

Gustatory Sweating: Clinical Implications and Etiologic Aspects

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Purpose: It was the aim of this study to provide detailed general information on the clinical picture of different kinds of gustatory sweating, including reevaluation of a series of patients who underwent parotidectomy, removal of the submandibular gland, or neck dissection.

Patients and Methods: This study summarizes the statements of 548 patients questioned about the occurrence of gustatory sweating after parotidectomy (n = 296), extirpation of the submandibular gland (n = 79), and neck dissection (n = 173).

Results: After parotidectomy, 45% of the patients had noticed gustatory sweating. In most of them (70%), the symptoms began within 6 months after surgery. Gustatory sweating developed in only one patient with submandibular extirpation (1.5%), and not at all after neck dissection. Most patients (52%) reported that the symptoms occurred independent of the kind of food ingested. These results show that the "masticatory component" is an important trigger for Frey's syndrome. Application of Minor's test localized gustatory sweating mainly in the region of previous parotid lobe removal, but also in other areas deriving their sensory supply from the auriculotemporal, greater auricular, and lesser occipital nerves. The size of the area affected by the sweating was similar after lateral and total parotidectomy. When evaluating clinical symptoms, subjective assessment by the patients seemed to play a major role. After submandibular extirpation and neck dissection, some patients reported gustatory sweating that was not verified by Minor's test.

Conclusion: There is general agreement that the cause of gustatory sweating is sympathetic or parasympathetic innervation of previously denervated sweat glands, initiated by gustatory triggers. The location of the "erroneous innervation" depends on the type of lesion. In cases after parotidectomy, misdirected parasympathetic regeneration is the model integrating all known factors into a rational concept. For didactic and systematic-pragmatic reasons, a clinically oriented classification of gustatory sweating (types I to III) seems to be useful.

The surgeon is confronted with gustatory sweating mainly after parotid gland surgery, most notably parotidectomy.^{1,2} It is widely assumed that this finding is caused by parasympathetic fibers regenerating after a lesion and, in the process of resprouting, making contact with sweat glands of the skin that are normally innervated by sympathetic fibers.^{1,2} As a consequence,

acetylcholine, the neurotransmitter active in both the sympathetic and the parasympathetic nervous systems, then triggers pathologic secretion from the sweat glands. The first descriptions of unilateral focal gustatory hyperhidrosis subsequent to a parotid abscess were provided as early as 1757 by Duphenix³ and in 1853 by Baillarger.⁴ As a separate diagnostic entity, the so-called auriculotemporal syndrome was described in detail by Frey.⁵ Gustatory sweating also has been reported to occur after extirpation of the submandibular gland.⁶

Several factors may be involved in lesions to parasympathetic fibers during operations on salivary glands: 1) the complete or partial removal of the gland in parotid or submandibular surgery during which these fibers are injured far in the periphery; 2) surgical dissection along the course of the facial nerve, which is accompanied by parasympathetic fibers, may result in their disruption during parotidectomy. Both factors may become involved after trauma, such as in fractures

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involving the temporomandibular joint⁷ or in salivary gland infections.^{3,4}

The area of pathologic sweating coincides in most instances partly or completely with the regional distribution of the auriculotemporal and greater auricular nerves. Gustatory sweating has even been observed in the external auditory meatus.⁸ Gustatory sweating may occur even without intraoperative excision of salivary gland tissue. Its occurrence after extirpation of cervical lymph nodes⁹ and after neck dissection^{10,11} also can be explained by innervation of cutaneous sweat glands by misdirected cholinergic parasympathetic nerve fibers. This happens when preganglionic parasympathetic fibers running toward the submandibular gland, or postganglionic parasympathetic fibers coursing to the sublingual gland, are damaged. The lesion is supposed to occur at the submandibular ganglion, which is manipulated during dissection of the lingual nerve.¹¹

It is generally assumed that the newly formed connection between the sweat glands and cholinergic parasympathetic fibers is only made possible by a preceding sympathetic denervation of the sweat glands, for instance, by surgical intervention (thereby disrupting the sympathetic fibers running in the auriculotemporal and greater auricular nerves) and during preparation of the skin flap.¹² After parotidectomy, Glaister et al¹³ even found the "reverse phenomenon" of pathologic saliva production caused by misdirected sympathetic fibers in the region of parotid remnants.

