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Vital Topic

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Olfactory Loss: Options for Measurement, Diagnosing, and Therapy

Although there may not always be a treatment option, it is important to determine the etiology of olfactory loss in order to provide appropriate counseling

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Olfactory loss is a significant clinical problem. More than two million Americans suffer olfactory impairment, and this accounts for at least 200,000 physician visits each year. There are safety concerns, such as no warning of fire, leaking gas, or spoiled food, but perhaps more important is the severely compromised quality of life that results.

The olfactory neuroepithelium is located within the olfactory cleft or nasal vault, along the cribriform plate, and the superior aspects of the nasal septum and superior turbinate. In contrast to the respiratory epithelium, the olfactory mucosa contains four cell types. The ciliated olfactory receptor is a bipolar neuron that provides nerve endings to the olfactory neuroepithelium. As such, these are directly exposed to the external environment. Microvillar cells are flask-shaped cells that have a neuron-like appearance, but their function at this point is not known. Supporting or sustentacular cells provide a supportive function to the olfactory neurons, as well as aiding in the enzymatic degradation of odorant molecules. Basal cells are progenitor cells, and are located close

to the lamina propria. In contrast to respiratory cilia that are motile, olfactory cilia are immotile. However, these cilia greatly increase the surface area for receptors and this facilitates odorant reception.

The precise nature of olfactory coding mechanisms is not clear. It is clear that humans are capable of distinguishing over 10,000 different odorants. It appears to be related both to selective tuning of olfactory receptors, as well as different adsorption characteristics of odorant molecules across the olfactory mucosa.

In describing olfactory loss, several terms should be defined. Normosmia is a normal sense of smell. Hyposmia is a diminished sense of smell, whereas anosmia is a complete loss of smell. Dysosmia is a disordered smell perception that is usually unpleasant and can be stimulated either by an environmental stimulus, which would be considered parosmia, or may occur spontaneously without an environmental stimulus, which is referred to as phantosmia.

In the diagnosis of olfactory loss, a thorough history is very important. This should focus on the well recognized

causes of olfactory loss that will be discussed below, such as a history of trauma, rhinosinusitis, or toxic exposure. In addition, it is important to objectively assess the degree of olfactory impairment. In the past this was very difficult, but now there are a number of methods available that can be utilized in a clinical setting. Perhaps the simplest and most straightforward approach is the University of Pennsylvania Smell Identification Test (UPSIT). This is a forced choice test that utilizes 40 microencapsulated odorants each on a scratch-and-sniff pad. The patient is asked to identify each odor from four possible alternatives. The number correct is the patient's score. A normal subject should be able to correctly identify 35–40 odorants, while only being able to identify 20–34 is consistent with hyposmia. Below 20 is consistent with anosmia. Based upon chance, a patient unable to smell should be able to correctly identify 25% or approximately 10 odorants. For those scores close to zero, one needs to be suspicious for the possibility of malingering.

It is important to keep in mind that many such patients with olfactory loss will present with complaints of taste impairment. This may be their presenting primary complaint. What they really mean is they have noticed a loss of flavor appreciation. Taste consists of four basic qualities, including salty, sour, sweet, and bitter. Our appreciation of flavor is comprised of taste, as well as olfactory input along with temperature and texture of the food. Therefore, when patients lose their ability to smell, everything tastes flat. While the majority of our patients have presented with both taste and smell complaints, only a very small number truly have a measurable gustatory deficit.

Looking through our first 339 patients presenting with olfactory loss, we see that the most common etiology is head injury, a prior viral infection, and nasal or sinus disease. These three categories account for 54% of our patients. 17% of patients remain idiopathic as far as the etiology for their smell loss. This simply demonstrates that there is still a great

deal we don't know about this problem. 5% described a history of toxic exposure that was believed to cause their loss. This is usually a sudden excessive exposure rather than chronic daily exposure over many years, and therefore, such an etiology should be readily discernable from the patient's history. Eight patients (2%) presented with a congenital olfactory loss. In only one patient did we feel the olfactory loss truly related to age. It is important to recognize that olfaction diminishes with age at about 69 or 70, and this can be correlated with appropriate age matched controls on olfactory test measures. The remainder of the patients had a variety of miscellaneous causes.

