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Caries Risk Assessment
Understanding—and Combating—Factors That Contribute to Risk
Pamela Maragliano-Muniz, DMD
PLUS:
The Dentist's Role in Diagnosing and Treating Halitosis
Stuart J. Froum, DDS; and
Kristina Rodriguez Solovey, DDS, MS
Kois Center Case: Transitional Resin Bonding
Sandra Hulac, DDS
Special Report:
Adhesive Dentistry
David S. Alleman, DDS; and
Simone Deliperi, DDS
ALL-BOND UNIVERSAL
p.656

CE 2

Diagnosis and Treatment of Halitosis

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Abstract

Diagnostic and treatment services for patient complaints of "bad breath" are currently being offered in many dental offices. There are no accepted standards of care for these services, and clinical protocols for the diagnosis and treatment of breath malodor vary widely. This article attempts to review the biological and psychological bases of patients' complaints of bad breath and to describe a clinical protocol for the evaluation and treatment of such complaints. This protocol resulted in a 99% success rate in eliminating objectively measured breath malodor. However, 24% of patients continued to believe that at least some of their bad breath persisted after treatment. The merits of various diagnostic procedures are discussed in light of the psychogenic component of the symptomatology of halitosis sufferers.

Learning Objectives

After reading this article the reader should be able to:

- identify if a patient is experiencing breath malodor, without the use of any specialized equipment.
- explain how to determine whether a patient's breath malodor is originating from the mouth or another part of the airway.
- describe the location, origin, and cause of most breath malodor.
- discuss how to distinguish between real and "imagined" halitosis.
- describe the difference between VSC generation in the mouth and VSC emission in the breath.
- describe the relationship between periodontal disease and bad breath.

Most adults and many children suffer from halitosis (bad breath). This affliction may occur occasionally, regularly, or chronically at specific times of the day or month.[1,2] For the purposes of this article, the terms *bad breath, halitosis,* and *breath malodor* all mean an unpleasant breath odor that is objectionable to others. Public awareness and concern for this phenomenon are evidenced by the support of an estimated $850 million mouthwash industry in the United States, despite reports that commercially available products have no significant effect on breath malodor.[3,4] Recent public opinion polls (taken between 1994 and 1995) have revealed that 55- to 75-million Americans consider bad breath a principal concern in social encounters.[5,6]

In general, physicians and dentists are poorly informed about the causes and treatments for halitosis. This article briefly reviews the current understanding of the etiologies of halitosis and introduces a clinical protocol for diagnosis and treatment. The clinical strategies and techniques for diagnosis and treatment described were drawn from the research methods and results of Tonzetich,[1,7-9] Preti,[10,11] Rosenberg,[4,12-14] Yaegaki,[15,16] Bosy[17] et al, and our experience in treating more than 3,000 patients presenting with chief complaints of "bad breath."

Studies on the etiologies of breath malodor agree that hydrogen sulfide (H_2S), methylmercaptan (CH_3SH), and dimethyl sulfide (CH_3SCH_3), collectively referred to as volatile sulfur compounds (VSC), are the principal odorants in bad breath.[1] Volatile sulfur compounds originate from the anaerobic bacterial degradation of sulfur-containing amino acids within the oral cavity. Therefore, it is reasonable for dentists to assume the responsibility for diagnosing and managing breath malodor. When systemic or other nonoral etiologies are

Table 1—Peak VSC Recordings and Organoleptic Assessments (ORG) of Patient in Figure 1

	VSC (ppb)	ORG
Mouth Air	890	4.0
Nose Air	105	0.5
Lung Air	125	1.0

Normal breath VSC is 50 to 150 ppb. ORG (scale of 0 to 5) were performed independently by 2 experienced judges and averaged: 0=no malodor; 1=barely detectable malodor; 2=mild malodor; 3=moderately offensive malodor; 4=strongly offensive malodor; 5=overwhelmingly offensive malodor.

Table 2—Peak VSC Recordings and Organoleptic Assessments (ORG) of Patient in Figure 2

VCS were measured with a modified sulfide monitor and recorded with a pen-writer (see text and Figure 3)

	VSC (ppb)	ORG
Mouth Air	<1000	5.0
Nose Air	130	0.0
Lung Air	150	0.5

Normal breath VSC are 50 to 150 ppb. ORG (scale of 0 to 5) were performed independently by 2 experienced judges and averaged: 0=no malodor; 1=barely detectable malodor; 2=mild malodor; 3=moderately offensive malodor; 4=strongly offensive malodor; 5=overwhelmingly offensive malodor.

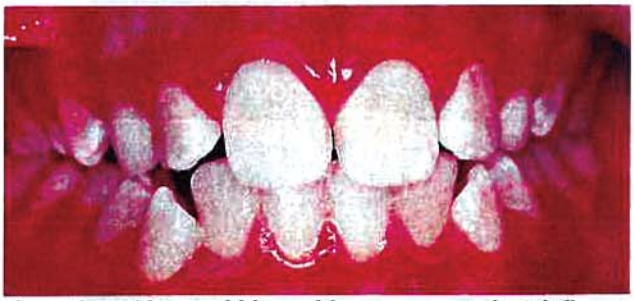


Figure 1—A 12-year-old boy with no apparent dental disease who has elevated VSC and breath malodor.

