NOTES FROM THE CHAIR
by Susan Blonshine, BS, RRT, RPFT

Individual Commitment to a group effort—that is what makes a team, a company work, a society work, a civilization work.
—Vince Lombardi

I had the opportunity to attend the first Congress on Integrated Clinical Systems Management in March. There were 150 attendees from eight different professional organizations that sponsored the Congress. The attendees represented middle- to high-level managers across the country. They were all there for one purpose: to build and learn management skills and strategies for a changing health care environment—one that will require honest teamwork across all professions. The message was clear that we need to break down the professional “silos” while preserving the value that each profession brings to the table. This Congress was really an inspiration and example of what can occur when all the professions come together for a common purpose. Our ability to work together and collaborate as health care professionals will define tomorrow’s system. The Congress will be offered again next spring in Dallas, TX, and I highly recommend it to those of you interested in remaining on the cutting edge of health care reform.

Our Bulletin focuses this month is pulmonary function testing. Dr. Peter Southorn, who hails from the Mayo Clinic and is currently serving as our section’s medical advisor, has written an excellent article on medical directors. We appreciate his support and contributions to this section. Our medical directors are essential members of the team. Their training and expertise in pulmonary diagnostics is critical to the success of all laboratories. Take the time to keep your medical director up-to-date on all technical advances. All medical directors should have copies of the AARC Clinical Practice Guidelines applicable to pulmonary diagnostics. The orientation process for medical directors should be as complete and thorough as it is for your therapists. Thank your medical directors for their time and keep them involved in the team.

Catherine Foss has contributed another superb article on plethysmography. These hints should help improve technique and the ease of use. This remains one of the tests we surround with mystery, but it is really not difficult to understand or use after the proper education is offered. This article will give you a few more tools to effectively utilize this testing modality. (The Summer 1996 edition of the Bulletin also contained several articles about whole body plethysmography, asthma, and airway resistance measurements.) Dan Draper has contributed an article on MIPS/MEPS testing that should prove informative to all, and Dr. Susan Harding has authored an article on gastroesophageal reflux in asthma that addresses issues relevant to pulmonary diagnostics.

Once again, I would like to encourage all section members to nominate a colleague for the Practitioner of the Quarter. (The form appears at the end of this issue.) There are many deserving members in this section!

It is not too early to begin planning for the AARC International Respiratory Congress in New Orleans, LA this December. Remember that this is our 50th anniversary! Our next issue will feature the program highlights for the Diagnostic Section.

Thanks to all whose individual commitment made this issue of the Bulletin another great team effort!

NOTES FROM THE MEDICAL DIRECTOR:
IMPORTANCE OF THE MEDICAL DIRECTOR TO THE PULMONARY FUNCTION LAB
by Peter Southorn, MD

I have had a professional relationship with several highly respected pulmonary function laboratories in my career, and a common feature of each has been capable leadership by the medical director. Through this individual’s qualifications, leadership ability, and personality, the lab’s work enjoyed kudos among the medical community and the integrity of its reports were universally appreciated as positively impacting patient care. Of course, an effective pulmonary function laboratory needs the professionalism of multiple people—technicians, supervisors, and the medical director. But in today’s environment of managed care and cost cutting, the importance of the medical director’s role is more relevant than ever.

Focusing on the medical director’s role, it is obvious that this individual has to wear multiple hats—administrative, supervisory, clinical, and last, but not least, education and research. The medical director is ultimately accountable to his/her medical colleagues and administrators for the accuracy and cost effectiveness of the tests performed. To accomplish these goals, he/she must be involved in developing the test procedures, including equipment selection; establishing lab policies pertaining to these tests; and ensuring that the methodology chosen meets national standards and is in compliance with state, federal, JCAHO, and other national mandates.
Working with his or her laboratory colleagues, the medical director must ascertain that patients are tested appropriately by competent, trained staff, and that equipment is calibrated and preventative maintenance is performed. The medical director is also responsible for the accurate and timely performance of tests and the quality of the interpretation of the test findings. A good medical director who has the respect of his/her colleagues will be sought out for advice concerning test selection and the use of the PFT facilities. Therefore, the medical director is an ambassador for the laboratory. This individual’s on-site presence serves as an important resource, particularly with patients who have complicated cardiac or respiratory conditions or who require medical interventions during the testing procedures. The medical director’s presence also ensures that patients and staff are at minimum risk in the laboratory.

From an educational point of view, the medical director has an important role in networking with his/her hospital staff, medical colleagues, and others to ensure that tests are used appropriately. This entails teaching not only staff, but residents, medical students, respiratory therapists, and other groups impacted by the work of the laboratory. Correct utilization and patient care depend on appropriate understanding of the physiologic basis behind the tests performed in the laboratory. In a teaching hospital setting, any research the laboratory performs must involve the medical director. Much remains to be done in this field, but the benefits are already apparent. For example, most enlightened physicians now understand how valuable spirometry is in the early detection of COPD.

In this account, I have given you my personal bias on why pulmonary function laboratories need a strong medical director. I believe the views I have expressed, however, are almost universally held by physicians. The credibility of the laboratory, its value, and the value placed on it, all depend on the respect that the medical director commands and his or her ability to successfully and consistently run the pulmonary function laboratory.

AARC Urges Caution As FDA Proceeds Toward CFC-Free Metered Dose Inhalers

The AARC recently provided comments to the Food and Drug Administration on their Advanced Notice of Proposed Rule Making regarding changes to regulations affecting the propellant in metered dose inhalers (MDIs).

The FDA is seeking to implement the Montreal Protocol, a pact that will ultimately result in a worldwide ban on chlorofluorocarbon (CFC) products. CFC ingredients are the propellants that are used to deliver medications to patients suffering from asthma, emphysema and other diseases through an MDI. Respiratory therapists are in the forefront of health care providers who educate and train patients to properly use MDIs.

The FDA is seeking to phase out current CFC products as CFC-free propellants become available. However, in a letter from AARC President Kerry George, the association warned the FDA about not removing current products from the marketplace until appropriate alternate products are widely available. In his letter to the FDA, George says, “We believe patients and the physicians who prescribe the MDIs must have a wide range of options until an equally wide range of CFC-free MDIs are available. Elimination of a particular active ingredient after 12 months of a CFC-free alternative, will not afford this necessary range of options.”

George also stated that Medicare has discontinued large and small volume nebulizers and hand-held ultrasonic nebulizers as covered devices. This policy “has the result of transferring hundreds of thousands of Medicare patients from clinically effective and appropriate MDI alternatives into using MDIs. We believe this will make the FDA’s goal of easing the transition to CFC-free MDIs more difficult, because usage and dependence on current MDIs have now tremendously been increased,” said George.

