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PRO/CON DEBATE

# Upper Airway Surgery Does Have a Major Role in the Treatment of Obstructive Sleep Apnea "The Tail End of the Dog"

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It is established that the causes leading to obstructive sleep apnea syndrome (OSAS) are usually multifactorial. These include a major anatomic component coupled with a yet to be identified central nervous system (CNS) imbalance.[1-2] Since the CNS derangements have eluded identification, as has the etiology of the syndrome, treatment has centered on enlarging the upper airway to allow for unobstructed sleep. The literature is replete with objective documentations using radiographs, CT and MRI scans, EMG, pressure monitors and fiberoptic imaging, all of which support anatomic involvement in nocturnal upper airway (UA) obstruction.[3-18] Furthermore, if OSAS is not an anatomic issue then upper airway and/or by-pass surgery (tracheotomy), or the use of continuous positive airway pressure (CPAP), would not be expected to ameliorate the problem. In fact, both surgery and CPAP improve the upper airway: surgery removes or repositions tissues and CPAP stents the airway open with a pressurized column of air. Therefore, in both cases treatments increase the size of the upper airway, decrease resistive breathing and improve or eliminate nocturnal obstructive events. Unfortunately, many of our medical colleagues would have you believe that CPAP is used all of the time by most patients and that surgery has little to offer over CPAP, dental splints and weight loss.

The goal for CPAP and surgery is to open the airway during sleep in OSAS. Both treatment approaches are primarily evaluated for objective outcomes by polysomnography (PSG). Since CPAP is graded as efficacious from these PSG's it is curious that surgery, which yields the same PSG outcomes for sleep and respiratory parameters, is usually not accepted by sleep medicine as equivalent in treatment efficacy. Is there some other mystic factor that CPAP provides over just opening the obstructed airway? Do you really not believe that surgery can make the airway bigger? If you do not then how do you explain improved PSG and excessive daytime sleepiness (EDS) results after surgery? Like it or not, the fact is that clinical outcomes for completed reconstructive surgery for OSAS are competitive with medical management. I refer to established surgery not investigational or fringe technology or techniques. For example we reported a study in Chest[19] fifteen years ago on 30 patients with severe OSAS (mean respiratory disturbance index [RDI] 72, lowest saturation [Low Sat] 61%) which directly compared nasal CPAP to maxillofacial surgery. All patients underwent baseline overnight attended PSG to document OSAS. Nasal CPAP titration studies were done for 2 consecutive nights. This cohort of 30 patients could not use the device long term and opted for surgery. A staged protocol was used for soft tissues and maxillofacial advancements. Six months following surgery overnight attended PSG was completed at the same center and then compared with night 2 of their previous CPAP results. Sleep and respiratory parameters compared were: RDI, Low Sat, $SaO_2$ falls <90%, Total Sleep Time, REM%, stage 3-4%, wake after sleep onset and change in BMI. Results showed *no statistical difference between CPAP and surgery for all variables*.

Our surgical literature is constantly attacked by a small group in sleep medicine as being of poor quality, with insufficient numbers of patients and of course seldom randomized or placebo controlled. But what exactly are you requesting and is it your role to guide our research? Most of those in sleep medicine have never experienced the responsibility of a surgical procedure. The cost in time and effort is substantially greater than in sleep medicine research, as is the patient responsibility. Seldom is there sufficient funding for large surgical studies which include operating room expenses, hospital care and lost work time for the patients. For example, we recently completed an investigation on radiofrequency (RF) complications (n=136) where the rate of complication was so low it would require a large study to address each specific factor of complications in a randomized controlled trial. To attain 90% power for any single one of these factors associated with a doubling of the rate of complications, a study would require 9500 patients. It is unlikely that a study of this magnitude could ever be conducted.[20] The data is clinically sound, but I am sure the study will be criticized by some since statistically we had insufficient power.

Furthermore, there may be an ethical issue for randomized placebo controlled trials (RCT) in some cases of sleep surgery. Withholding treatment or applying partial treatment protocols could

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delay the needed surgical treatment in patients and potentially expose them to serious health risks and quality of life decrements. This is especially true in light of the fact that we already know surgery can effectively treat sleep apnea by enlarging the airway. This is supported by PSG outcomes using the same metrics used to evaluate the efficacy of CPAP. To expect patients in these situations to go through this type of trial could be questionable.

