

The Use of Matrix Repatterning in the Management Upper Airway Obstruction (Snoring and Sleep Apnea)

A Preliminary Investigation

By Dr. George B. Roth

Introduction:

Upper airway obstruction (UAO) may be partial, in the case of snoring, or complete, in the case of obstructive sleep apnea. Narrowing of the upper airway, during sleep, causes this often serious condition, affecting millions of people worldwide. The uvula and soft pallet may come into partial or total contact on the back wall of the upper airway. When the contact is partial or intermittent, snoring (a loud vibration of these soft tissues) may result. The tongue may also drop posteriorly onto the back wall of the upper airway, coming into contact with the uvula and soft pallet, thus forming a tight blockage, preventing any air from entering the lungs. Respiratory effort on the part of the diaphragm and chest may cause the blockage to seal tighter. In order to breathe the person must arouse or awaken, causing tension in the tongue, and thereby opening the airway, allowing air to pass into the lungs. Apnea, sleep apnea or obstructive sleep apnea is defined as the cessation of breathing for 10 or more seconds while asleep¹. Traditional methods of treatment usually involve Continuous Positive Airway Pressure (CPAP) devices. Matrix Repatterning techniques were used in ten cases of long-standing, moderate to severe cases upper airway obstruction.

Key Terms: Matrix Repatterning, Upper Airway Obstruction (UAO), Airway Obstruction Test (AOT), Continuous Positive Airway Pressure (CPAP)

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Introduction

Partial upper airway obstruction and obstructive sleep apnea (obstructive sleep apnea) are caused by the narrowing of the upper airway while asleep. The uvula and soft pallet may come into partial or total contact on the back wall of the upper airway. When the contact is partial or intermittent, snoring (a loud vibration of these soft tissues) may result. The tongue may also drop posteriorly onto the back wall of the upper airway, coming into contact with the uvula and soft pallet, thus forming a tight blockage, preventing any air from entering the lungs. Respiratory effort on the part of the diaphragm, and chest may cause the blockage to seal tighter. In order to breathe the person must arouse or awaken, causing tension in the tongue, and thereby opening the airway, allowing air to pass into the lungs. Apnea, sleep apnea or obstructive sleep apnea is defined as the cessation of breathing for 10 or more seconds while asleep.

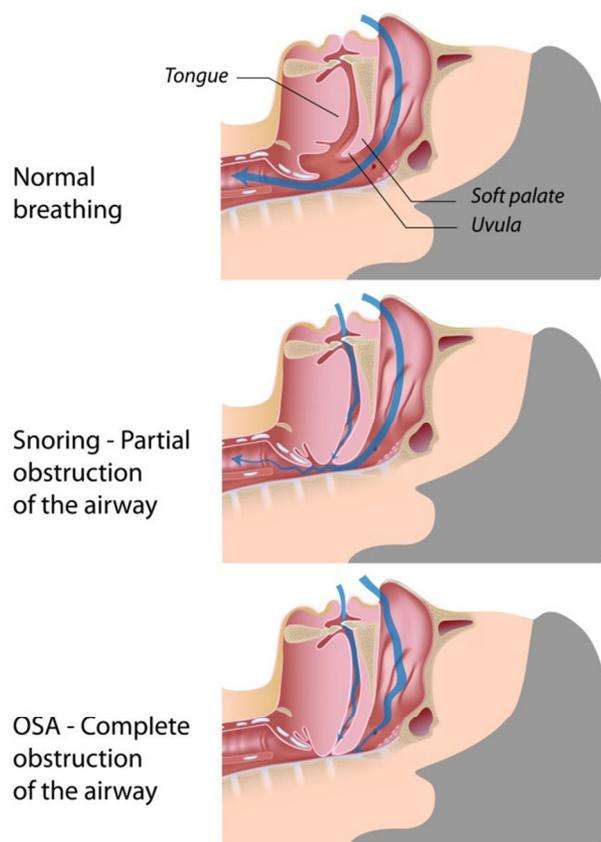


Figure 1: Normal Breathing and Airway Obstruction

Obstructive sleep apnea causes a drop in one's blood oxygen saturation (SaO₂) and an increase in the blood's carbon dioxide (CO₂). When the SaO₂ drops the heart will start pumping more blood with each beat. If the SaO₂ continues to drop the heart will start beating faster and faster. As the CO₂ increases the brain will try to drive the person to breathe. The effort and action of the abdomen and chest will increase. Eventually that action can become severe enough to cause an arousal, clearing the upper airway blockage, allowing the person to breathe. Then you go back to sleep and it happens all over again.

The American Academy of Sleep Medicine (AASM) rates the average number of obstructive sleep apnea events per hour as your Respiratory Distress Index (RDI). An RDI of 0 to 5 is normal; 5 to 20 is mild; 20 to 40 is moderate; over 40 is considered severe. An apnea event must last at least 10 seconds to be considered an event. It is not uncommon to see RDIs well above the 40. In some cases RDIs were well above 100, with events lasting as long as 90 to 120 seconds and SaO₂s going below 70% when normal is 95% to 100%.