He also urged the FDA to work with and educate Medicare policymakers on the appropriateness of this Medicare regulation.

Call For Nominations: Chair-Elect & Bulletin Editor

Nominations are now being accepted and encouraged from interested practitioners for the positions of Diagnostic Section chair-elect and Diagnostic Section Bulletin editor. Nominees must be AARC members and active members of the Diagnostic Section. Please send your nomination(s) to Carl Mottram or Susan Blonshine at the addresses listed on the last page of this issue.

ARCF “Silent Auction” Offers RC Managers The Chance To Acquire Equipment And Supplies At A Discount

In an effort to increase the amount of funds available for important research projects and other programs aimed at positioning the RCP for success in the managed care environment, the American Respiratory Care Foundation is planning to conduct its first-ever “Silent Auction” during the ARRC’s 43rd International Respiratory Congress, scheduled for December 6-9 in New Orleans, LA. All AARC members and officially registered attendees at the Congress will be eligible to bid onsite or they may participate in the pre-meeting bidding that will take place November 1-30.

While many of the items at the auction will be geared toward individual bidders, much of the inventory will consist of respiratory equipment and supplies designed to appeal to respiratory care managers working under increasingly restrictive budget constraints. Since opening bids for all donated items will be set at approximately 25% of retail value, the Silent Auction offers an outstanding opportunity for...
managers in all care settings to acquire much needed equipment at discounted prices.

RC managers or others with purchasing authority are encouraged to take advantage of this opportunity by working with their purchasing departments now to acquire the necessary purchase requisitions. In most cases, auction items will be shipped directly by the donor to the individual or institution with the winning bid.

A preliminary catalog of items will be included in the October issue of AARC Times to assist bidders in planning for the bidding process and to allow those unable to attend the Congress the opportunity to participate in pre-meeting bidding. A final catalog of items will be distributed at the meeting in December.

All funds raised by the auction will go directly into the ARCF’s unrestricted fund supporting educational grants, research projects, practice surveys, consensus conferences, and other philanthropic programs.

The Foundation is currently soliciting items for the auction from a variety of sources and plans to have a wide selection of products in all price ranges available for bidding. The solicitation of items for the auction will continue through September 30. Anyone wishing to donate an item (minimum estimated value of $100) may do so by contacting Brenda DeMayo at the ARCF Executive Office at 11030 Ables Lane, Dallas, TX 75229, (972) 243-2272.

Vladimir Miroslav, Childhood Anxiety, and Maximal Respiratory Pressures
by Daniel V. Draper

Daniel Draper is senior clinical applications specialist at SensorMedics Corporation

As a youth, I recall sitting in front of the television set during the Summer Olympic Games and marveling at the size and strength of the weight-lifters from the Soviet Union. They dominated the event, and no-one’s real surprise. Oh, these mutant humans—powerful behemoths that claimed fame solely by lifting very heavy metal objects off the ground, and then putting them back down again!

Color this historical anecdote with the prevailing notion of the times—that the Russians, as a unified nation, were driven solely by the war-mongering passion of world domination—and the reason for such sarcasm becomes apparent. Many of my age were, indeed, raised amidst the back-drop of a “Red Scare,” a deep set fear of the Soviets as an imminent threat to our peaceful free existence. Therefore, in order to trivialize their domination of this Olympic event, my friends and I would derogate the sport, punishing its participants with insults like “numbskull,” “bonehead,” and others far too colorful to list here.

At about the same time, the concept of aerobics was becoming popular. Countless publications were listing the benefits of aerobic exercise, while derogating the health and fitness benefits associated with weightlifting. Suddenly, the social order of beautiful people put as much emphasis, if not more, on the aerobically-trained athlete with the thin, svelte, zero-body fat physique. The bodybuilder somatotype was no longer the “picture of health,” much to the chagrin of many. It became easy to align overall fitness, endurance, and well-being with the aerobic athlete. Unfortunately, the converse opinion, i.e., that the bodybuilder was less fit, of limited endurance, and not as healthy, was largely incorrect.

And so we mercilessly slammed the Russian brutes—the bullies that we loved to hate—suggesting that their weightlifting prowess would surely lead them to an early grave. And the free world would be saved!

Concept of muscular strength and endurance

In the clinical setting, when we appraise the muscular endurance of the respiratory system, we typically consider the role of clinical exercise testing with a cycle ergometer or a treadmill, and possibly the MVV test as a quick assessment of the ventilatory apparatus’ ability to maintain a breathing sprint. The concept of employing tests of maximal inspiratory pressure (MIP) and maximal expiratory pressure (MEP) to discern the overall fitness and endurance of the ventilatory apparatus was a foreign one, as the MIPs and MEPs tests were considered more to be tests of the static power of the muscles of respiration, as opposed to tests that could divulge the ability of the respiratory muscles to sustain prolonged efforts (endure) associated with labored breathing—breathing against a “load.”

Yet individual muscle groups can be characterized by their group strength and endurance. These two indices of muscle function are not mutually exclusive. On the contrary, they are intimately related, such that one characteristic of muscle fitness cannot be considered without the other, at least not as muscles function as an integrated system in the human body. Since the MIP and MEP tests provide valuable data regarding the static strength of the respiratory muscles and their ability to overcome load (lift the weight), and since respiratory muscle strength and endurance are related, we can predict one’s ability to endure a breathing load for a period of time, whether it be a few minutes, a few hours, days, weeks, months, or even a lifetime.

The most typical breathing load mechanisms are expressed as an increase in inspiratory or expiratory resistance, or sheer fatigue associated with the increased ventilatory demands associated with carbon dioxide retention and the attendant hyperventilation that it stimulates. Patients experiencing these types of breathing loads are commonly encountered during the process of ventilator weaning. However, the MIPs and MEPs results are certainly not limited to the role of a weaning parameter.

The testing of maximal static pressures: The maximal inspiratory and expiratory pressures (MIPs and MEPs)

Let’s call this section “watch Boris lift a heavy weight with his lungs alone—no hands!” The measurement of maximal respiratory pressures has become a routine procedure in many pulmonary function labs. The MIP is a good index of the muscular strength of the diaphragm (the prime ventilatory driver muscle). The MEP measures the strength of the abdominal, intercostal, and other accessory muscles of ventilation.
Maximal inspiratory pressure test

In performing the maximal inspiratory pressure maneuver, the patient is typically seated (unless severely overweight). Spirometry and, ideally, static lung volumes should be conducted prior to the pressures test, with the results included for interpretation. The patient is instructed to exhale slowly and completely to residual volume (RV), seal the lips firmly around the mouthpiece, and then inhale with as much force as possible. A shutter mechanism is triggered to close at end exhalation, resulting in a near-complete occlusion. A small orifice, interposed between the patient and the shutter, remains open during shutter activation. The hole, often referred to as a Hyatt Leak or a Hyatt Trumpet (after its developer, Robert Hyatt), is designed to allow a very low flow rate, preventing the patient from sustaining pressure with his/her cheeks. Typically, multiple maneuvers are conducted, with an acceptable maneuver consisting of sustained volitional maximal effort for about 2 seconds without mouth leaks. The maximum MIP values are reported, either as the average of the largest (most negative) three efforts, or the largest two efforts that vary by less than 10%.

