The Pharyngeal Critical Pressure*  
The Whys and Hows of Using Nasal Continuous Positive Airway Pressure Diagnostically  

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**Abbreviations:** AHI=apnea/hypopnea index; CPAP=continuous positive airway pressure; NREM=nonrapid eye movement; Pcrit=critical pressure; Ps=pressure downstream to the collapsible segment; Pesoph=esophageal pressure; Pin=pressure within the segment; Pn=nasal mask pressure; Pout=pressure outside collapsible segment; Pus=pressure upstream to the collapsible segment; RUS=resistance of portion of tube upstream to site of collapse; UARS=upper airway resistance syndrome; UPPP=uvulopalatopharyngoplasty; Vmax=maximal inspiratory flow; Vmax= maximal flow

Obstructive sleep apnea is a disorder characterized by pharyngeal collapse and occlusion during sleep. The disorder affects between 2% and 5% of the middle-aged population and is associated with significant morbidity and mortality. The mechanism responsible for pharyngeal collapse during sleep remains uncertain. Investigators have identified both anatomic factors and neuromuscular control factors that may lead to increased pharyngeal collapsibility during sleep in patients with obstructive sleep apnea.

Although our understanding of the factors responsible for pharyngeal collapse during sleep is limited, treatments have been developed to oppose pharyngeal collapse during sleep in patients with obstructive sleep apnea. One such treatment, nasal continuous positive airway pressure (nasal CPAP), is often prescribed to offset the increase in pharyngeal collapsibility during sleep. When an appropriate level of nasal CPAP is prescribed, this treatment is highly effective at opening the pharynx during sleep regardless of the mechanism for elevated pharyngeal collapsibility. Patient compliance with nasal CPAP, however, is variable. Up to 35% of patients receiving nasal CPAP discontinue its use, and those who continue treatment do not use it consistently. Therefore, other effective treatments are needed for obstructive sleep apnea to complement or replace nasal CPAP in patients who do not tolerate its use.

Many treatments are known to improve the severity of obstructive sleep apnea. These treatments include the following: weight loss,16-18 protriptyline,19,20 uvulopalatopharyngoplasty,21-23 tongue-retaining devices,24,25 mandibular advancement devices26,27 and surgery,28 and electrical stimulation of the pharyngeal muscles.29,30 While each of these treatments offsets an increase in pharyngeal collapsibility during sleep, none of these treatments is universally effective in the manner of nasal CPAP. Furthermore, we are often unable to predict the effect of a treatment on the severity of obstructive sleep apnea in a specific patient. The development of a practical method for predicting the effect of a treatment on the severity of obstructive sleep apnea in a specific patient might guide the clinician's choice from the various alternatives to nasal CPAP.

To guide the clinician in selecting treatment for obstructive sleep apnea, we consider the relationship between obstructive sleep apnea and pharyngeal collapsibility. Current evidence suggests that pharyngeal collapsibility varies along a continuum from health (low collapsibility) to disease (high collapsibility). A primary goal of any therapy, therefore, is to decrease pharyngeal collapsibility to levels known to be associated with normal breathing patterns during sleep. Our approach will be to establish a quantitative basis for treating sleep apnea depending on both the degree to which pharyngeal collapsibility is elevated in a patient and the amount by which it is reduced with a specific treatment. To accomplish this, a physiologic basis for measuring pharyngeal collapsibility will be provided; the pharyngeal collapsibility of individuals with varying levels of pharyngeal airway obstruction during sleep will be examined; and the relationship between changes in pharyngeal collapsibility and changes in the severity of obstructive sleep apnea will be elucidated. From this

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In this discussion of the subject, we have adapted his approach to the purpose of describing the pharyngeal airway as a collapsible tube. The model (Fig 1A) consists of a tube passing through a sealed box. The tube is characterized by two rigid segments with a collapsible segment interposed between, within the box. The pressure in the box outside the collapsible segment is constant (Pout). In this discussion, we will assume that the segment within the box is so collapsible that whenever the pressure within the segment (Pin) falls below Pout, the segment collapses. Conversely, whenever the Pin exceeds the Pout, the segment opens. Thus, the Pin at the moment of collapse (Pin') is equal to the Pout. The Pin' is also known as the critical pressure (Pcrit) of the segment. Therefore, for the collapsible segment of this tube Pcrit=Pout.

