Taste Disorders in Acute Stroke
A Prospective Observational Study on Taste Disorders in 102 Stroke Patients

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Background and Purpose—The aim of the study was to assess whether and how frequently patients with acute first-ever stroke exhibit gustatory dysfunction.

Methods—We performed a 1-year prospective observational study. Gustatory function was assessed using the standardized “taste strips” test. In addition, we assessed olfactory function, swallowing, stroke location, comorbidities, and the patients’ medication.

Results—A total of 102 consecutive patients were enrolled (45 female, 57 male; mean age, 63 years); 31 of them (30%) exhibited gustatory loss and 7 (6%) had lateralized impairment of taste function. Predictors of impaired taste function were male gender ($P=0.003$), high National Institutes of Health Stroke Scale (NIHSS) score at admission ($P=0.009$), coexisting swallowing dysfunction ($P=0.026$), and a stroke of partial anterior circulation subtype (PACS) ($P=0.008$). In particular, in hypogeusic patients the lesion was most frequently localized in the frontal lobe ($P=0.009$). Follow-up examinations in 14 patients indicated improvement of taste sensitivity.

Conclusion—Taste disorders after stroke are frequent. A significant association was found for male gender, high NIHSS score, swallowing disorder, and PACS, particularly in the frontal lobe. Generally, taste disorders after stroke seem to have a good prognosis. (Stroke. 2005;36:1690-1694.)

Key Words: stroke ■ stroke management ■ taste

Among the functions of the sense of taste is the regulation of various aspects of nourishment, including sociocultural aspects. Despite this significance there are only few reports on taste disorders in stroke patients. One reason for this lack of systematic investigations may be that until recently, there were no standardized, validated tests of natural gustatory function that would also be commercially available. It is also important to note that detailed taste testing is time-consuming and, during the acute phase of stroke, concern about taste is overshadowed by the patient’s other serious and even life-threatening medical problems.

The aim of the present prospective observational study was to investigate whether and how frequently taste disorders occur in unselected, cooperative, first ischemic or hemorrhagic stroke patients, and whether these findings are associated with the type of stroke, its location, and clinical findings. In addition, a subset of patients was investigated for recovery of gustatory function.

Patients and Methods
Between August 2002 and August 2003, a prospective observational study enrolled all patients who had had an acute first-ever stroke (<7 days since onset of signs and symptoms) and were conscious and medically stable. Pre-existing taste disorders were dismissed based on an extensive, structured history of the patients. In addition, all patients received the question, “Do you have trouble tasting salt, sweet, sour, bitter?,” which excludes presence of taste dysfunction in 94%. Additional exclusion criteria were a transient ischemic attack, coexisting dementia, pure cerebellar stroke, and severe aphasia in which patients were conscious but unable to cooperate.

Clinical Assessment
The consecutive patients received a detailed neurological examination through an experienced neurologist (J.G.H.) to establish a diagnosis of stroke, clinical syndrome, pathological and etiological subtype of stroke, and functional effects of stroke as measured by the National Institutes of Health Stroke Scale (NIHSS). On admission, all patients underwent neuroradiological examination, preferably computed tomography examination. In most cases, imaging was repeated during the course of disease—preferably magnetic resonance imaging including diffusion-weighted imaging—to investigate size and location of the lesion in all patients. Stroke subtype was classified according to the TOAST and the Oxfordshire Community Stroke Project (OCSP) classification criteria, including partial anterior circulation syndrome (PACS), posterior circulation syndrome, total anterior circulation syndrome, and lacunar circulation syndrome. In addition, a neuroradiological specialist blinded to the results from gustatory testing (B.T.) localized the lesion according to...
its predominant neuroanatomical location (frontal, parietal, temporal, or occipital lobe; subcortical/thalamic location; brain stem). The intake of drugs with possible effects on taste was screened according to Ackerman and Kasbekar.17

In addition, swallowing impairment was diagnosed as “present” or “not present.”18 A buccal neglect was excluded,19 and the premorbid level of activity status was established.20 Dementia was excluded using the Mini Mental State Examination.14

Gustatory and Olfactory Testing
Testing of the chemical senses was performed by a single observer (C.S.) at the same time of day within a median of 3 days (range, 0 to 17 days) after symptom onset. All tests were performed at least 1 hour after intake of foods or drinks other than water.

