Does the Method of Inferior Turbinate Surgery Affect the Development of Empty Nose Syndrome

Steven M. Houser*

Department of Otolaryngology, Case Western Reserve University, 2500 MetroHealth Dr. MetroHealth Medical Center, Cleveland OH 44109, USA

Abstract: Empty Nose Syndrome (ENS) is a poorly understood iatrogenic syndrome that may follow surgery involving turbinate reduction. In the author's experience, procedures which spare the mucosa are less likely to create ENS. Mucosal damage, rather than the sheer volume of tissue loss, appears to be of critical importance in ENS. Turbinate reduction is often appropriate therapy, but conservative mucosal sparing techniques should be employed.

Keywords: Empty nose, turbinate, suffocation.

BODY

When considering the development of Empty Nose Syndrome (ENS) we must first define the condition we are discussing. ENS is a nasal breathing disorder characterized by paradoxical obstruction (i.e., a widely patent nose yet complaints of obstruction) after turbinate surgery. Dryness is a common feature as well, though this may relate more directly to the mucosal loss. Chronic pain afflicts some patients with ENS; it appears to be a distinct and separate entity.

While ENS suggests missing tissue, I am convinced the sensation of the mucosal surface is the most important factor in ENS. Some wish to define ENS solely on anatomic deficit but this fails to explain two groups of patients: 1) those that undergo total turbinectomy and yet do not have breathing complaints, and 2) patients that have undergone surface damage to the inferior turbinate and develop ENS symptoms despite relatively normal appearing anatomy. Having treated many patients for this condition I will strive to explain the issue and its development: how it occurs, how it can be prevented, and touch upon therapy.

The poor understanding of ENS is a reflection of airflow sensation itself being poorly understood. We do know that the nasal vestibule is the most sensitive area to airflow with the inferior meatus/turbinate being the next most sensitive, and less sensation above in the middle meatus [1, 2]. Indeed the olfactory mucosa itself has even been credited with some ability to sense airflow [3]. The nasal mucosa contains multiple receptors for airflow, temperature and chemosensation. In order to interact with the air and particulate matter these receptors are understandably situated at the mucosal surface. Any process that affects the mucosal surface is then capable of diminishing mucosal sensation. ENS is a loss of functional sensation to airflow, not simply a loss of mechanical volume.

Sensory nerves are necessarily injured with any surgical intervention; incising skin or mucosa will sever small, unnamed sensory nerves that will usually recover in time. Unfortunately, recovery does not always take place though. A study analyzing the rate of numbness at the site of post-auricular incisions revealed a 26% incidence of persistent numbness at 8 months after surgery, though only 3% found the numbness to be of clinical significance [4].

A permanently numbed region at a post-auricular site should minimally impact quality of life, but tissue within a nose responsible for sensing airflow can be more problematic. While oral breathing allows for oxygen saturation, the nose remains the most comfortable and natural means of respiration. Unless obstructed, the human nose is utilized 24 hours per day, every day. When nasal breathing is deranged the affected will be constantly reminded of their state without reprieve. Psychological stress logically follows.

The more mucosal surface that is affected will produce more sensory deficit, and more sensory nerve recovery must then take place with greater potential for incomplete repair. No direct studies have documented the incidence of ENS, but a study of subjects having undergone total inferior turbinectomy found a 22.2% rate of atrophy – very possibly representing ENS [5]. A total turbinectomy will target mucosa, submucosa, and bone; any sensory nerves included in the mucosa will be understandably damaged in the process, but fortunately they may recover as peripheral sensory nerves are capable of regrowth.

