



HELIUM ISO-FLOW STUDIES

by *Jim Harvey MS, RPFT, RCP*

Various studies have been performed during the last 40 years investigating the relationships between gas density and air-flow in a selection of obstructive disorders. These studies looked at the effect that different gases have on the shape and characteristics of flow-volume loops. These tests are referred to as helium iso-flow studies.

At lung volumes characterized by predominantly laminar flow patterns, which are generally in the airways having cartilaginous support, the expiratory flow is gas density dependent resulting in higher flows using lower density gas. In airways that do not remain patent during forced exhalation due to either obstruction or to emphysematic changes, the air flow is not gas density dependent, and flows for helium mixtures and room air are the generally the same.

Forced expiratory vital capacity maneuvers (FVC) using heliox, which is 80 percent helium and 20 percent oxygen, are performed, and their resultant flow-volume loops are superimposed. Remember, of course, that helium is less dense than either the nitrogen or oxygen of room air. The resultant flow-volume patterns and measurements have been interpreted as indications of early airway disease; however, there seems to have been no independent measure presented to verify this theory.

So, the question is whether this helium iso-flow test is a sensitive technique for the early detection of small airways obstruction. This question is still being asked in current studies. The following is a description of the helium iso-flow test as well as a summary of the debate.

The helium iso-flow study involves having the patient perform a flow-volume loop while breathing room air. The patient then takes three large breaths to total lung capacity through a demand valve or a volume displacement spirometer containing heliox. Immediately after the third inhalation of heliox, and without disconnecting from the breathing circuit, a flow-volume loop is performed. Both flow-volume loops obtained with the patient on room air and on heliox are superimposed and matched or lined

up at the point of residual volume or at the extreme right side of the flow-volume loop.

Typically, during the first portions of the flow-volume loop, the flows from the heliox loop are higher than the flows from the room air loop. The higher flow with heliox only applies to areas where the flow remains laminar, and in these areas the flow is dependent on gas density with higher flow rates in the heliox flow volume loops. The laminar or higher flows usually extend down to approximately the beginning of the 18th division of the bronchial tree.

During forced exhalation, as either the room air or heliox is pushed through airways that remain patent, there are higher observable flow rates as represented on the flow-volume loops. Then, at a certain point closer to the residual volume, the flows on both the room air and heliox curves begin to show identical flow rates. The volume that is exhaled after the flows from both loops become generally super imposable is referred to as the iso-flow or VisoV (volume with identical flow).

This lung volume is thought by many investigators to represent the gas flow through airways that do not remain patent at high trans-pulmonary lung pressures toward the end of forced exhalation, and in which the airways begin to collapse either due to lack of cartilaginous support or due to emphysematic changes. With the resultant airways collapse, there is non-laminar or turbulent flow. In these areas of the lung, the flow is gas density independent.

Laminar flow in the lung is due to the cartilaginous support in the airway. The respiratory tree has up to 28 generations or divisions of airways beginning with the trachea, which forks at its base to form two primary bronchi. Although the bronchi are histologically similar to the trachea, the arrangement of the cartilage in their walls is markedly different.

Bronchi, for example, do not have the C-shaped rings of cartilage present in the trachea. Instead, they have a series of partially circular plates of cartilage in their walls. These cartilage plates become fewer along the length of the bronchial tree as the primary bronchi divide and produce generations of smaller bronchi. Small bronchi continue to divide, giving rise to a series of airways of smaller and smaller caliber. When the airways are reduced to a diameter of 1 mm or less, they are called bronchioles.

After the terminal bronchiole, the following seven generations of dichotomous divisions are called respiratory bronchioles and serve the gas-exchanging parenchyma consisting of budding alveoli. The bronchioles have no cartilaginous support



Jim Harvey will be a featured speaker at the 9th annual Focus Conference May 14-16, 2009 Disney's Coronado Springs Resort Orlando, Florida

and are susceptible to collapse under positive trans-pulmonary pressure toward the end of forced exhalation. But in the healthy lung, as the patient forcefully exhales and the lungs are emptied, the bronchioles stay patent and the flow continues freely with mostly laminar flow.

If there are emphysematous changes in the lung involving the loss of elastic and reticular fibers from the release of excess trypsin from macrophages keyed by the response to particulate deposition, as in a smoker's lung, the bronchioles will not remain patent as the lung volume approaches residual volume (RV) and as the trans-pulmonary pressures increase. If these emphysematous changes are significant, the bronchioles will begin to collapse well before RV.

In the helium iso-flow maneuver, after the patient has taken in the three large breaths of heliox, the flow-volume loop is recorded. In the trachea, main stem bronchi and smaller bronchi, the cartilaginous support keeps the airways patent during the entire expiratory phase, even during the exhalation of the residual volume when the trans-pulmonary pressure is greatest, and the flow remains laminar. In a young, non-smoking, healthy person, all airways down to the alveoli remain patent throughout the flow-volume loop, resulting in almost no turbulent or decreased flow.

