



LUNG VOLUME AND OBSTRUCTIVE SLEEP APNEA

by Regina Patrick RPSGT

The most effective therapy for obstructive sleep apnea (OSA) is continuous positive airway pressure (CPAP) treatment. An OSA sufferer stops breathing intermittently during sleep. The cessation in breathing (i.e., apnea) occurs because pharyngeal structures such as the tonsils and adenoids are drawn into the upper airway and block (i.e., obstruct) airflow. CPAP treatment, by blowing pressurized air through the airway, counteracts the forces that draw pharyngeal structures inward. The pressure of the air is greater than atmospheric pressure. (Pressure greater than atmospheric pressure is called positive pressure.) Scientists have long described CPAP as an "air splint for the upper airway" believing that the pressurized air exerts

Lung volume may play a more significant role in upper airway patency than previously thought

an outward pushing force against the upper airway structures that is greater than the inward pulling force acting on the structures during an apneic episode. But recent studies suggest that CPAP treatment may not

prevent apnea by splinting the upper airway open but instead by increasing lung volume and putting pressure on the lower trachea.

The trachea is a rigid tubular structure. The lower region of the trachea breaks into a left and right branch with each branch supplying its lung with air. Above the trachea is the upper airway which extends from the rear portion of the nasal cavity to the larynx. The vocal cords of the larynx form a slitted cover over the trachea. The upper airway is somewhat tubular in shape with soft structures such as the adenoids, tonsils, and tongue forming its walls. Normally, the diameter of the upper airway increases during inhalation and decreases during exhalation. The changes in upper airway diameter during respiration is thought to primarily result from increased activation (during inhalation) and decreased activation (during exhalation) of pharyngeal dilator muscles such as the geniohyoid, sternohyoid, genioglossus, and stylopharyngeus muscles.

Scientists believe that impaired activation of the pharyngeal dilator muscles decreases their tone to the point that structures supported by these muscles are easily drawn into the airway during sleep in OSA sufferers. However, in 1988 Loyola University of Chicago scientist William B. van de Graaff demonstrated that traction on the lower portion of the trachea can increase the diameter of the upper airway regardless of dilator muscle activity.

In his study, van de Graaff measured the upper airway resistance (UAR) in tracheotomized dogs under two test conditions: 1) in the presence of and 2) in the absence of upper airway muscle activity. He measured UAR changes as the anesthetized dogs breathed

through a tracheostomy tube. The tracheostomy tube contained a stopcock by which he could block airflow. He found that at baseline, the mean inspiratory UAR was 31% less than the expiratory UAR but if the tracheostomy tube were closed, the inspiratory UAR fell even more to a mean of 55%. Van de Graaff then hyper-ventilated seven of the dogs to induce apneas (i.e., the dogs had no dilator muscle activity). During the apneic episode, he stimulated the dogs' phrenic nerve to trigger diaphragmatic contractions; each contraction caused the dog to inhale. The dogs' UAR fell by an average of 51% during these induced inspirations and by 63% when the tracheostomy tube was closed. To further determine to what extent the tone of upper airway muscles plays in the collapse of upper airway, van de Graaff severed nerve input to all of the upper airway muscles in six dogs. Whether the tracheostomy tube was open or closed, he found that the inspiratory UAR fell by 25% as the dogs spontaneously breathed.

To determine to what extent thoracic movement plays a role in upper airway resistance, van de Graaff severed all of the cervical muscles (e.g., longus coli and scalene muscles) and other structures (e.g., trachea and esophagus) that connect the thorax to the pharynx. This hindered the ability of the thorax to pull on the lungs. He noted that the dogs' UAR increased to the point that upper airway obstruction occurred. When he reestablished patency of the upper airway, the dogs did not make respiratory movements. He concluded from these results that 1) upper airway patency is possible even in the absence of dilator muscle activity meaning that these muscles are not the only factor in upper airway collapse and 2) thoracic forces which pull on the trachea play a role in upper airway patency.