Now consider the effect of Pout on flow through the collapsible segment. In Figure 1B, we have set the pressure within the box at +10 cm H2O (Pout=+10) and applied a pressure of +5 cm H2O upstream to the collapsible segment (Pus=+5). Under these conditions (Pin<Pout, +5<10), the segment remains occluded and there is no flow through the tube. There can be no flow as long as the Pus is below Pcrit (+10 cm H2O). We will refer to the circumstance of Pus<Pcrit as condition 1. This condition is analogous to zone 1 of the pulmonary vasculature of West et al. 35

Let us now examine the effect of an increase in Pus to a level above Pcrit (+15 cm H2O, Fig 1C). Because the Pus exceeds Pcrit, the collapsible segment opens and flow begins. In this example, let us assume that the pressure immediately downstream to the collapsible segment (Pds) remains below Pcrit (+9 cm H2O). Under these circumstances, the downstream end of the collapsible segment collapses or flutters to maintain its intraluminal pressure at +10 cm H2O. The reason for this phenomenon can be understood intuitively. If the intraluminal pressure at the site of

flow through collapsible tubes: the model

The study of pharyngeal collapsibility in obstructive sleep apnea has benefited from earlier research on other systems of collapsible biological tubes. Collapsible tubes are important biological conduits and their function modulates many physiologic events in man. Well-recognized examples include collapse of central veins entering the right atrium, 31 collapse of intrathoracic airways on forced exhalation, 32,33 collapse of the nasal alae at high inspiratory airflows, 34 collapse of pulmonary capillaries in lung zones 1 and 2, 35 and collapse of subendocardial capillaries at high levels of left ventricular end-diastolic pressure. 36 Explanations of the behavior of these varied biological conduits have been proposed using a simple model of flow through collapsible tubes. In recent years, this model has also been applied to the pharyngeal airway. Using the model, investigators have quantified differences in pharyngeal collapsibility during sleep among normal subjects, snorers, and patients with obstructive sleep hypopnea and apnea. Moreover, the model has been used to study the relationship between changes in pharyngeal collapsibility and changes in the severity of obstructive sleep apnea. The simplicity of the model makes it a potentially useful clinical tool for improving utilization of the many treatment alternatives to nasal CPAP.

The subject of flow through collapsible tubes has been discussed in a clearly written review by Green. 37

Analysis, we will develop a method to treat obstructive sleep apnea by reducing pharyngeal collapsibility "quantitatively." Throughout the discussion, we will attempt to identify deficits in our knowledge and to suggest potentially fruitful opportunities for clinical investigation. Finally, we will demonstrate how pharyngeal collapsibility can be measured in a clinical sleep laboratory using nasal CPAP.
collapse were to exceed +10 cm H₂O (Pcrit) and the site opened widely, the intraluminal pressure at the site would fall to below Pcrit because of the lower pressure in the rigid segment immediately downstream. The decrease in intraluminal pressure at the site to below Pcrit would cause it to collapse and the flow to cease. Cessation of the flow would cause the pressure at the site to become +15 cm H₂O (Pus), the site would reopen, and the sequence would begin again. Thus, when Pds is less than Pcrit, the downstream end of the collapsible segment collapses or flutters.

How does collapse of the collapsible segment affect flow through the tube? Collapse of the segment fixes the pressure at its downstream end at Pcrit. Therefore, the pressure gradient driving flow through the tube becomes fixed at Pus–Pcrit and remains independent of changes in Pds (Pcrit is the effective downstream pressure for flow as long as Pds<Pcrit). Because the pressure gradient driving flow is fixed, flow also becomes fixed and does not exceed a maximal level (Vmax) even if Pds falls further. The Vmax of the collapsible segment is given by the following equation:

Vmax=(Pus–Pcrit)/Rus  equation (a)

where Rus is the resistance of the portion of the tube upstream to the site of collapse. Because collapse of the collapsible segment limits flow through the tube at Vmax, it is termed the flow-limiting site (FLS). Therefore, when Pus exceeds Pcrit and Pds is less than Pcrit, collapse of the FLS fixes flow through the tube at Vmax. This circumstance will be referred to as condition 2. This condition is analogous to zone 2 of the pulmonary vasculature of West et al.35

Now consider the influence of alterations in Pus on the level of Vmax under model conditions 1 and 2. The relationship between Pus and Vmax for our model is illustrated in Figure 2. As long as Pus is less than +10 cm H₂O, there is no flow (condition 1, Figure 1B). When the Pus exceeds +10 cm H₂O (the Pcrit of the collapsible segment), Vmax increases linearly with Pus (condition 2, Figure 1C). From equation (a), the Pus at 0 flow is Pcrit and the slope of the relationship between Vmax and Pus is 1/Rus.