Maximal expiratory pressure test

The maximal expiratory pressure test (MEP) is performed as a reciprocal maneuver; that is, the patient inspires maximally to total lung capacity (TLC), then, after shutter activation, performs a maximal exhalation effort. The Hyatt leak disallows participation by the cheeks. The reproducibility and reporting criteria are the same as previously mentioned.

Ventilator Weaning

Because the MIP/MEP test relates to muscle strength and endurance, it is not surprising that the test is employed as an index of successful weaning from mechanical ventilators. In the intensive care unit, a MIP more negative than -30 cm H2O is a favorable indicator for weaning. The maximal isometric expiratory pressure (MEP) correlates reasonably well with coughing ability and muscle strength.

Relationship of Maximal Pressures to Lung Volume

There is a strong positive correlation between the forced vital capacity volume (FVC) and the MIP. Given that the MIP reflects the strength of the primary respiratory drive muscles, especially the diaphragm, as well as the muscles normally involved as primary expiratory drivers, this is not surprising. The limit to maximal inspiration (upper limit of FVC) is reached when the inspiratory muscles can no longer overcome the elastic recoil of the lung and the resistance of the chest wall to deformation. In addition, the lower limit of the FVC, or the residual volume, is largely determined by the strength of the expiratory muscles. In young adults, RV occurs during exhalation when expiratory muscle strength can no longer overcome the elastic recoil of the chest wall. In older persons, it appears that RV is determined primarily by the increased airways resistance at low lung volumes. Thus, the lower limit of the FVC depends more on the strength of the expiratory muscles and the subject’s maximal cooperation by exhaling completely for a prolonged period of time, than on overcoming the elastic recoil of the chest wall. MEP primarily measures the strength of the inspiratory muscles; however, since we measure MIP at RV, MEP is also affected by the strength of the expiratory muscles used to reach RV.

The MIP and MEP values become more meaningful, certainly from a clinical interpretation perspective, when it is referenced to actual lung volumes like the measured residual volume and total lung capacity. The relationship becomes very clear when one appreciates force velocity relationship of the muscle for a given degree of stretch. When a muscle is stretched to, at, or near its limit, its force velocity relationship is compromised. If you are handed a very heavy weight when your arms are fully extended and then asked to ‘curl’ the weight toward your chest, it will be difficult, as the muscle function is suboptimal at this initial degree of stretch. In this scenario, you may feel like the Olympic weight lifters representing the remote island of Bora Bora—not very strong. Now consider that the same weight is handed to you as your arms are flexed midway between full extension and full contraction. Curling the weight toward your chest is significantly easier, primarily because the muscles used to curl the weight have a more optimal force velocity relationship. You feel like Arnold as they slide the gold medal around your burly neck with the strains of “The Star-Spangled Banner” echoing from the rafters.

And so it is with the muscles of respiration. When the chest wall is deformed, for example by emphysema, the muscles are at suboptimal force-velocity relationships to effect efficient ventilation. They may be performing a sort of “ventilatory steal.” That is, they are ripping-off large
amounts of adenosine triphosphate (ATP) just to overcome the ventilatory loads—the constraints to normal ventilation. If too much ATP is being used just to support the process of ventilation, less is available to the other muscles to perform work. Evidence the dyspnea cycle—.the patient begins to work harder to ventilate, stealing ATP molecules from other essential and vital muscle groups. Since less ATP is available, and the body adapts to conserve the reserve ATP to support vital functions like cardiac and GI activity, very little is available to the working muscles of ambulation. So the patient gets tired climbing two flights of steps. He or she deconditions further. He or she steals even more ATP. Now the patient gets tired climbing one flight of steps. Further deconditioning. Soon it’s, “I think I’ll take the car to the local mailbox, 40 yards away.” Further deconditioning and loss of reserve. The end result, without a process to unravel the dyspnea cycle, is death.

The MIP and MEP values, when properly referenced, provide some indication as to the force velocity relationship characteristics of the muscles of respiration. By monitoring the MIP and MEP values over time, the ability of the ventilatory apparatus to overcome load is trended, providing useful data to the interpreter.

**Lung Volume Reduction Surgery**

MIP and MEP values are useful screening and follow-up parameters for lung volume reduction surgery (LVRS) candidates. Recall that the LVRS procedure is used to remove damaged lung tissue that acts as a nonventilating, physiological “sink.” When the “bad” lung parenchyma is excised, it is common for the chest wall to retract towards a normal volume, while the “good” lung tissue expands somewhat to fill the void. They meet, usually (but not always, for reasons that are now being widely studied), at a level characterized by a more optimal force velocity relationship for the muscles of respiration. Therefore, the load is diminished and the ventilatory steal is lessened, which effectively increases the ATP pool for all of the other vital muscle groups and the muscles of locomotion as well. The patients often report, at least initially, that they feel better. The results of the MIP and MEP tests, when compared pre-post surgical intervention, can serve to quantify the magnitude of the improvement in muscle strength and provide the interpreter with useful clinical research data about the efficacy of the surgery.

**Malnourishment**

Recall that malnourishment, an oft-associated clinical manifestation of the severe COPD patient, causes significant erosion of both diaphragmatic muscle mass and inspiratory drive function. Even a relatively small loss of weight below ideal body weight results in significant, and disproportionate, loss in the mass of the diaphragm. Performing the MIP and MEP tests in conjunction with indirect calorimetry, can be useful in quantifying the compromise to ventilation secondary to malnutrition, and providing data as to proper and effective replenishment of nourishment to restore the muscles of ventilation to a more optimal level of performance.
PRACTICAL TIPS FOR BODY PLETHYSMOGRAPHY TESTING
by Catherine Foss

Editor’s Note: This article was requested by a lab that just bought a new box, and had uncertainties regarding the use of equipment. It is generic to all vendors’ equipment.

