

OLFACTORY HALLUCINATION

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OLFACTORY HALLUCINATION (PHANTOSMIA)

Primary Disciplinary Field(s): Neurology, Otorhinolaryngology (ENT), Psychiatry

1. Core Definition

Olfactory hallucination, medically termed **phantosmia**, is a sensory disorder characterized by the perception of an odor in the absence of an external physical source. This phenomenon is distinct from true hallucinations in other sensory modalities in that the perceived odors are overwhelmingly described as unpleasant, noxious, or disgusting, often referred to clinically as **cacosmia**. The source content notes that these phantom scents are typically undesirable, frequently mimicking substances such as toxic gas, burning rubber, or decomposing organic matter, highlighting the distressing nature of the condition for the affected individual.

The core mechanism involves a malfunction within the olfactory system, where neural activity that typically corresponds to external stimulation occurs spontaneously or is triggered inappropriately. Unlike simple olfactory illusions, where a real scent is misinterpreted (parosmia), phantosmia involves a totally endogenous experience. The severity and persistence of these phantom smells vary widely among patients, ranging from brief, momentary episodes to constant, debilitating perceptions that drastically interfere with daily life, eating habits, and overall psychological well-being.

While smell perception originates in the nasal epithelium, the interpretation and experience of odor are mediated by complex pathways involving the olfactory bulb and cortical areas such as the piriform cortex, amygdala, and entorhinal cortex. Therefore, phantosmia is generally considered a central neurological phenomenon rather than a peripheral issue of the nose itself. Understanding this neurological locus is crucial for accurate diagnosis and management, as the hallucination often signals underlying pathology in the central nervous system, warranting immediate medical investigation.

2. Etiology and Associated Conditions

The causes of **phantosmia** are highly diverse, spanning neurological, psychiatric, and peripheral olfactory system disturbances. Neurological etiologies are among the most common and serious, particularly conditions affecting the temporal lobe. For instance, phantosmia frequently manifests as an **olfactory aura** preceding a focal epileptic seizure, especially in temporal lobe epilepsy. In these cases, the false smell sensation is a direct consequence of aberrant electrical discharge within the olfactory cortex, serving as a warning sign of impending seizure activity.

Other significant neurological causes include space-occupying lesions such as brain tumors, particularly those impinging upon the olfactory tract or uncus. Traumatic Brain Injury (TBI) can also

lead to chronic or intermittent phantosmia due to damage to the delicate neural structures involved in olfactory processing. Furthermore, neurodegenerative diseases like Parkinson's disease and Alzheimer's disease, which inherently involve the deterioration of central nervous system pathways, have been associated with altered olfactory function, including the development of phantom smells, often early in the disease progression.

Peripheral causes, though less common as the primary source of true phantosmia, can involve chronic nasal infections, severe sinusitis, or damage resulting from upper respiratory tract infections. In these scenarios, persistent inflammation or damage to the olfactory neuroepithelium may lead to disorganized or spontaneous firing of olfactory receptor neurons. Additionally, certain systemic conditions, including severe diabetes, kidney failure, and exposure to specific neurotoxic medications, can disrupt the neurochemical balance required for normal olfactory function, thereby inducing hallucinatory smells.

Psychiatric disorders, while less frequent primary drivers, can also present with olfactory hallucinations. Conditions such as schizophrenia or severe depression may occasionally include phantosmia as part of a broader spectrum of psychotic symptoms. However, it is clinically critical to rule out organic neurological causes first, as medically induced phantosmia is far more prevalent than purely psychiatric manifestations. The precise identification of the underlying trigger dictates the appropriate therapeutic strategy, emphasizing the need for comprehensive diagnostic screening.

3. Differential Diagnosis: Phantosmia, Parosmia, and Agnosia

Differentiating **phantosmia** from other forms of olfactory dysfunction is a critical step in clinical diagnosis. Phantosmia is defined by the perception of an odor that has no external stimulus. In contrast, **parosmia** (or dysosmia) involves the distortion or misinterpretation of a *real* external odor source. A parosmic patient might smell coffee and perceive it as rotting garbage, meaning the olfactory stimulus is present but is processed incorrectly into a highly distorted and usually unpleasant smell. This distinction is important because parosmia often results from the healing or miswiring of the olfactory nerve fibers, frequently following viral infections, whereas phantosmia suggests central neurological pathology or spontaneous neuronal firing.