As small airways disease takes hold either through smoking or advancing age, the cartilaginous supports begin to weaken, or during a forced expiratory maneuver as RV is reached, the airways collapse as a result of the loss of elastic lung recoil and the flows decrease as is demonstrated on the flow-volume loop. This is evident by the measurable increase in the helium iso-flow or VisoV, which is located in the later part of the flow-volume loop with similar flow rates.

Asthma, emphysema, and chronic bronchitis are flow obstructive disorders which cause an increase in VisoV by causing larger percentages of the FVC to be exhaled through narrowed airways with turbulent flow. In asthma and in chronic bronchitis, there may not be damage to cartilaginous structures, but with mucus and swollen and inflamed tissue obstructing the airways, the flows become obstructed with turbulent.

VisoV has been shown to increase with age because chronological aging results in a steady loss in reticular and collagenous fibers beginning generally after the age of 21. VisoV has universally been shown to significantly increase with smoking in young people, and dramatic increases are demonstrated as smoker related airway obstruction increases. The question is whether small VisoV changes in young people can be actually connected to increases in non-laminar or turbulent flow in damaged airways, and whether it can be used as an indication of the beginning of small airways disease in young smokers or in populations with chemical or air pollution exposure.

In patients with very severe emphysema, the VisoV point can begin almost immediately after the peak flow due to a mixture of flow from a combination of normal cartilage supported airways and from damaged airways. The VisoV can't distinguish between airway obstruction as found in asthma or bronchitis versus the obstruction caused in loss of elastic recoil found with emphysema.

This means that the variability found in test results can be blamed on the fact that there are different types of airway obstruction and, if caused by the daily variability often found in asthma and bronchitis, VisoV results will correspondingly be significantly different. Intra laboratory (within a single lab) data often has a reasonably low variability coefficient but inter laboratory data is more often significantly varied.

continued on page 31

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Helium Iso-Flow... Continued from page 25

A way to reduce the variability of VisoV measurements is to define a specific method of calculation. For example, when the volume down from TLC is measured, rather than using the commonly used method of measuring up from residual volume, most often very different results are obtained. The basic problem stems from the difficulty in attempting to mark an exact point where two somewhat noisy lines intersect.

If all measurements are made in one laboratory where only one technique is used, then the results are very reproducible. If the patients tested have multiple diagnoses then even the best intra-laboratory measurements will be off. In other words, if patients with beginning small air disease caused by emphysema are compared to patients with obstruction caused by chronic bronchitis, significant variability will result. Another factor that causes increased variability is the number of breaths of heliox inhaled before the test and the depth of breaths.

Finally there is a question as to what are the actual mechanisms that cause the changes in iso-flow segments of the flow-volume loop and their changes over time in an individual patient. No one really knows if the above described theories are valid during the latter sections of a flow-volume loop in normal and in obstructed patients because there is no corroborative evidence.

In conclusion, helium dilution studies can be accurate indicators of early airway obstruction in a well-run laboratory, but because of the complexity of the tests and the many factors which must be uniformly considered, the test is of questionable general clinical significance and currently is not normally used in clinical situations although it can be and is still used as a good research tool.

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Another Look... Continued from previous page

strongest predictor of asthma knowledge." For this reason, written materials and action plans should be written at a 5th -6th grade level. Taking the health literacy factor one step further, other studies suggest that low parental literacy is associated with worse asthma care measures in children. To this end, the new guidelines emphasize pediatric concerns and place a high premium on asthma education in schools because of its potential to reach a large number of children.

The National Asthma Educator Certification Board established an exam leading to the Asthma Educator-Certified credential. Today, respiratory therapists make up the majority of practitioners who hold this A-EC credential. Respiratory therapists are the logical providers to deliver aerosol device education and to teach MDI use. There are, however, many health care providers who have the knowledge and skill set to become effective asthma educators, including physicians, nurses and pharmacists. The NAEPP guidelines emphasize all caregivers should collectively and collaboratively teach and reinforce asthma self-management at every opportunity and across all care settings. The American Association for Respiratory Care has published a very well-received Guideline to Aerosol Delivery Devices and also sponsors continuing education opportunities for those who feel they may be in need of brush-up or review.

The real key to success is the partnership with the patient. It is a time-consuming endeavor and regrettably, third party payers seem not to recognize the inherent value of patient education. Nonetheless, we all have to believe that asthma self management education improves outcomes and will ultimately reduce overall costs. We owe it to our country's almost twenty million asthmatics and to our professional roles as health care providers.

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