1. Patient Maneuvers
   A. Demographics
      1. Measured accurate Height and Weight not asked from patient
      2. If unable to obtain height due to disability, measure arm span middle finger to middle finger; est. normal height Male = arm span/1.03 (if >/= 16 years old); est. normal height female = arm span/1.01 (if >/=16 years old)
      3. If testing a small child, the parent may hold the child in his/her lap for testing. Be sure to enter the combined weight of both the parent and child. The parent must hold his/her breath during each maneuver the child performs.
   B. Practice with the door open
      1. Explain
      2. Demonstrate
      3. Adjust mouthpiece/armpiece that holds mouthpiece so that the patient is neither rigidly upright nor slumping.
      4. Practice and correct any problems
      5. Ask if the patient has any questions
   C. Close the door: Check visually for any leaks in the box seal
   D. Thoracic Gas Volume (TGV)
      1. Wait a minimum of one minute for thermal equilibration (check vendor’s recommendations for any longer warm-up)
      2. Noseclips and a large flanged mouthpiece should be used; hands on cheeks or one hand under jaw, with thumb on one side of cheek and fingers on the other side. Do not prop elbows on any armrests.
      3. Wait for a minimum of four stable tidal breaths; if patient is anxious or breathing deeply, he/she is not at FRC. Work to calm and relax patient. If the patient is breathing irregularly, the end tidal average may not be correct.
      4. Have the shutter close at end tidal
         a. Coach the patient to pant gently, so loops are kept on screen; too deep will over-range the transducer.
         b. Pant at 50-100cc, at 1 hertz, which is 1 pant per second
         c. If system has auto shutter closure, do not coach patient to pant until the shutter closure is audibly heard.
         d. Do not “beat the computer” by getting ahead on keystrokes; allow the box to vent between efforts to prevent overheating. Wait for the computer prompt on screen for the next effort to begin.
      5. If performing a SVC linked with TGV
         a. Verify a stable end tidal breathing pattern and the location of shutter closure.
         b. Report the largest of three satisfactory VC maneuvers; the 2 largest would be within 5% or 100 ml, whichever is greater.
         c. Compare SVC with FVC; the SVC should be the same or larger than FVC.
      6. Perform a minimum of 5 reproducible efforts
   E. Airways Resistance
      1. After several stable tidal breaths, coach the patient to pant. The coaching instructions will need to vary with each patient. Coach to optimally obtain panting speed which will “close” the open shutter loop.
         a. Too fast will “open” the loop.
         b. Too slow will “open” the loop.
         c. Usually 2 - 3 Hertz
         d. Generally, severe COPD patients will require a slower panting speed than normal or restrictive patients.
      2. Coach the patient to keep pants small enough to visually keep the loops on screen. The open shutter loops should extend just beyond the + / - .5L/sec marks.
      3. Be sure to accumulate a minimum of 4-5 open shutter panting loops before closing the shutter.
      4. Strive to obtain no overlapping figure 8-shaped open shutter loops. Sometimes changing the panting speed and size will clear up this problem.
      5. The V-Pant is the volume the patient is panting at when the maneuver is performed. It is not the volume the patient is moving.
      6. Perform a minimum of 5 reproducible efforts
   F. Data Evaluation
      1. TGV
         a. Adjust the best fit line through the most linear portion of the TGV loop/line.
         b. Can also be measured point to point.
         c. If the patient performs a “good” effort, the results of both methods will come out the same.
         e. Use tangents that agree within 5% variance to average or 200 ml. Average 3 or more efforts.
         f. FRC from a plethysmograph should be greater than, or equal to, FRC measured by gas dilution methods of nitrogen washout or helium dilution. It should also be greater than the single breath VA measured during a DLCO test.
2. RAW
   a. Average 3-5 acceptable efforts
   b. Should be within 10% variance
   c. SGAW should be + / -.01 if mean is less than .18
   d. SGAW should be + / -.02 if mean is greater than .17
   e. Open shutter flows are measured between the +.05 and -.05 L/sec marks for the typical US method of measuring.
   f. If European Ulmer fit method is used, applicable predicteds must be used. This method measures point to point and has very different results in obstructed patients.
   g. Be consistent on methods used to evaluate graphic data between one effort to the next on a given patient.
   h. Suggestions
      1. If expiratory is down on the computer’s screen, measure on the right side of the open shutter loop, unless the section there is curved; then measure on the left.
      2. Train all lab staff to measure and adjust consistently the same way.
      3. If encountering an upper airway obstruction, hysteresis will be seen in the + / -.5L/sec section. Adjust the tangent to best fit as the patient line crosses the + / -.5 L/sec lines

3. QA
   a. Suggest a “print screen” of graphic records to save in patient’s chart.

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GASTROESOPHAGEAL REFLUX IN ASTHMA

by Susan M. Harding, MD

Asthma is exacerbated by multiple triggers which produce bronchial smooth muscle contraction, mucous production, and ignite an inflammatory response resulting in cell recruitment and the release of inflammatory mediators. Despite anti-inflammatory agents and bronchodilators, asthma continues to impair the quality of life of millions of Americans. Trigger avoidance, patient education, and asthma medications are key elements of asthma therapy. One common, often overlooked trigger of asthma is gastroesophageal reflux (GER). Anti-reflux therapy can improve asthma outcome in many asthmatics with GER.

Prevalence of GER in asthmatics

Sir William Osler first reported that asthmatics, “learn to take their large daily meal at noon in order to avoid nighttime asthma, which occurred if they ate a full supper.”

Gastroesophageal reflux symptoms are common in asthmatics. Field et al. noted that 77% of asthmatics reported heartburn, 55% complained of regurgitation, and 24% experienced swallowing difficulties. In the week prior to completing the questionnaire, 41% of asthmatics noted reflux-associated respiratory symptoms and 28% used their inhalers while experiencing GER symptoms. Importantly, some asthmatics have significant GER without esophageal symptoms. In difficult-to-control asthmatics, GER was clinically “silent” in 24%.

Asthmatics also have a high incidence of esophagitis and abnormal esophageal acid contact times. Sontag et al. evaluated 186 consecutive adult asthmatics with endoscopy and esophageal biopsy, finding that 43% had evidence of esophagitis or Barrett’s esophagus. Using 24-hour esophageal pH monitoring and esophageal manometry in 104 consecutive asthmatics and 44 controls, the same group observed that 82% of asthmatics had higher than normal esophageal acid contact times. Also, asthmatics, compared to controls, had low esophageal sphincter (LES) pressures and more frequent reflux episodes. Multiple studies now show that GER is quite prevalent in the asthma population. Gastroesophageal reflux needs to be considered in every asthmatic.

Table 1: Esophageal Findings in Asthmatics

<table>
<thead>
<tr>
<th>Symptom or Test Result</th>
<th>Percentage of Asthmatics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heartburn2*</td>
<td>77%</td>
</tr>
<tr>
<td>Regurgitation2</td>
<td>55%</td>
</tr>
<tr>
<td>Dysphagia2</td>
<td>24%</td>
</tr>
<tr>
<td>“Clinically Silent” GER3**</td>
<td>24%</td>
</tr>
<tr>
<td>Esophagitis4</td>
<td>43%</td>
</tr>
<tr>
<td>Abnormal Esophageal pH Values5</td>
<td>82%</td>
</tr>
</tbody>
</table>

*Numbers respond to references.
**GER - Gastroesophageal reflux.