Finally, let us examine the effect of increasing Pds above Pcrit on flow through the tube. In Figure 1D, the pressure downstream from the collapsible segment is increased to +11 cm H₂O. Because the pressure throughout the collapsible segment is now greater than the Pcrit, the collapsible segment will open widely. There will be no flow limitation (as in condition 2) and the flow through the tube will be determined from the following equation:

V=(Pus–Pds)/R  equation (b)

where R is the resistance of the entire tube between the upstream and downstream reference points. We will refer to this circumstance as condition 3. This condition is analogous to zone 3 of the pulmonary vasculature of West et al.35

THE PHARYNGEAL AIRWAY AS A COLLABILSIBLE TUBE

The advantage of modeling the pharyngeal airway as a collapsible tube is that the model can be used to examine the factors causing airflow obstruction even though the precise mechanisms are not fully understood. At present, much remains to be learned about the interactions of the pharyngeal muscles that maintain the pharyngeal airway patency during sleep. Nevertheless, if the pressure-flow relationships of the pharyngeal airway are described empirically by a simple model, then that model can be used to organize our thinking and to predict pharyngeal airway function, regardless of the physiologic mechanisms responsible. Although at first glance, the upper airway does not resemble a tube running through a box, its pressure-flow relationships are remarkably similar to those of the model that we have presented. In the following discussion, we encourage the reader to focus on the similarity between empirically observed flow through the pharyngeal airway during sleep and that predicted by the model of flow through collapsible tubes.

Figure 2. This figure demonstrates the effect of progressively increasing the pressure upstream to the collapsible segment (Pus, Figs 1B+C) on maximal flow through the tube (Vmax). Until the Pus exceeds +10 cm H₂O (Pcrit), the tube remains collapsed and occluded (condition 1). Above +10 cm H₂O, Vmax and Pus are linearly related with a slope of 1/Rus as long as the pressure downstream from the collapsible segment is less than Pcrit (condition 2).
Fluttering during pharyngeal collapses is associated with the limitation of inspiratory flow and closely resembles model condition 2 (Fig 1C). In contrast to the preceding two groups who demonstrate complete and partial pharyngeal airway obstruction during sleep, individuals who breathe normally during sleep have a pharyngeal airway that is widely patent like a collapsible tube under condition 3 (Fig 1D). Thus, obstructive sleep apnea, snoring, and obstructive hypopnea, and normal breathing resemble the three levels of patency in collapsible tubes.

To understand the behavior of the pharyngeal airway during obstructive apnea, let us compare the airflow patterns of the pharynx to those of the model. If the pharyngeal airway during obstructive apnea resembles condition 1 of the model (Fig 1B), then it behaves like a collapsible tube when $P_{us}$ is less than $P_{crit}$. For the pharyngeal airway during inspiration, $P_{us}$ is atmospheric pressure. Therefore, the $P_{crit}$ of the pharyngeal airway during obstructive apnea must be greater than atmospheric pressure. What are the determinants of the $P_{crit}$ of the pharyngeal airway? Referring back to the model, we observe that the $P_{crit}$ is equal to the $P_{out}$, the pressure surrounding the collapsible segment of the tube. To apply this model to the pharyngeal airway, we can think of the pharyngeal airway wall as a thin, very collapsible mucosal membrane (ignoring its muscle mass). The $P_{out}$ of the pharyngeal airway consists of the pressures exerted on the airway by the pharyngeal muscle (resulting from its mass and the pressure resulting from its contraction) and the tissues surrounding the pharyngeal airway. Therefore, the pharyngeal airway $P_{crit}$ is a pressure that is equal to the pressure exerted on the pharyngeal airway by these same structures. Because the $P_{crit}$ is equal to the pressures tending to collapse the airway, it is an index of pharyngeal airway collapsibility (the greater the $P_{crit}$, the more collapsible the airway). Thus, during obstructive apnea, the airway is occluded because the $P_{crit}$ of the pharyngeal airway is greater than atmospheric pressure. The pharyngeal airway is too collapsible to remain open at atmospheric pressure.