It was the aim of this study to provide detailed information on the clinical picture of gustatory sweating based on a thorough reevaluation of our own patients. Besides a general description of symptoms, we present data on the localization of Frey syndrome after parotidectomy, after extirpation of the subman-

dibular gland, and after neck dissection. The data derived from our patients form the basis of a discussion of certain etiologic aspects. To better differentiate between the manifold pathologic states connected with gustatory sweating that were described in the literature, a classification system based on clinical appearance is suggested.

Patients and Methods

The 548 patients included in this study responded to a questionnaire sent to them. Questions concerned the time when symptoms were noticed, trigger mechanisms, and the nature of complaints. One hundred eighty (60.8%) of the original 296 patients who had a parotidectomy, 68 (86.1%) of the 79 patients in whom the submandibular gland had been removed, and 109 (63%) of the 173 patients who had a neck dissection returned the questionnaire. Of the group of neck dissection patients, nine underwent radical neck dissection and 164 underwent functional neck dissection. Among the 109 patients that underwent a total of 161 neck dissections who answered the questionnaire, the submandibular gland had been extirpated simultaneously in 24 cases. Most of our investigated patients (85%) were treated for laryngeal, oropharyngeal, or hypopharyngeal cancer. Based on our therapy regimen, dissection of the level I lymph nodes, including removal of the submandibular gland was not usual in these cases, in contrast to patients with cancer of the oral cavity (15%).

Minor's test¹⁴ was performed in 37 patients with Frey's syndrome (Fig 1). This number included 19 patients after parotidectomy (22 operated sides, 14 lateral and 8 total parotidectomies), seven patients after extirpation of the submandibular gland, and 11

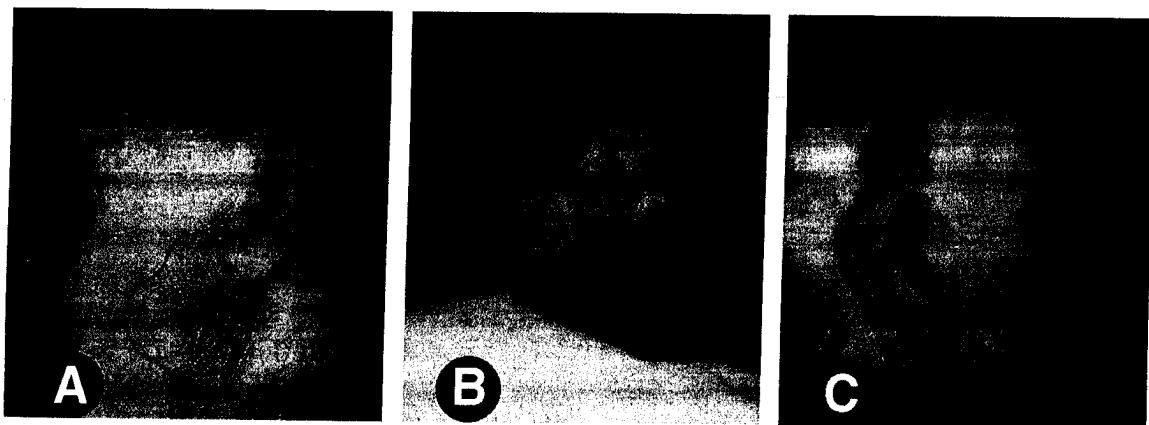


FIGURE 1. Examples of a positive Minor's test, in A,C, patients after parotidectomy who also showed sweating in regions outside the area of previous lobe surgery. C, Even the area supplied by the lesser occipital nerve may be involved. After mandibular fracture with concomitant damage to the parotid, the area of sweat production, B, lies relatively far anteriorly.

