Once again, the New Year has rushed in as a time for renewal and resolutions. I challenge all diagnostic practitioners to make 2001 the year that they become involved in a research project in their area of expertise. Wouldn’t it be wonderful to have a large contingent of diagnostic presentations included in the Open Forum abstracts and poster presentations when we convene December 1-4 in San Antonio, TX, for the AARC International Congress? Think about your daily practice. Are there things you could share, investigate, or study? Or, look beyond the daily routine. Is there something you have always wanted to know more about? Now is the time to be inquisitive and stimulate those brain cells!

Here are some ideas:
• Case studies (look for something special or out of the ordinary)
• Retrospective data review
• Prospective research
• Database analysis
• Instrument evaluation
• Instrument comparison

If you need help getting started, check out the following references: Research for the Health Professional, A Practical Guide, by Diana Bailey, published by F.A. Davis Company; and PDQ Statistics, by Geoffrey Norman and David Streiner, published by Mosby. You might also find support from a research or education department at your institution, and some state societies have research specialty sections to assist practitioners with the advancement of research as well.

This Bulletin our featured author is Dr. Monica Kraft, from National Jewish Medical and Research Center, who shares with us the results of studies she and others have done on the distal lung in asthma. Dr. Kraft recently received a PECASE award at the White House. PECASE, which stands for Presidential Early Career Award, is the highest honor bestowed by the U.S. government on young professionals at the start of their independent research careers. Let’s let Dr. Kraft be an example for all of us in advancing the field of pulmonary research.

If I can be of assistance to you, please contact me through my e-mail address listed on page 2. Happy New Year!
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Editors’ Note: Dr. Kraft was recent honored at the White House, where she received the prestigious PECASE (Presidential Early Career Award) award, the highest honor bestowed by the U.S. government on young professionals at the start of their independent research careers.

Data from the last three decades have suggested that the distal lung, which includes the airways < 2 mm and the lung parenchyma, contributes to asthma pathogenesis. This concept was first evaluated in an animal model by Macklem and Mead (1,2), which showed that the peripheral resistance, Rp, was too small to detect above 80% of vital capacity but increased at lower volumes to 15% of the total lung resistance at 1% of vital capacity. These data confirmed work by Weibel et al., which demonstrated that the cross sectional area of the small airways was significantly larger than that of the central airways (3). Thus, the distal airways were dubbed the “quiet zone” of the lungs by Mead in 1970 (4).

Due to the challenges raised in evaluating the small airways of the lung in living humans, this region has not been studied at the same level of detail as the larger airways. However, a significant amount of pathologic data do exist from autopsy specimens (5-9). Investigation in chronic, stable asthmatics is starting to be performed in regard to the small airways and lung parenchyma (10,12). We have shown that in nocturnal asthma, significant parenchymal inflammation is present and increases at night (11,12). Significant parenchymal inflammation has also been shown in severe asthma (10).

However, the functional significance of this inflammation in living patients with asthma is not clear. Wagner and colleagues, employing a bronchoscope in humans, showed that peripheral airway resistance (Rp) was increased in subjects with mild asthma and normal spirometry as compared to non-asthma controls (13,14). The conductance (1/Rp) inversely correlated with hyperresponsiveness (13). These investigators extended these observations by assessing small airways hyperresponsiveness by directly administering histamine and isoproterenol into the small airways using the wedged bronchoscope technique (14). Again, the baseline Rp was greater in asthmatics with normal spirometry as compared to normal controls. More histamine was required in normal subjects to cause a 100% increase in Rp (log PC100) than in asthmatics. In asthmatics, the PC100 correlated with whole lung responsiveness to histamine (r=0.847, p < 0.005). Isoproterenol completely reversed the increase in Rp in normal controls but not in the asthmatic subjects, possibly secondary to edema and airway closure.

Woolcock and colleagues also evaluated the peripheral lung by determining the dynamic compliance in four subjects with asthma, five subjects with chronic bronchitis, and eight normal controls (15). This was performed by first measuring static compliance via an esophageal balloon, then asking subjects to breathe at increasing frequencies up to 120 breaths/minute. Dynamic compliance was calculated by dividing the volume change by the pressure change between points of zero flow on the tracing. In subjects with asthma and chronic bronchitis, there was a significant frequency dependence of dynamic compliance. This was particularly seen in the asthmatics, where dynamic compliance fell to 25% or less of static compliance at the highest frequency. After isoproterenol, less frequency dependence was appreciated, particularly in the asthmatics, but in no case did compliance become independent of frequency. Thus, the frequency dependence suggests that some regions of the lungs are filling and emptying at different rates than other regions, which can produce ventilation-perfusion mismatching.