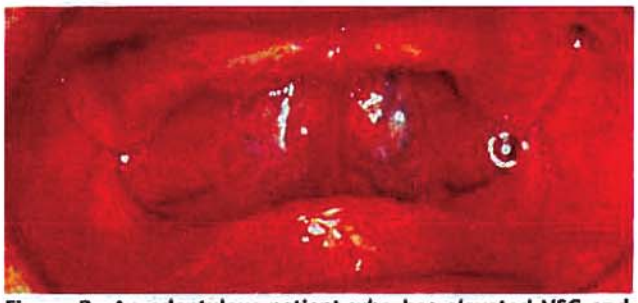


Figure 2—An edentulous patient who has elevated VSC and breath malodor but shows no evidence of periodontal disease.

suspected, dentists must be prepared to make appropriate medical referrals. There are many common nonoral diseases cited in the literature for which halitosis can be a symptom.[18,19] However, halitosis typically occurs late in the pathogeneses of these diseases when other more obvious or more urgent symptoms are present.[18,20,21] Rapid onset and progressively intensifying breath malodor are suggestive of an infective process, possibly secondary to carcinomas or other localized pathologies in the airway.[18,20] However, in our experience for patients with a *chief* complaint of long-standing, chronic halitosis, there is, almost without exception, either an oral origin or no halitosis at all.

Oral Origins of Breath Malodor

Tonzetich[1] showed that incubated whole saliva produces a putrid odor and that hydrogen sulfide, methylmercaptan, and, to a lesser extent, dimethyl sulfide, are the principal responsible malodorants. When fresh saliva was filtered, the incubated supernate alone produced very little VSC. Saliva filtrate was shown to contain dead epithelial cells, live and dead bacteria, white blood cells, other blood elements, and food debris, all of which are rich in proteins, peptides, and free amino acids. Through a series of systematic studies, Tonzetich[1] and coworkers established that the malodorous volatiles produced by incubated whole saliva evolve from anaerobic bacterial activity on sulfur-containing amino acids derived from degraded proteins present in salivary filtrate. They also observed that the incubated saliva of patients with periodontal disease produced a more rapidly developing, intense malodor and evolution of VSC. Volatile sulfur compounds that evolved from substrates high in the amino acid cystine were high in hydrogen sulfide, while VSC that evolved from high methionine substrates were high in methylmercaptan.

Direct measurements of breath volatiles using gas chromatography-flame photometric detection confirmed that the distribution of VSC generated from incubated saliva is similar to that which can be identified in malodorous human-mouth air. Kostelc et al[22] and others[15,23] have shown that patients with periodontal disease produced more breath malodor and mouth-air VSC than patients with healthy periodontiums. However, it has been reported that periodontal disease is not a prerequisite for the production of high levels of orally generated VSC or oral malodor as measured organoleptically.[17,24] In our practice/clinic, periodontal disease is not a routine finding. We routinely see a

Table 3—Results of Patient Treatment for Breath Malodor

Patients Examined Pretreatmt	Patients With Breath Malodor Pretreatmt	Patients Treated	Patients Examined Posttreatmt	Patients With Breath Malodor Posttreatmt
2837	2243	2228	923	5

Pretreatment and posttreatment examinations were performed after 6 to 18 hours of food and oral hygiene deprivation. Patients were defined as having breath malodor if they had an organoleptic breath malodor score of >2, a peak VSC >180 ppb, a VSGT <30 minutes, or third-party corroboration.

variety of dentally healthy children, young adults with no clinical evidence of periodontal diseases, adults with inactive and/or well-controlled periodontitis, and totally edentulous patients who have high levels of oral malodor. Some of them have extremely intense breath malodor and extremely high VSC in their mouth air (Figures 1 and 2) (Tables 1 and 2). Yaegaki[15] and others[1,25,26] identified the dorsoposterior surface of the tongue as the principal location for intraoral generation of VSC and breath malodor. It is well established that anaerobic bacteria capable of producing VSC are routinely recoverable from this location.[27-31] The benefits of tongue cleaning for controlling dental plaque and breath malodor have been extolled in the literature for decades.[25,26,32,33]

The incidence, intensity, and duration of episodes of breath malodor can vary considerably in an individual, depending on a number of conditions.[1,8,25] Using a modified industrial sulfide monitor, called the Halimeter™,[a] (Figures 3 and 4), Rosenberg[14] demonstrated a diurnal cycle for oral emission of VSC. Tonzetich et al[34] showed that oral VSC production in women varies in accordance with the menstrual cycle. The concentrations of VSC in saliva[35] and, perhaps, the surface soft tissue of the mouth[36] exceed those of the breath. Because VSC are permeable in soft tissue and toxic to gingival connective tissue,[36] a major role for VSC has been postulated for the pathogenesis of periodontitis (Figure 5).[23,24] Within this context, the VSC associated with oral malodor may constitute only a relatively small proportion of the intraoral VSC that volatilize when the VSC in saliva and soft tissues exceed saturation. If, in fact, the saliva and oral soft tissues store significant quantities of VSC by absorbing them before they volatilize, then it is likely that the concentration of VSC in mouth air at any moment may not always indicate the instantaneous rate of VSC generation within the mouth.