**Similarities Between Pharyngeal Airway Obstruction and the Model**

The pharyngeal airway of man during sleep demonstrates three distinct levels of patency that parallel the three conditions of the model of flow-through collapsible tubes discussed above. Individuals with obstructive sleep apnea have a pharyngeal airway that is completely occluded during sleep. The appearance of the human airway during an obstructive apnea is illustrated in Figure 3 by three midsagittal views of the pharyngeal airway of a patient with obstructive sleep apnea. Figure 3A illustrates his patent pharyngeal airway during wakefulness. Figures 3B and 3C demonstrate the progressive collapse of his pharyngeal airway during sleep. The appearance of the completely occluded pharyngeal airway in Figure 3C resembles the appearance of the collapsible segment of the model in condition 1 (Fig 1B). Unlike subjects with obstructive apnea, individuals who snore have a pharyngeal airway that collapses and flutters with inspiration during sleep. The fluttering of the airway, snoring, or obstructive hypopnea, is associated with the limitation of inspiratory flow and closely resembles model condition 2 (Fig 1C). In contrast to the preceding two groups who demonstrate complete and partial pharyngeal airway obstruction during sleep, individuals who breathe normally during sleep have a pharyngeal airway that is widely patent like a collapsible tube under condition 3 (Fig 1D). Thus, obstructive sleep apnea, snoring, and obstructive hypopnea, and normal breathing resemble the three levels of patency in collapsible tubes.

**Modeling the Response to Nasal CPAP in Obstructive Sleep Apnea**

Now let us examine the effect of an increase in nasal pressure ($P_N$, analogous to $P_{us}$) above the pharyngeal $P_{crit}$ in a patient with an obstructive apnea. When we raise the $P_N$ from atmospheric pressure to a pressure above the pharyngeal $P_{crit}$, the pharyngeal airway is no longer occluded and airflow resumes when the patient inspires. If $P_N$ is slightly above $P_{crit}$, the pressure downstream from the collapsible segment of the pharynx (laryngeal pressure) will fall below $P_{crit}$ during inspiration. Under these conditions, as the patient inspires, collapse of the pharynx will lead to flow lim-
4B Figs

Finally, at higher levels of $P_N$, inspiratory flow and downstream pressure parallel each other (Fig 4E). This suggests that at higher levels of $P_N$, the laryngeal pressure does not fall below Perit during inspiration, and inspiratory flow limitation does not occur (condition 3). Therefore, as $P_N$ is increased during sleep in a patient with obstructive sleep apnea, the upper airway passes through the three levels of patency observed in a collapsible tube.

The response of inspiratory airflow to nasal CPAP administration can be examined in more detail to define a relationship of $V_{\text{Imax}}$ to $P_N$ for the obstructive sleep apnea patient (similar to the relationship of $V_{\text{Imax}}$ to $P_{\text{Us}}$ in the model, Fig 2). Smith and associates\textsuperscript{39} examined the relationship of $V_{\text{Imax}}$ to $P_N$ in six patients with obstructive sleep apnea. They consistently observed a linear relationship between $V_{\text{Imax}}$ and $P_N$ that intercepted the x-axis at Perit (defined as the value of $P_N$ at $V_{\text{Imax}}=0$, Fig 5). Thus, in obstructive sleep apnea patients, the pressure-flow relationships of the pharyngeal airway are similar to those of a simple collapsible tube and can be used to define a Perit in obstructive apnea that is greater than atmospheric pressure.

