Diagnostics Bulletin
May/June '00

American Association for Respiratory Care

Notes from the Chair

by Catherine M. Foss, BS, RRT, RPFT

I am pleased to introduce one of our two Diagnostic Section medical directors from the AARC's Board of Medical Advisors, Dr. William Peruzzi, in this issue. In a "Notes from the Medical Director" column, Dr. Peruzzi gives us his background in pulmonary medicine, critical care, and anesthesia, and also shares his experience in diagnostic and critical care research issues. We are pleased to have a physician of his caliber join our ranks in the Diagnostic Section.

Our two feature articles this issue highlight diagnostic areas that many practitioners are involved with: flow volume loops and bronchoscopy. Deb White, from St. Louis, takes us back to the basic art of interpreting spirometry graphics based on physiology. Her spirited writing style invites you to challenge your interpretation skills. This article is a great synopsis of common and unusual shapes seen in FVLs and the causes behind these graphic findings. Save this one for your archives – you'll definitely want to pull it out for review when students rotate through your lab. (But if you don't want to save the paper copy, remember that you can always call up past *Bulletin* issues from the Diagnostic Section area on the AARC web site – www.aarc.org.)

Our other major article comes from Dr. W. G. Petersen from Scott & White Clinic and Memorial Hospital in Temple, TX. He has written a great review for us concerning causes of low saturations that occur during and after bronchoscopy procedures. Some of the situations he describes can also occur during other pulmonary diagnostic procedures, so this article is pertinent to all practitioners, even those not currently involved in bronchoscopy or conscious sedation. His article brings to mind the NCCLS guideline for arterial blood gases, which was revised in 1999. Practitioners need to be aware of the limitations and usefulness of both pulse oximetry and arterial blood gases with co-oximetry.

In other news, there are several diagnostics-related developments in the works that you'll want to keep track of over the next few months. Watch the AARC web site for diagnostic updates on the following:

- The upcoming NCCLS guideline for pulse oximetry.
- Two new guidelines, one on the six-minute hallwalk and the other on cardiopulmonary exercise, from the ATS Committee on Proficiency Standards for Clinical Pulmonary Function Laboratories. The updated guidelines will be published in RESPIRATORY CARE and should be available on the AARC web site soon. ■

Notes from the Medical Director

by William Peruzzi, MD, FCCM

Currently, I am an associate professor of anesthesiology and chief of the section of critical care medicine in the department of anesthesiology at Northwestern University Medical School in Chicago, IL. I also serve as medical director of both the department of respiratory care and the blood gas laboratory at Northwestern Memorial Hospital. Our laboratory is responsible not only for blood gas analysis and other laboratory determinations, but metabolic cart measurements as well. I was appointed to the AARC's Board of

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Medical Advisors by the Respiratory Care Committee of the American Society of Anesthesiologists.

I obtained my undergraduate education (BS) and earned a Master of Science degree in biology/neuroscience from John Carroll University in Cleveland, OH. My MD degree was obtained from Ohio State University, where I also completed my internship and residency in the department of anesthesiology. Following my residency, I moved to Chicago, where I became a critical care medicine fellow in the department of anesthesiology at Northwestern University Medical School under the direction of Dr. Barry Shapiro. I am currently completing requirements for a Master of Science degree in health care management from the Harvard School of Public Health.

Since beginning my academic career, my research interests have included mechanical ventilatory support of victims of spinal cord injury, AIDS, and the acute respiratory distress syndrome. Also, we have conducted research in the areas of blood gas analysis in the laboratory and clinical (point-of-care) settings and metabolic function in acute injury. My team is currently involved in research similar to the above, and we have recently added, or are in the process of adding, minimally invasive hemodynamic monitoring, cerebral oximetry, and technical interface issues facing metabolic measurements in the intensive care unit.

I am very pleased to be involved with the Diagnostics Section of the AARC, and I look forward to interesting and enlightening interactions. ■

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11030 Ables Lane
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(972) 243-2272
FAX (972) 484-2720
e-mail: info@aarc.org

Kelli Hagen
AARC communications coordinator

Debbie Bunch
Bulletin managing editor

Edwards Printing
Bulletin typesetting

Section Chair
Catherine M. Foss, BS, RRT, RPFT
11276 Sandy Creek Ct.
South Lyon, MI 48178-9396
(734) 936-5246
FAX (313) 763-2059
Home FAX (248) 486-3974
e-mail: cfoss@umich.edu

Bulletin Editors
Pauline Wulbrecht, RPFT
Scott and White Hospital
c/o Pulmonary Laboratory
2401 South 31st Street
Temple, Tx 76508
(254) 724-2114
FAX: (254) 724-2497
pwulbrecht@swmail.sw.org