To measure the rate of VSC generation within the mouth, we developed the HaliTest™,[b] a modified, anaerobic bacterial-growth medium enriched with cystine and methionine and a trace of lead acetate, which undergoes a color change when exposed to VSC. We observed the time it takes for a standardized sample of tongue coating, collected on a cotton swab and submerged in the medium, to produce a barely observable color change. We interpreted this time to be a measure of the rate of VSC generation by the tongue coating (VSGT). The less time it took for the color change to occur, the faster the rate of VSC generation. We then compared rates of VSC generation from tongue-coating samples, mea-

[a] Interscan Corp, Chatsworth, CA 91313

[b] ProFresh, Inc, Philadelphia, PA 19104

Figure 3—The modified sulfide monitor (Halimeter™) (below) and the pen-writer (above) used for measuring VSC.

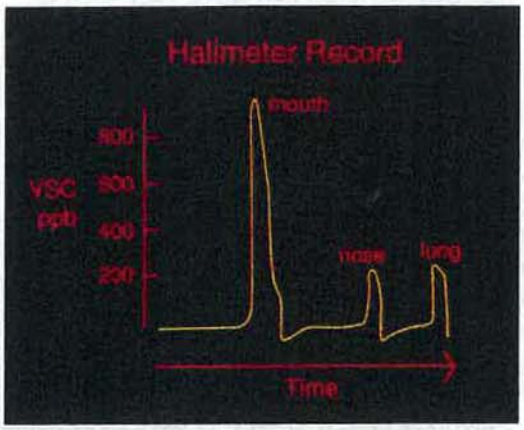


Figure 4—Pen-writer record of VSC in mouth, nose, and lung air demonstrating an oral origin for elevated VSC. Normal breath VSC is 50 to 180 ppb.

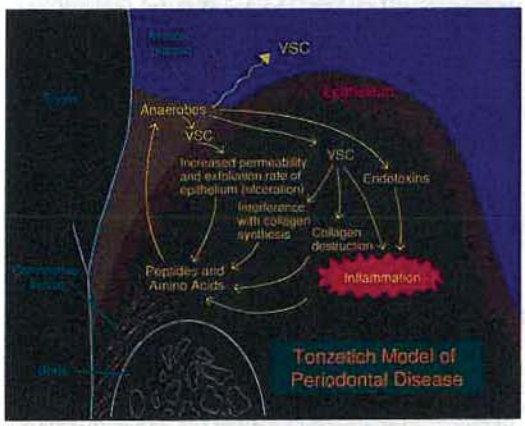


Figure 5—Schematic model for the pathogenesis of periodontitis proposed by Tonzetich and coworkers.[23,24] Aerobic supragingival plaque promotes oxygen depletion and anaerobic bacterial growth in the gingival sulcus. Volatile sulfur compounds produced from available proteins by anaerobes cause increased epithelial exfoliation and permeability, which permit access for bacterial endotoxins and VSC to underlying connective tissue. Ensuing inflammation and destruction of collagen by VSC result in elevated peptides and amino acids in the gingival fluid, thus providing more substrate for anaerobic bacterial VSC production.

sured by this simple technique, with mouth air VSC (VSC_m) and organoleptic malodor assessments (ORG_m) in patients preconditioned by at least 6 hours of abstention from oral activities (eating, oral hygiene, etc). We also recorded the results of our attempts to verbally confirm episodes of bad breath perceived by friends or relatives of 215 patients.

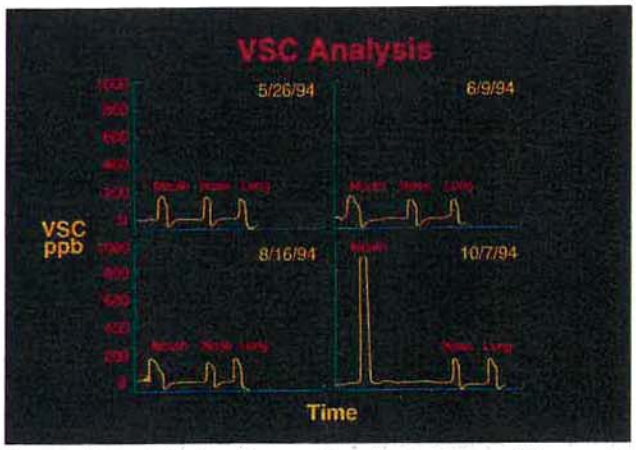
Patients with organoleptic scores that we judged significant (ORG_m >2) or elevated mouth-air VSC (VSC_m >180) had high rates of VSC generation from tongue coating (VSGT <30 min). Many had high rates of VSC generation from tongue coating (low VSGTs) but normal VSC_m and low ORG_m at the time of testing. However, for each of those patients, friends or relatives confirmed the patient's bad breath at least occasionally. This means that even patients who had fasted and exhibited no evidence of breath malodor at a particular evaluation may have at least occasional episodes of perceptible bad breath. We could not confirm episodes of bad breath with friends or relatives for any patient with a VSGT of 50 or above (low rates of VSC generation).

