



Diagnostics

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Bulletin

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Diagnostics Specialty Practitioner of the Year: Dale Mayers, RRT

The Diagnostics Section is proud to honor Dale Mayers, RRT, as our 2002 Specialty Practitioner of the Year.

As clinical resource specialist for pulmonary diagnostics at St. Mary's Hospital in Grand Junction, CO, Dale has distinguished himself on the job not only through his skills as a diagnostician, but also by sharing those skills with other members of the respiratory therapy staff. According to co-worker Jane Wild, "Each month, Dale expands on a topic of interest to all staff. He has targeted blood gas analysis, simple spirometry and MRI and CT interpretation." These sessions, which are generally attended by all of the 28 RTs in the department, provide important insights that enable practitioners to incorporate diagnostics into direct patient care.

In addition to the educational sessions, Dale works to increase the services available through the pulmonary diagnostics service at the hospital. "Dale has also been intimately involved in the development and expansion of several pulmonary diagnostic procedures here at St. Mary's," says Jane Wild. "He freely works on interdepartmental teams, and with the respect of five staff pulmonologists, has incorporated nutritional metabolic assessments for our ventilator patients, with documented decreased length of stay."

Dale will receive the award at the 2002 AARC International Congress in Tampa Bay, FL, in October. Congratulations, Dale! ♦

Diagnostics Play a Key Role in the NLHEP Guidelines

by Gretchen Lawrence, BA, RRT, FAARC, NLHEP liaison to the AARC

Did you know that:

- Smoking is listed as a significant factor not only in chronic lung disease but also in stroke, cancer, and heart disease?
- At least 20% of smokers will develop airflow limitations that will impact their quality of life?
- Spirometric abnormalities predict all-cause mortality?

As a member of the Diagnostics Section, you are most likely employed in a pulmonary diagnostic laboratory, testing those who may be finding out for the first time that they have airflow limitations related to these and other disease states. Wouldn't it be nice if you didn't have to be the "bearer of bad news" to these patients and their doctors?

The National Lung Health Education Program (NLHEP) guidelines strongly suggest that the first line of defense is a good offense - and the primary care physician (PCP) is in the best position to identify and treat these patients early in the course of their disease, many times long before they present with symptoms. However, in current practice, the PCP may not ask the important but very simple questions:

- Are you 45 or older and/or have a smoking history or environmental exposure?
- Do you have a chronic cough, sputum production, and/or shortness of breath?

If the patient can answer "yes" to any of these questions, he/she should have a simple spirometry test - a test that can be performed in the PCP's office, if the PCP has the right equipment, a well-trained staff, and knows how to interpret the numbers. And the only numbers that the doctor needs to know how to interpret are the FEV1, FEV6, and the ratio between the two. As always, good patient effort and reproducibility are the keys to validity.

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Clinical Effects of Flow Limitation

by Michael Snow, RPFT, RCPT

Expiratory flow limitation (EFL) is an area of increased focus not only in the pulmonary function laboratory but also in the critical care units. The presence of EFL can dramatically impact clinical interventions by dictating the pattern of response to bronchodilators and the development of intrinsic positive end-expiratory pressure (PEEPi). As a result, flow limitation can cause impaired muscle function, increased inspiratory work and adverse hemodynamics for patients on mechanical ventilation.¹

By definition, expiratory flow limitation is the inability to increase expiratory flow by increasing pleural and alveolar pressure at a given lung volume. During a forced vital capacity maneuver, after the peak expiratory flow, increasing effort does not increase expiratory flow, as shown in Figure 1. This classic example of expiratory flow limitation provides the basis for reproducibility assessment between efforts. Patient effort, driving pressure, airway resistance and elastic recoil are the most important factors determining maximal flow.^{2,3} The characteristic shape of a flow volume loop is determined by patient effort in association with minimized airway resistance and the contribution of elastic recoil to the driving pressure. As flow limitation is reached, the flow volume loop becomes independent of patient effort and elastic recoil begins to aid in resisting airway compression.