Joyce Canterbury RPFT, MSHA
National Jewish Center
1400 Jackson St.
Denver, CO 80206
(303) 398-1533
FAX (303) 398-1607
canterburyj@njc.org

Internet Coordinators
Susan Blonshine
sblonshine@aol.com

Steve Nelson
sbnelson@kansascity.com

Medical Advisors
Robin Elwood, MD
(405) 271-4351

William Peruzzi, MD
(312) 926-2537

All My Life's a Circle

by Deborah White, RRT, RPFT, chief technologist, pulmonary function laboratory, St. Louis Children's Hospital at Washington University, St. Louis, MO

Those of us who have been working in PFT labs for a significant portion of our lives are frequently asked, "How can you do the same test over and over and still have enthusiasm and joy for your job?" There are as many replies to this question as people who ask, but today I would answer, "It's those amazing flow-volume loops . . . You gotta love'em!" I thought it would be fun to present a series of patients whose flow-volume loops range from completely normal to amazingly, and indeed, dramatically abnormal. So dramatic, in fact, that we won't even need numbers to analyze these curves.

WHAT . . . pulmonary function tests without an FVC and FEV_1? Yes! Just the flow-volume loop. The beauty of this simple test is that it has the power to reveal so much important information regarding the dynamics of airflow – both intrathoracic (within our lungs), as well as extrathoracic (upper airways not under the influence of pleural pressure).

Unfortunately, the physics of airflow is NOT so simple. Robert E. Hyatt, R. Drew Miller, Leo Black, and colleagues wrote a series of articles in the late '60s and early '70s that are still the gold standard for airflow dynamics, the mechanics of forced expiration, and the effect of upper and lower airway lesions on the shape of the flow-volume loop. [1-4]

"Flow limitation" is a term that should be in the vocabulary of all pulmonary function technologists. It is the maximal flow that can be forced through a tube (or an airway), beyond which further increase in driving pressure does not yield increased flow[2]. The caliber of the airway and the resistive forces working on that airway determine the rate of flow through the airway.

Flow limitation can occur during breathing on inspiration and/or expiration, and the location of that limitation can be anywhere within hundreds of airways within the lungs, or outside of the lungs in the "upper airways." Our limitation in understanding this phenomenon is that we can only measure flow at one point – the mouth – and this flow is the sum total of flow through the myriad airways comprising our respiratory tract.

In the spirit of keeping this discussion something less than a dissertation, visualize a normal flow-volume loop. Think about the SHAPE of the curve. I will have to make one important disclaimer for this entire discussion. We have to assume that the flow-volume loops presented are performed from total lung capacity and that maximal effort was exhibited on both expiration and inspiration. This assumption should not be made lightly. Over-interpretation of PFTs in the face of poor patient effort can lead to many undesirable consequences, including incorrect diagnosis and/or over-treatment .

During exhalation, the peak expira-

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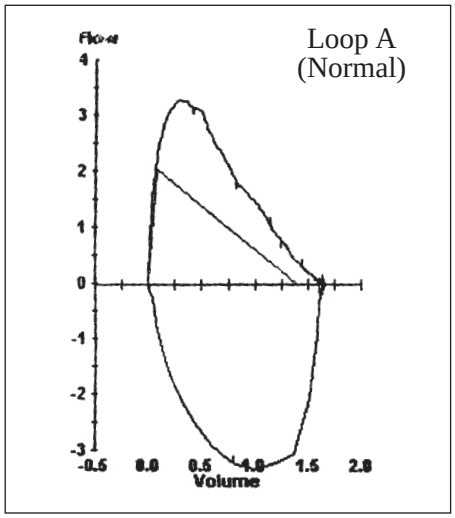

