Auris Nasus Larynx xxx (2015) xxx–xxx

Contents lists available at ScienceDirect

Auris Nasus Larynx

journal homepage: www.elsevier.com/locate/anl

# Posttraumatic olfactory dysfunction

Daniel H. Coelho, Richard M. Costanzo *

*Department of Otolaryngology – Head and Neck Surgery and Department of Physiology and Biophysics, Virginia Commonwealth University School of Medicine, Richmond, VA 23298-0146, USA*

ARTICLE INFO

*Article history:*
Received 16 July 2015
Accepted 26 August 2015
Available online xxx

*Keywords:*
Trauma
Olfactory
Smell
Dysfunction
Anosmia
Hyposmia
Implant

ABSTRACT

Impairment of smell may occur following injury to any portion of the olfactory tract, from nasal cavity to brain. A thorough understanding of the anatomy and pathophysiology combined with comprehensively obtained history, physical exam, olfactory testing, and neuroimaging may help to identify the mechanism of dysfunction and suggest possible treatments. Although most olfactory deficits are neuronal mediated and therefore currently unable to be corrected, promising technology may provide novel treatment options for those most affected. Until that day, patient counseling with compensatory strategies and reassurance is essential for the maintenance of safety and QoL in this unique and challenging patient population.

© 2015 Elsevier Ireland Ltd. All rights reserved.

## 1. Introduction

We use our sense of smell every day – to prepare and enjoy foods, to appreciate the fragrances of flowers or wine, to detect and avoid noxious or hazardous odors, to maintain personal hygiene, and in many other complex and subtle ways that effect social and intimate interactions with others. Loss of smell can greatly impair these activities, thereby interfering with both quality of life (QoL) as well as activities of daily living (ADLs). Unfortunately, olfactory dysfunction is particularly common following head trauma, often with substantial negative consequence [1]. Not all injury to smell is complete, and patients can have alterations in detection (i.e. hyposmia, anosmia) or in identification (i.e. parosmia, phantosmia). A comprehensive understanding of the anatomy, pathophysiology, epidemiology, clinical presentation, evaluation, treatment options, and prognosis of posttraumatic olfactory impairment is therefore critical to the successful management of this unique patient population.

## 2. Anatomy

The olfactory system is the primary and most sensitive system for detection and identification of odors. The peripheral elements of this system are found in approximately 22 $cm^2$ of pseudostratified columnar respiratory epithelium in the superior recesses of the nasal cavity, between the septum and the middle turbinates and below the cribriform plate. This area, termed the olfactory cleft, contains roughly 6 million bipolar olfactory receptor cells [1]. These highly specialized cells possess many unique characteristics. Firstly, they continuously regenerate from horizontal and globose basal cells located within the olfactory neuroepithelium [2] throughout one's lifespan and even following trauma. This regenerative capacity allows the basal cells to mature into neurons which can then grow back into the olfactory bulb and even re-establish functional connections [2–6]. Secondly, these bipolar receptor cells act both as a sensory receptor and as the first-order neuron that projects directly into the brain – also unique amongst sensory systems [1]. Thirdly, each receptor cell expresses a single odorant receptor gene derived from a single allele. Roughly 1000 distinct transmembrane G-protein olfactory receptor genes have been identified which represent 3–5% of the entire mammalian genome – a discovery for which Drs. Linda Buck and Richard Axel were awarded the 2004 Nobel Prize in Physiology [7]. Although each cell only expresses one gene, the cells which express a particular gene are distributed throughout the olfactory epithelium in a random fashion [7–9]. Central integration of spatial coding is what allows for unique odor perception.

In addition to the bipolar receptor cells, a variety of other vital cells and structures comprise the olfactory neuroepithelium. Sustentacular (supporting) cells as well as the cells of Bowman's

* Corresponding author.
*E-mail addresses:* daniel.coelho@vcuhealth.org (D.H. Coelho), richard.costanzo@vcuhealth.org (R.M. Costanzo).

http://dx.doi.org/10.1016/j.anl.2015.08.006
0385-8146/© 2015 Elsevier Ireland Ltd. All rights reserved.

Please cite this article in press as: Coelho DH, Costanzo RM. Posttraumatic olfactory dysfunction. Auris Nasus Larynx (2015), http://dx.doi.org/10.1016/j.anl.2015.08.006

glands produce the mucous that is critical to the maintenance of the epithelium and transduction of odorants from the gaseous phase to the aqueous phase of the epithelium. Disruption or alteration of the mucous quantity or viscosity may affect olfactory function.

After passing through the basal lamina of the epithelium axons from the olfactory receptors types begin to converge with axons of other receptor types. These nerve groupings course through the cribriform plate of the ethmoid bone and comprise the fila of cranial nerve I (CN I). Once through the cribriform these bundles go on to form the first and outermost concentric layer of the olfactory bulb. Within the next deeper layer of the bulb, axons converge and synapse onto dendrites of second-order neurons (mitral and tufted cells) forming oval-like structures known as glomeruli.

Upon leaving the olfactory bulb via the olfactory tract, the second-order olfactory neurons synapse in the areas of the primary olfactory cortex. This area is composed of the olfactory tubercle, the pyriform cortex, the amygdala, the periamygdaloid complex, and the entorhinal complex. A secondary olfactory cortex is located in the orbito-frontal region of the brain. Many connections between the primary and secondary cortexes travel via the mediodorsal nucleus of the thalamus; however some connections exist directly between the olfactory bulb and cortical areas – yet another unique characteristic among the sensory systems. All of these areas perform critical roles in the central processing of odor perception including learning, memory, integration of visual and gustatory inputs, and the assignation of emotional significance [10].