We have interpreted these findings to suggest that at a particular testing visit, the HaliTest™, compared with organoleptic or instrumental assessments of mouth air, is a better predictor of which patients have an actual, day-to-day breath malodor problem. Within this conceptualization, a high rate of intraoral VSC generation results in an episode of breath malodor only when the quantity of VSC in the mouth exceeds the capacity of the saliva and soft tissue to absorb them into solution.

We also found that many patients who had high VSC_m had low ORG_m and vice versa. This observation was previously attributed to the general unreliability of organoleptic scoring.[13] It is not uncommon for a patient whose breath odor is significantly offensive according to multiple judges to have a normal peak VSC measured by the Halimeter™. It is well established that the proportion of CH_3SH to H_2S in VSC varies considerably.[15] Tonzetich[25] demonstrated that the air concentration threshold for organoleptic objectionability of CH_3SH (0.5 ng/10 mL) was one third that of H_2S (1.5 ng/10 mL). Because the Halimeter™ is approximately twice as sensitive to H_2S as it is to CH_3SH (verbal communication, March 1994, Shaw M, Interscan Corp., Halimeter™, manufacturer) it is possible that patients with high CH_3SH relative to H_2S in their mouth air may produce normal Halimeter™ measurements despite significant organoleptically detectable breath malodor. Conversely, patients with low CH_3SH relative to H_2S may have elevated Halimeter™ measurements and no organoleptically discernible breath malodor.

Imaginary Halitosis

When dealing with patients seeking professional care for halitosis, one must be prepared to differentiate between the patients who emit above-average malodor, those who emit average or near-average malodor but are more sensitive to it, and those who emit below-average or no malodor but believe that their breath is offensive (imaginary halitosis). In the former two cases, treatment of the organic causes of malodor is war-



Organoleptic Assessments	5/26/94	6/9/94	8/16/94	10/7/94
mouth air	0	1	1.5	4.5
nose air	0	0	0	0
lung air	0	0	0	0
tongue sample	1	1	0.5	5
floss sample	0	2	0	3
appliance	0	3	2	4
HaliTest™	Neg	60	120	15

Figures 6A and 6B—Testing results collected from a 22-year-old woman on 4 different occasions. All tests were performed under the same previsit preparation conditions: (a) VSC recordings of mouth, nose, and lung air on four different dates; (b) corresponding ORG using the 0 to 5 scoring system and HaliTest™ scores (VSGT). The patient demonstrated significant mouth air malodor, elevated mouth air VSC, and a high rate of VSC generation from tongue coating (HaliTest™ <30) on October 7, 1994, only, which was the first day of her menstrual cycle. However, even multiple occasions of negative testing for breath malodor may not justify a diagnosis of "imaginary halitosis."

ranted; in the latter, it is not. That is not to say that sufferers of imaginary halitosis cannot be helped. There are many patients who complain of chronic bad breath for whom no objective evidence of breath malodor can be identified.[12,18,37,38] Olfactory reference syndrome[39,40] is a recognized psychiatric condition. It has been described as a somatization of some psychological distress resulting in a belief that an offensive odor emanates from some body part, usually the mouth. This condition interferes with normal social interactions for fear of offending others with breath malodor and has been described in the psychiatric literature for more than 100 years.[37,40] Affective disorders and schizophrenia were reported to develop in patients whose initial complaints were limited to malodor, and some success has been found in treating olfactory reference syndrome with tricyclic antidepressants and the antipsychotic chlorpromazine.[37,38,41] We have had some preliminary success treating this condition with the serotonin reuptake inhibitor Zoloft®[c] (verbal communication, March 1995, E Schweitzer, MD, Hospital of the University of Pennsylvania).

[c] Roerig Division Pfizer Incorporated, NY, NY 10017

Breath malodor can be episodic[14] and may be provoked or diminished by a variety of transient conditions[1,9] that cannot always be controlled. Consequently, an incorrect diagnosis of imaginary halitosis can result if a patient's evaluation is limited to a single occasion of VSC and organoleptic assessment. We have found that many patients who suffer mild or occasional episodes of breath malodor may test negative for elevated VSC and/or organoleptically determined malodor at a particular office visit, even if previsit conditions are imposed that enhance breath malodor (Figures 6A and 6B). Therefore, we have established guidelines to avoid mistaking mild or occasional breath malodor problems for imaginary halitosis. A diagnosis of imaginary halitosis is considered *only* if all the following conditions are met after repeated testing under various properly selected conditions conducive to the production of breath malodor:

1. Breath malodor cannot be organoleptically identified, *and* above-normal levels of VSC cannot be demonstrated instrumentally in any breath samples taken from various airway locations.
2. A sample of tongue coating is demonstrated to have a poor ability to convert bioavailable sulfur to VSC (VSGT >60).
3. The patient cannot provide reliable third-party verification of his/her bad breath.