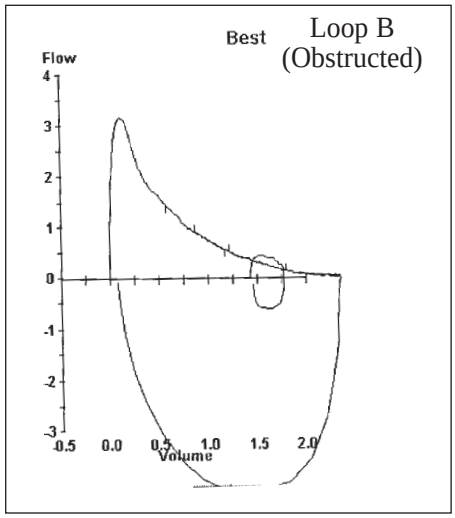

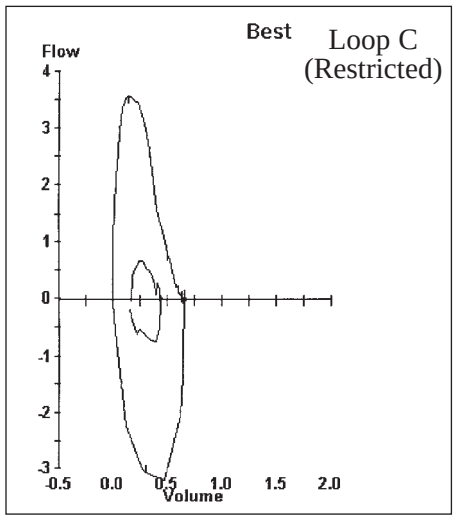

tory flow and the flow generated during the first 25% of the FVC maneuver are both influenced by elastic recoil of the fully expanded lung and patient effort. A rapidly rising flow and a sharp "peak" are the hallmarks of this portion of the expiratory curve. From a pediatric "bent" we say that the "mountain" should be tall and steep – no rounded or "wimpy" mountains allowed. Flow from "mid" and lower lung volumes is relatively effort-independent, meaning that the intrinsic elastic recoil of the lung is primarily responsible for the pressure gradient or driving pressure that continues to reduce airway caliber and limit flow.

The translated version: pushing harder and harder doesn't improve flow. Actually, pushing too hard may limit flow in patients with malacic airways or with airways subject to dynamic compression. This phenomenon is known as negative effort dependence and can be seen frequently in pediatric patients, as well as elderly patients or patients with COPD.

We are all familiar with the shape of a "normal" flow-volume loop and the slope of descending flow during expiration (Loop A). Contrast this to the "scooped out" appearance of a patient with significant intrathoracic airway obstruction from asthma (Loop B), and the very sharp, steep slope of the expiratory limb in a patient with fibrotic lung disease (increased lung recoil, but no intrathoracic airway obstruction), as seen in Loop C.

In many PFT labs, the emphasis is entirely on the expiratory limb. The inspiratory limb takes a backseat or is a non-issue all together. This is truly unfortunate, because the inspiratory limb yields completely different information than the expiratory limb. It's our window on the upper airways, from the pharynx to larynx (including the very important vocal cords) to the proximal portion of the trachea. Ignoring the inspiratory limb is like looking at an elephant from the front and assuming the rear view is the same!

Why has the inspiratory loop been so neglected? Why are there no standards for reproducibility and acceptability as there are for the expiratory maneuver? The answer is that the inspiratory loop is COMPLETELY effort-dependent, and it can be very hard to get a patient, pediatric or adult, to give maximal and consistent effort for the entire inspiratory maneuver.

Inspiration is an energy-consuming process, and the muscles of inspiration (i.e., the diaphragm and intercostals) must overcome the resistance of dilating airways. Since the time required for a maximal inspiration is much shorter, the action happens quickly. $FIV_1/FIVC$ may not be available (depending on patient age or size) and does not have the power to detect flow limitation that the FEV_1/FVC has. Although flow at various volumes (i.e., FIF_{25}, FIF_{50}, FIF_{75}) are measured, these are highly variable, especially when total inspiratory time is one second or less. Comparisons of inspiratory flowpoints to expiratory counterparts is valid for the mid-portion of the curve only.

The most widely accepted parameter is the FEF_{50}/FIF_{50} , with a normal ratio expected to be < 1.0. However, this is based on a very small study by Rotman et. al.[5], with little supporting evidence, and the parameter is too variable for consideration as a epidemiologic tool.[6] Compounding the problem is the observation that a patient's maximal inspiratory loop may not follow a maximal expiratory loop. Indeed, the best inspiratory loop is often generated following a submaximal FVC or an SVC maneuver.[7] "Mixing and matching" the best expiratory loop to the best inspiratory loop is a consideration but is time consuming and may be software-limited. Nevertheless, the shape of the curve (minus all the numbers), and the reproducibility within a testing session, are exceedingly meaningful.

I promised some unusual and amazing flow-volume loops, so let's take a look and try to apply some of the above physiology to the shape of these interesting curves. Consider Patients 1, 2, and 3 and their respective flow-volume loops:

Patient 1 is a 12-year-old boy who presented with cough and a very unusual speech pattern. He spoke in short gasping sentences and complained of shortness of breath while speaking. This young boy has classic vocal cord dysfunction. Note that the expiratory loop is entirely normal, but the inspiratory loop is clipped for the entire maneuver, and the FEF_{50}/FIF_{50} appears markedly abnormal (even without the numbers).

