Obstructive Sleep Apnea (OSA) is one of many sleep disorders. It is caused by a collapse of the pharyngeal airway as a result of both Anatomic and Physiologic pathologies. A recent study shows OSA being a heterogeneous disorder. Although anatomy is an important determinant, abnormalities of nonanatomic traits are also present in most patients with OSA. The Study confirms that nonanatomic features play an important role in 56% of patients with OSA. Some of these nonanatomic features being: 36% of patients with OSA had minimal genioglossus muscle responsiveness during sleep, 37% had a low arousal threshold, 36% had high loop gain etc.¹

Unfortunately most in our profession treat OSA with little to no consideration of physiology. Mandibular Advancement Devices (MAD) are fabricated by today’s dentists with arbitrary and sometimes excessive protrusions violating a patient’s neuromuscular, TMJ, cervical, and in many instances, airway physiology. This leads to a less than favorable outcome.
There are many definitions of success for oral appliance therapy (OAT) - some very strict, and others very liberal. Morgan et al defined success as a: >50% decrease in the Respiratory Disturbance Index (RDI) and post treatment RDI <20 events per hour. Is this really the best our profession can achieve?

There are some anthropomorphic, physiological, and polysomnographic predictors of successful oral appliance treatment outcomes that are mentioned in the literature:

Female, lower age, lower body mass index (BMI), smaller neck circumference, lower baseline AHI, supine-dependent OSA, primary oropharyngeal collapse of the upper airway during sleep, larger retropalatal airway space, decreased distance between the hyoid and mandibular plane, decreased distance of Sella-Hyoid, narrow SNB (sella–nasion–B point) angle, and a wider SNA (sella–nasion–A point) angle.

More recent studies find adjusted neck circumference with supine REM sleep respiratory events more predictive of OAT success.

All published papers about Mandibular advancement devices (MAD) for OSA show one thing in common: that the treatment protocol, bite registrations, and MAD titrations are aimed anatomically in trying to keep a patent airway, with little to no physiologic considerations. Hence, many times this ends with some serious undesirable side effects such as a worsening of the Apnea-Hypopnea-Index (AHI), and an unmasking of central apnea, and cervical vertebrae displacement etc. We are not treating phantoms nor corpses...we are treating human beings and treating anatomy and physiology.

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**CLINICAL PREDICTION OF SLEEP APNEA: ANC (ADJUSTED NECK CIRCUMFERENCE)**

<table>
<thead>
<tr>
<th>Neck circumference</th>
<th>High blood pressure + 4 cm</th>
<th>Snoring + 3 cm</th>
<th>Observed apnea + 3 cm</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 43</td>
<td>low probability</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>43 - 48</td>
<td>moderate probability (4 to 8 times as probable)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt; 48</td>
<td>high probability (20 times as probable)</td>
<td></td>
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</tr>
</tbody>
</table>

Therefore, any type of arbitrary and aggressive protrusive techniques of bite registrations, without objectively taking into consideration the neuromuscular physiology, and/or any type of an oral appliance design that does not respect oropharyngeal reflexes that modulate vital functions, like respiration and deglutition, will lead to an OAT with a poor outcome - or even failure.

With a physiologic NMD approach TENS will optimize muscle fiber length, which is crucial to muscle function according to the Nobel prize winning work of Huxley.

This theory, which explains how the muscles create internal forces is that known as “Sliding filaments” developed by Huxley (1957) and based on the model of Hanson and Huxley (1955). It stipulates that, during muscle contraction, the fine Actin filaments slide between the thick myosin filaments.

AN OPTIMAL MUSCULAR FUNCTION, (Recruitment, activation, etc.), IS PRODUCED WITH an OPTIMAL PHYSIOLOGICAL LENGTH of MUSCLE FIBRES.
Optimal muscle fiber length becomes extremely important in OSA patients in order to open their airway. A fatigued stomatognathic muscle will collapse easily.

Literature shows clearly how OSA sufferers have their genioglossus muscle fatigued, with more Type II fiber content, which is reversed by CPAP therapy. These same genioglossus muscles in respiratory distress need healthy masseter recruitment in order to open the airway. In fact, masticatory muscles, including masseters, are considered respiratory muscles.

In PNMD (Physiologic Neuromuscular Dentistry) we respect muscle physiology with TENS and we measure muscle physiology with EMG/CMS unlike mechanistic methods of jaw manipulation (George Gauge, CR etc.).

A judicious choice of MAD (MicrO2), which Respects physiology of respiration in particular A.J. Miller’s the Lingual-Hypoglossal reflex combined with our physiologic approach takes DENTAL SLEEP MEDICINE to the next level.

“Our patients are REAL.”

They are neither corpses nor phantoms. It’s time for our profession to treat them as such, and get rid of all mechanistic approaches because they deserve the best treatment which is THE PHYSIOLOGIC APPROACH TO TREAT OSA.

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