Unlike the discomfort we associate with traditional dental diseases, the discomfort caused by halitosis is totally psychological. The social and sexual taboos associated with halitosis originate in antiquity. Commercial mouthwash and "breath-freshener" advertising reinforces and exaggerates the stigma of bad breath to the public. Such repeated exposure to the social anathema of halitosis may account for the high level of disproportionate concern about bad breath that we have observed in patients who present for halitosis treatment. This phenomenon can occur with real or imagined halitosis, and it appears in two forms, which often coexist: (1) an exaggerated perception by the patient of the intensity of the halitosis; and (2) an excessive preoccupation with the personal consequences of the halitosis. Patients who manifest these symptoms often exhibit inappropriate behavior that interferes significantly with their everyday lives. Halitosis sufferers commonly identify their halitosis as the cause for limited ca-

reer choices, poor job performance, loss of jobs, failed marriages, impotency, social isolation, inability to concentrate, and thoughts of suicide. Therefore, any protocol for evaluating patients for complaints of halitosis, whether real or imagined, must include criteria that reveal the degree to which a patient may be inappropriately preoccupied with or psychologically impaired by his/her condition.

Documentation and Diagnosis

The following is a protocol that we have developed for the diagnosis and management of complaints of bad breath:

1. *Visit Preparation*

In preparation for their first visit to the office, patients are instructed to abstain from: food, breath fresheners, and oral hygiene for 6 to 12 hours; smoking for 12 hours; scented cosmetics for 24 hours; onions, garlic, and spicy foods for 48 hours; and antibiotics for 3 weeks.

2. *Halitosis History*

After reviewing the medical history, a thorough halitosis history is taken, which is divided into six sections—each designed to reveal specific objective and subjective information about the patient and his/her complaint. Some of the issues raised in the interview may be emotionally charged, and patients may resist open and frank discussion about them. Therefore, it is important that the interviewer establish a relaxed and communicative atmosphere at the outset. The six sections of the halitosis history are described briefly below:

Age and circumstances of onset of halitosis—When and how did the patient first become aware that he/she had halitosis? If the onset was recent and/or accompanied by other physical signs or symptoms, one might suspect the origin to be a systemic or localized respiratory disease. If first recognition can be traced to a particular comment from another person, the degree to which this initial awareness is supported by subsequent events becomes important. Among our patients, it is not uncommon for a comment received in childhood to trigger a lifelong debilitating preoccupation with halitosis.

Current methods used by the patient for judging his/her halitosis—Patients rely on three methods for assessing or knowing about their halitosis: (1) self-perceived taste or odor; (2) body language, including verbal and nonverbal "hints" from other people; and (3) direct verbal corroboration from other people. In our experience, only direct verbal corroboration is objectively reliable evidence of a breath malodor problem. Patients who rely heavily on the first two methods sometimes have difficulty perceiving improvement when their breath malodor is eliminated.

Blood-borne malodorant volatiles—Ethnic diets that regularly include a large amount of garlic and other mercaptan- or thiol-containing foods can result in conspicuous and prolonged episodes of breath malodor. These and other dietary volatiles, such as ethanol, are rapidly absorbed into the bloodstream through the gastrointestinal tract and are carried to the lungs where they volatilize and enter the breath.[18] Other potential sources of blood-borne malodorant volatiles are metabolic disorders, such as trimethylaminuria,[11] amino acid absorption deficits,[42] and medications.[18] Patients with these conditions may also present with complaints of bad taste and/or body odors.[43]

Conditions that can result in elevated salivary proteins—Breath malodor production is enhanced by elevations in the saliva concentrations of the VSC precursors, cystine and methionine, whether they appear as free amino acids or in peptide configu-

Figures 7A, 7B, and 7C—Anaerobic bacterial glossitis: (a) tongue coating; (b) removal of tongue coating; (c) ulceration of dorsoposterior tongue surface was revealed after removal of tongue coating. All oral soft-tissue surfaces were equally debrided with a 20-ppm molecular ClO_2 irrigant administered with a Prophy-Jet30®. Sodium bicarbonate powder was not used with the irrigant.

ration.[1] Periodontal disease, xerostomia, oral ulcers, rampant caries, poor oral hygiene, or oral inflammations associated with excessive use of irritants, such as tobacco and some commercial breath fresheners, can be expected to result in elevations in salivary proteins. The cyclic variations in oral VSC production associated with the menstrual cycle[34] (see Figure 6A) may also correspond to variations in salivary protein concentrations.

Inaccurate perception of the intensity of halitosis—In our experience, many of the patients who seek treatment for bad breath have, to varying degrees, exaggerated perceptions of the frequency and intensity of their episodes of breath malodor. The level of exaggeration can be estimated by asking the patient to assess the intensity of his/her own malodor at the moment and then comparing this self-assessment to organoleptic assessment(s) by the examiner(s). A patient's estimate of the distance from which his/her halitosis can offend people is also helpful in this regard. Estimates of up to 3 feet are probably realistic, whereas estimates of more than 5 feet suggest exaggeration.