**Perit and the Spectrum of Pharyngeal Collapsibility**

Using the model of a simple collapsible tube, we can also predict the pressure-flow relationships of the normal pharyngeal airway during sleep. In contrast to...
obstructive sleep apnea patients, normal individuals have patent pharyngeal airways without inspiratory flow limitation during sleep. In other words, at atmospheric pressure, their pharyngeal airways are in condition 3. If the pharyngeal airway of normal individuals also behaves like a collapsible tube, then the Pcrit of their airway must be substantially below atmospheric pressure. To test this hypothesis, Schwartz and associates\(^4\) decreased the nasal pressure of normal individuals during sleep. Normal subjects slept in supine position while wearing a nasal mask attached to a vacuum source. The PN was progressively lowered during nonrapid eye movement (NREM) sleep while inspiratory flow, PN, and Pesoph were measured. With progressive lowering of PN, each subject demonstrated snoring (inspiratory flow limitation, condition 2). Figure 6 demonstrates a plot of V\(_{\text{max}}\) against PN for one of the normal subjects. As PN was decreased, V\(_{\text{max}}\) decreased in a linear fashion until it fell to zero as the PN approached the Pcrit of the pharyngeal airway (−9 cm H\(_2\)O). Below Pcrit, there was no inspiratory airflow (condition 1) and the normal individual resembled a patient with obstructive sleep apnea (Fig 7). For the group of normal subjects, the Pcrit was −13.5±3.4 cm H\(_2\)O. Therefore, similar to the pharyngeal airway in obstructive sleep apnea, the pressure-flow relationships of the normal pharyngeal airway can be predicted by a model of flow through a collapsible tube. The two airways differ only in the values of their Pcrit: Pcrit in patients with obstructive sleep apnea being greater than atmospheric pressure (a more collapsible pharyngeal airway) and Pcrit in normal subjects being subatmospheric (a less collapsible pharyngeal airway).

How can differences in Pcrit explain the varied degrees of pharyngeal obstruction during sleep? From the work of Schwartz and associates,\(^4\) individuals with a Pcrit of below −8 cm H\(_2\)O have normal inspiratory airflow during sleep. As Pcrit increases above −8 cm H\(_2\)O, airflow pressures fall below Pcrit during inspiration causing pharyngeal collapse and flow limitation. Under conditions of flow limitation, V\(_{\text{max}}\) decreases directly with PN−Pcrit (equation [a], PN=atmospheric pressure). As Pcrit increases, PN−Pcrit decreases and V\(_{\text{max}}\) decreases until it becomes zero at PN=Pcrit (Pcrit=atmospheric pressure). Therefore, the clinical progression from normal airflow to snoring, obstructive hypopnea, and apnea should be paralleled by a progressive rise in Pcrit. Figure 8 demonstrates the values of Pcrit determined for normal individuals\(^4\) and for groups of snorers, obstructive sleep hypopnea patients, and obstructive sleep apnea patients.\(^4\) From these data, it is evident that a spectrum of Pcrit values exists beginning with obstructive sleep apnea patients whose values of Pcrit are at or above atmospheric pressure, followed by patients with obstructive hypopnea whose Pcrit values range from atmospheric pressure to −4 cm H\(_2\)O, followed by snorers with values between −4 cm H\(_2\)O and −8 cm H\(_2\)O and normal individuals with values below −8 cm H\(_2\)O. Thus, increasing degrees of pharyngeal obstruction during sleep result when increasing (less subatmospheric) values of Pcrit progressively limit the level of maximal flow through the pharynx during sleep.

If the correlation between increasing levels of pharyngeal Pcrit and increasing levels of sleep-related

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**Figure 5.** This figure demonstrates the relationship between maximal inspiratory flow (V\(_{\text{max}}\)) and nasal mask pressure (P\(_N\)) for the breaths identified with arrows in Figure 4B through D. Above the pharyngeal critical pressure (Pcrit) of +1.2 cm H\(_2\)O, P\(_N\) and V\(_{\text{max}}\) are linearly related. The relationship of V\(_{\text{max}}\) to P\(_N\) in this figure is similar to the relationship of V\(_{\text{max}}\) to Pus in Figure 2.