Another related condition is **olfactory agnosia**, which is the inability to correctly name or classify an odor despite the physical ability to detect and distinguish it. In this scenario, the patient can register the presence of a smell--unlike in anosmia (total loss of smell)--but the cognitive recognition and associative processing required for identification are impaired, typically due to damage in higher cortical areas. Neither agnosia nor anosmia involves the experience of a phantom smell, underscoring that phantosmia is unique in its hallucinatory character.

The clinical history helps distinguish these conditions. Patients reporting phantosmia often describe

smells that suddenly appear and disappear, sometimes triggered by environmental changes or specific body movements, and are almost universally unpleasant. Patients with parosmia, however, report that all, or many, normal odors have become distorted and offensive. Because peripheral olfactory damage often leads to recovery phases involving parosmia, the timeline and preceding events (e.g., recent severe colds or COVID-19 infection) are important clues for the clinician determining the exact nature of the olfactory disturbance.

4. Clinical Presentation and Characteristics

The clinical presentation of **olfactory hallucinations** exhibits several characteristic features that help clinicians classify the disorder. The perceived odors are highly specific and often monochromatic; that is, the patient typically reports experiencing the same phantom smell repeatedly, rather than a variety of smells. Common descriptors include burning toast, cigarette smoke, chemical fumes, sulfur, feces, or rancid meat. This consistency suggests a fixed neural pathway or focus generating the spontaneous signal.

The duration of the episodes is highly variable. In cases linked to focal seizures, the phantosmia is typically brief (lasting seconds to a few minutes) and serves as an aura, rapidly preceding motor symptoms or loss of consciousness. In non-epileptic, idiopathic phantosmia or phantosmia linked to chronic sinusitis, the episodes may last longer, sometimes hours, or even persist constantly for weeks or months, creating a severe burden on the patient's psychological state and ability to function socially.

Furthermore, the intensity of the phantom smell can range from a barely noticeable background presence to an overwhelming, dominating sensation. Importantly, phantosmia is almost always bilateral (perceived in both nostrils), though in cases involving localized damage (such as a tumor affecting one olfactory bulb), the sensation may be reported as unilateral. Unilateral phantosmia is a particular red flag for a serious localized lesion and necessitates urgent neuroimaging.

Unlike visual or auditory hallucinations in psychosis, patients experiencing phantosmia usually maintain insight; they recognize that the smell is not real and that others cannot perceive it. This preservation of reality testing differentiates neurological phantosmia from psychotic hallucinations, although the lack of control over the distressing sensation still leads to significant anxiety and distress. The presence of headache, visual disturbances, or memory lapses accompanying the hallucination strongly suggests a central neurological cause, such as migraine aura or seizure activity.

5. Neurological Basis and Pathophysiology

The underlying pathophysiology of **olfactory hallucination** centers on the aberrant activation of the primary olfactory processing centers in the brain. The olfactory system is unique in that primary

sensory neurons project directly to the primary olfactory cortex (the piriform cortex) without first synapsing in the thalamus, a feature that contributes to the strong emotional and memory associations tied to smells.

In many cases of phantosmia, the spontaneous firing is localized to the piriform cortex or the uncus, a region of the temporal lobe. Abnormal excitability in these areas can generate the perception of an odor without input from the olfactory bulb. When phantosmia is the prodrome of epilepsy, it represents a specific type of simple partial seizure originating in these deep temporal lobe structures. The unpleasant quality of the phantom odor is often attributed to the close anatomical relationship between the piriform cortex and the amygdala, a brain region central to processing fear and negative emotions.

Recent research suggests that changes in neuromodulatory systems, particularly those involving dopamine and serotonin, may also contribute to the genesis of phantom smells, particularly in cases linked to psychiatric conditions or chronic drug use. However, the most robust mechanism remains the spontaneous generation of action potentials in the peripheral receptors (peripheral phantosmia) or, more commonly, the central misfiring within the primary or secondary olfactory cortices (central phantosmia).