Disproportionate concern about halitosis—The degree to which a patient's concern about his/her halitosis interferes with normal, daily activities reveals the appropriateness of the patient's adaptation to his/her condition. Patients may disclose that their halitosis is interfering substantively with their ability to work, participate in social activities, express affection, or concentrate. Some admit to thoughts of suicide. Some patients betray an excessive preoccupation with their halitosis by having undergone unreasonable numbers of invasive dental and/or medical tests and procedures in search of a cure.

3. *Testing*

Volatile sulfur-compound concentrations in mouth, nose, and lung air are recorded with a Halimeter™, a modified sulfide monitor first described by Rosenberg et al[13] and a pen-writer, Linear model 1101[d] (Figure 3). According to the manufacturer, the sensitivity of the monitor is 0-1900 ppb to H_2S with a 60% response to equal concentrations of methylmercaptan.[3] The Halimeter™ output signal is adjusted with a variable potentiometer so that a 500-ppb response by the Halimeter™ produces a 5-cm deflection of the pen-writer set at a sensitivity of 500 mV/cm. Before each recording, the monitor is adjusted to zero for ambient room air. Samples of mouth, nose, and lung air are collected in plastic bags (6 5/8" x 5 7/8") through flexible straws (7 5/8" x 15/64" diameter) and fed into the Halimeter™ immediately on collection. Because we have found no differences in the recordings obtained from mouth air fed directly from the mouth to the Halimeter™ through a vented straw and those obtained through the bag collection method, we generally feed air directly to the Halimeter™ from the mouth through a vented straw for convenience. Detailed instructions for preparation, sample collection, VSC measurement, and recording with the Halimeter™ and pen-writer are available through Interscan Corp.[a] or ProFresh, Inc.[b] Figure 4 is an example of a recording obtained in this manner. Peak VSC values for each sample are recorded in the patient's chart.

Separate organoleptic assessments of oral, nasal, and pulmonary air are performed and recorded independently by two operators. A 0 to 5 scoring system is used. "0" indicates no perceptible malodor; "1," barely detectable malodor; "2," slightly offensive malodor; "3," moderately offensive malodor; "4," strongly offensive malodor; and "5," overwhelmingly offensive malodor. Oral air is assessed immediately after a 3-minute incubation period during which the patient keeps the lips sealed and breathes through the nose. Then the patient opens the mouth wide and breathes out slowly through the mouth. The operator, having positioned his/her nose 4 to 6 inches from the patient's open mouth, assesses the odor of the patient's mouth air (ORG_m) with a series of two to four strong, but brief, stacattolike sniffs. The patient then pinches the nose closed for 3 minutes while breathing through the mouth. The operator repeats the assessment procedure on the initial air sample expelled

[d] Linear Instrument Corp, Irvine, CA 92718

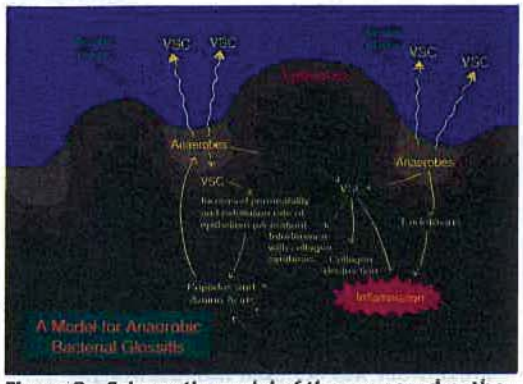


Figure 8—Schematic model of the proposed pathogenesis of anaerobic bacterial glossitis and related breath malodor. Superficial, aerobic plaque on the dorsoposterior surface of the tongue imposes oxygen depletion and promotes anaerobic bacterial growth in deeper plaque layers, especially between papillae. VSC produced from degradation of salivary proteins by anaerobes cause increased epithelial permeability, which permits access for bacterial endotoxins and VSC to the underlying connective tissue. Ensuing inflammation and collagen destruction result in elevated levels of peptides and amino acids at the tissue surface, thus providing more substrate for anaerobic bacterial generation of VSC. When the concentrations of VSC in soft tissue and saliva reach saturation, they volatilize and may be perceived as bad breath. Compare with Figure 5.

through the nose (ORG_n). The patient continues to expel air slowly while the operator turns his/her head away from the patient and sniffs room air to avoid olfactory adaptation to any perceived odor by reacclimating his/her sense of smell to ambient room air. As the patient approaches the end of exhalation, the operator turns back to the patient and assesses the last sample of air that the patient can force out of the lungs through the nose (ORG_1). The two operators' scores for each air sample are averaged and recorded in the patient's chart.