**Figure 6.** This figure demonstrates the relationship of maximal inspiratory flow (V\(_{\text{max}}\)) to nasal mask pressure (P\(_N\)) for a normal subject during NREM sleep. The plot contains data from several experimental trials (progressive reductions of nasal mask pressure). Each point represents a mean value of V\(_{\text{max}}\) (with SD bars) for several breaths at a particular P\(_N\). Open symbols represent samples from stage 2 sleep, while closed symbols represent samples from slow-wave sleep. The relationship of V\(_{\text{max}}\) to P\(_N\) in this figure is the same as the relationship of V\(_{\text{max}}\) to Pus in Figure 5 and V\(_{\text{max}}\) to Pus in Figure 2 (reprinted with permission\(^4\)).
airflow obstruction holds true, then we should be able to predict the pharyngeal Pcrit in a newly described syndrome of airway obstruction during sleep. Since the characterization of Pcrit in snorers and patients with obstructive sleep hypopnea by Gleadhill and associates,42 Guilleminault and associates43 have described the upper airway resistance syndrome (UARS). The UARS combines clinical features of both snoring and obstructive sleep hypopnea. Like patients who snore, patients with UARS demonstrate mild inspiratory flow limitation during sleep without recurrent oxyhemoglobin desaturation. Like patients with obstructive sleep hypopnea, patients with UARS experience sleep fragmentation and daytime sleepiness that resolves with nasal CPAP use. Because inspiratory airflow is only mildly reduced in the UARS, we would predict a Pcrit near that of snorers (−6 cm H2O). Nevertheless, patients arouse recurrently suggesting a level of Pcrit closer to patients with obstructive hypopnea (−2 cm H2O). We postulate that the range of Pcrit for patients with UARS is −2 cm H2O to −6 cm H2O, intermediate between that of snorers and that of patients with the sleep hypopnea syndrome. Although the Pcrit in patients with UARS has not yet been determined, its measurement would contribute to our understanding of the spectrum of pharyngeal collapsibility from health to disease.

Pcrit and Therapy for Obstructive Sleep Apnea

From the discussion of the pharyngeal airway as a collapsible tube, normal individuals have a differential of at least 8 cm H2O between PN (atmospheric pressure) and pharyngeal Pcrit (<−8 cm H2O). As the differential between PN and Pcrit decreases with increasing Pcrit, inspiratory flow limitation develops and V1max decreases causing snoring, obstructive hypop-
ne, and obstructive apnea. In contrast, we predict that airflow obstruction will be abolished and levels of inspiratory airflow will return to normal in obstructive sleep apnea, when a pressure differential of 8 cm H₂O between PN and pharyngeal Pcrit is reestablished. Therefore, effective therapy of obstructive sleep apnea requires that a sufficient gradient from PN to the site of pharyngeal collapse be achieved.

The reestablishment of the normal pressure gradient between PN and pharyngeal Pcrit can be accomplished by one of two means. First, PN can be increased to a level of 8 cm H₂O above Pcrit using nasal CPAP. Because Pcrit in hypopneic and apneic individuals usually lies between −4 and +4 cm H₂O (Fig 8), nasal pressures between +4 and +12 cm H₂O usually provide adequate relief of pharyngeal airway obstruction. Alternatively, the differential between PN and Pcrit can be widened by decreasing the Pcrit. Thus, it should be possible to eliminate the sleep fragmentation of obstructive sleep apnea (hypopnea) with treatments that decrease Pcrit to below −4 cm H₂O and to eliminate snoring with treatments that decrease Pcrit to below −8 cm H₂O. Studies of the effects of uvulopalatopharyngoplasty (UPPP) and weight loss on Pcrit support the concept that a change in the severity of obstructive sleep apnea accompanies a reduction in Pcrit below these threshold levels.

Schwartz and associates examined the relationship between Pcrit and the response to UPPP. UPPP is a surgical procedure designed to alleviate pharyngeal obstruction during sleep by the removal of tissue at the level of the velopharynx and oropharynx. It has long been recognized that the response of pharyngeal obstruction to UPPP is quite variable. Although some patients experience a marked reduction in the frequency of their disordered breathing events (apnea/hypopnea index, AHI), others are unaffected by the procedure. To determine whether a therapeutic response to UPPP is dependent on a reduction in pharyngeal Pcrit, the preoperative and postoperative Pcrit of 13 patients who underwent UPPP for obstructive sleep apnea was determined. Of the 13 patients, 6 responded favorably to UPPP with a mean reduction in AHI of 84% while 7 patients were classified as nonresponders. Figure 9 demonstrates that responders to UPPP could not be differentiated from nonresponders by their preoperative Pcrit (−0.8 ± 3.0 and 1.1 ± 1.6, respectively). Following UPPP, patients who responded demonstrated a mean decrease of their Pcrit of 6.5 cm H₂O. Moreover, patients whose pharyngeal Pcrit decreased to below −4 cm H₂O following UPPP reduced their AHI to a normal level. In contrast, nonresponders had no significant change in Pcrit following UPPP. Thus, patients who respond to UPPP demonstrate a change in pharyngeal collapsibility manifested as a change in Pcrit. The study also supports the hypothesis that an effective treatment for obstructive hypopnea and apnea must decrease the Pcrit to near −4 cm H₂O, the level separating patients with obstructive hypopnea from those with asymptomatic snoring (Fig 8).