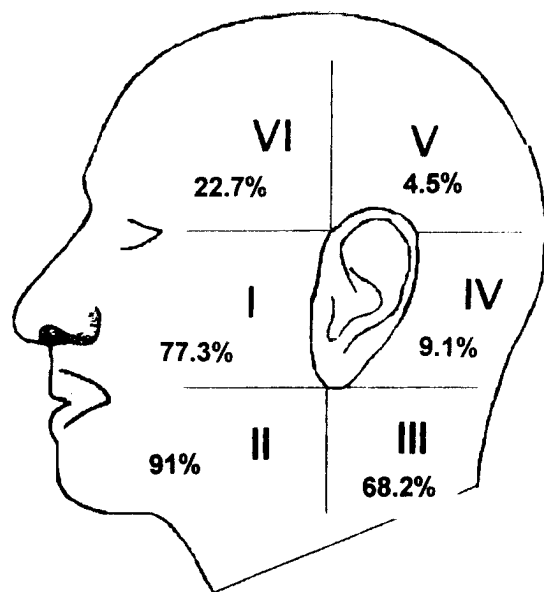


FIGURE 2. Schematic representation of the areas selected for evaluation based on various regions of sensory innervation of the preauricular region. Regions I and VI correspond to the area innervated by the auriculotemporal nerve; regions II, III and IV are supplied by the anterior and posterior branches of the greater auricular nerve; and region V is innervated by the lesser occipital and auriculotemporal nerves. Percentages give the frequency of postparotidectomy gustatory sweating in the various regions.

patients after neck dissection. For Minor's test, a solution containing 15 g iodine, 100 mL castor oil, and 900 mL ethanol was applied to the skin of the affected side. Then this area was powdered with starch. Patients were asked to eat an apple and in positive cases about 30 seconds later the treated skin areas showed a deep blue color, indicating the reaction between the two basic components (starch, iodine) of this test.

For an exact determination of the area of gustatory sweating, the periauricular region in the confines of the various regions of sensory innervation¹⁵ was divided into six fields (Fig 2). Because of the severity of symptoms, the previously noted 19 patients (22 treated sides) who had had a parotidectomy were treated with botulinum toxin. The criteria for treating were a strongly felt reduction of life quality and the desire to be treated. The dosages used depended on the extent of the sweating skin area and varied between 2.5 and 100 units of Botox (Allergan Inc, Irvine, CA). In all treated patients, gustatory sweating ceased completely within 2 days, and the average duration of this positive effect was 17.3 months.^{16,17} Before botulinum injection, the area showing gustatory sweating was divided into fields of 4 cm² each that facilitated assessment of their size. Where applicable, one of three different statistical tests was used

(χ^2 test for contingency tables, Fisher's exact test, unpaired *t* test), as specified in the Results section.

Results

GENERAL DATA ON SYMPTOMS AND TRIGGERS

Eighty-one (45%) of the 180 patients with parotidectomy returning questionnaires reported the occurrence of Frey's syndrome. The different pathologic conditions are summarized in Table 1. Among the clinical symptoms stated, 52 patients (63%) suffered from "dripping sweat," sometimes combined with other complaints, and "sweating" was reported by 41 (50%) patients. Lesser symptoms of "erythema" and "local hyperthermia" were noticed by 30 (36%), and 25 (31%) patients, respectively. Other complaints were formication in 16 cases (19%) and a swelling sensation in 11 cases (13%). "Burning" (5 patients = 6%) and "yellowish discoloration of hair" (3 patients = 4%) were rarely mentioned.

Fifty-six (70%) of these 81 patients stated that the symptoms had developed within the first 6 months after surgery. Gustatory sweating noticed for the first time as late as 1 year postoperatively was reported by five patients (6%). In 43 cases (52%), Frey's syndrome occurred regardless of the kind of food ingested.