An assessment of the relative capacity of the patient's tongue coating to convert bioavailable sulfur to VSC is obtained by gently drying the dorsoposterior surface of the tongue with a gauze square or air syringe, collecting a sample of dorsoposterior tongue coating on a cotton swab 1 to 3 cm anterior to the lingual cecum, and then plunging the cotton swab into HaliTest™[b]. The operator then records in the chart the time required for a yellow-brown coloration, which indicates evolution of VSC, to first appear on the cotton swab. At room temperature, a time of 30 minutes or less is considered a strong positive; 31 to 90 minutes, a weak positive; and more than 90 minutes, a negative test result.

Separate organoleptic assessments (0 to 5) of intraoral appliances, tongue coating, and interdental samples are performed by a single operator and then recorded in the chart. Dental appliances are removed, dried to remove saliva, and assessed organoleptically for malodor. Next, a 2 x 2 gauze square is applied with finger pressure to the midline of the dorsoposterior border of the tongue and drawn anteriorly for about 1 inch. The gauze is removed and immediately assessed organoleptically for malodor. Finally, Superfloss 7™[e], an absorbent dental floss, is drawn interproximally between all of the posterior teeth. A separate piece of floss is used for each sextant and is immediately assessed organoleptically and discarded.

4. *Examination*

Ears, nose, and oropharynx are examined with an otoscope and tongue depressor for evidence of acute or chronic upper respiratory diseases or conditions commonly associated with complaints of bad breath (tonsilloliths, postnasal drip, nasal polyps, etc).[18] Dental radiographs (panoramic x-ray and bite-wings) are taken, and a complete dental examination is performed. During these examinations, particular attention is given to conditions that may cause patients to perceive bad tastes or odors, such as endodontically infected teeth, cryptic tonsils, or defective dental restorations. It is not usual for such conditions to be a source of bad breath (as perceived by others), but they may promote or intensify a patient's concern about bad breath.

5. *Diagnosis*

With the information obtained from the malodor history, testing, and examination, a diagnosis is proposed. In virtually all apparently healthy patients who have breath malodor, the cause of malodor is the degradation of host tissue proteins by anaerobic bacteria on the dorsoposterior surface of the tongue. We have called this condition "anaerobic bacterial glossitis." We feel this term, which implies infection, is warranted because of the clinical signs of inflammation and the frequency of frank ulceration of the tongue surface that accompanies this condition (Figures 7A through 7C). The apparent similarities between anaerobic bacterial glossitis and periodontitis are striking. Consequently, we have proposed a model for the pathogenesis of anaerobic bacterial glossitis (Figure 8) that parallels the model for the pathogenesis of periodontal disease depicted in Figure 5.

The most important and challenging aspects of diagnosis are estimating: (1) the degree to which the patient produces breath malodor (this includes estimating the frequency and intensity of episodes of breath malodor); (2) the degree to which the patient's perception of his/her breath malodor is exaggerated; and (3) the degree to which the patient's concern about bad breath provokes inappropriate behavior or preoccupation with the condition. The relative contributions of these three factors to the patient's overall level of discomfort with his/her condi-

[e] Oral-B, Redwood City, CA 94065

Table 4—Patient Results of Treatment for Breath Malodor

Patients responding to a posttreatment questionnaire 4 to 20 weeks after treatment were asked, "Do you feel there has been a significant improvement in your breath odor problem?"

Total Responding	Responding "Yes"	Responding "Somewhat"	Responding "No"
1343	1047	243	53

tion must be considered when formulating a treatment plan.

Treatment

The objectives of treatment are elimination of the patient's breath malodor and the discomfort the patient experiences in situations in which breath malodor was a problem in the past. While these two objectives are somewhat interdependent, success in achieving the former is not always followed by success in achieving the latter. Treatment for breath malodor consists of three phases: (1) elimination of all breath malodor; (2) instruction of the patient in objective methods for the evaluation of his/her breath malodor; and (3) monitoring the patient's progress in divesting him/herself of the social apprehension and psychological discomfort associated with having bad breath.

Orally generated breath malodor is caused by VSC that volatilize from the saliva and oral soft tissues. Elimination of breath malodor is directed toward reducing the oral generation of VSC and removing volatilized and nonvolatilized VSC from the mouth. For these purposes, an intraoral liquid-air spray device, Prophy-Jet® 30[f], and an ultrasonic intraoral dental cleaner, BOBcat® Ultrasonic Scaler[f], have been modified to deliver a 20-ppm molecular chlorine dioxide irrigant, ProFresh™ Irrigant[b], for debridement and deodorization of the hard and soft tissues of the mouth. Sodium bicarbonate is not used

[f] Dentsply International Inc, York, PA 17405

with the Prophy-Jet® 30. A spray deflector is attached to the Prophy-Jet® 30 handpiece to localize the impact of the spray to a small area and to prevent the spray from causing gagging when debriding posterior oral soft-tissue surfaces. The tip of the Prophy-Jet® 30 nozzle is positioned about 1 cm from the intraoral tissues at approximately a 45-degree angle and moved in a slow, circular motion over all accessible soft-tissue surfaces (Figures 7A through 7C). Particular attention is given to the circumvallate region of the tongue. Bleeding can be expected from this area as a result of the spray. Supragingival calculus is removed, and interdental areas are flushed with molecular chlorine dioxide (ClO_2) irrigant using the Cavitron TFI®-1000 insert.[t]