Many of our medicine colleagues have condemned surgery for OSAS and have used out- dated-published surgical investigations to justify their beliefs. Consider the fact that patients are commonly told that "surgery is not successful and has many complications and at best improvement is less than 50 percent". This is absolutely a misconception and unfair to the surgeon and the patient. Take for example the comprehensive review paper by Sher et al[21] sponsored by the then American Sleep Disorders Association (ASDA). They meticulously reviewed and did a systematic metanalysis of the surgical literature on OSAS from 1966-1993 (29 years). The resultant surgical effectiveness from part of the report has been used by our sleep medicine physicians as proof that surgery is "< 50% effective". This effective percentage was cited only for uvulopalatopharyngoplasty (UPPP). Surgery for OSAS is "NOT" a UPPP. This is "NOT" the mid 80's; it is 2005. If other regions such as the tongue base are obstructed an isolated UPPP would not be expected to be successful by today's surgeons. The data in the remaining metanalysis which included tongue base obstruction procedures, alone or combined with UPPP, is seldom referenced. These combinations have very acceptable cure rates that improve or eliminate clinically important outcomes of excessive daytime sleepiness (EDS), performance, cardiovascular and accident risks, co-morbidities and survival.[22-30]

I would further remind the reader of Wright et al[31] (1997) who in a systematic review of the research evidence on CPAP concluded "The effectiveness of continuous positive airways pressure in improving health outcomes has been poorly evaluated". Only after this paper was published did serious sleep research appear to address this issue and this was *sixteen years* after CPAP was first applied in patient care for OSAS. As expected, over time sleep medicine principles have improved, as have those of surgery and there is no question that CPAP has efficacy, at least in the laboratory or when used nightly, all night.

In addition, there is an overwhelming misconception in sleep medicine that surgery is complicated, risky, and painful with poor clinical outcomes. The fact is our clinical outcomes for reconstructive surgery for OSAS are competitive with medical management. Current established surgical procedures offer reconstruction of the airway, usually in a phased manner (phase I and Phase II). In phase I which includes contemporary conservative treatment of the nose, palate and tongue, the cure rates are a mean of approximately 60%-70%.[32-48] When OSAS is stratified for severity (n=239) from mild to severe, outcomes for mild OSAS cases (RDI<20) are as high as 77% and in severe OSAS cases (RDI>60) as low as 42%.[49] Bi-maxillary advancement (Phase II) is often used if there is incomplete treatment in Phase I. The cure rate in Phase II when done in a systematic fashion and treatment is fully completed is ≥ 90%.[40,41,46,49-53] To suggest that surgery has no place in the treatment of OSAS rings hollow since you would have to have blinders on to believe that enlarging the airway surgically does not have the potential to treat OSAS.

Surgeons world wide usually encourage and request medical treatment (CPAP) first as the primary modality since it is conservative and reversible. However, not all university or private medical sleep centers have adopted a reciprocal combined approach. In the field of sleep, surgeons are generally relegated to the very end of most treatment protocols for patients with OSAS, which I consider and call "*the tail end of the dog*" When we finally see the patient they have usually failed medical management such as sleep hygiene, weight loss, nasal pressure devices, dental splints and pharmacologic remedies. The patient's major problem in medical management is compliance and those physicians who treat OSAS know these limitations but often do not admit to them. Hence patients are generally *not referred* by a sleep center but are self referred to a surgeon.

There may be merit in medicine's more conservative approaches as the risks, discomfort, healing process and lost work time sometimes associated with surgery are not a factor with medical intervention. This assumes that you truly believe and can show that conservative treatments will control the airway constantly, every night, all night, during sleep. If not, then perhaps medical treatments are ***not less risky*** for OSAS patients due to compliance issues. Consider Sher et al [54] who as far back as 1985 reported on patients who were carefully selected for UPPP and demonstrated by post operative polysomnography (PSG) that 72% of patients had marked improvements in their mean apnea index (AI). Eighty seven percent of these patients showed greater than 50% reduction in AI. I agree that in this day and age these outcomes would not equate to a cure. However, compare outcomes for just UPPP surgery alone in this study to a self reported 50% nightly success of CPAP usage, and even pessimistic views of surgery are not that bad. In the case of surgery, patients have their 50% reduction every night without compliance issues. Also consider the fact that in 1985 these were reasonable surgical outcomes. Yet again old data such as this is frequently cited by our medical colleagues. Surgeons do not consider these reasonable outcomes today and it is inappropriate to suggest so. Cure rates are stringently defined and include all PSG metrics and EDS (Table 1).

It is not acceptable to tolerate partial or non usage of any device prescribed for health protection when the patient may be exposed to serious risks by such actions. At least for those patients who have fully completed surgery there follows a full nightly benefit whereas CPAP is only *effective* if used all night, every night. Even though CPAP *efficacy* can be demonstrated in an investigation under ideal conditions, *effectiveness* continues to be poor and unproven due to compliance issues.