*Address correspondence to this author at the Department of Otolaryngology, Case Western Reserve University, 2500 MetroHealth Dr. MetroHealth Medical Center, Cleveland OH 44109, USA; Tel: 2167785791; Fax: 2167787868; E-mail: shouser@metrohealth.org

E-ISSN: 2308-7978/15

Procedures damaging less mucosa, and hence require less regrowth of sensory fibers, should be less likely to cause ENS. Excision of a piece, but not the whole, turbinate, will also target mucosa, submucosa, and bone. Partial turbinate excision should be less problematic than total excision, but one must also consider the location of tissue excised as being important as well. Inferior meatal tissue appears to be more important for sensation than middle meatal, therefore more ENS might result from trimming the inferior turbinate over the middle turbinate. The author has found ENS from partial inferior turbinate excision more common from head than tail excision; this suggests the location of resection relative to the airflow and nerve location is also important.

Procedures that target submucosal vasculature of the inferior turbinate but damage surface tissue to reach their goal would be expected to be problematic according to the ideas explained herein. Such techniques include laser or surface electrocautery. Having read many operative notes of ENS patients I am convinced these techniques are frequently at fault. The patients may have relatively normal looking anatomy, but deranged breathing results from sensory deficit. The mucosa appears to heal, but the nerves embedded within might never manage to recover. Mucosal sparing techniques are a safer option.

One would expect that procedures that target bone, and spare mucosa, to be potentially safe in regards to ENS. Caution must be taken though, when honestly assessing the mucosal impact of any turbinate procedure. An inferior turbinate submucosal bone excision should cause minimal mucosal impact, but in reality redundant mucosa is often trimmed back, and vigorous bleeding often requires cautery to the site that damages mucosa and nerves. The author has treated multiple patients that underwent this procedure; their descriptions of prominent crusting postoperatively suggested mucosal damage. Keep in mind that submucosal bone resection appears mucosal sparing in artwork, but might not be in actual practice. Of note, turbinate outfracture alone has not been observed to cause ENS to date. The mucosal damage during outfracture is so minimal that nerve damage should be exceedingly rare.

Procedures that target the submucosal tissue with minimal impact to the overlying mucosa should be less capable of producing ENS, and the author's experience bears this out. Submucosal radiofrequency treatment, including Coblation™, and microdebrider reduction are very rare causes of ENS. Any technique can be used to excess though, even if mucosal sparing, e.g., submucosal monopolarcautery or Coblation™ may inadvertently damage considerable mucosal surface, and submucosal microdebrider use can also readily excise mucosa if care is not taken. One ENS patient had undergone Coblation™ reduction per operative note, yet described six weeks of crusting requiring many debridement sessions; the author suspects considerable mucosal damage necessitated the cleaning sessions hence the patient underwent a Coblation™ session much different than typical. Other ENS patients produced operative notes describing submucosal reduction per microdebrider yet their prominent tissue loss suggested a more aggressive use of the microdebrider had taken place.

I have been treating patients with ENS for over 15 years. I have refined my thought process on the subject to go from being purely structural, to engage in an explanation relying upon functional sensation to airflow. The author has found that performing a simple "cotton test" by placing moistened cotton in the deficient area to shift airflow toward virgin (undamaged) tissue can bring significant relief, and subjective improvement with a cotton test is now considered essential to diagnose ENS. Success with cotton can then be simulated on a permanent basis with submucosal implants at the location(s) suggested by the cotton. As of last tallies in 2015, I have performed 94 implant procedures on 64 different patients (ranging from 1 to 5 procedures per