Considering those patients who present with a traumatic loss, the pathogenesis is believed to relate to a coup contracoup injury. Therefore, it is most often seen after a frontal or occipital blow. Presumably there is a shearing effect that severs the olfactory fila as they pass through the cribriform plate. Less often, it may relate simply to a contusion or hematoma. These patients will typically present with a loss of smell that began immediately following an episode of trauma, although the event need not have been associated with loss of consciousness. Since the olfactory receptors have the capacity to regenerate, recovery is theoretically possible but rarely seen. Our experience and other studies have demonstrated a 30% recovery rate, which seems to occur within the first six months. It is very likely that these patients have experienced a contusion and not a true severing of the olfactory nerve. After this point, further recovery is unlikely.

Regarding a viral-induced olfactory loss, again the true pathogenesis is unknown. However, the virus seems to directly invade the olfactory epithelium, as degenerative changes have been demonstrated. The olfactory loss usually follows an upper respiratory infection that patients will often describe as having been particularly severe. These patients more often complain of dysosmia, and that can be a useful diagnostic clue. Although no effective therapy has been demonstrated, we have found over a period of time that most of these patients do demonstrate some recovery. While little improvement is seen in the first year, over 3–5 years we have found 90% of patients will demonstrate some recovery. In 67% of cases, this was significant. Therefore, this is useful prognostic information that can be provided to patients.

Lastly, I would like to consider those patients who have lost their smell secondary to nasal and sinus disease. There are essentially four situations where nasal and sinus pathology may result in an olfactory loss. The first is anatomic obstruction, and of course the most common would be a deviated septum. However, we have never seen a patient who had a measurable loss of smell secondary to a septal deformity. Therefore, further exploration should be made in patients presenting with an olfactory loss rather than diagnosing just a deviated septum. Secondly, neoplastic disease can certainly cause an olfactory loss. However, such patients will typically develop other symptoms before complaining of a loss of smell. Inflammatory disease will be the most common cause of olfactory loss and will be discussed in more detail below.

The last category is surgical trauma. Interestingly, the incidence of olfactory loss after traditional nasal and sinus surgery is very low, although objective measurements of olfaction have not typically been utilized. With the development of endoscopic sinus surgery, more recent reports are beginning to appear looking at the incidence of olfactory loss. Data is scarce but thus far suggest an incidence of anywhere between 1–3%.

We looked at a series of 53 patients with nasal and sinus pathology causing olfactory loss. The average age was 52, the mean UPSIT score 18.9 (consistent with anosmia), and the mean duration of symptoms 6 years. Regarding symptoms, only 32% complained of nasal obstruction. 59% had a history of chronic sinusitis. 59% had a history of allergies. 23% had a history of asthma. Although fluctuation and olfactory loss is consistent with an inflammatory etiology, only 45% of these patients described fluctuation and therefore, such a history cannot be relied upon to make the diagnosis of an obstructive or conductive olfactory loss.

In these patients, physical examination becomes very important in order to determine whether the loss may relate to a prior viral infection or active nasal and sinus pathology. In many of these cases, pathology will be present within the ostiomeatal complex, or perhaps the nasal vault, and this may not be apparent simply with anterior rhinoscopy. Many of these patients will not have symptoms suggestive of sinusitis. A nasal endoscopic exam, therefore, becomes very valuable. We found in this series of 53 patients that if anterior rhinoscopy alone had been performed,

the diagnosis would have been missed in 48% of the cases. These are patients, remember, that presented primarily because of an olfactory loss. CT scans are very useful, but are not necessarily indicated in the presence of a negative endoscopic exam. In most cases, we will obtain an MRI scan of the head only when we have not been able to identify a clear etiology by either history or physical examination.

To further verify the presence of an obstructive or inflammatory etiology causing olfactory loss, a trial of steroids may be worthwhile. While topical nasal steroids rarely reverse such a loss, a brief trial of systemic steroids can be very effective. We found that 84% of patients in whom systemic steroids were utilized loss. Again, such an improvement is usually temporary, but verifies that the loss is reversible and, therefore, obstructive. A viral-induced loss or traumatic loss will not respond to such steroid administration.

In conclusion, it is very important to consider olfactory loss as a serious problem and do a thorough examination to determine its etiology. While in many cases, effective therapy may not be available, appropriate counseling is extremely valuable to these patients. In addition, in those cases of loss secondary to nasal and sinus disease, certainly effective treatment is available.