Patient 2 is a 15-year-old female

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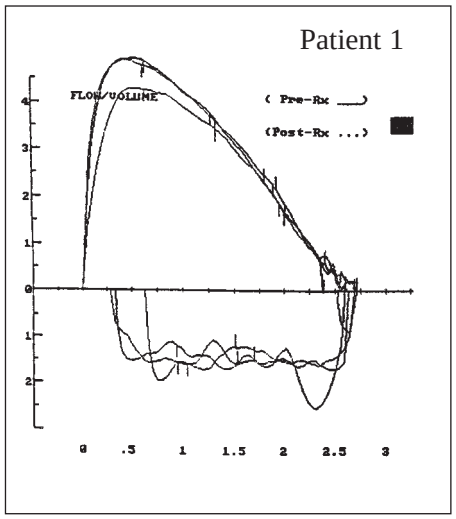
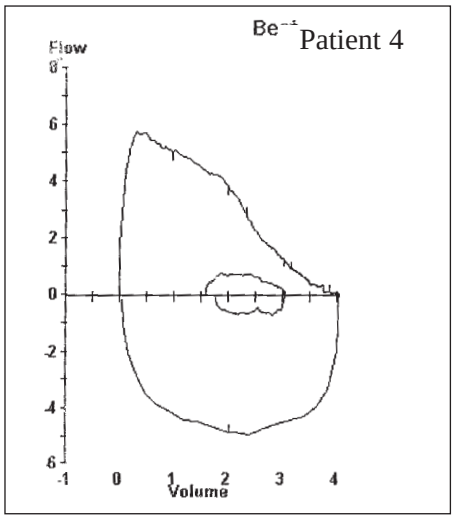
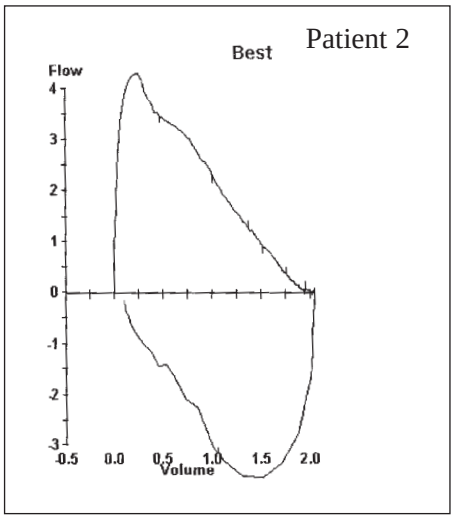
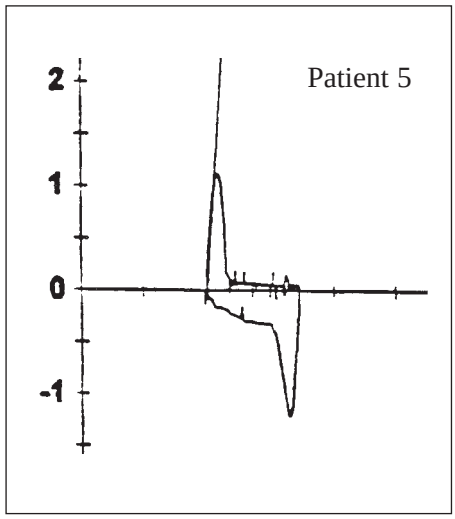
"Life's a Circle" continued from page 3

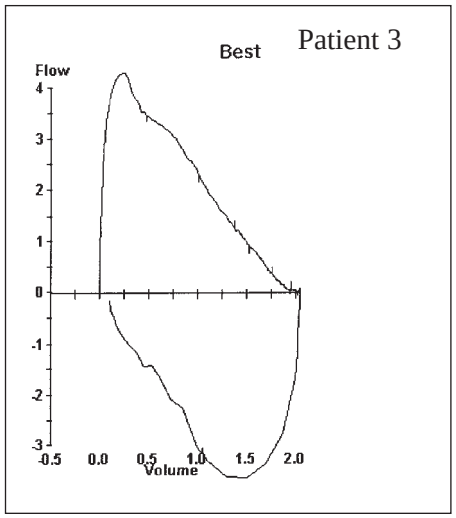
seen in our pulmonary clinic for rapid onset of shortness of breath during exercise. Once again the expiratory loop is absolutely normal. The inspiratory loop has an FEF_{50}/FIF_{50} less than 1.0, and there is only one inflection point. Is this inspiratory loop normal? Probably not, and it is likely part of the "spectrum" of vocal cord dysfunction. Stressing this child with an exercise or a methacholine challenge might evoke more clipping of the inspiratory curve.