Does asthma predispose patients to develop reflux?

The pathophysiology of gastroesophageal reflux is quite complex. Factors controlling the competence of the gastroesophageal junction and the lower esophageal sphincter (LES) are altered during an asthma attack. Normally, gastric pressure is positive in relation to pleural and esophageal pressure because the latter structures are located in the chest. With increased airflow obstruction during an asthma attack, very negative pleural pressures can increase the pressure gradient between the thorax and the abdominal cavities, predisposing the patient to reflux acid into the esophagus. In addition, the crura of the diaphragm, through which the esophagus exits the thoracic cavity, contributes to the LES pressure. Overinflation and air trapping may lead to flattening of the diaphragm, further impairing the anti-reflux barrier. In clinical studies, Moote et al.
showed that methacholine administered to asthmatics to induce airflow obstruction produced longer periods of reflux.⁶

Bronchodilator medications may also decrease the competence of the gastroesophageal junction. Theophylline is a methylxanthine which increases gastric acid secretion and lowers LES pressure.⁷ However, it is debatable whether these findings have clinical implications. Hubert et al. performed a randomized, double-blind crossover study in asthmatics and administered oral theophylline or placebo for seven days while assessing symptoms, pulmonary function tests (PFTs), and 24-hour esophageal pH tests.⁸ Although PFTs improved on theophylline, no differences were observed between the two groups with respect to number of reflux episodes, total duration of reflux, and the number of reflux episodes lasting more than five minutes.⁸

b-adrenergic agents also have been implicated. Studies show that orally or intravenously administered b₂-adrenergic agents decrease LES pressure.⁷ However, Schindlbeck et al. found that inhalation of albuterol either by metered dose inhaler or nebulizer had no significant effect on GER or esophageal motor function.⁹ Thus, it is unlikely that inhaled b₂-adrenergic agents significantly worsen GER in asthmatics.

In the largest study to date, Sontag et al. studied 44 controls and 104 adult asthmatics with 24-hour esophageal pH monitoring.⁵ Asthmatics had significantly more acid reflux than controls. However, the 74 asthma patients receiving theophylline, b₂-adrenergic agents, and/or prednisone did not have any more reflux than the 30 asthmatics not taking bronchodilators.⁵

More studies are needed to further understand why GER is more prevalent in asthmatics than in control populations.

**Pathophysiology of GER: Associated bronchoconstriction**

There are two potential mechanisms whereby esophageal acid triggers airflow obstruction in asthmatics. These include a vagally mediated reflex, where acid in the esophagus stimulates acid sensitive receptors, resulting in bronchoconstriction and microaspiration of gastric contents into the upper airway.

Many studies support a vagal mechanism. In a dog model, esophageal acid caused an increase in respiratory resistance which was ablated with bilateral vagotomy.⁷ In human studies, Mansfield et al. observed in asthmatics with reflux that total respiratory resistance increased 10% with esophageal acid.¹⁰ In another study, Wright et al. monitored 136 subjects before and after esophageal acid infusions and noted reductions in airflow and arterial oxygen saturation with esophageal acid which were not present with atropine pre-treatment.¹¹ However, others report conflicting results, including a study of 15 nocturnal asthmatics, where esophageal acid caused no significant change in airflow resistance.¹²

We performed a series of studies evaluating the pathogenesis of esophageal acid induced bronchoconstriction. Using esophageal infusions of normal saline and acid, we showed that peak expiratory flow rates (PEF) decreased with esophageal acid in normal controls, asthmatics with GER, asthmatics without GER, and subjects with GER alone.¹³ Esophageal acid clearance resulted in improvement in PEF in all groups except in the asthma with GER group. This bronchoconstrictor effect was not dependent on proximal esophageal acid exposure, a prerequisite for microaspiration.¹⁴ Subsequently, we infused esophageal acid while the subjects remained in the supine position. Again, esophageal acid caused a decrease in PEF and an increase in specific airway resistance in asthmatics with GER, which didn’t improve despite esophageal acid clearance.¹⁴ Vagolytic doses of atropine given before esophageal infusions partially ablated the bronchoconstrictor response, further supporting the role of a vagally mediated reflex.¹⁵

Microaspiration of esophageal acid into the upper airway can also cause significant bronchospasm. Convincing experiments were performed by Tuchman et al. in a cat model. They infused 10 ml of acid into the esophagus, noting a 1.5 fold increase in total lung resistance compared to a five-fold increase with 0.5 ml of tracheal acid.¹⁶ Furthermore, the esophageal acid bronchoconstrictor response occurred in only 60% of the cats versus 100% of the cats with tracheal acid.¹⁶ Interestingly, the effect of tracheal acid on total lung resistance was abolished after bilateral surgical vagotomy, so even in the aspiration model, the vagus nerve plays a role.¹⁶

The importance of microaspiration was also shown in humans. Jack et al. monitored simultaneous tracheal and esophageal pH in four severe asthmatics.¹⁷ Esophageal acid caused an 8 liter-a-minute decrease in peak expiratory flow rate. However, if both esophageal and tracheal acid were present, peak expiratory flow rates decreased an average of 84 liters a minute.¹⁷ So, episodes of tracheal microaspiration were associated with significant deterioration in pulmonary function.¹⁷

In summary, esophageal acid causes bronchoconstriction by a vagally mediated reflex. However, if microaspiration is present, there is further augmentation of this bronchoconstrictor response. It is interesting that the vagus nerve plays a role in both mechanisms.

**Table 2: Symptoms of Gastroesophageal Reflux in Asthmatics**

<table>
<thead>
<tr>
<th><strong>Esophageal Symptoms</strong></th>
<th><strong>Heartburn</strong></th>
<th><strong>Regurgitation</strong></th>
<th><strong>Dysphagia</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Extraesophageal Symptoms</strong></td>
<td><strong>Sore Throat</strong></td>
<td><strong>Choking</strong></td>
<td><strong>Hoarseness</strong></td>
</tr>
<tr>
<td><strong>Worsened Asthma Symptoms With</strong></td>
<td><strong>Eating</strong></td>
<td><strong>Reflex Symptoms</strong></td>
<td><strong>Supine Position</strong></td>
</tr>
<tr>
<td><em><em>Using an Inhaler While Experiencing GER</em> Symptoms</em>*</td>
<td><strong>Clinically Silent</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*GER - Gastroesophageal reflux.*
Diagnosing GER in asthmatics

The patient history is extremely important in diagnosing GER in asthmatics. Table 2 illustrates esophageal and extraesophageal symptoms of GER. Unfortunately, many physicians do not routinely inquire about heartburn, regurgitation or dysphagia. Worsened asthma symptoms may be noticed after eating a high fat-containing meal or foods which lower the LES pressure, including chocolate, peppermint, caffeine or alcohol. Also, inquire if asthma symptoms are associated with reflux episodes, or if a patient uses his/her inhaler while experiencing reflux symptoms. If the patient’s history is consistent with GER, no further diagnostic studies are necessary and a trial of aggressive anti-reflux therapy should be started.