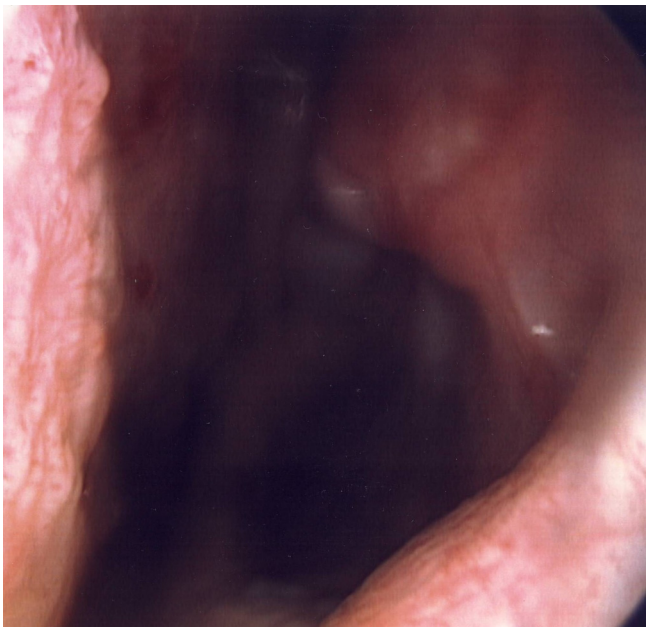

Figure 1: Left nasal cavity with inferior turbinate mostly resected.

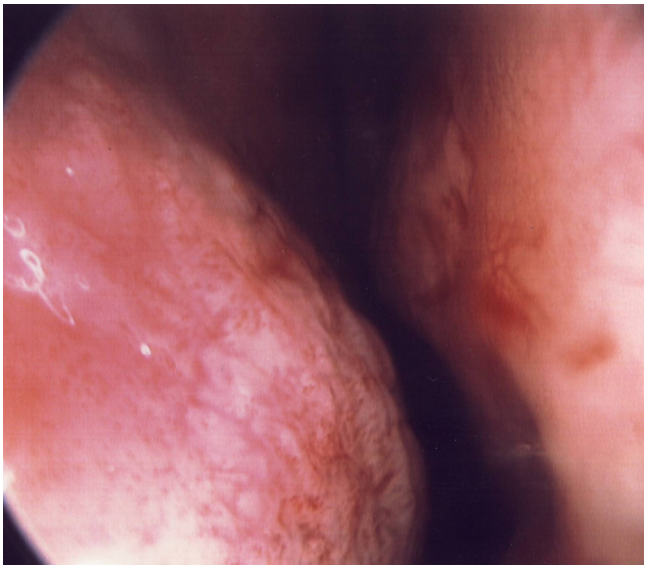

Figure 2: Left septal submucosal implant.

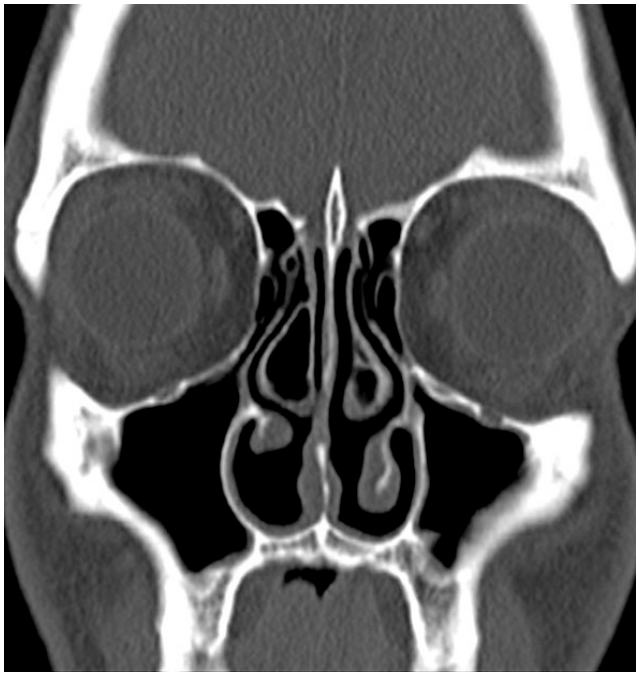

Figure 3: Right sided ENS.

patient); Acellular dermis (e.g., Alloderm™) is typically used, though autologous tissue could be utilized. Submucosal implantation took place into the septum 43 times (Figures **1** and **2**), the lateral wall 52 times (Figures **3** and **4**), and directly to the inferior turbinate's 21 times. I have also performed 36 submucosal injections of liquefied acellular dermis (i.e., Cymetra™) to the inferior turbinates in an office setting (no blindness has occurred, though care is taken). Nearly all implanted/injected patients report an improved sensation of breathing and less dryness, though these issues are hard to quantify.