However, in others, the syndrome was triggered by food of a particular taste or consistency. Among the types of food responsible for the attacks were, alone or in combination, "hard food" (31 patients = 38%), food with a very sour taste (30 patients = 36%), hot food (22 patients = 27%), and very spicy food (17 patients = 21%). Sweets (11 patients = 13%), cooking smells (4 patients = 5%), drinking (5 patients = 6%), and ice cream (3 patients = 4%) were mentioned less frequently. Nineteen patients (10.6%) who suffered very greatly from the sweating symp-

Benign Lesions	Number	Malignant Lesions	Number
Pleomorphic adenoma	80	Adenoidcystic carcinoma	11
Warthin tumor	47	Mucoepidermoid carcinoma	7
Chronic infection	14	Lymphoma (non-Hodgkin)	4
Cyst	4	Squamous cell carcinoma	3
Histiocytoma	1	Metastases (of squamous cell carcinoma)	3
Lymphangioma	1	Acinic cell carcinoma	1
Lipoma	4		
Total	151		29
Frequency Frey's syndrome	70 (46%)		11 (37%)

toms were given appointments for botulinum toxin therapy.

LOCALIZATION AND EXTENSION OF GUSTATORY SWEATING AFTER PAROTIDECTOMY

Minor's test was performed in all 19 patients (22 operated facial sides) before receiving botulinum toxin therapy. Close inspection showed that the area showing discoloration after provocation by gustatory stimulants was predominantly localized where a parotid lobe dissection had been carried out (Fig 2). The hirsute area of the head (region IV, Fig 1) and the upper, middle, and lower retroauricular regions (regions III to V), areas where the sweat gland-equipped skin had not been touched, also showed the typical bluish color in some instances (Fig 2).

Table 2 shows the data indicating the color development in the various areas and their combinations. Area VI was involved significantly more often in patients after total parotidectomy ($P = .039$, Fisher's exact test). No such difference could be found for the other areas or their combinations. Area sizes were only slightly different between patients with a lateral (mean: 49.4 mm²; min, 16; max, 96 mm²) and a total parotidectomy (mean: 52.4 mm²; min, 28; max, 84 mm²).

INCIDENCE OF GUSTATORY SWEATING AFTER EXTIRPATION OF THE SUBMANDIBULAR GLAND

Seven (10%) of 68 patients in whom the entire submandibular gland had been extirpated had noticed gustatory sweating. However, the Minor's test was positive in only one patient (1.5%, Fig 3). The area of pathologic sweating corresponded exactly to the skin overlying the previous site of the extirpated gland. The patient had noticed abnormal sweat production for the first time 1 month after surgery. There was a discrepancy between the results of the Minor's test and the subjective complaints in the other patients.

INCIDENCE OF GUSTATORY SWEATING AFTER NECK DISSECTION

Of the 109 patients available for follow-up, 11 (10%) indicated the occurrence of gustatory sweating. Fif-

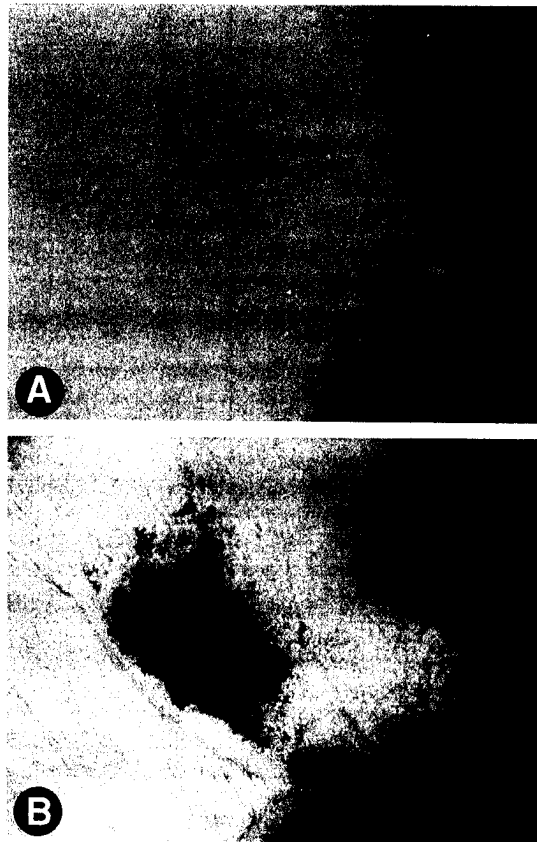


FIGURE 3. Minor's test in a patient after submandibular gland extirpation A, before and B, after gustatory stimulation. The discoloration of the submandibular region is clearly visible.

teen neck dissections had been performed in these 11 patients. They all belonged to the group of functional neck dissection.¹⁸ In two of them, the submandibular gland had also been removed. The starch-iodine test was negative in all patients studied. Here too, a discrepancy existed between subjective (patients' complaints) and objective data (Minor's test). Also, both neck dissections that included the submandibular gland had failed to produce Frey's syndrome.