Table 3: Diagnostic Tests for Gastroesophageal Reflux*

<table>
<thead>
<tr>
<th>Test</th>
<th>Specificity</th>
<th>Sensitivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>24-hour Esophageal pH</td>
<td>95%</td>
<td>93%</td>
</tr>
<tr>
<td>Endoscopy with Esophagitis - Grade 1</td>
<td>96%</td>
<td>68%</td>
</tr>
<tr>
<td>Barium Esophagram</td>
<td>93%</td>
<td>65%</td>
</tr>
<tr>
<td>Esophageal Manometry with LESP** &lt; 10mm Hg</td>
<td>84%</td>
<td>58%</td>
</tr>
<tr>
<td>Positive Bernotein Test</td>
<td>86%</td>
<td>77%</td>
</tr>
</tbody>
</table>

*Reference number 19.
**LESP = Lower esophageal sphincter pressure.

Some asthmatics with GER may not experience esophageal reflux symptoms, and esophageal testing is required to identify these patients. Table 3 reviews the sensitivity and specificity of esophageal tests available to diagnose GER. Twenty-four-hour esophageal pH testing is considered the gold standard and has a sensitivity and specificity of greater than 90%. The American Gastroenterological Association’s Medical Position Statement on the Clinical Use of Esophageal pH Recording recommends testing asthmatics suspected of having reflux triggered asthma. Twenty-four-hour esophageal pH testing also allows correlation of asthma symptoms with reflux episodes. Referral to a gastroenterologist is recommended for patients in whom empiric therapy of GER is unsuccessful or in those patients who have symptoms suggesting complicated GER, such as esophagitis, esophageal structure, Barrett’s esophagus, or neoplasm.

Does anti-reflux therapy improve asthma outcome?

Since esophageal acid is a trigger of asthma, aggressive anti-reflux therapy should improve asthma symptoms and pulmonary function. In difficult-to-control asthmatics requiring more than 10mg of prednisone QOD, anti-reflux therapy and inhaled corticosteroid use were the two most helpful interventions in converting them into easier-to-control asthmatics. However, previous medical trials using antacids or H2-receptor antagonists at standard doses have reported only mixed results, as illustrated in Table 4. Referring to a gastroenterologist is recommended for patients in whom empiric therapy of GER is unsuccessful or in those patients who have symptoms suggesting complicated GER, such as esophagitis, esophageal structure, Barrett’s esophagus, or neoplasm.

Table 4: Medical Trials of GER-Related Asthma*

<table>
<thead>
<tr>
<th>Author</th>
<th>Number Patients</th>
<th>Number Controls</th>
<th>Tx</th>
<th>Asthma Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kjellen et al.7</td>
<td>31</td>
<td>31</td>
<td>Antacids/Alginic acid</td>
<td>54% improved</td>
</tr>
<tr>
<td>Goodall et al.7</td>
<td>18</td>
<td>Placebo crossover</td>
<td>Cimetidine 200 mg QID for 6 weeks</td>
<td>Increased PEF and decreased asthma symptoms in 78%</td>
</tr>
<tr>
<td>Harper et al.7</td>
<td>15</td>
<td>——</td>
<td>Ranitidine 150 mg BID for 8 weeks</td>
<td>Decreased symptoms and improved PFT’s over entire group</td>
</tr>
<tr>
<td>Nagel et al.7</td>
<td>15</td>
<td>Placebo crossover</td>
<td>Ranitidine 450 mg a day for 1 week</td>
<td>No difference</td>
</tr>
<tr>
<td>Ekstrom et al.7</td>
<td>24</td>
<td>Placebo crossover</td>
<td>Ranitidine 150 mg BID for 4 weeks</td>
<td>Mild decrease nocturnal symptoms and decreased MDI use</td>
</tr>
<tr>
<td>Depla et al.7</td>
<td>1</td>
<td>——</td>
<td>Omeprazole 20 mg a day for 3 months</td>
<td>Complete relief of symptoms</td>
</tr>
<tr>
<td>Meier et al.21</td>
<td>15</td>
<td>Placebo crossover</td>
<td>Omeprazole 20 mg BID for 6 weeks</td>
<td>29% increased FEV1 by 20%</td>
</tr>
<tr>
<td>Ford et al.22</td>
<td>11</td>
<td>Placebo crossover</td>
<td>Omeprazole 20 mg a day for 4 weeks</td>
<td>No difference</td>
</tr>
<tr>
<td>Harding et al.18</td>
<td>30</td>
<td>——</td>
<td>Omeprazole 20-60 mg/d; documented acid suppression</td>
<td>73% increased PEF or decreased symptoms by 20%</td>
</tr>
</tbody>
</table>

* GER = Gastroesophageal reflux
† QID = Four times a day
§ BID = Twice a day
‡ PFT’s = Pulmonary function tests
¶ PEF = Peak expiratory flow rate
¶¶ MDI = Metered dose inhaler
** FEV1 = Forced expiratory volume at one second

We performed a trial where daily asthma symptoms, peak expiratory flow rates, and spirometry were assessed before and after three months of acid suppressive therapy with omeprazole. Asthmatics with GER who had a greater than 20% improvement in asthma symptoms and/or a 20% improvement in peak expiratory flow rates with three
months of acid suppressive therapy were considered asthma responders by a priori definitions. Seventy three percent were asthma responders. Responders reduced their asthma symptoms by 57%, improved their peak expiratory flow rates by 9%, and had significant improvement in pulmonary function tests, including the FEV1, forced expiratory flow during the middle half of forced vital capacity (FEF25-75%), and peak expiratory flow rate, with acid suppressive therapy. Also, nearly one-third of asthmatics with GER required more than 20mg of omeprazole a day to control GER. Two variables predicted asthma improvement. The presence of regurgitation at least once a week and/or excessive proximal acid reflux by 24-hour esophageal pH testing predicted asthma improvement with a 100% sensitivity and a positive predictive value of 79%. Thus, documented acid suppression with proton pump inhibitors can improve asthma in nearly 75% of asthmatics with GER. This response rate approximates the asthma improvement rate with anti-reflux surgery.

Combined results of surgical therapy trials reported to date show that 34% of asthmatics were free of asthma symptoms post-operatively, 42% were improved, and 24% were unchanged. Study design flaws include the lack of a control group, poor documentation of airflow obstruction pre- and post-operatively, and no proof that reflux was controlled in the post-operative state.

