International Tinnitus Journal, Vol. 5, No. 2, 135–140 (1999)

Gustometry of Diabetes Mellitus Patients and Obese Patients

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Abstract: The sensation of adequate taste detection can be associated with satisfaction of food intake. The impairment of taste detection may be associated with the development of obesity. Taste detection is determined hereditarily, but it can be influenced also by the occurrence of neuropathy. To find an explanation for these phenomena, we investigated 73 patients with diabetes mellitus (DM) 2 (i.e., non-insulin-dependent DM); 11 patients with DM 1 (i.e., insulin-dependent DM); 12 obese patients (body-mass index >30) without DM; and 29 control patients. All subjects underwent electrogustometric examination with Hortmman's electrogustometer. During this examination, we obtained electrical thresholds of taste by stimulating appropriate parts of the tongue. We stimulated the apex, middle, and near tongue radix areas on both sides. The resulting value is the average on the left and right sides of the mentioned areas. We considered a value of less than 40 μA to be normal. Values in excess of 100 μA are considered as hypogeusia. Values between 40 and 100 μA are taken as borderline, and ageusia is in excess of 500 μA.

According to these criteria, in the DM 2 group, we found 40% of patients with hypogeusia, whereas in the DM 1 group, we found 33% of patients; 25% of patients were in the obese group. Among normal subjects (people without obesity or DM), no hypogeusia was found. We found ageusia in 5% of patients with DM 2, in 3% of patients with DM 1, and in 14% of obese patients. Among normal subjects, we found no ageusia. These results support the hypothesis that diminished taste detection can evoke hyperphagia and later obesity.

Keywords: diabetes mellitus; gustometry; obesity

Already known are many pathological states in which disorders related to taste preference and sharpness can be found. Many diseases can be included in this group of pathological states (e.g., alcohol abuse and cigarette smoking). Taste disorders can occur also as a side effect of drugs and radiotherapy [1]. For example, essential hypertension is associated with a significant disruption in taste (partially specific for salt sense distinction). This finding is relatively important. A recently published study showed that individuals with hypertension ingest more salty fluids than do normotensive control groups. A higher preference for salt, especially in the early phases of life, may have an influence on later food intake habits and may be a factor also in the emergence of hypertension [2]. However, a nonspecific increase in the awareness of taste also exists, occurring among patients with adrenal gland insufficiency.

Many published studies also have addressed the taste sensation among patients with diabetes mellitus (DM) and among obese patients, in comparison with a normal population. Diabetic patients, although not systematically followed for this disorder, seem to be very susceptible to taste disorders. We can consider many mechanisms, but a specific cause for taste sense disruption still is unknown. Antihypertensive drugs given to hypertensive diabetic patients are known also to have a deleterious effect on taste sensation. Among other drugs known to have a negative influence on taste sensation are sulfonylureas, also frequently used among diabetic patients. Additionally, an important association has been found between taste sensation degeneration and the degenerative complications of diabetes—specifically peripheral neuropathy and microalbuminuria, which are early signals of microangiopathy [1]. According to

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other theories, lowered taste sensation can be reflected also in generalized defects of glucose receptors, which includes both pancreatic B cells sensitive to glucose and tongue taste cells. Lowered taste sensation may lead to an increase in glucose intake (sweetened food and drinks) because, to elicit the same taste sensation, a greater amount of glucose would have to be ingested [2]. The disruption of taste sensation has a great influence on diabetic patients, not only in that it leads to a certain decrease in the quality of food ingested but that it may lead to changes in the quantity of food ingested, especially saccharides and salt. These factors later may influence metabolism and blood pressure [1].

The center of interest of many research projects also has been obese patients. These patients also have been followed up for their state of taste sensation, the preference of specific tastes, evaluation of specific tastes, and the like, in an effort to determine possible factors related to the etiology of obesity. Sweet taste sensation has been shown to be dependent on age, gender, and the level of obesity. With age, taste sensation gradually deteriorates. Published articles also have described sweet taste sensation preference differences among obese patients and normal-weight control individuals [3]. The results of individual studies in these areas differ greatly.