What is considered safe and acceptable CPAP compliance? Kribbs et al[55] investigated CPAP compliance in 35 subjects by evaluating *frequency and duration* of use. Regular use of CPAP was defined as 4 hrs per day on at least 5 days a week (≥ 70% of the time 5/7 of 7 days). Sixteen of 35 met regular use criteria (46%). Only 2 of 35 (5.7%) used it for 7 hrs per day ≥ 70% of

**Table 1**—Powell - Riley definition of surgical responder or cure

1-4 below or 5*

1. AHI ≤20 and or at least a reduction of 50% for any AHI below 20**
2. Oxygen saturation ≥ 90%
3. Normalization of Sleep Architecture
4. Resolution of EDS
5. Equivalent to CPAP titration night*

** If the AHI is 15 then it must drop to 7.5 which is a Reduction of 50%

days. But let us examine the math for *frequency and duration*. Compliance in this investigation was defined as a duration of 4 hrs per day at a frequency of at least 5 days which results in 20 hrs of CPAP usage a week. Yet average mean adult sleep time is approximately 7-8 hours nightly so at 7 hrs x 7 days = 49 hrs/week. Four hours per day for 5 of 7 days represents only 20 hrs a week of 49 hours weekly which is only 40 % of a patient's needed sleep. Many authors have since enshrined and adopted this standard of "adequate CPAP use". How can this be OK? What of sleep debt, EDS, intermittent hypoxemia, hypertension, cardiovascular sequela and a constellation of other serious problems that afflict our OSAS patients when the airway is unprotected?

Engleman et al[56] investigated CPAP use for mild OSAS using a randomized placebo-controlled crossover trial on 34 patients with mild severity (AHI 5-15) and sleepiness over a 4 week period on CPAP and then 4 weeks on placebo. They reported effective CPAP (on and at pressure) use averaged 2.8 ±2.1 hours per night by compliance meter. Though this study did show weak improvement in symptoms over the placebo group, it failed to demonstrate improved objective neurobehavioral metrics. It also highlights the poor compliance found in a group of symptomatic patients with mild SAHS. The dose response in this study might be expected to be minimal with these short hours but perhaps just enough to give the patient a false sense of security. The question is, if patients with mild OSAS do not use or benefit from CPAP why are they continually offered this treatment?

It is a stark reality that compliance issues are swept under the rug by sleep medicine and may in some cases be a detriment to the patients since there is evidence in your literature that clearly suggests unacceptable compliance for many patients at risk. For instance, the compliance to CPAP previously reported as acceptable varies as 4 hrs 70% of days[55] or by reported mean nightly hours of 2.8 hrs,[56] 5.5 hrs,[57] 4.3 hrs,[58] and 3.7hrs.[59] This should not be considered adequate treatment since the utilization is so short. These reports are from good researchers and under investigational conditions where you would expect the best compliance outcomes. An eye opener is seen in McArdle et al[59] who in their study reported average use of CPAP of 3.7 hrs or more per night and states "The rates of use quoted above refer to time the machine is running, not time at the preset therapeutic pressure". Do you really think your patients are using CPAP more than in these clinical trials where they have constant assistance? These standards may very well paint an inappropriately optimistic outlook for CPAP effectiveness in real life outside the laboratory. It should be a major research focus of all sleep medicine physicians to define exactly what amount of CPAP usage is associated with clinically important improvements in cardiovascular risk, mortality, performance, and symptoms. Without this information, it is difficult to argue any clear-cut benefits of CPAP over any other treatment.

Why are surgeons not more involved in treating patients with poor compliance? What happens to these patients? This disconnect is particularly bothersome, because even minor radiofrequency surgery alone may produce comparable, or even superior, outcomes to CPAP in this group of patients.[28] These surgical benefits include consistent improvement across symptomatic, physiological, anatomical, and objective performance outcomes, and they persist long term.[27]

Medical management *does not* offer a chance for cure. Instead it is only a treatment and then only if it is consistently used. Kribbs et al[60] has further reported that just one night off CPAP reverses all the gains derived from sleeping with the device. It would seem that ideally proper compliance requires an individual patient to use CPAP all their sleep hours, every night, 7 days a week. The fact that this does not generally happen should be of concern to the patient and treating physician. This is not to say that occasional usage of a CPAP device has not led to some measure of improvement but the same could be said for improved but still incomplete control in surgery. Marin et al[61] investigated long-term cardiovascular (CV) outcomes in OSAS with, and without CPAP treatment, and reported that with CPAP compliance for severe OSAS (AHI > 30) CV risks were reduced. The criterion for *compliance* was defined as CPAP usage of > 4 hours nightly. However, comorbidity was equal in untreated patients and in those treated with CPAP. Again what of EDS and the pathophysiologic derangements during sleep when CPAP is only used for half of the night? Furthermore, as the airway narrows resistance increases. Hence, it is highly possible that snoring (a sign of partial airway obstruction) along with frank obstructions and resistive breathing pathologically stretches the delicate soft tissues and may even further damage the tissues of the airway accelerating the rate of OSAS severity.

