The letter was rejected for publication because there appeared to be no plausible causative factor. Because I could not find anything in the literature to support the claim, the editorial decision served to confirm my belief that a causal relationship did not exist. Unfortunately, the patient remained unconvinced and brought the matter to litigation.

During the process of discovery, I obtained a copy of a letter written to the patient's husband from a Robert H. Jenkin, M.D., a taste-and-smell specialist from Washington, D.C. In the letter, Jenkin related the patient's symptoms to the anesthetic she had received. Specifically, he blamed halogenated hydrocarbons as being capable of inducing taste and smell distortions, although the patient had received a halogenated ether. In addition, Jenkin referred to the textbook Otalaryngology as a source to support his view. Of course, Jenkin is the author of the chapter "Olfaction in Human Disease" in the book. The only references to anesthesia or surgery as causative factors are found in table 1 on page 3 and table 4 on page 18. These tables were compiled from the results of a survey carried out by the author. Clearly, almost 70% of cases of taste and smell dysfunction are related to postinfluenza (24.8%), idiopathic (19%), head trauma (15.4%), and allergic rhinitis (10.3%). 3.5% of patients experienced symptoms after systemic surgery. It would seem that the evidence implicating anesthesia as a cause for the patient's complaints is circumstantial at best.

The lawsuit was dismissed with prejudice. These events have made me realize how important it is for anesthesiologists to be aware and wary of what our colleagues in other disciplines are telling our patients about anesthesia.

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Reference


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In Reply.—The patient to whom Adelman refers in his letter was evaluated and treated for disorders of taste and smell that she stated followed cyscoscopy performed under general anesthesia. She reported all fruits, dairy products, meats, and coffee tasted and smelled spoiled or rotten, which, she said, caused her to decrease her food intake, initiating a 15 pound weight loss and a significant change in her normal lifestyle. She is one of 359 among the 3,512 patients (or 1.8%) whom I have evaluated over the past 19 yr at the Taste and Smell Clinic, Washington, D.C., who complained of some persistent dysfunction of taste and/or smell that they stated followed a surgical procedure associated with a general anesthesia.

Table 1 summarizes the taste and smell changes observed in these 59 patients. Each patient came to the Clinic with a variety of taste and smell dysfunction complaints that followed a surgical procedure and general anesthesia. Each patient noted on awakening from anesthesia and regained composure, including eating and drinking, that a significant alteration in their taste and/or smell function had occurred that encompassed either loss of taste or distorted function. No patient had an associated cornea, head injury, or underlying disease process (e.g., endocrine, oncologic, nutritional, or infectious). None was taking any drug associated with permanent sensory dysfunction, and no other cause of the dysfunction could be ascertained despite a complete history, physical examination of head and neck, clinical laboratory studies, and radiographic studies, including computed tomography or magnetic resonance studies of skull and brain, as previously described in detail. The anesthetic agents administered vary greatly but included, in some combination, commonly used agents and procedures, preanesthetic medication (a sympathetic or para sympathetic blocking agent, a short-acting barbiturate, phenothiazine, benzodiazepine), intravenous induction (ketamine, propofol, benzodiazepine), inhalation anesthesia (nitrous oxide with oxygen, halothane, enflurane, isoflurane), and succinylcholine. Taste and smell acuity were measured by standard forced-choice, three-stimuli, staircase drop and sniff tests for taste and smell, respectively, with determination of detection and recognition thresholds and magnitude estimation for four tastants (NaCl, sucrose, HCl, urea) for taste and five odors (pyridine, nitrobenzene, triphenol, as well as maleol, methanol) for smell, as previously described. Each patient reporting loss of taste or smell acuity had both subjective loss of function and impaired detection and/or recognition thresholds and/or impaired magnitude estimation for one or more tastants or odors. Distortion of function, dysgeusia and dysosmia, for taste and smell, respectively, was estimated quantitatively by questionnaire such that allagusia or allomia (unpleasant taste or smells generated by the presence of food or drink) or phantagonia or phantosmia (unpleasant tastes or smells occurring in the absence of external stimuli) could be established. Changes in flavor perception were assessed quantitatively by questionnaire. All loss of acuity and distortion of function affected either the entire taste system and/or olfaction in both nares. The patients were arbitrarily divided into two groups (1) those in whom changes followed surgical procedures not involving the cranial area and (2) surgical procedures in which the cranial area was involved but excluding any procedure directly involving the nose (e.g., submucous resection, nasal polypectomy) or craniotomy and subsequent procedures associated with involvement of olfactory nerves, bulbs, or temporal lobes.