The use of volume history response in humans can also shed light on parenchymal hysteresis, a reflection of tissue resistance. Hysteresis refers to the fact that more transmural pressure is required to keep the airway at a certain dimension (and volume) on the inflation limb as compared to the deflation limb (Figure 1). The lung parenchyma and the large airways are highly interdependent mechanically, such that changes in the size of each occur simultaneously (16), and each also has hysteresis. Most of the pressure volume hysteresis is due to the parenchyma, which in this setting also includes the terminal airways and is attributable to the surfactant acting at the gas-liquid interface (17). In addition, there is an effect on hysteresis produced by opening and closing of the extremely peripheral airways that is related to

The Distal Lung in Asthma: Is It Important?
by Monica Kraft, MD, National Jewish Medical and Research Center

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“The Distal Lung” continued on page 3

“Palliative Care” continued from page 1

conference were: how to assess and treat dyspnea in terminally ill patients, how to talk to families about death and dying in the ICU, the nuts and bolts of withdrawing life support, and the role of respiratory therapists in palliative care. The two Journal issues contain the formal papers presented by the faculty, the often spirited discussions following each presentation, and an insightful conference summary.

According to Dr. David Pierson, Journal editor, “although almost nothing has previously been published on this specific aspect of palliative care, respiratory care clinicians have long played a vital role in this arena. The material presented in these special issues breaks new ground, and is sure to become a valuable and practical reference source for everyone participating in the care of patients with severe respiratory disease.” For more information on the conference, visit the Journal’s website, www.rcjournal.com.
“Distal Lung” continued from page 2
to intrinsic tone of small airway smooth muscle and contractile elements of alveolar ducts (17).

The use of deep inhalation and the ratio of maximal to partial flow rates at the same volume illustrates the concept of airways and parenchymal hysteresis (17,18) (Figure 2). A maximum to partial (M/P) flow ratio greater than one means that flow increased after a deep inhalation, suggesting that airway size is greater after a deep inhalation and airway hysteresis exceeds that of the parenchyma. Therefore, a deep inhalation produced bronchodilatation. A low M/P ratio indicates that parenchymal hysteresis exceeds airway hysteresis, thus large airway size will decrease after a deep inhalation. In this case, a deep inhalation produced bronchoconstriction.

An observation that may shed light upon these disparate responses to deep inhalation has been shown in patients with mild asthma. When airway obstruction was induced by hyperventilation with cold air (19), methacholine (20), or histamine (21), a deep inhalation most often resulted in lessening of obstruction that was transient (bronchodilatation). The degree of improvement was proportional to the degree of induced obstruction. This observation is compatible with an increase in airway hysteresis and is possibly due to constriction of large airway smooth muscle. In contrast, when the airway obstruction is spontaneous, a deep inhalation results in bronchoconstriction, opposite to the effect seen in induced obstruction. The hypothesis suggested by Ingram is that the response to a deep inhalation might be indicative of the degree of peripheral inflammation. Piss and colleagues assessed the volume history response and bronchoalveolar lavage (BAL) eosinophils, histamine, and protein (22). As the M/P ratio decreased, indicative of increased parenchymal hysteresis, the BAL eosinophils, protein, and histamine increased. Other explanations of this observation may be stimulation of contractile elements in the parenchyma, as reported by Dolhnikoff et al. and Fredberg et al. (23) (24), and transudation of edema or inflammatory exudate into the peribronchial space. Therefore, the response to a deep inhalation may be helpful in determining the balance of airway and parenchymal hysteresis, and may determine which process dominates. If a deep inhalation results in bronchodilatation, then large airway hysteresis dominates. If it causes bronchoconstriction, parenchymal and small airway hysteresis dominates.

Although this focuses upon the physiologic aspects of the distal lung, inflammation in the distal lung units in asthma has been assessed primarily via autopsy studies and surgical specimens. Recently, there have been three studies published where transbronchial biopsies have been performed in chronic, stable asthma (10-12). Most of the studies from autopsy and surgical specimens focus upon the large and small airways, whereas the studies employing transbronchial biopsy describe lung parenchymal inflammation. They demonstrate that significant distal lung (both small airway < 2 mm in diameter and lung parenchyma) inflammation exists to correlate with physiologic changes noted.