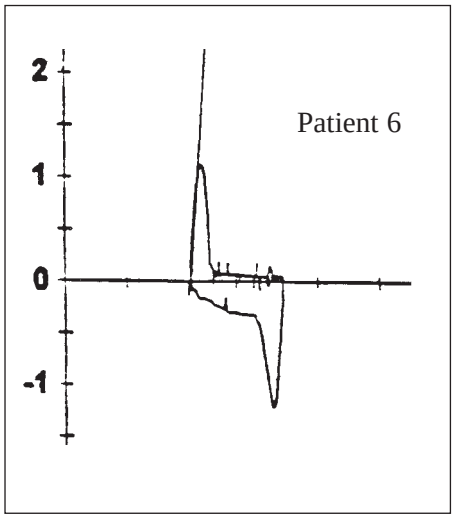












Patient 3 also has a normal expiratory loop, but the inspiratory loop has two points of inflection. Multiple inflection points, with rapid changes in flow, are often seen, and may be a phenomenon also associated with vocal cord dysfunction. Evaluating the FIF_{50}, or any other single flowpoint, can lead to erroneous conclusions. This reinforces the point that using one parameter does not do justice to the entire inspiratory loop. Until better standards are developed, look at the contour of the entire inspiratory loop! At least ask the questions: "Does this patient have reason to have an extrathoracic or upper airway obstruction? Does the shape of this inspiratory loop tell me something about the flow dynamics of this patient's upper airway?"

The first two flow-volume loops above (Patients 4 and 5) are from teenage adolescents with cystic fibrosis; the third (Patient 6) is from an eight-year-old female who developed bronchiolitis obliterans.

From total lung capacity, all patients are able to generate significant peak flow from their upper airways. Patient 4 exhibits a rather common, mildly obstructed expiratory loop. The first 75% of exhaled volume has a "normal" convex shape. Only the last 25% of expired volume takes on a concave or "scoopy" appearance and is associated with a decrease in the $FEF_{25-75\%}$.

Often a reduction in $FEF_{25-75\%}$ is referred to as "small airway disease." This term may be a misnomer. Although in this setting the patient is very likely to have obstruction in her smaller airways, the correct terminology is "reduced flow from low lung volumes." Why the distinction? As we will see shortly, the $FEF_{25-75\%}$ can be severely reduced and still not associated with any obstruction of the small airways.

Now consider Patient 5. The point of flow limitation is much different than in Patient 4. Although small airway obstruction is undoubtedly involved, it

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Figure (7)A

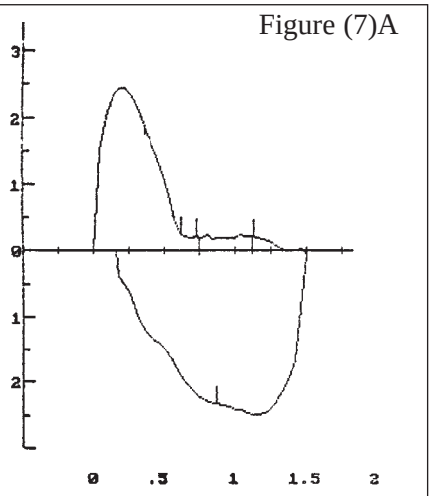
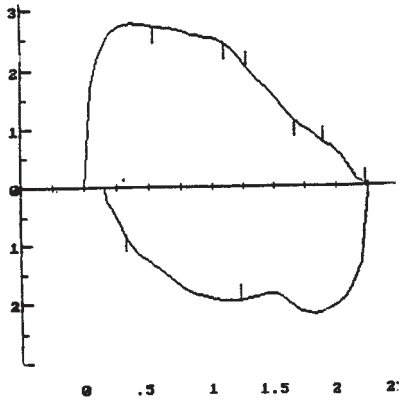