Larrain et al. compared cimetidine 300mg QID to surgical therapy (Hill procedure) in non-allergic asthmatics with GER. After six months, asthma medication scores decreased significantly in both treatment groups versus the placebo group. Asthma was considered improved in 76% of the surgically treated group, 74% of the medically treated group, and 36% of the placebo group.

**Therapy of reflux in asthmatics**

Since aggressive anti-reflux therapy can significantly improve asthma outcome in approximately 75% of asthmatics with GER, all patients should have a therapeutic trial. All patients should be educated on lifestyle therapy. Lifestyle therapy includes smoking cessation, avoiding large meals (especially four hours before bed), elevation of the head of the bed, and weight loss, if overweight. Asthmatics with GER should eat a low-fat diet and avoid foods which decrease the LES pressure, including alcohol, chocolate, peppermint, and caffeine. If possible, medications which decrease the LES pressure should be avoided, including theophylline.

Medical therapy includes antacids, H2 antagonists (cimetidine, ranitidine, nizatidine, and famotidine), proton pump inhibitors (omeprazole and lansoprazole), and prokinetic agents (bethanechol, metoclopramide, and cisapride). Bethanechol is contraindicated in asthmatics because it can induce bronchospasm. Also, metoclopramide has a 20% to 50% incidence of side effects, including fatigue, restlessness, tremor, Parkinsonism, and tardive dyskinesia. Surgical interventions include the Nissen, Toupet, and Belsey fundoplications, and the Hill gastropexy. Newer laparoscopic techniques decrease hospital time; however, their long term efficacy is unknown.

Aggressive initial therapy of GER in asthmatics should include a proton pump inhibitor (omeprazole or lansoprazole), since they provide better acid control than H2 antagonists or prokinetic agents. A trial of omeprazole 20 mg BID should be initiated and continued for three months. This dose may be necessary because Harding et al. noted that omeprazole 20mg a day did not suppress esophageal acid in 27% of asthmatics with GER. During the therapeutic trial, patients should monitor their asthma symptoms and daily peak expiratory flow rates. Anti-reflux therapy is considered successful if there is a 20% decrease in asthma symptoms or daily corticosteroid dose, or a 20% improvement in peak expiratory flow rates. If the patient improves, then chronic maintenance therapy should be considered (see below). If treatment is unsuccessful, then either the patient’s asthma is not triggered by GER and aggressive anti-reflux therapy can be discontinued or GER was not adequately controlled. In this case, 24-hour esophageal pH testing can be performed while the patient is on anti-reflux medication. If GER is not controlled on omeprazole 20 mg BID, then the dose should be increased to 40 mg BID and a prokinetic agent such as cisapride should be added.

Maintenance therapy can include a proton pump inhibitor or a high dose of a H2 antagonist. Prokinetic agents are usually used in combination with acid suppressive agents. All patients requiring a proton pump inhibitor for chronic therapy should have the option of surgery discussed, especially in younger patients, since there are still unanswered questions about the long term safety of proton pump inhibitors. Optimal surgical candidates include those with LES hypotension and normal esophageal motility.

Laparoscopic fundoplication is best employed in patients with uncomplicated reflux disease. There are no studies evaluating the cost/benefit analysis of medical versus surgical therapy for long term therapy of GER-associated asthma.

In conclusion, GER is a significant trigger of asthma. Since aggressive anti-reflux therapy can improve asthma outcome in up to 75% of asthmatics with reflux, all asthmatics with reflux should receive a three month trial of acid suppressive therapy. Further research is needed to better understand the pathophysiology of reflux-induced bronchoconstriction and to determine the optimal therapeutic management of GER in asthmatics.

**References**


**SPOTLIGHT CORNER:**

**WORKING IN AN OFFICE-BASED LAB**

Editor’s Note: This section is dedicated to highlighting labs and/or practitioners throughout the nation who are initiating or participating in innovative programs.

Mary Kay Collins, RRT, RPFT, works in a six-pulmonologist, office-based laboratory in Spokane WA, along with one other RCP. Before being seen by the physicians, patients are seen in the office’s PFT lab, where many receive pre- and post-bronchodilator spirometry and airways resistance. DLCO and lung volumes are often performed as well.

Mary Kay and her associate plan their time very efficiently so as to avoid downtime or patient delays. Frequently, she will start one patient’s spirometry, give a bronchodilator, then have the patient wait in the PFT lab waiting room while the next patient is brought in and started. Then the first patient is finished while the second patient waits for his or her bronchodilator to achieve peak effect. While giving bronchodilator treatments, Mary Kay utilizes the time to educate and examine patient technique for inhaler use.

After the patient sees the physician, he or she comes back to the lab to review the ordered medications and receive follow up training, if necessary. At this time, Mary Kay and her associate set up any home care needs, such as oxygen and home nebulizers, that are ordered. Mary Kay also fits EKGs and treadmill cardiac stress tests into her PFT schedule. However, cardiopulmonary exercise studies with gas exchange, and methacholine challenges are sent to the local hospital-based labs because the high time commitment for those tests do not allow them to fit into the sched-
ule of a small, busy lab.

Since attending a seminar sponsored by National Jewish Hospital and Medgraphics about a year ago, Mary Kay has been performing RAW on a consistent basis. Through the seminar, she obtained advanced training and theory on body plethysmography and the benefits that these data bring to the physician on interpretation. Indeed, Mary Kay had to educate the physicians in the group after she returned from the seminar, as they did not have a good working knowledge of $R_{AW}$ and $\Delta S_{AW}$ and their interpretation. After attending lectures conducted by Mike Snow, Bob Brown, and Sue Blonshine at the seminar, Mary Kay was confident in her ability to explain her new skills and knowledge to the physicians.

Mary Kay enjoys working in a physician-based lab, where the doctors are always available if questions or problems arise. Physicians also appreciate the arrangement, because when it comes time for them to interpret the tests, the technicians are right there if they have any technical questions. Mary Kay believes her patients like the physician-based lab as well, because it is non-threatening and more comfortable than a hospital lab. Also, the patient sees the same staff with each return visit. Mary Kay strives to have the patient tested in the same equipment each time they return to the office as well. The lab is currently equipped with three devices, one with helium dilution, one with $N_2$ washout, and one with body plethysmography, including $D_{CO_2}$.

Mary Kay makes it a point to participate in continuing education activities whenever she gets the chance. She is also an active supporter of her profession at the state level and tries to attend one national conference a year. All these activities are performed on her own time.

Mary Kay Collins has shown great dedication to the diagnostics specialty, and I hope that everyone in the field will be inspired to follow her example. When we continually broaden our horizons as she has done, our profession and our specialty reap the rewards.