Symptoms:

Most prominent symptoms are snoring, not breathing while asleep, excessive daytime sleepiness and obesity. Other symptoms include lack of concentration, forgetfulness, uncharacteristically irritable, anxiety, depression, mood and/or behavioral changes, morning headaches, disorientation at awakening and loss of sexual interest.

Diagnosis:

Diagnosis is typically made by a physician specially trained in sleep medicine. After a physical examination of the upper airway and an interview, if it is determined that the patient may have a sleep disorder, he or she will be asked to take a polysomnogram (sleep test). Most sleep centers and labs monitor 16 different sleep parameters including EEG, EKG, eye movement, chin movement, air flow, chest effort, abdomen effort, SaO₂, snoring and leg movement. Each parameter serves to help the physician make a correct diagnosis.

Conventional Treatment:

Continuous Positive Airway Pressure (CPAP) is the most common conventional treatment for obstructive sleep apnea. CPAP flow generators develop a constant, controllable pressure to keep the upper airway open so that the patient can breathe normally. CPAP is effective on 95% of patients with obstructive sleep apnea. The units are reliable, quiet and efficient and come in a variety of sizes and shapes, as shown in Figure 2.

Controlled pressure is induced through the nasal passage, holding the soft tissue of the uvula and soft palate and the soft pharyngeal tissue in the upper airway in position so the airway remains open while the patient descends into the deeper stages of sleep and REM sleep. The pressure acts much in the same way as a splint, holding the airway open.

There are typically three methods of inducing the pressure and airflow into the nasal cavity: nasal masks, nasal pillows and nasal seals. The most common used is the nasal mask. Nearly all CPAP manufactures make at least one style of nasal



Figure 2: CPAP: using pressurized air to open the upper airway

mask, most make two or three different ones. Nasal pillows are small, oval shaped latex rubber prongs that fit into the opening of the nostril. They are held in place by a shell that is attached to the headgear. When fit properly they are very comfortable and seldom leak. Nasal seals fit against the opening of the nostril and are held in place by a special frame attached to the headgear.

Matrix Repatterning:

Matrix Repatterning is a revolutionary manual approach, which addresses the *primary* sources of dysfunction in the connective tissue-fascial system in an efficient and effective manner. It incorporates objective and reproducible methods, based on a scientific foundation of structural pathophysiology. Treatment is gentle and painless, and results in global biomechanical reorganization and postural stabilization, encouraging the body towards normal, pain-free function. It is currently in use by physical therapists, chiropractors, physicians, osteopaths, athletic trainers, massage therapists and veterinarians on six continents and twelve countries around the world.

Matrix Repatterning uses a scanning procedure, which allows the practitioner to determine the location of so-called *primary restrictions*, followed by mechanical testing to determine specific vectors of fascial tension. Treatment is generally applied manually with light force directed into the restriction barriers. Matrix Repatterning is based on a revolutionary, new model of the underlying structure of organic tissue – the *Tensegrity Matrix* – which explains the complex interrelationship of all the structural components of the body. It extends the basic concept of the tissue response to injury, beyond the level of joint, muscle and ligament, to include all structures of the body as potential sources of dysfunction₂.

The tensegrity matrix model of the body, as elaborated by Stephen Levin, M.D.₂ and Donald Ingber, M.D., Ph.D.₃, among others, holds that the body tissues are composed of interconnected *tension icosahedra* (complex triangular trusses), which inherently provide a balance between stability and mobility. This structural model explains many of the observed phenomena related to body support, movement, response to stress and trauma, as well as the effects of therapeutic interventions. According to Ingber, a key investigator who has proven the existence of this structural model at the cellular level (see Figure 3), this model explains the biomechanical properties of the entire body. Other researchers have also confirmed the existence of this interconnected structural network within the extracellular matrix₅. According to Ingber, a key investigator who has proven the existence of this structural model at the cellular level:

“The principles of tensegrity apply at essentially every detectable size scale in the human body. At the macroscopic level, the 206 bones that constitute our skeleton are pulled up against the force of gravity and stabilized in a vertical form by the pull of tensile muscles, tendons and ligaments. In other words, in the complex tensegrity structure inside every one of us, bones are the compression struts, and muscles, tendons and ligaments are the tension-bearing members.”