Discussion

Our results concerning the time after which Frey's syndrome develops after surgery and the frequency of "vascular symptoms" are in good agreement with those of other authors.¹⁹ Table 1 summarizes the frequency of the different pathologic conditions. The analysis showed that there was no significant difference in the incidence of Frey's syndrome in surgically treated benign and malignant lesions. However, in this series of patients, an increased incidence of Frey's

TABLE 2. FREQUENCY OF COLOR DEVELOPMENT IN VARIOUS AREAS

Area	n	%	Combination of Areas	n	%
I	17	77.3	I/II	3	13.6
II	20	91.0	I/II/III	7	31.8
III	15	68.2	I/II/III/IV	2	9.1
IV	2	9	I/II/III/VI	2	9.1
V	1	4.5	I/II/VI	2	9.1
VI	5	22.7	I/VI	1	4.5
			II/III	4	18.2

syndrome was noticed compared with our previous investigation.²⁰

Most patients reported an independence of the occurrence of gustatory sweating from the quality of the taste of the food. The most frequent statement made with regard to food consistency was that "hard food" elicited sweating. This confirms that, as noted by others,^{21,22} the "masticatory component" seems to play a major role as a trigger. Ice cream or beverages hardly ever elicited the syndrome in our patients.

The size of the area of gustatory sweating after parotidectomy varied greatly, but even relatively small, circumscribed, areas caused discomfort in the affected persons. Neither field size nor amount of sweat production seemed to determine the degree of suffering, and the subjective feeling was the determining factor. This pronounced subjectivity was substantiated by the patients' impression of having developed gustatory sweating after submandibular gland extirpation or neck dissection, when the objective measurement by Minor's test failed to confirm actual sweating. Conversely, gustatory sweating nearly always developed after parotidectomy² but was not noticed by all patients as a problem needing treatment, a fact that also points to a strong subjective component. Likewise, when the symptoms recur after treatment of gustatory sweating by botulinum toxin injection, it goes unnoticed by some patients.¹⁷

Not all regions affected by sweating coincided with the regional distribution of the auriculotemporal nerve. It receives its parasympathetic fibers from branches connected with the facial nerve and provides the temporal skin anterior to and above the ear through the superficial temporal branches. The other regions (areas II to V) receive their sensory supply through the anterior and posterior branches of the greater auricular nerve and partly through the lesser occipital nerve, representing the dorsal portion of area V. In principle, although very rare according to our results, these nerves may serve as guiding structures along which regenerating parasympathetic fibers find their way toward the skin. This phenomenon was described by Laage-Hellmann¹² for the auriculotemporal and greater auricular nerves. The area supplied by the lesser occipital nerve was not involved. As far as we know, we are the first to describe such an involvement. The mechanism denervating the sweat glands in this region is unknown because the lesser occipital nerve is not damaged. It might be speculated that innervation is maintained or reestablished by competing parasympathetic and sympathetic fibers, but two other mechanisms seem much more likely. Either there exists an "expanded area of representation" of the auriculotemporal nerve that would also permit sensory supply to more cranial-dorsal skin areas or, most likely, sympathetic fibers accompanying blood vessels nourishing

the relevant regions of the skin were injured intraoperatively. Such vessels could be the occipital artery and its branches, and a lesion of sympathetic fibers close to their branching off from the external carotid artery seems possible during parotidectomy.