Peripheral phantosmia, related to damage in the olfactory epithelium, involves a kind of "short circuit" where damaged neurons fire randomly. However, central phantosmia often involves complex neural network imbalances. For instance, in severe migraine, cortical spreading depression, which is a wave of transient neuronal depolarization, may transiently affect the olfactory cortex, leading to a brief hallucinatory odor as part of the migraine aura. This illustrates the diverse ways in which neural excitability can manifest as a phantom smell perception.

6. Diagnostic Procedures

Diagnosing **olfactory hallucination** requires a systematic approach aimed at ruling out serious underlying organic causes. The initial step involves a detailed medical history focusing on seizure activity, head trauma, exposure to toxins, and the precise nature and timing of the phantom smell. A comprehensive ENT examination is necessary to rule out peripheral causes such as chronic sinusitis, nasal polyps, or localized inflammation that might be contributing to peripheral irritation.

The cornerstone of diagnostic testing for central causes is **neuroimaging**. Magnetic Resonance Imaging (MRI) of the brain, often with dedicated sequences focusing on the temporal lobes, is essential to identify structural abnormalities such as tumors, abscesses, or signs of stroke that may be compressing or irritating the olfactory pathways. If epilepsy is suspected, an Electroencephalogram (EEG) is required to detect abnormal electrical activity, especially if the phantosmia serves as a seizure aura.

Specialized olfactory testing, known as **olfactometry**, can also be employed, though its primary use is to quantify smell loss (anosmia or hyposmia) or distortion (parosmia). In phantosmia, olfactometry helps confirm that the patient's overall ability to perceive and identify real odors may be intact, isolating the symptom as a pure hallucination rather than a pervasive distortion. Further blood work may be necessary to screen for metabolic or systemic causes, such as vitamin deficiencies or endocrine disorders that could affect nervous system function.

7. Treatment and Management

The treatment strategy for **phantosmia** is entirely dependent on identifying and addressing the underlying etiology. If the hallucination is symptomatic of a serious condition like a brain tumor or ongoing seizure activity, the primary focus is treating that underlying pathology, whether through surgical resection of the lesion or pharmacological management of epilepsy.

When phantosmia is related to epilepsy, **anti-epileptic drugs (AEDs)**, such as carbamazepine or valproate, are the first line of treatment. These medications work by stabilizing neuronal membranes and reducing abnormal excitability in the olfactory cortex, thereby suppressing the phantom odor generation. Successful seizure control almost invariably eliminates the associated phantosmia.

For cases where a specific neurological cause cannot be definitively identified (idiopathic phantosmia) or when the condition is secondary to chronic peripheral irritation, treatments are aimed at symptom control. Localized treatments, such as saline nasal rinses or specific topical anesthetic sprays (e.g., lidocaine applied to the olfactory cleft), have been reported in some instances to temporarily disrupt the sensory input and provide relief. In severe, chronic cases refractory to other treatments, neuromodulation techniques or low-dose psychotropic medications (like certain antidepressants or atypical antipsychotics) may be considered to modulate the central nervous system activity responsible for the perception.

8. Psychological Impact and Quality of Life

The persistence of **olfactory hallucinations**, particularly the perception of profoundly disgusting odors, exerts a substantial negative impact on a patient's quality of life. Unlike other sensory hallucinations, which can often be ignored or rationalized, the sense of smell is fundamentally linked to survival mechanisms (detecting spoiled food or danger) and comfort, making a constant, unpleasant phantom smell extremely intrusive and stressful.

Patients often develop significant psychological distress, including anxiety, depression, and social isolation. Eating becomes challenging because the phantom smell interferes with the taste of food, leading to appetite suppression, weight loss, and nutritional concerns. Social situations are frequently avoided, as the individual may fear that they are emitting the terrible smell they perceive,

leading to withdrawal and a feeling of alienation.

Counseling and cognitive behavioral therapy (CBT) are often integrated into the management plan, even when the underlying cause is neurological. CBT can help patients develop coping strategies, minimize the emotional reaction to the phantom odor, and manage the associated anxiety and depressive symptoms. Validating the patient's experience is crucial, as the highly subjective nature of the symptom can sometimes lead to misdiagnosis or dismissal of the severity of their distress.

Further Reading

[Phantosmia \(Wikipedia\)](#)

[Olfactory Dysfunction: Phantosmia, Parosmia, and Anosmia \(Academic Article\)](#)

[Phantosmia: Overview and Treatment \(Cleveland Clinic\)](#)

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