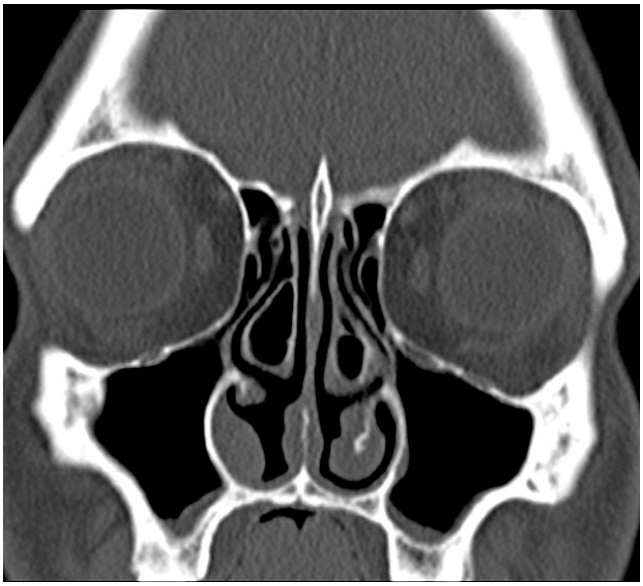

Figure 4: After right lateral wall implantation of acellular dermis.

CONCLUSION

ENS, fortunately, is extremely rare. The degree of mucosal damage relates more directly to the development of ENS than the volume of excised tissue. Most patients will regrow nerves at a surgical site and not go on to develop ENS. Turbinate surgery should only be considered if medical management (e.g., allergy medications in the face of allergic rhinitis) has failed. Using the least invasive process to effect airway improvement in the face of turbinate enlargement is then wise. Turbinate outfracture appears safe, as do conservative submucosal reduction procedures. Turbinate excision should be restricted to disease processes that prohibit lesser approaches (e.g., cerebrospinal fluid leak repair may necessitate middle turbinate sacrifice). Turbinate reduction approached via mucosal damaging procedures (i.e., surface cautery and laser reduction) should be abandoned in favor of safer techniques.

REFERENCES

[1] Clarke RW, Jones AS, Charters P, Sherman L. The role of mucosal receptors in the nasal sensation of airflow. Clin Otolaryngol Allied Sci 1992; 17(5): 383-387. http://dx.doi.org/10.1111/j.1365-2273.1992.tb01679.x

[2] Wrobel BB, Bien AG, Holbrook EH, *et al*. Decreased nasal mucosal sensitivity in older subjects. Am J Rhinol 2006; 20(3): 364-368. http://dx.doi.org/10.2500/ajr.2006.20.2862

[3] Grosmaitre X, Santarelli LC, Tan J, *et al*. Dual functions of mammalian olfactory sensory neurons as odor detectors and mechanical sensors. Nature Neuroscience 2007; 10(3): 348-54. http://dx.doi.org/10.1038/nn1856

[4] Frampton SJ, Pringle M. Cutaneous sensory deficity following post-auricular incision. The Journal of Laryngology & Otology 2011; 125: 1014-1019. http://dx.doi.org/10.1017/S002221511100199X

[5] Passàli D, Lauriello M, Anselmi M, Bellussi L. Treatment of the inferior turbi-nate: long-term results in 382 patients randomly assigned to therapy. Ann Otol Rhinol Laryngol 1999; 108(6): 569-575. http://dx.doi.org/10.1177/000348949910800608

Received on 19-10-2015 Accepted on 30-11-2015 Published on 16-12-2015

DOI: http://dx.doi.org/10.12970/2308-7978.2015.03.02.3