The significant difference between involvement of area VI after total and lateral parotidectomy may be explained by the more extended lesion of the auriculotemporal nerve and its branches after total parotidectomy, with subsequent sympathetic denervation of larger portions of the skin.¹² However, resprouting of parasympathetic axons without guiding structures also could permit the regenerating fibers to reach skin areas outside the normal field innervated by the auriculotemporal nerve.

Berini and Gay⁶ and McEwen and Sanchez,²³ as well as this study, found gustatory sweating after extirpation of the submandibular gland, although this seems to be a very rare occurrence. Only a single case of gustatory sweating was found after 206 operations by Berini and Gay⁶ and after 68 operations by us. Of course, these results have to be interpreted with caution because gustatory sweating may be present without being noticed by the patients. It is not clear why it is so rare after submandibular gland extirpation. This operation certainly damages the integrity of the parasympathetic innervation, but regeneration does not elicit the same effects as parotidectomy. Several reasons may be responsible for this phenomenon: the submandibular gland lies deeper under the skin than does the parotid; undermining the skin during surgical dissection is much less rigorous than in parotidectomy, which reduces the degree of sweat gland denervation, and the platysma may act as a natural barrier. This assumption is substantiated by the finding that, after parotidectomy, "barriers" against parasympathetic fibers such as the "superficial musculo-aponeurotic system"^{24,25} or others were successfully introduced to prevent gustatory sweating.²⁶ Lastly, a "guiding structure" such as the auriculotemporal nerve is missing in the vicinity of the submandibular gland, making it much more difficult for resprouting parasympathetic fibers to find their way to the skin.

The absence of gustatory sweating after neck dissection in our patients can be explained by the technique used.¹⁸ The lingual nerve, as a rule, was not dissected, and thus the danger of injuring the submandibular ganglion was avoided. For the reasons previously stated, simultaneous extirpation of the submandibular gland does not enhance the chances for development of Frey's syndrome.

The theoretical concept of the cause of gustatory sweating after operations with combined sympathetic-parasympathetic lesions was presented in the introduction. However, gustatory sweating has also been described as a consequence of sympathetic-sympa-

thetic lesions in the cervical region, such as after sympathectomy.^{27,28} In trauma or after resection of sympathetic ganglia containing axons controlled by thermal or gustatory stimuli, preganglionic and postganglionic fibers are damaged. Preganglionic sympathetic fibers that are controlled by gustatory stimuli contain acetylcholine as a neurotransmitter; aberrant regeneration after disruption therefore may lead to atypical sympathetic innervation of sweat glands resulting in Frey's syndrome when these fibers are excited by stimulants. Because our technique of neck dissection does not involve resection of the cervical sympathetic ganglia, the lack of gustatory sweating in our patients is not surprising. Another cause for Frey's syndrome has been suggested to be an exclusively sympathetic efference of a preformed reflex arc.²⁹ This would exclude parasympathetic involvement after parotidectomy. The model leaves open a number of anatomic questions pertaining to gustatory control; acetylcholine is supposed to be secreted in increased amounts in the parotid remnants left behind after surgery^{9,28} and to subsequently stimulate the denervated, and thus supersensitive, sweat glands. In our opinion, this explanation is not valid because it leaves the contact of neurotransmitter with the effector to chance by implying simple diffusion.

A further point frequently discussed in the literature is the possible existence of a preformed reflex arc.²⁹ This surmise is supported by the occurrence of gustatory sweating in normal persons, and the degree is supposed to depend on the excitatory level of the reflex arc. Such a model would help to explain why Frey's syndrome develops after lesions of the central nervous system, for instance, in encephalitis,²⁷ and the nature of the changes involved. In this context, a "shift" of the reflex threshold could play a role. Other hypothetical central nervous system mechanisms might be the formation of new connections between both functional systems (the thermoregulatory and the gustatory) during regeneration, and the induction of plastic processes in more distant brain centers after peripheral lesions (eg, after parotidectomy). Findings derived from study of the motor system³⁰⁻³² show that a number of cortical changes, for instance after damage to the facial nerve, can be demonstrated.