Figure (7)D



Best Figure (7)B

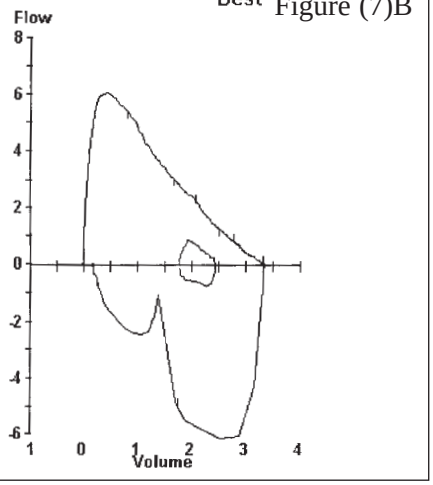


Figure (7)E

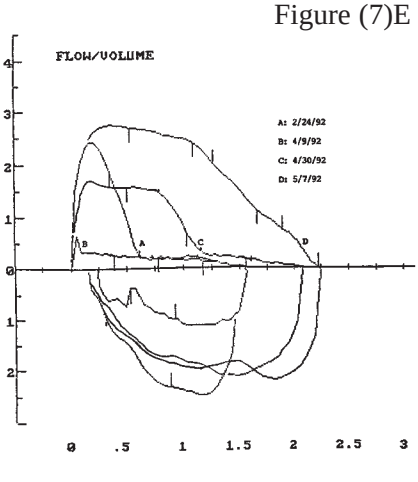
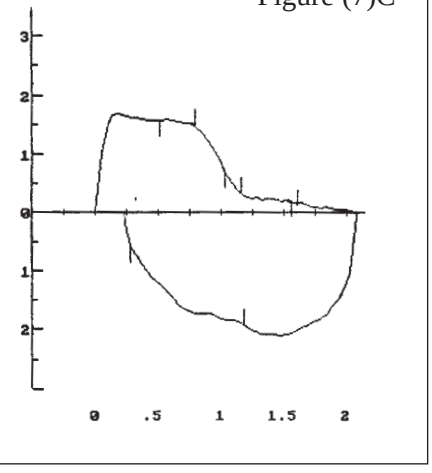


Figure (7)C



is masked by obstruction occurring at a higher level. The rapid drop in flow is the hallmark of dynamic compression of a more proximal central airway. The site of flow limitation, or "choke" point, is also known as the equal pressure point. The shape of this severely obstructed expiratory loop is often called the "L" sign.

Patient 6 not only demonstrates the "L" sign on expiration, but, remarkably, has a "reverse" L sign on her inspiratory loop. Again, dynamic collapse of a central malacic airway produces flow limitation; however, the site of this limitation overrides into the extrathoracic airway – most likely, the upper trachea. Patient 6 is able to generate a relatively normal inspiratory loop because her extrathoracic airway is less compromised.

Figures (7)A through (7)D are a series of flow volume loops for Patient 7, a teenage female who underwent bilateral lung transplantation. Figure (7)E is a compilation of all the loops superimposed on each other.

What an amazing series of loops! This patient had a multitude of airway complications at the anastomotic sites (where the lobar bronchi of the transplanted lungs were surgically attached to her native lobar bronchi). Figure 7(A) was obtained after the patient had some dishissance or breakdown of one of the anastomotic sites. Notice again the "L" shape, or the plateauing of flow during expiration due to loss of integrity of this central airway. The inspiratory loop is maintained because the extrathoracic airway is normal.

Figure 7(B) was obtained one month later when the patient further decompensated. On bronchoscopy it was determined that both anastomatic sites were grossly malacic. A stent was placed in one of the lobar bronchi, and PFTs obtained subsequently.

The FV loop in Figure 7(C) clearly shows improvement in flow through the major airway with the stent; however, the unstented bronchus is still dynamically compressed during forced exhalation. The clipping of the upper portion of the expiratory loop is due to the fixed nature of the stent.

Guess what Figure 7(D) represents? Two stents are better than one! The malacic segments in both lobar bronchi are stented, with a resultant FV loop that is near normal. Once again, the stents act as fixed resistors and the flow-limiting segments responsible for the blunting of the peak flow on inspiration and expiration.

Above are two very abnormal flow-volume loops. How are they similar? How are they different? Does the shape of the curve help us identify the location of the lesion?

The expiratory limb of Patient 8 looks almost impossible, but, indeed, is that of a real eight-year-old. The severely reduced flow throughout the entire expiratory maneuver (including peak flow) tells us that a central airway is again involved. Is it intrathoracic or extrathoracic? Although the inspiratory loop is not entirely normal, it is relatively spared as compared to the expiratory loop. Flowrates are 2-3 times higher than the expiratory counterparts.

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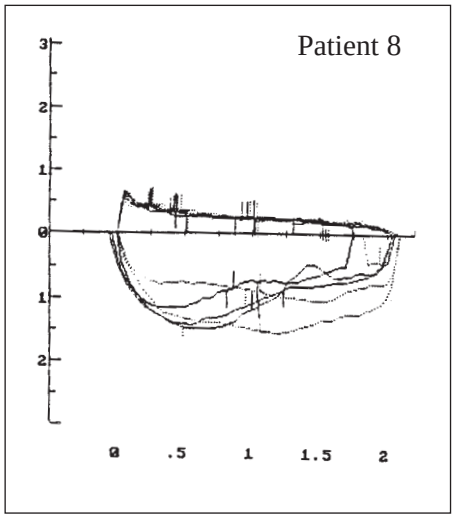
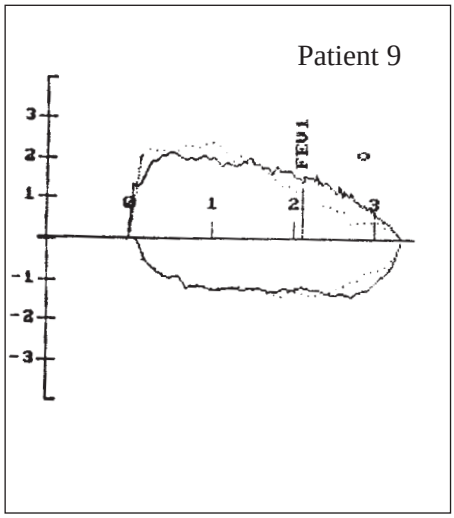
"Life's a Circle" continued from page 5





Therefore, our best guess is that this lesion is in a large central airway that is still intrathoracic.