According to some studies, obese patients may have a higher preference for sweetened food than do normal-weight individuals, or they may prefer a higher sweet-taste sensation intensity. This higher preference for sweeter food substances is considered a possible factor related to the etiology of obesity and may lead to an increase in sweet food and high-energy substance intake [4]. However, according to other authors, most normal weight-control subjects and moderately overweight subjects appraise sweet fluids as agreeable or neutral. Obese subjects appraised the same fluids as less agreeable or neutral and preferred fluids with a lower concentration of sugar [3].

Other researchers divide factors influencing food intake—and with that also the relative energetic balance—into two categories. The first category includes external (preabsorbtive) sensory signals (e.g., visual, olfactory sensory signals) and other food characteristics. The other category includes internal metabolic signals (e.g., glucose levels and other hormonal and metabolic changes). This division of sensory and metabolic factors in reality is not as artificial as it may seem: Sensory signals can initiate metabolic changes and, conversely, internal states of the organism can modify reactions to sensory signals. Evidence also exists that both changes in body weight and energetic intake and hypoglycemia may influence the preference of specific signals arising from food (e.g., the preference of sweet taste or specific smells) [5].

OBJECTIVE

The aim of this study was to determine electrogustometric values among examined patients with DM type 1 (i.e., insulin-dependent DM [IDDM]); DM type 2 (i.e., non-insulin-dependent DM [NIDDM]); obese patients without diabetes; and control-group individuals neither obese nor diabetic, and to compare results among the groups. Another aim was to compare our results with the results of other similar published studies. The number of patients in our study does not allow us to claim 100% validity, but our results can come sufficiently close to reality.

PATIENTS

A total of 125 patients were examined (40 men, 85 women; average age, 46.3 years). Individuals were grouped into four categories. The first contained 73 patients with NIDDM (26 men, 47 women; average age, 57.74 years). The second group contained 11 patients IDDM (4 men, 7 women; average age; 47.55 years). The majority of diabetic patients were examined within a 3-year span during a stay at the second internal medicine clinic at the faculty hospital of Kralovské Vinohrady. Some patients and information regarding their health state were provided by the neurological clinic in Bílina under the care of Dr. B. Beneš. Among the patients, we imposed the following parameters: individual duration of diabetes, individual drug therapy in use, body-mass index (BMI), systolic and diastolic blood pressures, maximal and minimal glucose levels during hospitalization, and the presence of diabetic complications (polyneuropathy, retinopathy, microangiopathy, and neuropathy). All such information was determined from patient documentation.

The third group contained a total of 12 obese patients without DM (4 men, 8 women; average age, 49.67 years). These patients visited the obesity clinic at the second internal medicine clinic at the faculty hospital of Kralovské Vinohrady on an outpatient basis.

The fourth group contained 29 control subjects. All information regarding these individuals was made available by the neurological clinic of the Bílina hospital. All were volunteers, some being hospital personnel. Specific information regarding all patients can be found in Table 1.

ELECTROGUSTOMETRIC EXAMINATION

The examination of taste sensation was performed using electrogustometry. The electrode instrument (5 mm) was attached constantly to the tongue: on the tip and middle portions and close to the radix and always

Table 1. Four Categories of Study Individuals

Subject Types	Number (%)	Age (yrs) Mean	Age (yrs) Standard Deviation
IDDM	11 (8.8)	47.55	16.06
NIDDM	73 (58.4)	57.74	13.99
Obesity	12 (9.6)	49.67	11.95
Control	29 (23.2)	25.62	9.49

on both sides of the tongue. All point area examinations were repeated. The electrode elicited a nonpainful, taste-stimulating impulse (1-sec duration), to be characterized by patients either as acidic or bitter or as a tingling similar to that of a battery charge to the tongue. Patients without any taste sensation disruptions were able to detect low-intensity current. Among patients with taste sensation disruption, we recorded a higher threshold value—electrogustometric threshold (EGT)—in response to current intensity. The spectrum of possible current intensities ranged from 0 to 500 μA. Examinations always were begun at low values and gradually increased the intensity until patients sensed the signal. Patients never were informed of, and could not determine externally, the intensity of current administered. Values documented as the EGT were considered as the lowest perceptible current intensity, that being always an average of both sides of the given region of the tongue. Thus, a lower EGT is proportional to better taste sensation. The duration of examination for each individual was approximately 10 minutes, which included an explanation of the test. A normal value (that found among healthy individuals) was taken to be an EGT recording of 40 μA, values above 100 μA were considered as marked hypogeusia, and values between 40 and 100 μA were considered borderline values. A value that exceeded 500 μA was considered as ageusia, meaning that a patient did not sense any impulse even at maximal levels.