The deodorizing and antibacterial characteristics of molecular ClO_2 in aqueous solutions are well known.[44-46] It is safe and nontoxic in solutions in the range of concentrations described here.[44,45] The physical and chemical properties of molecular ClO_2 are different from those of so-called "stabilized chlorine dioxide." Stabilized ClO_2 is a solution of sodium chlorite that, by definition, contains no significant amounts of molecular ClO_2.[44] Various commercially available stabilized ClO_2 mouthrinses contain no chlorine dioxide when measured spectrophotometrically in the laboratory. Chlorine dioxide deodorizes by oxidizing malodorous sulfides and thiols (VSC) to nonodoriferous salts.[46] It also oxidizes other malodorous volatiles, such as aliphatic amines, phenols, and alcohols.[44,45] Chlorine dioxide's high solubility in nonpolar solvents,[46] as well as its affinity for cell surfaces,[46] suggest it may concentrate itself in plaque and on soft-tissue surfaces. These properties may enhance the deodorizing effect of ClO_2 because a large proportion of oral VSC is dissolved in soft tissue, saliva, and plaque. The antibacterial effect of ClO_2 is not well understood but presumably derives from interference with protein synthesis and alterations of membrane permeability to electrolytes within bacterial cells.[44,45] Chlorine dioxide also may reduce VSC generation by oxidizing sulfur-containing, free amino acids and peptides,[46] which are the precursors of VSC.

After professional debridement and irrigation of the soft tissues with aqueous chlorine dioxide, the patient is introduced to a regimen that is designed to prevent recurrences of breath malodor. The purpose of the regimen is to mechanically remove surface plaque and pellicle so that all potentially accessible anaerobic sites in the mouth become exposed directly to the oxidizing effects of ClO_2. Patients are instructed to brush their teeth, cheeks, lips, and palate for 1 minute with ⅓ oz of an 8-ppm molecular ClO_2 mouthrinse, such as Profresh™ Mouthrinse[b]. This is followed by flossing, tongue cleaning of the posterior third of the dorsoposterior surface of the tongue with a tongue blade, and a final 30-second rinse, taking care to saturate the dorsoposterior surface of the tongue with the ClO_2 mouthrinse. This regimen, performed once in the morning and again in the evening, is sufficient for nearly all individuals to maintain complete control of breath malodor 24 hours per day after undergoing the in-office antiseptic debridement and irrigation.

After treatment and maintenance instruction, patients are instructed to keep a log of organo-

leptic breath odor assessments performed by family members or friends at 2 or more randomly selected times each day for 2 to 4 weeks. This exercise is important for patients to "unlearn" previous, unreliable methods of breath odor self-assessment. Without such direct, cognitive feedback from other people, many patients cannot achieve confidence that their breath odor is controlled.

Two to 4 weeks after treatment, the patient returns for retesting under the same pretesting conditions that produced the greater level of breath malodor of the 2 visits. Attention is given to the time of day, time without food, drink, or oral hygiene, and, for women, day of the menstrual cycle. The patient is instructed to bring the assessment log to this visit for review. If episodes of breath malodor persist after treatment, the log is helpful in determining the periodicity of such episodes so that appropriate alterations in the timing and/or frequency of the patient's maintenance regimen can be recommended.

Results

Of the 2,837 patients who visited our clinic between February 1993 and February 1995, verifiable breath malodor was evident for 2,243. Because many of these patients traveled considerable distances to our clinic, we were unable to perform posttreatment assessments on all of them. Of the 923 patients who presented with verifiable breath malodor for whom posttreatment organoleptic and VSC assessments were performed, breath malodor was eliminated in 918 (99%) (Table 3). A patient was identified as having no breath malodor if all organoleptic assessment scores were below 1.0, all VSC measurements were below 180 ppb, tongue coating VSC generation time exceeded 60 minutes, and no reliable confirmation from a third party could be obtained.

When a posttreatment follow-up questionnaire was mailed to 1,343 patients between 4 and 20 weeks after in-office treatment, 1,047 (78%) indicated that they had experienced "significant improvement" in their breath odor as a result of treatment and maintenance. Another 243 (18%) indicated "somewhat improved" while 53 (4%) indicated "no improvement" (Table 4). Of the 53 who indicated no improvement, only 1 could provide reliable third-party confirmation of a persistence of breath malodor. Many of these patients received no in-office, posttreatment evaluation.

Conclusion

Bad breath is a major concern for many people. Because it nearly always originates in the mouth, it can and should be diagnosed and treated professionally by dentists. There is no known "stand-alone" product solution for halitosis, nor do traditional standards of dental or periodontal care eliminate the problem. Recent developments in the understanding of the etiologies of breath malodor have spawned new techniques for its assessment and management. This article outlined a clinical protocol for diagnosing and treating chronic halitosis that is highly effective and reliable and consistently produces patient satisfaction.

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