BINGO! This child had a very rare mucoepidermal carcinoma 2 cm above her carina. Amazingly, she was sent to our division with a diagnosis of asthma. Of note, the $FEF_{25-75\%}$ of this loop would be severely reduced, but this is NOT "small airway disease."

Patient 9 also demonstrates an expiratory limb that is markedly obstructed, with a blunted peak flow and diminution of flow throughout the remainder of the expiratory phase. However, the inspiratory limb of this FV loop looks more like a mirror image of the expiratory loop. Curves that have this square or rectangular appearance are referred to as "square wave" FV loops and denote "fixed obstruction." The stricture does not permit dilatation on inspiration and also acts as the "choke point" during expiration. The flowrates during inspiration and expiration are very similar, and are relatively maintained throughout the cycle. These lesions involve the large central airways and may be intrathoracic, extrathoracic, or show a component of both. In this case, the 14-year-old male suffered from tracheal stenosis secondary to a long intubation and scarring of the trachea.

In summary, classification of obstructing airway lesions was first proposed by Miller and Hyatt[3-4]. They include (1) variable *extrathoracic obstruction* as demonstrated by Patients 1, 2, and 3, as well as (2) *variable intrathoracic obstruction* as seen in Patients 4, 5, 6, 7, and 8. Patient 9 is an example of the third classification, *fixed obstruction*, which may be intrathoracic or extrathoracic in origin. Keep in mind that the site of obstruction does not have to be limited to one airway or one portion of the lung. Multiple sites of obstruction yield flow-volume loops that may show combinations of intrathoracic and extrathoracic components.

So . . . time to vote! Which of the flow volume loops above is the most amazing? My vote: ALL of the above! There is no such thing as a boring flow-volume loop. When one considers the complicated physiology and airflow mechanics responsible for producing such a simple circle – well, that's amazing!

On a personal note, back in the late '60s and early '70s when Hyatt, Miller, Black, et. al., were writing the landmark papers on flow volume loops, one of my all time favorite singer/songwriters was on the top the music charts. Harry Chapin wrote a wonderful, simple song called "All My Life's a Circle." For those of you who remember the melody, here's my rendition of the refrain. Feel free to sing along . . .

All my life's a circle
Peak flow to RV.
And then we bring it 'round again,
Back to TLC.

All my life's a circle
And I can't tell you why
I love those amazing flow volume loops,
They keep on rollin' by!

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The Lowdown on Low Saturations During Bronchoscopy

by W. G. Petersen, MD, FCCP, Scott & White Clinic and Memorial Hospital, Texas A&M University Health Science Center, College of Medicine, Temple, TX

A 43-year-old woman with relapsing polychondritis is undergoing fiberoptic bronchoscopy to evaluate the patency of endobronchial stents. She receives a standard preoperative preparation with meperidine 50 mg and atropine 0.6 mg IM. Topical anesthesia of the nasal and oral pharynx is accomplished with 20% Benzocaine spray. During the procedure she receives an additional 7 mg of Versed because of agitation.

Inspection of the airway reveals that the stent in the left mainstem is nearly

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"Bronchoscopy" continued from page 6

occluded with granulation tissue. This is cleared with a YAG laser; however, topical epinephrine 1:20,000 is required to achieve hemostasis, with a total of 17 cc. Because of strenuous coughing throughout the bronchoscopy, a total of 35 cc. of 1% lidocaine is administered via the bronchocscope. All seems to be going well until just three minutes after the bronchoscope has been removed when the digital oximeter reveals a declining saturation to 68%. Changing from nasal cannula to a 50% Venti-mask does not result in any significant improvement. What has gone wrong?

Bronchoscopy has changed significantly over the last 25 years. In keeping with the established practice of rigid bronchoscopy it was initially thought that fiberoptic bronchoscopy would require deep anesthesia, intubation, and hospitalization for post-procedure observation. However it rapidly became apparent that fiberoptic bronchoscopy was amenable to conscious sedation on an outpatient basis. Yet the very advances that led to ambulatory fiberoptic bronchoscopy have also necessitated increased vigilance.

Digital oximetry has become standard monitoring in conscious sedation. Transient decreases in the SaO_2 are commonly seen as a consequence of coughing, particularly in the setting of underlying lung disease. However, sustained saturations below 88% should be viewed with concern. Rapid consideration of the various factors that produce hypoxia should lead to prompt corrective action.