Patient age in the group undergoing cranial surgical procedures was significantly less (P < 0.01, Student's t test) than in the group undergoing noncranial procedures (cranial group 19-68 yr, noncranial group 49-82 yr). Dysfunction was persistent after onset in each patient, and mean length of time from dysfunction onset to first visit to the Clinic (noncranial group 1 month–24 yr, cranial group 1 month–42 yr) was similar in each patient.
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1 month–42 yr) was similar in each patient group (P > 0.10, Student’s t test). A similar incidence of impairment was present in each patient group with respect to loss of taste, smell, and flavor acuity (P > 0.10, t2). There was uniform loss of smell and flavor acuity after tansillectomy in which the olfactory area was not surgically involved. In addition, a large number of patients, after various abdominal surgical procedures, also had taste, smell, and flavor loss, including smell and flavor loss in all patients who underwent genitourinary procedures (including the patient who is the subject of this letter). Dysosmia, although more frequent in the arbitrarily defined noncranial procedure group, was not significantly different between group (P > 0.10, t2). The group who underwent ear surgery is unique in that each patient experienced dysgeusia after the procedure, which may be more related to surgical manipulation of the chorda tympani nerve in the middle ear than to effects of anesthesia.

During the early course of these studies, the cause of taste and smell dysfunctions reported by individuals was difficult to define in a specific manner. However, as the continuum of patients appeared, with the repetition of similar history in the face of a diversity of surgical procedures not involving either gustatory or olfactory systems, it became apparent that the common thread relating these taste and smell changes was that each patient had undergone general anesthesia. Such changes have not been observed after spinal or local anesthesia, except for taste and local sensory changes after anesthesia and extraction of third molar teeth4 and smell and flavor loss after anesthesia and surgical procedures directly involving upper nasal airways and paranasal sinuses,4 which may be related to direct damage to olfactory epithelium and olfactory receptor cells. It is clear that drugs used to induce anesthesia in these patients were multiple and varied, making it difficult to focus on one step or drug in the complex series of procedures used to induce or maintain anesthesia.

Comparison of patients who developed taste and smell dysfunction after general anesthesia with those who experienced these symptoms because of the variety of common causes previously described2 indicates significant differences in demographics and in characteristics of the sensory defects found. Among patients after general anesthesia, there are significantly more men than women (68% vs 32%, respectively) contrasted with this distribution in the general group (45% vs 55%) (P < 0.01, χ2, table 1). Although incidence of taste loss, dysgeusia, and dysosmia was similar in both groups, incidence of smell loss (dysosmia) was significantly greater after anesthesia (90%) than in the general group (78%; P < 0.01, χ2) as was incidence of flavor loss (93%) compared to the general group (82%; P < 0.01, χ2). In addition, patients who complained of these sensory changes after anesthesia reported their onset sooner after the initiating event than did general group patients, although the time between onset of their dysfunction and presentation to the Clinic did not differ from that of general group patients. These differences suggest that patients who develop taste and smell dysfunction after general anesthesia exhibit demographic and sensory dysfunction changes that distinguish them from the general group of patients who experience taste and smell dysfunction from the more common causes previously described.2

Although incidence of these dysfunctions comprises about 2% of patients who presented to a clinic devoted exclusively to evaluation and treatment of taste and smell problems, incidence of these changes among the population of patients who undergo general anesthesia is small. These changes, when they occur, however infrequent, interfere significantly with the quality of the patient’s life and are unexpected and usually unexplained phenomena that follow an otherwise successful surgical procedure. Lack of knowledge or understanding of the significance of these changes on the part of the physician can exacerbate the problem and enhance anxiety and animosity on the part of the patient.

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