RESULTS

All patient parameters are available in Table 2. The average number of years patients inflicted with NIDDM was 13 years. Of those subjects, 45% were treated conservatively through diet, 30% with oral antidiabetic therapy, 5% with a combination of oral therapy and insulin, and 20% with insulin alone. The average BMI value was 32.02 kg/m^2, average systolic pressure was 149.79, and diastolic pressure was 82.71 mm Hg. Average maximal glycemia concentrations during hospital stay were 14.93 mmol/liter, and minimal glycemia concentrations were 6.78 mmol/liter. Polyneuropathy was present in the majority of patients (51.2%); 37.5% had retinopathy, 12.5% had microangiopathy, and nephropathy was not followed up.

Table 2. Patient Parameters

Parameter	n	Mean ± σSD
Electrogustometry threshold (mA)		
DM 1	11	139.39 ± 119.14
DM 2	73	158.75 ± 148.42
Obesity	12	184.86 ± 193.37
Control	29	53.08 ± 14.10
Body mass index		
DM 1	5	22.94 ± 2.60
DM 2	32	32.02 ± 5.30
Obesity	7	39.59 ± 4.35
Control	—	—
Systolic pressure		
DM 1	3	136.67 ± 20.82
DM 2	24	149.79 ± 30.23
Obesity	5	142.00 ± 14.83
Control	—	—
Diastolic pressure		
DM 1	3	80.00 ± 10.00
DM 2	24	82.71 ± 10.53
Obesity	5	86.00 ± 5.48
Control	—	—
Maximum glycemia		
DM 1	8	19.23 ± 2.21
DM 2	40	14.93 ± 1.82
Obesity	—	—
Control	—	—
Minimum glycemia		
DM 1	8	4.08 ± 1.15
DM 2	40	6.78 ± 1.00
Obesity	—	—
Control	—	—

DM = diabetes mellitus.

The average number of years that patients were afflicted with IDDM was 20 years; all were treated with insulin. The average BMI value was 22.94 kg/m^2, average systolic pressure was 136.67, and diastolic pressure was 80.00 mm Hg. Average maximal glycemia concentration during hospital stay was 19.23 mmol/liter, and minimal glycemia concentration was 4.08 mmol/liter. Polyneuropathy was present in 37.5% of patients, 37.5% had retinopathy, 12.5% had microangiopathy, and nephropathy was not followed up.

Among those obese patients without diabetes, the average BMI was 35.59 kg/m^2, systolic blood pressure was 142.00, and diastolic pressure was 86.00 mm Hg. Average EGT values among the control group were 53.08 μA, among DM 1 patients 139.39 μA, among DM 2 patients 158.75 μA and among obese patients 184.86 μA (Fig. 1).

The results of patient electrogustometric examinations are displayed in Table 3. Patients are divided into

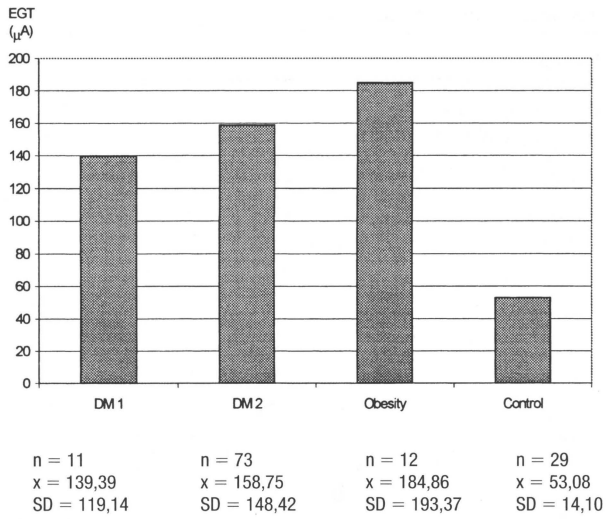

Figure 1. Patient distribution according to results of electrogustometric examination.

their primary categories: NIDDM, IDDM, obese, and controls. The results are expressed as a percentage of the patients of a given column. For example, during the measurement of EGT values in the area of the radix of the tongue, our NIDDM patients were subdivided so that 17% were found to have EGT values up to 40 μA (normal values), 37% had values between 41 and 100 μA (borderline values), 26% were in the range of 101 and 300 μA (hypogeusia), 12% occupied the range of 301 to 500 μA (major hypogeusia), and 8% were measured with ageusia. The patients in the other groups are evaluated in the same manner, as are specific tongue-field areas. As the table includes a large number of values, another table (Table 4) does not contain values divided according to specific tongue-field areas but only single values representing the average of all three areas (Fig. 2).