### NCCLS guidelines target cardiopulmonary diagnostics

In an effort to bring greater standardization to the clinical laboratory environment, NCCLS reviews and approves guidelines for a wide range of laboratory procedures, including those involving cardiopulmonary diagnostics. Recently approved guidelines pertaining to the pulmonary lab include—


NCCLS also works with other organizations to develop guidelines and standards for the industry as a whole. The American National Standards Institute has recently approved the following pulmonary-related NCCLS documents as American National Standards—

- C21-A, Performance Characteristics for Devices Measuring $P_{O_2}$ and $P_{CO_2}$ in Blood Samples; Approved Standard.
- C27-A, Blood Gas Preanalytical Considerations: Specimen Collection, Calibration, and Controls; Approved Guideline.

In addition, NCCLS has several guidelines in development that may be of interest to RCPs working in the clinical lab—

- GP24 Total Cost Management: To complement the exiting NCCLS guideline on cost accounting, this new guideline will provide an overview of the basic principles of total cost management and guidance on planning and adjusting budget allowances in a capitated environment. The guideline will present a system for the estimation of the direct and indirect costs of laboratory health care and for the evaluation of financial alternatives.
- GP25, Cost Analysis at the Point of Care: This project will address calculation of the cost of use and the cost benefit of various testing methods and instruments utilized at the point of care, as compared to those traditionally used in the clinical laboratory. The resulting guideline will be a companion document to NCCLS's AST2 (Point-of-Care IVD Testing) and GP11 (Cost Accounting) guidelines.

For more information about these or other NCCLS guidelines, call (610) 688-0100. (Source: NCCLS)

### Add your name to the list: RCPs can assist AHCPR by signing up to review grant applications

Since its inception in the late 1980s, the Department of Health and Human Service’s Agency for Health Care Policy and Review (AHCPR) has provided important funding and oversight for a wide range of research efforts aimed at identifying best medical practices. Every year, hundreds of health professionals across the nation assist the agency in that goal by serving as reviewers in the peer review of re-
search grant applications. If you would like to add your name to the list, please forward a current curriculum vitae to: AHCPR, Office of Scientific Affairs, Attention: Bonnie Edwards, 2101 East Jefferson Street, Suite 400, Rockville, MD 20852, or fax your CV to Bonnie Edwards at (301) 594-0154. (Source: Research Activities, 3/97)

AARC Comments On Salary Equivalency Guidelines

The AARC recently provided comments on the proposed Salary Equivalency Guidelines issued by the Health Care Financing Administration (HCFA). The proposed guidelines, which would cover physical therapy, speech language pathology, occupational therapy, and respiratory therapy, were published in the March 28, 1997 Federal Register.

The AARC expressed concerns about three aspects of the proposed rules—

1. The compression of the registered respiratory therapist (RRT) and the certified respiratory therapy technician (CRTT) professionals with non-credentialed workers into one generic category of “respiratory therapy.”
2. The methodology and data sources used to determine the proposed respiratory therapy salary equivalencies.
3. The disregard of added costs imposed by respiratory therapy’s unique Medicare transfer relationship between a hospital and a skilled nursing facility (SNF).

In a letter to Bruce Vladeck, HCFA administrator, from AARC President Kerry George, the AARC went on record as opposing the adoption of the salary equivalency guidelines.

In commenting on the single level respiratory therapy category, George said, “The single category does not account for the higher level of compensation an RRT receives, nor the propensity for SNFs to utilize the advanced RRT practitioner with experience.”

In addition, the methodology used to determine salary equivalency rates for all of the therapy professions does not represent an equitable calculation. “The Medicare transfer agreement requirement limits the efficiency of providers in contracting for respiratory therapy services; a unique set of circumstances not faced by other therapy professions,” said George. “This must be addressed in the regulation.”

Visit AARC on the Internet—

http://www.aarc.org

RESOURCE PANEL UPDATE

An updated version of our Resource Panel appeared in the Fall issue of the Bulletin, but we are still looking for additional qualified members to add to the list. If you would like to participate in the panel, fill out the form below (we ask that you limit yourself to ten topics or less) and return it to one of the Bulletin editors at the addresses listed on the last page of this issue. Another update will be coming soon.

___ New Panel Member  ___ Returning Panel Member w/Changes

___ Please drop my name from the panel

Name:___________________________________________
Title: ____________________________________________
Institution:_______________________________________
Complete address(es) (work and/or home)
________________________________________________
________________________________________________
________________________________________________
________________________________________________
________________________________________________
Phone(s): ________________________________________
Fax:_____________________________________________
E-mail (if available) _______________________________

TOPICS

Pulmonary Diagnostics
___ Spirometry
___ Lung volumes
___ Airway Mechanics
___ Diffusing Capacity
___ Steady State Diffusing Capacity
___ Blood Gas, Electrolyte and Hemoximetry Analysis
___ Point of Care Testing
___ Bronchoscopy
___ Sweat Chloride Testing
___ Conscious Sedation
___ Cardiopulmonary Exercise Testing
___ Airways Challenge Testing
___ Pulmonary Mechanics and Occluding Pressures
___ Sleep Disorders
___ High Altitude Simulation
___ Ventilatory Drive

Critical Care Pulmonary Diagnostics
___ Indirect Calorimetry
___ Noninvasive Cardiac Diagnostics

Pediatric and Neonatal Care
___ Neonatal, Infant, Toddler, and Pediatric Pulmonary Diagnostics
___ Pediatric Bronchoscopy
___ Research
___ Occupational Health
___ Administrative Management
___ Rehabilitation & Education
___ Patient Focused Protocols
___ Clinical Practice Guidelines

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Please check the type of accreditation visit you are reporting:

- Pathology & Clinical Laboratory Services
- Home Care
- Hospitals
- Long Term Care

What was the surveyors’ focus during your last site visit?

What areas were cited as being exemplary?

What suggestions were made by the surveyors?

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

Please offer any additional comments about the site visit that will be helpful to others. (use additional sheet if necessary)
Don’t forget to make your nominations for the Diagnostics Outstanding Section Member of the Quarter award. The winner of each Outstanding Section Member of the Quarter award will be featured in an article in the Bulletin and our Specialty Practitioner of the Year will be chosen from these four winners. The winner of the Specialty Practitioner of the Year award will be honored during the Awards Ceremony at the AARC 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 Outstanding Section Member of the Quarter because

_______________________________________________________________________________________________________

_______________________________________________________________________________________________________

_______________________________________________________________________________________________________

_______________________________________________________________________________________________________

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_______________________________________________________________________________________________________

Nominee ___________________________________________ Your Name ______________________________

Hospital/School _______________________________________ Hospital/School __________________________

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 the last page of this issue.
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