Research into the effect of weight reduction on pharyngeal collapsibility has modified our approach to weight reduction for the treatment of obstructive sleep apnea. Weight loss is known to reduce the AHI in obstructive sleep apnea patients. In 15 moderately obese patients with moderate to severe obstructive sleep apnea, Smith and associates demonstrated a 45% reduction in the NREM sleep AHI following a 9% body weight loss. Patients varied widely, however, in their responses to weight loss. Some patients completely eliminated their disordered breathing while others remained unchanged. Because weight loss is often difficult to achieve and it does not consistently reduce the AHI, physicians have not used weight loss as a primary treatment for obstructive sleep apnea.

To better understand the relationship among weight loss, airway collapsibility, and the frequency of disordered breathing events, Schwartz and associates studied the effect of weight loss on the pharyngeal Pcrit.
of patients with moderate to severe obstructive sleep apnea. They found that each patient who lost weight demonstrated a decrease in $P_{\text{crit}}$, and that the change in $P_{\text{crit}}$ was roughly correlated with the amount of weight lost (Fig 10). For the group of 13 patients who lost a mean 17% of their body weight, $P_{\text{crit}}$ decreased by 5 cm H$_2$O. In contrast, 13 patients who did not lose weight demonstrated little change in $P_{\text{crit}}$. From these findings, they concluded that weight loss affects obstructive sleep apnea by reducing the collapsibility of the pharyngeal airway roughly in proportion to the amount of weight lost.

If weight loss consistently reduces the collapsibility of the pharyngeal airway, why is the response of the AHI to weight loss so variable? The answer emerges as we examine the relationship between $P_{\text{crit}}$ and AHI (Fig 11). Those patients with the greatest reduction in AHI began with low values of pharyngeal $P_{\text{crit}}$ that fell below $-4$ cm H$_2$O after weight loss. Patients whose AHI did not fall significantly began with higher values of $P_{\text{crit}}$ that remained substantially above $-4$ cm H$_2$O after weight loss. From these findings, it is clear that two principal factors, the initial pharyngeal $P_{\text{crit}}$ and the amount of weight lost, determine the therapeutic response to weight loss in patients with obstructive sleep apnea.

From the study of Schwartz and associates, we may conclude that the response to a given decrease in body weight depends largely on the initial value of $P_{\text{crit}}$. A loss of 10% body weight (a change in body mass index of 3 to 4 kg/m$^2$ in a patient with an initial body mass index of 35 kg/m$^2$) should produce a decrease in $P_{\text{crit}}$ of 2 to 4 cm H$_2$O (Fig 10). If a patient's initial $P_{\text{crit}}$ is $-2$ cm H$_2$O, then this amount of weight loss should result in a $P_{\text{crit}}$ of $-4$ to $-6$ cm H$_2$O. The resulting value of $P_{\text{crit}}$ is in the range of asymptomatic snoring and the patient should experience an improvement in his or her symptoms. In contrast, if the same patient's initial $P_{\text{crit}}$ was $+4$ cm H$_2$O, a loss of 30% body weight (10 to 12 kg/m$^2$, Fig 10) would be required to achieve the same result. Recognizing the difficulty in achieving this weight loss, one may conclude that the patient will probably not benefit clinically from a weight loss program as the sole treatment for obstructive sleep apnea.