In normal subjects, increased ventilatory demand is met by expanding the tidal volume within the constraints of the maximal flow volume loop. During maximal exercise, tidal volume approaches the baseline inspiratory capacity. Expiratory flows may reach or even exceed the maximal FVL envelope. In the absence of flow limitation, the lung volume may also shift to facilitate larger tidal volume and flows. Respiratory rate response is flow limited. In other words, minute ventilation doesn't improve if the increased tidal volume requires more time to exhale. The forced expiratory volume for one second (FEV1) is the maximal volume that can be exhaled in one second and represents an upper limit for the tidal volume and respiratory rate. As shown in Figure 2, once the tidal volume reaches the IC or FEV1, flow limitation can be clearly seen on exercise flow volume loops. Comparison baseline and exercise flow volume loops can clearly show the onset of EFL as long as end-expiratory lung volume is measured periodically.^{4,5,6}

In patients, several mechanisms can reduce the maximal expiratory flow that can be achieved. Subjects with EFL secondary to obesity present the characteristic pattern on the FVL shown in Figure 3. The flow limitation at rest presents no opportunity to shift the end-expiratory lung volume. The only mechanism available to respond to ventilatory demands is increasing the tidal volume within the maximal flow volume loop. These patients may have normal or slightly reduced functional residual capacities but the flow limitation causes a marked decrease in expiratory reserve.

Airway obstruction results from a reduction in airway cross-sectional area that is caused by bronchospasm, inflammation- or compression. Dynamic compression, as seen with emphysema and cystic fibrosis, occurs when increased effort causes the airways to compress due to reduced elastic recoil and structural airway support. Significant airway obstruction can cause flow limitation at rest or with even minimal exercise. A common response to increased airway obstruction is to expand the airways by shifting to a higher volume and dramatically improving the patient's subjective sense of well-being at rest. Unfortunately, higher lung volume limits the ability to increase tidal volume and therefore significantly increases dyspnea on exertion. Airway compression can lead to the paradox of flow actually decreasing with increased effort when increased ventilation is most needed, as shown in Figure 4.⁷

After bronchodilators, the primary effect may be to relieve overinflation, since even small increases in expiratory flow at the lung volume corresponding to tidal volume can cause substantial reductions in the functional residual capacity or end-expiratory lung volume. This type of flow limitation creates what are referred to as "volume" responders. While the bronchodilators may not significantly reverse expiratory flow limitation, they allow the limitation to occur at a lower absolute lung volume. These patients subjectively feel better because their absolute lung volume is reduced, but they may not show significant FEV1 or even FVC responses. However, they will show specific conductance, specific resistance and even FRC or RV/TLC responses.^{8,9,10}

Expiratory flow limitation in mechanically ventilated patients requires special attention to expiratory time to prevent PEEPi. If the expiratory time is insufficient, dynamic

hyperinflation and PEEPi will result from breath stacking. While the tidal volume can be increased, the EFL will require proportionally increased expiratory time which, in turn, limits the respiratory rate, increases inspiratory work of breathing and leads to hemodynamic changes. Because of this effect, it is essential to detect flow limitation.

Methods for detecting expiratory flow limitation

Of the three primary methods that have been used to assess EFL, the most accurate is measuring the isovolume transpulmonary pressure/flow relationship.¹¹ If increasing pleural pressure at the lung volume corresponding to the tidal volume does not increase expiratory flow, expiratory flow limitation is present. The drawback of this method is that it requires an esophageal balloon and the cooperation of the subject. It is probably more appropriate for a research laboratory.

Another method involves comparing partial or full maximal to resting tidal flow volume loops or M/P ratio.¹² If the tidal expiratory flow matches or exceeds the maximal flow at comparable lung volumes, EFL is demonstrated. It is important to control volume and time history in the preceding inspirations when comparing the two maneuvers. To limit thoracic gas compression, comparisons between resting and slightly increased efforts are made. The difference between maximal and submaximal efforts has previously been used to detect FEV1 differences in patients with emphysema.¹³ While this technique is simple, the drawback is that it does require subject cooperation. Exercise flow volume loops are an example of this type of method.