Because of the multitude of possible pathologic states that may be instrumental in causing gustatory sweating, we believe that the introduction of a classification based on and characterizing the different clinical situations encountered is advisable (Table 3). An advantage of such a classification system would be the possibility to use the aforementioned pathologic and anatomic models as a basis for a simplified, yet unequivocal, discussion of the subject. In other words, we need some kind of standardization of assessment when communicating about gustatory sweating.

Type	Pathologic State	Pathologic Basis
Type I Sympathetic/ para- sympathetic	Salivary gland surgery, salivary gland diseases, neck dissection	Lesion of thermoregulated sympathetic fibers + a lesion of gustatory-controlled parasympathetic fibers
Type II Sympathetic	Sympathectomy, neck surgery with sympathetic (trunk) lesions, cervical and cephalic trauma	Lesion of thermoregulated sympathetic fibers + a lesion of preganglionic (cholinergic) gustatory-controlled sympathetic fibers
Type III Central	Normal persons, feeble persons, central nervous diseases, eg, encephalotrophy, encephalitis, spinal processes such as syringomyelia, emotional stress	Central nervous lesion of thermoregulated or (?) gustatory regulation centers, low reflex threshold of gustatory sweating

In conclusion, there is some general agreement on the cause of gustatory sweating. Its symptoms are caused by sympathetic or parasympathetic innervation of previously denervated sweat glands, initiated by gustatory triggers and stimulated by the neurotransmitter acetylcholine. The location of the "erroneous innervation" depends on the type of lesion. A number of hypotheses have been offered to explain gustatory sweating after parotidectomy, but none of them has been proved. In our opinion, misdirected parasympathetic regeneration is the model that best integrates all known factors into a rational concept.

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Discussion

Gustatory Sweating: Clinical Implications and Etiologic Aspects

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The authors present a vast amount of information and discussion related to the pathophysiology of Frey syndrome. Their conclusions regarding the mechanisms responsible for the clinical aspects of this syndrome, although complete, lack supportive scientific evidence based on their study. The stated aim of the study is to discuss the clinical aspects of gustatory sweating, and through a questionnaire they were able to gather information regarding the general complaints of patients, mainly after parotidectomy. To this reader it is evident that many of the patient's complaints are not related to the commonly understood underlying neurophysiologic mechanisms of gustatory sweating. Complaints such as erythema, local hyperthermia, swelling sensations, burning, flushing, buzzing, shooting pain, etc, would more likely have a pathologic neurosensory or vasomotor mechanism, and should not necessarily be included as being related directly to gustatory sweating. The validity of their questionnaire related to the incidence of Frey syndrome is suspect

given that 45% of those questioned were thought to have gustatory sweating. The general literature would suggest an overall incidence of around 30%, which is about what this study demonstrates if you only include patients with complaints of sweating. However, the information gained through this questionnaire does help to delineate the nature of long-term morbidity after parotidectomy, even if not all of these symptoms can be related directly to Frey syndrome.^{1,2}

The authors showed strong evidence that botulinum toxin is an effective treatment for gustatory sweating, even though this was not an actual stated goal of their study. Numerous recent studies confirm the success of this treatment, many of which have much longer follow-up intervals and patient numbers. This study can be added to the body of information that supports botulinum toxin as a safe and effective treatment for gustatory sweating. This simple and efficacious treatment of gustatory sweating may well decrease the relative importance of preventing, understanding, and contemplating this surgical complication from a clinical standpoint.³

The authors nicely map out the involved areas of gustatory sweating through standard starch-iodine testing (Minor's test). This is useful information in determining what areas might be involved, and in which areas to concentrate treatment and preventative measures. Such localization

could influence the placement of anatomic barriers aimed at treating gustatory sweating such as superficial musculo-aponeurotic system flaps or alloplastic interpositional materials.

Overall, this article presents a great deal of information about the nature of morbidity after parotidectomy, even if not all such complaints can be categorized as part of Frey syndrome. The incidence of gustatory sweating is low after submandibular gland excision, and even lower after neck dissection, as again related in this article. The long-term results of botulinum toxin, and the significance of mapping out gustatory sweating by area, await further investigation.

Both of these topics have only been "touched on" in this report.

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