. This same desire to use familiar and comfortable language is reflected in the images invoked in the seven container metaphors, the twelve mechanical metaphors, and the four somatic metaphors. On the other hand, as also emphasized by Hodgkin, the use of mechanical metaphors points out the potential inadvertent downside of metaphoric communication. While the role of technology in our culture is widely accepted and revered by many, the use of mechanical images by physicians in their communication with patients is potentially dehumanizing and antithetical to physicians' desire to transmit sensitive information in a humane manner.

While offering a catalog of metaphors that we hope help clinicians communicate with their patients, our study has several important limitations. First, the sample from which we sought responses was small and derived from one institution. We hope this study will spur others to describe and expand this small collection and to point out other experiences in which the use of metaphors can enhance clinical practice. Our limited sample has other implications. A survey of clinicians in other institutions might reveal additional metaphors not described here; these might refine our conclusions and, with that, our understanding of how language shapes medicine as a cultural practice.

A second potential limitation is that our exploratory study does not address several important questions involving the effectiveness of the metaphors. Specifically, as Spiro and colleagues have pointed out in the setting of educating medical students, metaphors can detract from clinical explanation by oversimplifying or by causing misunderstanding. To more fully understand and to test the communicative value of metaphors in medicine, it would be important to assess both the patients' ratings of the value of metaphorical communication and the patients' understanding of clinical issues after receiving typical explanations (for example, with conversation, text, and diagrams) versus receiving explanations based on metaphors. Furthermore, our study invites further inquiry about the process by which physicians choose metaphors (for example, when to use analogy, simile and metaphor) and the metrics by which physicians assess their effectiveness in communicating with patients.

Overall, our study shows that pulmonary and critical care physicians at our institution use metaphors widely and explicitly to enhance how they communicate complex clinical concepts to patients. We offer this catalog of metaphors and our conclusions to colleagues in the hope that others will find them valuable as communication tools. We also invite other to expand on this inventory, to use metaphors consciously and conscientiously, and to optimize ways to communicate effectively with patients.

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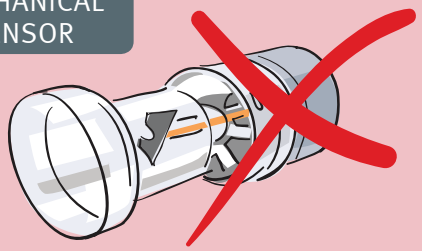

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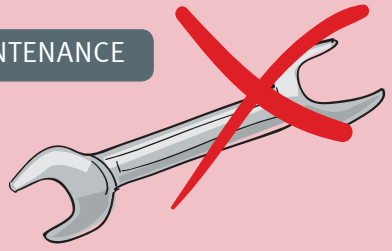

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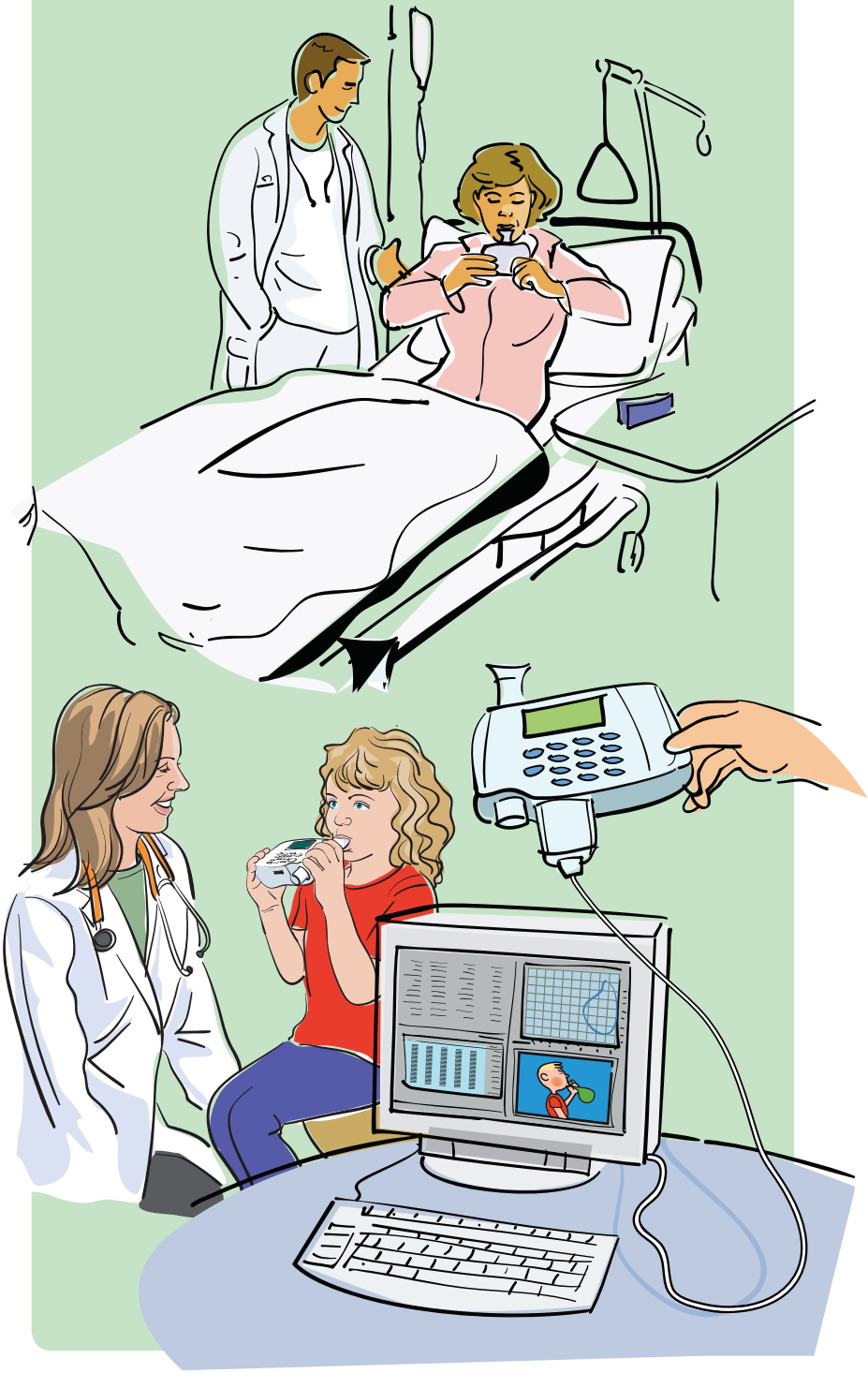

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