In addition to the olfactory system, the trigeminal system plays an important role in the detection of chemical stimuli, though not in odor discrimination. Fibers of CN V innervate the entire nasal cavity and can sense tactile, pain, temperature, and noxious stimuli – oftentimes resulting in reflexive responses that lead to mucous production, mucosal congestion, sneezing, etc. that can indirectly effect olfaction [1]. Because of its deeper, protected course and bilateral innervation, CN V is more resistant to injury and may even be spared in cases of severe injury. Therefore a patient's subjective inability to detect strong noxious stimuli (i.e. ammonia) may provide a clue to malingering.

## 3. Pathophysiology

Olfaction requires a patent nasal airway, intact nasal mucosa with appropriate mucous coating, and intact neural pathways from the nasal cavity to the higher cortical processing centers. As such, any posttraumatic olfactory dysfunction comes from the disruption of any or all of the following components: 1) sinonasal tract, 2) shearing of the olfactory nerves at the cribriform plate, 3) focal trauma to the olfactory bulb, diffuse injury to primary or secondary olfactory cortex, or injury to the connections between central olfactory structures (Fig. 1).

### 3.1. Sinonasal tract disruptions

Nasal bone or midface fractures can distort the normal airflow, thereby creating a conductive loss of smell by preventing the odorants from reaching the olfactory neuroepithelium. The more extensive the injury (i.e. LeFort fractures), the higher the incidence of smell loss [11]. Soft tissue injuries within the nasal cavity itself leading to blood within the cavity, mucosal or septal hematoma, edema, and scar formation (from the initial injury or from resuscitative airway support) may all distort anatomy and therefore function [12]. Furthermore, injury may lead to blocked sinonasal outflow resulting in rhinosinusitis and inflammatory changes that can further limit airflow or mucous character. As disruptions of the sinonasal tract are the most amenable to medical or surgical treatment, such potential etiologies should not be overlooked, even in cases of complex injury.

### 3.2. Olfactory nerve injury

The small foramina where the olfactory nerve fila traverse the cribriform plate are sites particularly prone to injury. The mechanism by which this happens is usually from either direct bony disruption of the ethmoid roof or cribriform plate, or from a rapid shift in position of the brain relative to the skull base. This latter mechanism is commonly seen following abrupt deceleration experienced in motor vehicle collisions or even ground level falls to the occiput. The olfactory bulb and brain are mobile within the intracranial compartment, insulated by the cerebrospinal fluid (CSF) within the dura. However, the olfactory receptor axons are fixed within the cribriform foramina. This type of injury can result in complete shearing or significant stretching of the olfactory fibers that subsequently result in axonal degeneration [13]. These injuries, commonly referred to as *coup-contracoup* injuries, are frequently severe and bilateral.

### 3.3. Central lesions

Injury to any central component or connection of olfaction can lead to dysfunction. In the most dramatic form, this may include contusion, but frequently is due to edema, hemorrhage, or hematoma. In 1985 Levin first suggested that such central injury could result in olfactory dysfunction, but was unlikely to lead to complete anosmia [14]. Further work by Yousem elaborated on this notion, proposing that the extensive and bilateral connections to the olfactory cortex were likely to protect against anosmia [15]. Nonetheless, the olfactory bulbs sit precariously inferior to the frontal lobe and superior to the fixed cribriform plate and particularly vulnerable to compressive forces or secondary ischemia. Isolated injury to the olfactory bulb can occur in the absence of involvement of other brain structures, suggesting a potential increased susceptibility from these mechanisms [13,16].

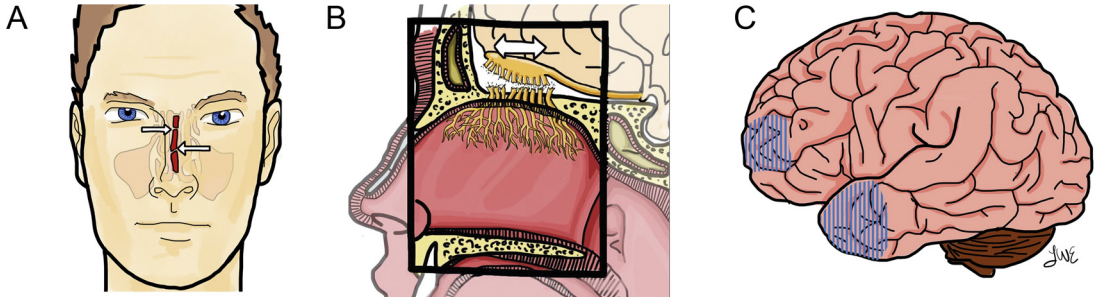


**Fig. 1.** Mechanisms of post traumatic anosmia. (A) Sinonasal tract disruption (conductive loss), (B) Olfactory nerve injury – transection, (C) Central lesions – cortical contusions.
Adapted from Costanzo and Zasler [27].

Please cite this article in press as: Coelho DH, Costanzo RM. Posttraumatic olfactory dysfunction. Auris Nasus Larynx (2015), http://dx.doi.org/10.1016/j.anl.2015.08.006

Download English Version:

https://daneshyari.com/en/article/8755127

Download Persian Version:

https://daneshyari.com/article/8755127

Daneshyari.com