

The Smell of Burnt Toast: A Case Report

Walter B. Shelley, MD, PhD, Toledo, Ohio

E. Dorinda Shelley, MD, Toledo, Ohio

A 71-year-old woman awoke one morning to find that she perceived all aromas, odors, and fragrances as smelling like burnt toast. Over the next three years, numerous studies and therapeutic trials failed to elicit the cause of her dysosmia or to provide relief. Finally, the demonstration of small infarcts as seen on a brain MRI suggested that an infarct near the olfactory pathway was responsible for the sudden onset and the 11 year persistence of her unique paromia.

A 71-year-old woman awoke one morning smelling smoke, but no smoke was found. As the day progressed, she realized that all aromas, odors, and fragrances smelled like burnt toast. This perception has continued, seriously interfering with her enjoyment of foods and leading gradually to a 25-pound weight loss. Her sense of taste has remained intact.

Her general health has been good, with her only medications being occasional naproxen and chlordiazepoxide. A neurologic examination, general physical examination, and extensive examinations of her nose, throat, teeth, and ears showed normal results. Blood studies were also normal, including a serologic test for syphilis, thyroid scan, and serum immunoglobulin E. Her electrocardiogram results were normal, as were sinus roentgenograms, and a computed tomography scan. An electroencephalogram, with and without challenge with pungent odors (coffee grounds, onion, and cantaloupe), showed a normal pattern.

A magnetic resonance imaging (MRI) scan of the brain showed no specific abnormality in the region of the olfactory tracts, but revealed focal areas (2 to 3 mm in size) of high signal density in the white matter of the cerebral hemispheres, which were interpreted as small focal infarcts. A repeat MRI scan one year later demonstrated similar findings. Using Doppler studies, the carotid artery circulation was found to be normal.



FIGURE 1. "Everything smells like burnt toast."

After failed attempts at treatment by four other physicians during the first 3 months of her condition, we conducted short therapeutic trials over the following 3 years, which were also unsuccessful. The medications she had taken included acyclovir; biotin; doxepin; guaifenesin; L-lysine; intramuscular methotrexate; metronidazole; pentoxifylline; phenytoin; pimocid; propranolol; intramuscular triamcinolone acetonide; vitamins A, B₁₂, and E; and zinc sulfate. Local anesthesia of the olfactory epithelium provided no relief, and discontinuing the use of naproxen and chlordiazepoxide produced no change. A follow-up investigation by phone 11 years after the onset of her condition revealed no change in her dysosmia, and her general health had remained excellent.

At least 1000 different receptor proteins are expressed by the 10 million neurons in human olfactory epithelium. These receptor neurons are unique in their ability to regenerate every month and to lie bare, protected only by mucus.¹ They are the starting point for the conversion of odorants into sensation, either pleasurable or protective. Volatile water-soluble odorants initiate impulses that go to a synaptic jungle

From the Medical College of Ohio Ruppert Health Center, Toledo, Ohio.

REPRINT REQUESTS to 3120 Glendale Avenue, Toledo, Ohio 43614-5809 (Dr. W.B. Shelley).

(glomeruli) in the olfactory bulb and then pass down the olfactory tract to the orbitofrontal cortex where they are processed. The uncus (anterior part of the temporal lobe) is considered a major site in odor processing; hence, the condition, “uncinate fits” is characterized by spells of abnormally perceived odors. The odor information path travels through the limbic system of the brain, where it can evoke memories and emotions, and ultimately reaches the hypothalamus.

A score of conditions can induce the inability to smell (anosmia) or the misidentification of odorants (dysosmia or parosmia).^{2,4} In our patient, we were able to rule out the common causes of dysosmia: head injury, Alzheimer’s disease, Parkinsonism, depression, hallucinations, drug reaction, and infectious disease.⁵ The abrupt onset, persistence for 11 years, and MRI findings of small infarcts favored the diagnosis of dysosmia secondary to a tiny infarct in a critical site of the processing pathway in the temporal lobe.⁶ The use of 14 drugs and three nasal sprays failed to alter the unpleasant odor of burnt toast. The patient elected not to undergo nasal mucosa denervation, since she had already been denied the primitive pleasure of fragrances. To denervate the olfactory epithelium would rob her of the protective function of her chemical sensory system. Although she has a nasal alarm system that does not work properly, at least it still works.

REFERENCES

1. Shipley MT, Smith DV: The olfactory system. In, *Neuroscience in Medicine* (Conn PM, ed), p 521. Philadelphia, J.B. Lippincott, 1995.
2. Schiffman SS: Taste and smell losses in normal aging and disease. *JAMA* 278: 1357-1352, 1997.
3. Zilstoff K: Parosmia. *J Laryngol Otol* 80: 1102-1104, 1966.
4. Zatorre RJ, Jones-Gotman M, Evans AC, *et al*: Functional localization and lateralization of human olfactory cortex. *Nature* 360:339-341, 1992.
5. Doty RL, Yousem DM, Pham LT, *et al*: Olfactory dysfunction in patients with head trauma. *Arch Neurol* 54: 1131-1140, 1997.
6. Rousseaux M, Muller P, Gahide I, *et al*: Disorders of smell, taste and food intake in a patient with a dorsomedial thalamic infarct. *Stroke* 27: 2328-2330, 1996.