In 2002 Peker et al[29] reported a 7 year prospective study on cardiovascular disease (CVD) and OSA. In this study, the risk of developing CVD was increased in middle-aged patients with OSA. They additionally reported a much greater incidence in CVD when stratified to incomplete treatments using CPAP or UPPP. Compliance was assessed using objective CPAP time counters. Incomplete compliance was defined as < 50% of the individual's estimated sleep time. Surgical UPPP cases were evaluated for acceptable outcomes efficiency after a follow up sleep study where the oxygen desaturation (OD = oxygen saturation ≥ 4%) was below 30 events per night of sleep. Thirty six percent (36%) of the group using CPAP had adequate use (64% did not) in contrast to 50% of the UPPP group. Peker et al,[29] found that half of UPPP patients also experienced dramatic reductions in cardiovascular risk, at a slightly better rate than CPAP patients when one includes inadequate users.

More recently Weaver et al[30] reported on survival of veterans with sleep apnea who were prescribed CPAP or underwent surgery (UPPP). This was a retrospective cohort study (1998-2002) which included all veterans in Veteran Affairs Facilities (n= 20,826 patients). Survival was as follows: 1339 (7.1%) of 18,754 CPAP patients and 71 (3.4%) of 2,072 UPPP patients respectively were dead at the end of the study period ($p$<0.001). This data was adjusted for age, gender, and race, year of treatment initiated and for comorbidity. Although they were unable to adjust for OSA severity and CPAP usage they did adjust for comorbid conditions with the Charlson Comorbidity Index which includes 19 conditions. After these adjustments, CPAP patients had a higher probability of being dead at any time relative to the UPPP patients. Their conclusion in part was "Surgical therapy for sleep apnea provides better survival than provision of CPAP therapy to all comers". They further recommended that OSA patients who do not use or inadequately use CPAP seek surgical therapy.

It is intuitive that incompletely or untreated patients may be exposed to some degree of decrements in quality of life, health and survival. Unfortunately, the degree of control is yet to be defined, and so until we have that metric complete, treatment should be considered our goal.

I have no doubt that some will argue that there are methodology problems in all of these studies as can be said with the other CPAP literature and for that matter with all medical or surgical literature. Remember too that a UPPP alone was used in the above studies where surgery was compared to CPAP. We now have surgical procedures of varied invasiveness for all regions of the upper airway (nose, palate, base of tongue) with clinical outcomes that are far better than those from an isolated UPPP. For patients who use their CPAP fully and are satisfied with the treatment results, it is appropriate to downplay surgical therapy. However, for the more common situation where the patient struggles to use CPAP for even a fraction of his sleep time, surgical therapy should at least be considered.

Whose responsibility is it to follow through with patients to assure the best health outcome? Certainly you cannot expect the responsibility to be solely laid on the patient especially if they have not been educated about all of the possible options of treatment in an unbiased manner. It is a well known fact that medication compliance is commonly poor and to use this excuse to absolve your professional responsibility is weak. This fact should not be used as a justification to accept poor CPAP compliance, because surgical treatment alternatives presently exist for OSAS. The risks of not thoroughly advising and following patients with OSAS are great, and could potentially lead to accidents and/or death of the patient or others. This could be due to sleepy driving or directly from the co-morbidities of the syndrome. It is far more prudent to discharge the responsibility and educate patients to all of the possible treatment options including the pros and cons as well as the risk of no treatment.

The surgeon's role in OSAS should be a major one. It is time for the surgeon to bring a breath of fresh air to our patients that is not from a CPAP device. Medicine's lack of recognition and support for surgery as a treatment option is not justified. It leaves those patients who have failed or are not compliant to CPAP with weight loss and dental splints, both of which are second and third string options. We are tired of the "tail end of the dog" as patients and sleep medicine would benefit by our skills. This is a disorder of the upper airway and it falls squarely in the domain of Otolaryngologist-Head & Neck Surgeons, not just based on our anatomic and physiological expertise, but also based on successful surgical outcomes which sleep medicine refuses to acknowledge and continues to scorn. What is there to be gained by such a negative attitude towards surgery? Sadly I cannot fathom a good answer to this question. What we do as surgeons is logical, caring and in the best interest of the patient and field of sleep, and to think anything less is a serious error in judgment.

Sleep medicine should not leave the limitations of CPAP hidden behind their masks forever as sooner or later CPAP compliance will surface as a potentially serious issue for both the physician and the patient. It is time to recognize and support surgical management of OSAS and establish a relationship with a university surgical center or private surgeon who will provide proper and realistic treatments for OSAS. In this manner parity will allow for both medicine and surgery to have major, yet different roles in OSAS, thus improving the quality of life and survival of our patients.

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