*Donald E. Ingber, M.D., Ph.D.,
“The Architecture of Life”*

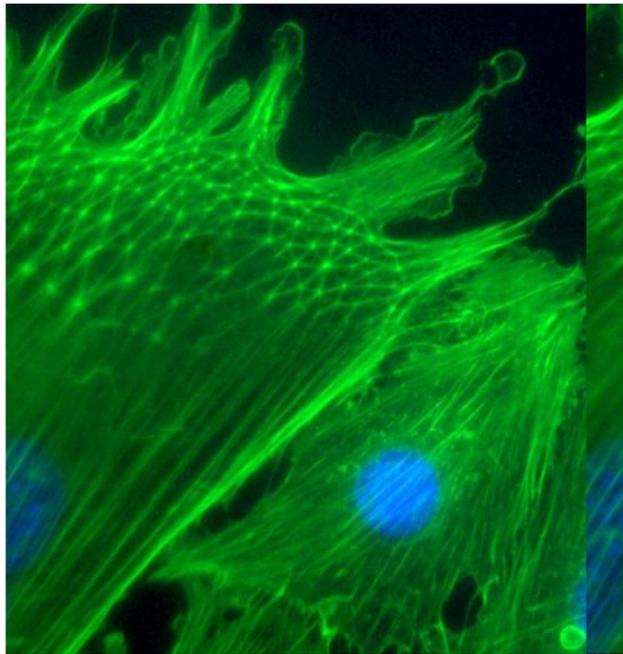
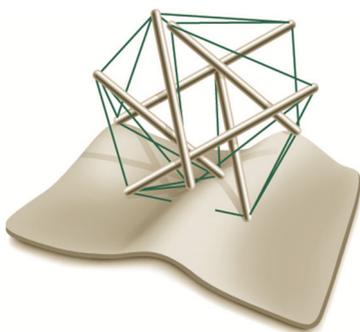
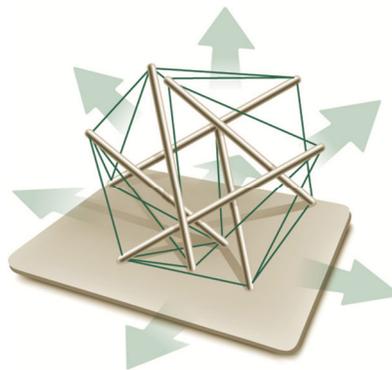


Figure 3: Cytoskeleton Showing Tensegrity Structure



NORMAL MOLECULAR STRUCTURE
Flexible, Balanced



RESTRICTED MOLECULAR STRUCTURE
Rigid, Expanded

Based on:
"The Architecture of Life"
Scientific American 1998

When the ability of the tissues to adapt or compensate becomes overwhelmed by mechanical or physiologic stress, the fascial system responds by altering the patterns of tension and elasticity. The tensegrity matrix explains the physiologic changes, which manifest in injured or strained tissue. The apparent fibrosis of muscle and fascia can be seen as an altered electro-mechanical relationship at the molecular level. The matrix is thus converted from a neutral, flexible form to a strained, high-energy, linearly-stiffened and expanded mode as shown in Figure 4.

Figure 4: Normal and Restricted molecular structure

Structural Implication of Upper Airway Obstruction:

The upper airway is constructed of the hard and soft palate above, the posterior pharynx, the tongue, and the epiglottis at the level of the tracheo-esophageal junction below. There are several mechanisms of partial or complete obstruction (see below):

Obstruction may occur by approximation of several structures and tissues. This may include the soft palate retracting toward the posterior pharyngeal wall, the soft palate descending to approximate the posterior aspect of the tongue, the tongue retracting toward the posterior pharynx, or descending to approximate the epiglottis. These tissues may deviate from their functional positions within the upper airway due to a number of structural dysfunctions, including cranial vertex or occipital trauma, leading to descent and/or protraction of the cranial base, along with the maxillary portion of the roof of the upper airway. Vertex compression, in our studies has also shown a tendency to lead to radial expansion of the upper cervical vertebrae (an intraosseous deformation), leading to loss of anterior/posterior dimension of the upper airway. Facial trauma may cause deviation of the maxilla or mandible. Hyperflexion injury of the cervico-thoracic spine may also induce an approximation of the posterior tissues toward the tongue and epiglottis. This is common in motor vehicle collisions (rear end or front end) and in falls onto the back of the head or upper back. Visceral lesions in the thoracic and/or abdominal cavities may also create mechanical traction on the esophageal fascia, affecting the position of the tongue in relation to the structures listed above. Several other mechanisms of structural dysfunction are also currently under investigation.

Assessment:

In cases of upper airway obstruction, a specific airway obstruction test (AOT), developed by the author, was also used to verify partial or complete obstruction. This involves placing the patient in an accentuated position of upper cervical hyperflexion or moderate extension, along with varying degrees of rotation or lateral flexion. The ease or difficulty of breathing, along with the amount of airflow turbulence noise was recorded for each position. The dysfunctional structural patterns associated with snoring and sleep apnea (see above), were then evaluated, using the standard Matrix Repatterning assessment.

Method of Treatment:

A maximum of four treatments to resolve these patterns were administered over a maximum period of two months for ten patients with moderate to severe upper airway obstruction. Two of these cases were previously diagnosed with significant sleep apnea, as verified by sleep studies.

Results:

AOT was improved significantly in 80% of the cases. Patients (and spouses, or sleeping partners) reported a cessation or a significant improvement in snoring in 70% of the cases. The two individuals, diagnosed with sleep apnea, reported they were able to sleep through the night without the assistance of a CPAP machine, on which they were previously dependant. These findings suggest that structural dysfunction may play a role in the development of upper airway obstruction and that Matrix Repatterning procedures may be beneficial in the management of these conditions. The findings suggest that a randomized controlled trial within a broader population base might be justified.

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