In summary, evaluation of physiologic and pathologic studies suggests that the distal lung units participate in the pathogenesis of asthma. However, the challenge lies in proving the ability to assess their structure and function noninvasively. Physiologic studies of lung hysteresis reveal important relationships between the airways and parenchyma and potentially provide a way to assess which compartment of the lung is contributing the most significantly in a given asthmatic. However, the precise site of obstruction is still controversial in these studies. Bronchoscopic measurements of peripheral resistance offer a more direct, but still invasive, method to evaluate this compartment of the lung. Imaging studies are particularly exciting, as they are noninvasive. Before these techniques can be widely used, they must be validated through pathologic and physiologic techniques, such as biopsy and bronchoscopic peripheral resistance measurements. Although challenging, the endeavor to evaluate the distal lung is definitely worth the effort, as several studies have illustrated that if the distal lung is participating significantly in asthma pathogenesis in a given patient, there are clinical implications (25,26). Therefore, the distal lung in asthma is anything but quiet.

**References**

Figure 1. All four panels plot normalized volume (A, of alveoli alone, B, of airways alone; C, D, alveoli as solid lines and airways as dashed lines) vs. transpulmonary pressure (PTP). Arrows on lines show direction of volume change. It is assumed that PTP equals transmural pressure of airways as well as alveoli (parenchyma). Horizontal lines in A and B indicate the volume difference at the same PTP, showing a higher volume during deflation. Vertical lines indicate pressure difference at a given volume, showing a lower PTP at the same volume during deflation. Panel C shows airway (dashed line) hysteresis relatively greater than parenchymal (solid line) hysteresis. With airway size as the dependent variable, panel C shows airway hysteresis greater than that of the parenchyma and indicates that airways are larger at a given lung volume after a deep inflation. Panel D shows airway hysteresis less than that of the parenchyma so that following a deep inhalation, airways are smaller at a given lung volume. (Adapted from reference 17.)

Figure 2. Forced exhalations, first begun from approximately 60% of vital capacity (partial maneuver) and quickly followed by inhalation of total lung capacity (plotted at 100% vital capacity) before a second forced exhalation (maximal maneuver). At approximately 30% vital capacity maximal and partial expiratory flows were compared to give M-P ratios. On the right the M-P ratio is <1.0, indicating a constrictor response to a deep inhalation. The reverse is shown on the left. (Adapted from reference 17.)

Figure Legend

AARC Wants to Know Your Top Five Areas of Concern

The AARC is currently seeking input from section members regarding the top five areas of concern unique to our specialty area. Please mail, email, or fax your top five concerns related specifically to the specialty (not to the AARC or the practice of respiratory care in general) to: Kelli Hagen, 11030 Ables Lane, Dallas, TX 75229, email: hagen@aarc.org, FAX (972) 484-2720 or (972) 484-6010. The Association will utilize our input in determining priorities for the coming year.
The AARC is currently seeking information on JCAHO accreditation site visits. Please use the following form to share information from your latest site visit with your colleagues in the Association. The information will be posted immediately on the AARC web site at http://www.aarc.org/members_area/resources/jcaho.html and will also be featured in the *Bulletin*.

**JCAHO Accreditation Report**

Accreditation visit you are reporting (choose one):

- [ ] Home Care
- [ ] Hospital
- [ ] Long Term Care
- [ ] Pathology & Clinical Laboratory Services

**Inspection Date:**

**Facility Name:**

**Contact:** (Please provide name and email address.)

**1.** What was the surveyors’ focus during your site visit?

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**2.** What areas were cited as being exemplary?

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**3.** What suggestions were made by the surveyors?

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**4.** What changes have you made to improve compliance with the guidelines?

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Additional comments:

Mail or fax your form to:
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FAX (972) 484-2720
Specialty Practitioner of the Year

Don’t forget to make your nominations for the 2001 Diagnostics Specialty Practitioner of the Year. This honor is given to an outstanding practitioner from this section each year at the AARC’s Annual Convention.

The recipient of this award will be determined by the section chair or a selection committee appointed by the chair. Each nominee must be a member of the AARC and a member of the section.

Use the following form to send in your nominations for this important award:

I would like to nominate ____________________________ for Diagnostics Specialty Practitioner of the Year because

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Nominee: ____________________________

Your Name: ____________________________

Hospital: ____________________________

Hospital: ____________________________

Address: ____________________________

Address: ____________________________

City, State, Zip: ____________________________

City, State, Zip: ____________________________

Phone: ____________________________

Phone: ____________________________

Mail or FAX your nomination to the section chair at the address/number listed on page 2 of this issue.