Table 3. Results of Electrogustometric Examinations

	Normal		Hypogeusia		Ageusia
EGT (μA)	**20–40**	**41–100**	**101–300**	**301–500**	**>500**
Near radix of tongue					
DM 2	17%	37%	26%	12%	8%
DM 1	18%	27%	37%	18%	
Obesity	33%	17%	17%	8%	25%
Control	14%	86%			
Middle					
DM 2	15%	39%	30%	12%	4%
DM 1	18%	46%	18%	18%	
Obesity	25%	25%	17%	17%	17%
Control	21%	79%			
Apex					
DM 2	20%	39%	29%	9%	3%
DM 1	27%	55%	9%		9%
Obesity	50%	33%		17%	
Control	28%	72%			

EGT = electrogustometric threshold; DM = diabetes mellitus.

Table 4. Patient Distribution According to Results of Electrogustometric Examination

	Normal		Hypogeusia		Ageusia
EGT (μA)	**20–40**	**41–100**	**101–300**	**301–500**	**>500**
DM 2	17%	38%	29%	11%	5%
	55%		40%		
DM 1	21%	43%	21%	12%	3%
	64%		33%		
Obesity	36%	25%	11%	14%	14%
	61%		25%		
Control	21%	79%			
	100%				

EGT = electrogustometric threshold; DM = diabetes mellitus.

DISCUSSION

Although electrogustometry is used widely, it is not a physiological test. The mechanism by which an electric stimulus elicits a taste sensation is not exactly clear. For this reason, chemical gustometry also exists, based on a physiological stimulus. Chemical gustometry uses four basic taste sensations (sweet, salty, acidic, and bitter), both in mixed and singular forms. It has been shown that diseases that affect taste sensation influence all four basic forms of taste. However, performing chemical gustometry is not easy, and its results depend on the subjective appraisal and nature of the individual being examined. Electrogustometry is simple, nonpainful,

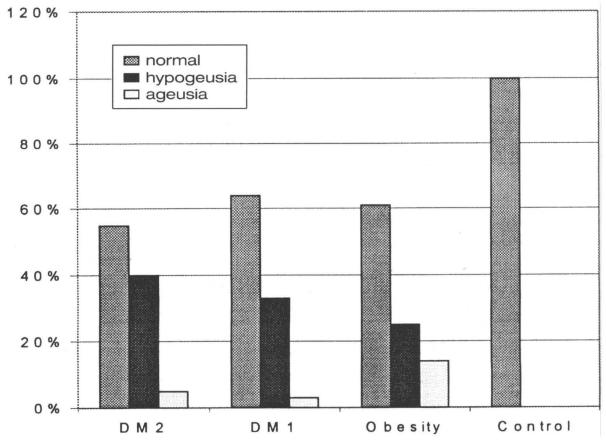

Figure 2. Mean figures of electrogustometric threshold as valid for given groups of patients.

easily performed, and reliable. The results of electrogustometry and chemical gustometry are very similar (although not consistently). The values of electrogustometry expressed as a threshold always increase relative to higher degrees of taste sensation degeneration [1].

The study performed by Le Floch et al. [1] proved that among diabetic patients, EGT values increased with the passage of time, whereas during the same period, the EGT values among the control group did not change. These results could not be explained in the context of gender, BMI, the consummation of alcohol, or cigarette smoking. To a certain degree, these results correlated with the incidence of hypertension and the use of antihypertensive drugs, which was more frequent among patients in the diabetic patient group. Generally, EGT values were not influenced by glycemia and glycosylated hemoglobin values with fasting at the time of examination. From this finding, a direct influence of blood glucose levels on taste can be said to be unlikely. More importantly, an association between EGT and degenerative complications was found. Similar degeneration in taste sensation, renal function, retinopathy, and neuropathy were found. This finding forces the consideration of a common instigating mechanism.