In the studies of weight loss and UPPP, we observe two principles that should guide the clinician in treating obstructive sleep apnea. First, a decrease in $P_{\text{crit}}$ is necessary to treat obstructive sleep apnea successfully without nasal CPAP. Second, a decrease in AHI to normal levels can be expected when the $P_{\text{crit}}$ decreases to below $-4$ cm H$_2$O. These observations suggest that to apply a treatment for obstructive sleep apnea effectively, we must know how much $P_{\text{crit}}$ falls in response to the treatment. Depending on the magnitude of this decrease in $P_{\text{crit}}$, the response of a particular patient to the treatment can be predicted based on the patient's pretreatment value of $P_{\text{crit}}$. This knowledge will enable clinicians to offer their patients greater certainty about treatment response and to effectively combine treatments to decrease a patient's $P_{\text{crit}}$ to below $-4$ cm H$_2$O. In this way, quantifying

**Figure 9.** This figure demonstrates the relationship between the disordered breathing rate (DBR, synonymous with AHI) and $P_{\text{crit}}$ for 13 patients who underwent UPPP. A line connects pre-UPPP and post-UPPP symbols for each patient. Consistent reductions in $P_{\text{crit}}$ and DBR were seen only in responders. Reductions in $P_{\text{crit}}$ to below $-4$ cm H$_2$O resulted in complete resolution of sleep disordered breathing (reprinted with permission).

**Figure 10.** This figure demonstrates the relationship between the change in weight (represented by body mass index) and the change in $P_{\text{crit}}$ for 26 patients with obstructive sleep apnea/hypopnea. Patients who lost weight demonstrated a rough correlation between the amount of weight loss and the change in $P_{\text{crit}}$ ($p=0.056$). The usual care group did not lose weight (reprinted with permission).
patients, the Pcrit can be approximated during a nasal CPAP study by including the PN among the monitored parameters of the polygraph tracing. When PN is below Pcrit, the pharyngeal airway is occluded and there is no fluctuation of mask pressure with respiration (Fig 4A). When the PN is raised above Pcrit and respiratory airflow begins, mask pressure fluctuates with respiration (Fig 4B). By raising the PN in 1 to 2 cm H2O increments during stage 2 sleep, the Pcrit of the airway can be established between the last PN at which mask pressure does not fluctuate with respiration and the first PN at which the fluctuations are apparent. The simplicity of this approach should make an approximation of Pcrit available to most sleep clinicians.

The clinical investigator will assume an important role in acquiring data about the effect of specific interventions on pharyngeal Pcrit. For example, Meurice and associates have compared the Pcrit with the mouth closed to the Pcrit with the mouth open in normal sleeping subjects and found a 4 cm H2O increase in Pcrit (increased pharyngeal collapsibility) with the mouth open. Similar investigations are needed on a variety of treatments for obstructive sleep apnea. At present, little is known about the effects of protriptyline, oral devices, and surgical mandibular advancement on the Pcrit of patients with obstructive sleep apnea. Furthermore, although we routinely advise patients to avoid alcohol and sedative hypnotics before sleep, little is known about the effects of alcohol and benzodiazepines on Pcrit. By routinely measuring the change in Pcrit as an outcome parameter in a variety of therapeutic trials, clinical investigators can collect the data needed to facilitate this change in approach to the management of obstructive sleep apnea.

Adding the pharyngeal Pcrit to the list of measured outcomes will also increase the sensitivity of therapeutic trials. At present, the outcomes that we accept for demonstrating a treatment’s clinical efficacy are a reduction in the frequency of disordered breathing events, an improvement in oxygenation, and an improvement in sleep architecture. With these outcomes, only therapies that reduce Pcrit to near −4 cm H2O, the level needed to convert hypopnea to snoring, will demonstrate efficacy. By unknowingly selecting patients with high pretreatment values of Pcrit, or by testing treatments that decrease Pcrit modestly (but may be well tolerated and combine well with other treatments), clinical researchers may not recognize the full potential of a new treatment. Measurement of Pcrit before and after treatment will provide a quantitative assessment of the effect of the treatment on pharyngeal collapsibility, the basic problem for obstructive sleep apnea patients.

In summary, we have applied a model of flow

**FIGURE 11.** This figure demonstrates the relationship between NREM DBR (DBR=disordered breathing rate, synonymous with AHI) and Pcrit in a group of weight loss and nonweight loss (usual care) patients with obstructive sleep apnea/hypopnea. A line connecting preintervention and postintervention symbols for each patient is shown. For weight loss patients, reductions in Pcrit to below −4 cm H2O resulted in complete resolution of sleep-disordered breathing (reprinted with permission).
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