The third method consists of applying small, negative pressure during tidal expiration that widens the pressure gradient. By measuring tidal flow volume loops with and without the negative expiratory pressure, the presence of EFL is confirmed by increased flows with negative expiratory pressure. Negative expiratory pressure widens the pressure gradient and permits increased expiratory flows. This method has been compared to isovolume pressure/flow curves and does not require patient cooperation.¹⁴

Patients with airway obstruction present a significant clinical challenge. Frequently, they are treated symptomatically without a clear understanding of what the clinical intervention is hoping to achieve. In addition to clinical complexities, vague treatment outcomes create stress in a managed care environment. Detection and measurement of expiratory flow limitation can not only improve the understanding of the pathophysiology but also provide objective evidence to drive outcomes. ♦

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FIGURE 1

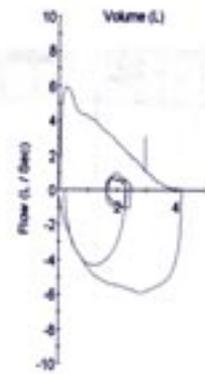


FIGURE 2

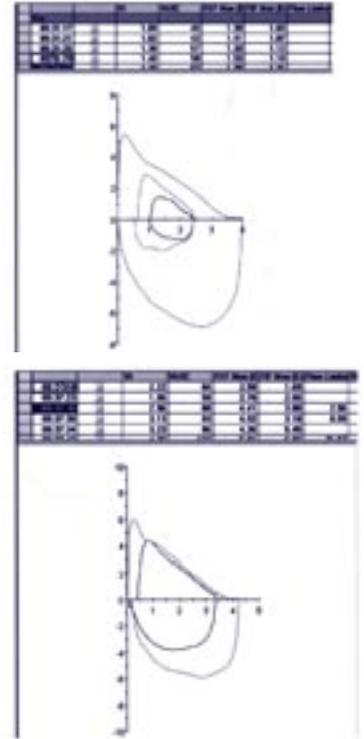


FIGURE 3

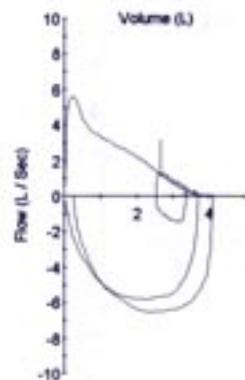
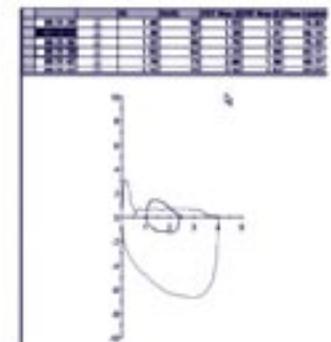


FIGURE 4



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DIAGNOSTICS PLAY A KEY ROLE IN NLHEP GUIDELINES

Given this information, what can you, the RT working in the pulmonary lab, do to support the NLHEP guidelines? Here are some ideas:

- Encourage the doctors who send patients to you for testing to become familiar with the NLHEP guidelines. Become a NLHEP "expert" and share the literature.
- Develop and implement a smoking cessation program in your hospital. Talk to school children and large employee groups about smoking and health.
- Set up a "case finding" spirometry program and screen those who fit the criteria. Test fellow employees and the public who come to your facility. You can conduct this testing in the local mall, senior citizens center, and churches and synagogues. How about at the YMCA, local health clubs, and area pharmacies?
- Help your local PCPs set up a quality testing program in their offices. Train their staffs. Provide ongoing education. Or consider setting up a "mini-lab" in a physicians' office building, making it convenient for the doctors to send patients for testing. Put small posters in their exam rooms to remind them to ask the questions outlined above.

And finally, to become an "expert," put the following on your "read and share" list:

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- Strategies for Preserving Lung Health and Preventing COPD and Associated Diseases. The National Lung Health Education Program. Chest 1998;113(2Suppl): 123S-163S (reprinted in Resp Care, 1998;43[3]).
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- Chronic Obstructive Pulmonary Disease Surveillance - United States, 1971- 2000. Centers for Disease Control. Aug 2002/Vol. 51/No. SS-6.

To find out more about NLHEP, visit their web site at www.nlhep.org. Or contact me directly at gl-lungs@swbell.net. ♦