



THE MYSTERIOUS CLOSING VOLUME

by Jim Harvey MS, RPFT, RCP

Closing volume is a mysterious test which is performed as single breath nitrogen washout. The "closing volume" aspect of nitrogen washout is presently given little or no attention in the pulmonary function lab. But it is worth looking back at this measurement for, at least, the sake of historical background.

Closing volume is defined as the lung volume at which dependent lung zones cease to ventilate, presumably as a result of airway closure or compression. Although the predictive value of closing volume has never been well established, it is thought that the closing volume measurement may indicate the degree of obstructive airway disease at early stage or the point of early small airway disease, before it can be gleaned from flow volume loop data. The fact is, the proposed airway mechanics at the point of the closing volume are completely hypothetical since there is no possible method to actually see or measure what happens at the instant of closing volume in a living person. In other words, our common

and accepted interpretation of the test results makes sense but can not be proved.

Before analyzing the test a few words about respiratory anatomy are in order. There are approximately 300 million alveoli in the lung separated by thin septa which contain a tightly laced network of blood capillaries.

The respiratory tree supplying these alveoli with air and blood supply has approximately twenty three generations of divisions. The 19th, 20th, and 21st are called respiratory bronchioles and are different from preceding bronchioles in that their epithelium changes from pseudostratified ciliated columnar to squamous, nonciliated. The bronchioles preceding the respiratory bronchioles are completely encircled with reticular, elastic, and collagenous fibers which support the lumen from not collapsing. The few generations of bronchial tubes before the respiratory bronchioles begin to lose their complete circling of connective fibers and the respiratory bronchioles themselves have only partial fibers around them which allow them to collapse with external pressure. This tendency of the respiratory bronchioles to collapse is augmented by the fact that they consist of squamous cells. The large airways have cartilaginous support which provides rigidity and stability. The respiratory bronchioles are held open by elastic fibers extending through the parenchyma and are also held open by surfactant, which helps control the pressure-alveolar diameter relationship.

The single breath nitrogen washout test which includes the closing volume measurement, is performed with a demand valve, or reservoir bag, a pressure differential or other type pneumotachograph, microprocessor or chart recorder, and a nitrogen analyzer. Performance of the test requires measurement of inspired/expired vital capacity, the monitoring of nitrogen concentration close to the mouth, and the monitoring of flow. Lung volumes and percent nitrogen are displayed on the Y and X axes respectively of an X-Y recorder or microprocessor. Exhaled flow is monitored and controlled at 0.5 L/sec. Appropriate valves are necessary to allow the switching from room air to 100% oxygen.

The test begins with the patient exhaling to RV, and then inspiring to TLC of 100% oxygen from a reservoir or demand valve. Immediately the patient begins to exhale slowly and evenly at a flow of 0.5 L/sec. To achieve this flow the patient can be coaxed or visually follow a displayed flow meter. The nitrogen analyzer follows the nitrogen gas and it is graphed on the X axis and the volume is monitored by the pneumotach or spirometer and is graphed on the Y axis. Volume is plotted against nitrogen concentration on a graph.

The measurement of closing volume requires the creation of a gradient in the concentration of a marker gas (oxygen) from top to the bottom of the lung. The inspired oxygen dilutes the nitrogen already in the lung. As all alveoli are not equally diluted, due to the difference in size of the alveoli at residual volume from top to bottom lung zones, the alveoli in the lower lung zones undergo approximately one to one and a half time the volume increase as alveoli in the upper zones. This creates a relatively higher nitrogen concentration in the upper lung zones. The reason the alveoli in the upper lung zones are less expandable, is that since they have a lower pressure gradient. They are more fully expanded to begin with. The alveoli in the lower zones have a gravitational pressure gradient and are normally less expanded. On inspiration, a larger portion of the inhaled gas, in this case 100% oxygen directs to the upper zones. Of course, the above described condition gradually varies from top to bottom of the lung field.

There are four described phases as the patient exhales from TLC to RV. Phase I consists of 100% oxygen returning from the larynx and pharynx and trachea and larger airways or anatomical dead space. Phase II is mixed oxygen and lung nitrogen. The nitrogen concentration rises abruptly as the anatomical volume is depleted. Phase III is mixed alveolar and small air-

Closing volume is defined as the lung volume at which dependent lung zones cease to ventilate



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way oxygen from the single breath and residual nitrogen. This combination of gases produces a plateau since the relative concentrations of both gases change very slowly and evenly. Phase IV is an abrupt increase in nitrogen until RV.

The slope of phase III is an indication of the distribution of ventilation and is measured as change in percent nitrogen concentration per liter of lung volume. The slope in healthy, young individuals is approximately 1%. It is interesting that there are small bumps or oscillations in the slope of phase III which are thought to be small changes in alveolar nitrogen concentration as the capillary blood pulses through the alveolar walls. The oscillations are thought to be caused by the microscopic obstructions caused by the pulses in the capillaries. With cases of increasing obstruction or decreased ventilation, the slope of phase III will increase and in very severe cases will be indistinguishable from and actually become an extension of phase IV.

Here we finally go into the theory of the mechanics of phase IV or the closing volume. We saw previously that the alveoli in the lower lung fields are more distensible and also have a higher pressure or gravitational gradient against them. Also on inhalation, they inflate more than alveoli in the upper lung fields. This means that after inhaling 100% oxygen from RV to TLC, the highest concentration and volume of nitrogen is located in alveoli of the upper lung fields since a higher percentage of oxygen traveled to the lower lung fields. At the same time, upon forced exhalation and approaching RV, there is more positive pressure exerted against the small airways and alveoli of the lower lung fields so that if there is any structural compromise in small airway structure from either smoking damage or from age changes, the small airways in the lower lung fields will begin to close first which results in a higher proportion of exhaled gas to come from the upper lung fields, which in turn results in a marked rise in the exhaled nitrogen percent of phase IV.

In normal lungs at resting lung volumes, the transpulmonary pressure is positive, which distends the airways. The intra-pleural pressure is felt on the walls of the airways as a compressing force. Intrapleural pressure and transpulmonary pressure are not equal in all parts of the lung, due to a gravity determined gradient of approximately 0.25 cm H₂O/cm of vertical lung height. This causes the pressure on the walls of the airway to become positive in the lower areas of the lung at low lung volumes. As the pressure on the walls exceeds the pressure within the airway, the airway will narrow. In healthy, non smoking, young adults there may not be narrowing or closing of airways as RV is approached. But with smoking damage present or very early small airway disease, the closing of airways might begin well before RV is approached. In a normal healthy, young adult there may be no closing volume or closing may begin at 90% of the VC. In an older adult airway closure may begin after 70 or 75% of the VC has been expired.

Thirty years ago closing volume measurements were done with the idea of disclosing beginning small airway disease in young people, as an adjunct to flow volume loop analysis, and as a measurement of distribution of ventilation. Even though the test results seem to correlate with other markers for beginning small airway disease, the mechanics of closure are completely hypothetical and are assumed, since there is no possible way in a living person to observe the closing effect directly.

Jim Harvey MS, RPFT, RCP works in the Pulmonary Function Laboratory at Stanford Hospital and Clinics in Palo Alto, and teaches Pulmonary Function at Skyline College in San Bruno, California.