Perros et al. [6] studied patients with newly diagnosed NIDDM. As do other researchers in this field, they believed that peripheral neuropathy (including taste nerve tracts) and microangiopathy (in taste buds) may be responsible for the degeneration of taste sensation. They added that this mechanism is not likely among patients with newly diagnosed diabetes, because such patients do not have clinical signs of microvascular disruption. These authors suggested another hypothesis: that the taste receptor among NIDDM patients is defective (worsened taste was partially reversible). As another possibility, they supposed a modifying influence of refined sugar on taste. Affected patients were prescribed a diet reduced in these types of sugars as basic therapy. However, no correlation was found between measured taste parameters and the amount of sugar consumed. A change in oral flora may be another factor influencing taste, but no significant difference was found in this parameter [6].

In regard to the comparison of obese patients and control subjects, the majority of studies did not find any significant differences. However, none of the reviewed studies used electrogustometric examination. If gustometry was performed, it was the chemical form. Obese and healthy individuals showed no differences in the occurrence of the so-called sweet tooth, and those in that group do not seem to be more sensitive to external influences (tested saccharide taste and the smell of bitter almonds) [5].

The results of the study performed by Grinker et al. [7] are similar. They compared obese and normal control subjects, divided obese subjects into four categories (according to age and the time in which obesity emerged), and compared results between those in the groups. They did not find any differences in the ability to detect low fluid concentrations of saccharide, neither between the obese groups nor between obese and healthy patients. They further followed the influence of the color of sweet fluid on the ability to determined its sweetness (concentration). They used colorless and red-colored fluids, both having the same sugar concentration. Neither in this experiment did they find a difference between obese and healthy subjects. Both groups evaluated the red fluid as sweeter. These authors considered the presence of other possible factors among obese patients, applicable during feeding. These authors referred to an interesting finding by P. Dunker (1939) that white chocolate tastes less chocolate (less sweet) than does classic dark chocolate [7].

The determinations of Le Floch et al. [1] regarding useful EGT values in relation to the later occurrence of neuropathy is very important to us. Their specificity is very high, their sensitivity and ability to predetermine negative values is low. A great contribution is that with the use of electrogustometry as a routine screening method among diabetics, we can obtain information relevant to progressed but reversible future complications. In this way, we can identify early those patients who are in danger of future degenerative complications [1].

CONCLUSION

Our study showed that the gustometric threshold among DM and obese patients is significantly higher than that of patients in the control group ($p = .003$). This means that, as opposed to those in the control group, the DM and obese patients have a worse taste sensation. However, no significant differences were seen between members of the other patient groups (NIDDM, IDDM, and obese subjects).

Table 4 shows that 21% of the control group subjects had normal EGT values; the remainder (79%) had borderline values. The authors of other similar studies have assumed a 5% rate of hypogeusia among the healthy population [1]. None of the subjects in our control group could be included in this group, a restriction attributable to the relatively young average age of our control subjects. Fifty-five percent of the patients with NIDDM had normal or borderline values, 40% had major hypogeusia, and 5% had total ageusia, which is relatively high.

In comparison, 64% of the subjects in the IDDM group had values of normal or borderline. Hypogeusia was measured among 33% of these patients and (less frequently), ageusia was measured in 3%.

The obese patients were fairly nonhomogeneous. The number of patients with normal or borderline EGT values was 61%. Normal EGT values (up to 40 μA) was found among 36% in this group, which is higher than the control group. Major hypogeusia was found in 25% of the patients. Of all the groups, this group registered the highest number of ageusic subjects: 14%. However, a comparison to other published results cannot be performed, as all other reviewed literature regarding taste sensation was performed using chemical gustometry.

Evaluation of the general relationship between EGT and other factors shows that EGT correlates with age. This result was determined similarly by all other published studies. Furthermore, results correlated with the incidence of hypertension. This finding would confirm the hypothesis that hypertension and antihypertensive drugs may play roles as etiological factors in the development of taste sensation disruption. Results also correlated with the incidence of neuropathy but, as opposed to other published work, this correlation was not significant. Also, no correlation was found between the duration of diabetes; in other published work, this correlation sometimes could be made. The correlation between EGT and the duration of diabetes among NIDDM patients (see Fig. 1) is controversial, as the date of diagnosis does not necessarily have to correspond with the actual emergence of the disease.

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