With this in mind, Harvard sleep researcher Raphael C. Heinzer and associates hypothesized that increasing lung volume, which increases the pulling force on the trachea, would make the upper airway more patent and decrease the amount of air pressure needed for CPAP treatment in people with OSA. Heinzer et al. used 17 OSA subjects who had already been prescribed CPAP therapy. They had the subjects sleep in a head-out rigid shell. This device is a cylindrical tank that fits over one's body except for the head. The air pressure within the shell is raised to a pressure greater than atmospheric pressure (i.e., positive extrathoracic pressure) or lowered to a pressure less than atmospheric pressure (i.e., negative extrathoracic pressure) inducing the person's lungs to deflate or expand, respectively. The subjects went to sleep within the shell with their CPAP set at the prescribed pressure. Once the subject was in non-rapid eye movement (NREM) sleep, the researchers reduced the CPAP

heart failure (CHF) or other exacerbations may no longer require oxygen therapy. This has to be considered with regard to patient age, the type and severity of the primary pulmonary disease, presence of any co-morbidity and other patient-related variables.

Since most pulse oximetries are taken during the day with the patient awake and sitting upright, consideration should also be given for an overnight pulse oximetry study. This test may provide some valuable data. With the patient sleeping in a supine, prone or lateral position, ventilation/perfusion (V/Q) imbalance or mismatch may occur affecting overall oxygenation. In addition, during sleep there may be a slowing of the respiratory rate, decrease in a patient's tidal volume and possible periods of apnea that may be suggestive of sleep apnea. All of these will impact a patient's blood oxygen level. Before discontinuing a patient's oxygen therapy, it might be advisable to perform an overnight oximetry. The test is easy to perform and the data provided will give you a better picture of the patient's cardiopulmonary status.

Equipment capable of performing overnight pulse oximetry is readily available. Many home care companies are now able to provide this service through CMS (Centers for Medicare and Medicaid Services) and HIPPA approved programs and independent testing facilities (IDTFs) using tamper-proof oximeters and soft-ware. However, home care companies offering overnight oximetry must comply with very strict guidelines pertaining to patient set-up and data retrieval. Downloads of overnight testing are sent directly to the independent laboratory which in turn sends test results in the form of an extensive report to the prescribing physician or health-care provider. This data is useful in determining if the patient qualifies for nocturnal oxygen. It also provides valuable baseline data on the patient that can be referred to at a later date.

On the economic side of the coin, we really do not save healthcare dollars if patients with any type of chronic cardiopulmonary condition constantly go to the emergency department (ED) and are admitted to the hospital for care. In 2006, Medicare estimates indicated that the average length of stay (LOS) in a hospital for a COPD patient was 5.2 days. The average cost of treating these patients was over \$4600 per day resulting in an average cost of close to \$24,000 for each hospitalization. Home care with prescribed oxygen and aerosol therapy coupled with RT follow-up and education is far less expensive and can result in fewer ED visits, hospitalizations and reduced healthcare expenditures overall.

Thomas Petty, MD and associates, in several landmark studies (1980 and 2000) on nocturnal and continuous oxygen therapy in COPD patients, demonstrated greater survival rates in patients who kept active (high walk) and used oxygen continuously. Consequently, we have to ask ourselves the following questions. Why are we in such a hurry to wean these patients off of oxygen? Are we doing our patients any disservice when we discontinue a therapy that has been proven to help their cardiopulmonary condition? The economic argument does not appear to be valid and neither is the aesthetics issue of being on oxygen or having the equipment in the home. Home oxygen systems are safe, and for patients who qualify, the newer oxygen conserving devices are light-weight, easy to operate and provide hours of oxygen if properly used. RTs need to look at the greater picture of improved patient care which can result in reduced hospitalizations and an improved health-related quality of life for patients afflicted with chronic lung disease.

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