A significant percentage of hypoxic episodes experienced during fiberoptic bronchoscopy will result from airway obstruction. This may arise at different anatomic levels, some quite obvious, others not. Laryngospasm most often occurs with the initial attempts at passage of the bronchoscope through the vocal cords. This will usually be evidenced by inspiratory stridor and the visual confirmation of closure of the cords. Laryngospasm may be relieved by the additional application of topical lidocaine and supplemental intravenous sedation. Passage of the scope through the cords should not be attempted until the laryngospasm has been resolved. Persistent laryngospasm should constrain further attempts at fiberoptic bronchoscopy.

Conversely, deep levels of sedation may induce sufficient laxity of the pharyngeal structures to produce airway obstruction above the level of the cords. This is more likely to be a problem in those patients with anatomic features of obstructive sleep apnea: thick neck, narrow posterior pharynx, large tongue, pendulous soft palate, and floppy epiglottis. This situation can be remedied by slight hyperextension of the neck, removal of the patient's head from the pillow, and a jaw thrust by the assistant.

On occasion, the level of sedation may centrally suppress the ventilatory drive even in the absence of airway obstruction. Obviously, this is best avoided by the judicious use of sedatives and narcotics. Stimulation of the nasopharynx or bronchi with the scope may produce sufficient sensory input to increase depth and rate of ventilation. However, the full depth of sedation may not be appreciated until after the bronchoscope is withdrawn at the end of the procedure. Reversal agents (Narcan and Romazicon) must always be immediately available in the bronchoscopy suite.

The diameter of most fiberoptic bronchoscopes is on the order of 5-6 mm, and thus they do not obstruct a significant cross-sectional area of the trachea or bronchi. However, it should be remembered that the contralateral mainstem may become obstructed by secretions or blood unbeknownst to the bronchoscopist. Likewise, indolent bleeding in the sedated patient may lead to post-procedural bronchial obstruction. This may not be detectable by auscultation of the chest or fluoroscopy. The bronchoscopist should carefully inspect all airways at the termination of the procedure to remove any residual debris and observe for continued bleeding. Prompt re-inspection of the airways should be considered for the patient who develops significant hypoxia during the recovery period.

The total volume of the large airways is relatively small, about 150 cc. Thus the patient may rapidly asphyxiate with relatively small volumes of endobronchial bleeding. Significant hemorrhage is often treated with scope tamponade of the involved segmental bronchus and application of topical epinephrine. If bleeding is not rapidly controlled, the patient should be turned with the bleeding lung in the dependent position. This will hopefully prevent obstruction of the contralateral mainstem.

Bronchospasm of the smaller airways cannot be appreciated visually by the bronchoscopist. All patients should be queried as to a prior history of asthma. Those patients with a history of reactive airways should be pretreated with inhaled dilators. Rapid auscultation should be performed in those patients experiencing a persistent decrease in the arterial saturations to assess for wheezing.

One of the more feared complications of fiberoptic bronchoscopy is pneumothorax. Although most often associated with the performance of transbronchial biopsy, this can occur following the use of the brush catheter. The pneumothorax can be either abrupt or insidious in onset. If the patient is supine on the fluoroscopic table, the first sign of air in the pleural space may be detected at the base rather than the apex (deep sulcus sign). Small pneumothoraces may often be asymptomatic. Those producing significant hypoxia will usually be large and produce dyspnea as well.

Finally, it should be remembered that decreased arterial saturations may also be due to significantly impaired cardiac output. Hypotension with hypoxia can rapidly lead to a critically unstable patient. Blood pressure should promptly be assessed anytime there is persistent depression of the saturations. Arrhythmias, deep sedation, myocardial ischemia, and pneumothorax may all result in hypoxia due to impaired cardiac output.

What about our 43-year-old woman? This particular patient experienced one of the lesser known complications of fiberoptic bronchoscopy. The topical anesthetics have virtually all been associated with the formation of meth-hemoglobin. This will be suspected when the patient evidences unimpaired ventilation with stable cardiac parameters, yet the digital oximeter continues to be quite low. Arterial blood gases will typically register high pO_2 with low pCO_2 due to hyperventilation. Arterial saturations must be measured with co-oximeter, which will reveal the elevated level of methhemoglobinemia. Methylene blue can be administered intravenously if indicated.

In summary, fiberoptic bronchoscopy remains a relatively innocuous procedure well-suited to performance on an outpatient basis. However, arterial saturations must be monitored closely, both during and after the procedure. Persistent hypoxia can arise from a number of causes. Rapid assessment of the patient will usually reveal the problem and lead to successful resolution. ■

Specialty Practitioner of the Year

Don't forget to make your nominations for the 2000 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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Mail or FAX your nominee to the section chair at the address/number listed on page 2 of this issue.