The taste test was based on filter paper strips.12 These strips of filter paper were impregnated with 4 concentrations of the 4 basic taste qualities. They were placed on the left or right side of the anterior third of the tongue, resulting in a total of 32 trials. Before each administration of a strip, the mouth was rinsed with water. The tastes were presented in increasing concentrations. Taste qualities were applied in a randomized fashion at each of the 4 levels of concentration. With their tongue still extended, patients were asked to identify the taste from a list of 4 descriptors plus carbonaceous, i.e., sweet (cake), sour (lemon), salty (salt shaker), and bitter (black coffee). The taste score was the sum of correctly identified strips. Normative values indicate taste impairment (normogeusia versus hypogeusia/ageusia) at scores <16 for subjects younger than 61 years and <14 in subjects older than 60 years.12

Olfactory function was assessed using the “Sniffin’ Sticks” test kit, which comprises 3 individual tests of olfactory function (odor threshold, discrimination, and identification). Scores of the 3 individual tests are summed to the “Threshold Discrimination Identification score,” which is a reliable means to estimate the degree of olfactory function.21

Controls
Despite the fact that normal values have been published for both olfactory and gustatory tests, we also examined healthy volunteers (n=24; 9 male, 15 female; median age, 63 years; range, 26 to 75 years) to re-investigate the proposed limits between normogeusia and hypogeusia/ageusia. None of these healthy controls had scores compatible with hypogeusia/ageusia or a marked side difference (left–right difference of >30% in taste score); the mean taste score was 21.5 (SD 4.7). All controls had normal olfactory function.

Follow-up Examinations
Follow-up examinations were performed 3 months after the incident in a subset of patients with hypogeusia (n=7) or normogeusia (n=7) diagnosed at the first visit.

Statistical Analysis
Associations between gustatory function and other variables were investigated using contingency tables and the χ² test. To evaluate the severity of the stroke, we compared the mean of NIHSS in both groups with the Mann–Whitney U test; t tests were used to compare means of unpaired samples. A forward logistic regression analysis was performed with gustatory function (normogeusia versus hypogeusia/ageusia) as the dependent variable. Variables in this analysis were selected from univariate analysis when they reached a level of significance of <0.10. Analyzed variables included age group, sex, major risk factors (arterial hypertension, current smoking, diabetes mellitus, and alcohol consumption), intake of drugs known to impair taste, type of stroke (hemorrhage versus ischemia), TOAST etiologic subtype, baseline NIHSS, olfactory function, and swallowing function. Whereas stroke location according to OCSP criteria was included in this logistic regression analysis, because of the small sample sizes of the subgroups, the frequency of hypogeusia and of normogeusia in relation to the neuroanatomical location of the stroke were analyzed using χ² tests. Results were considered significant at P<0.05. All statistical analyses were performed with the SPSS software 12.0 (SPSS Inc).

Results
A total of 102 consecutive patients were prospectively investigated; 4 additional patients were not included because of the assumption of a pre-existing taste disorder. The mean age of the sample was 63.3 years (SD 12.8 years); 57 patients were male and 45 were female. The stroke type was ischemic in 86 cases and hemorrhagic in 16 cases. The median NIHSS was 4, ranging from 0 to 13, ie, patients were slightly to moderately affected by the stroke. All subjects had a Mini Mental State Examination ≥23, with a mean of 28.3 (SD 1.7), excluding the presence of major cognitive dysfunction.

Distribution of the clinical stroke syndrome (classified according to OCSP criteria) was in 31 cases a PACS, in 32 cases a lacunar circulation syndrome, and in 39 cases a posterior circulation syndrome.16 In clinical tests, 12 patients exhibited a swallowing disorder, and 10 patients had olfactory dysfunction. Based on gustatory testing, 31 patients (30.4%) were hypogeusic/ageusic and 71 patients (69.6%) were normogeusic.

In Tables 1 and 2, the patients’ characteristics are presented separately for the 2 groups with “hypogeusia/ageusia” or “normogeusic,” respectively. Using a univariate analysis, a significant association was found for the factors male gender, NIHSS score, coexisting swallowing disorder, and olfactory function. For the OCSP classification criteria, P=0.079 was found.

Differences in the frequency of hypogeusia and normogeusia were found for the stroke location according to neuroanatomical criteria (P=0.009). When comparing individual neuroanatomical areas to all other areas, the frequency of hypogeusia was significantly higher after a frontal lobe lesion (frontal lobe lesion: 59% hypogeusia [16 of 27 cases]; lesions in all other brain areas: 20% hypogeusia [15 of 74 cases]; P<0.001).

Accompanying diseases (diabetes mellitus, arterial hypertension, or intake of drugs) or TOAST subtype, hemorrhagic subtype, premorbid level of activity, alcohol consumption, and smoking had no significant effect on gustatory or olfactory function. However, as listed in Table 3, the independent predictive variables for a taste disorder were male gender (odds ratio [OR], 0.17; confidence interval [CI], 0.055 to 0.54; P=0.003), NIHSS at admission (OR, 1.29; CI, 1.07 to 1.56; P=0.009), coexisting swallowing disorder (OR, 0.21; CI, 0.05 to 0.82; P=0.026), and PACS subtype in the OCSP classification criteria (OR, 0.19; CI, 0.05 to 0.65; P=0.008).

Lateralized differences in taste function were detected in 7 patients. In 2 patients the deficit was ipsilateral to a pontine lesion, in 2 patients contralateral to a supratentorial lesion, and in 3 patients ipsilateral to a supratentorial lesion.

In 7 hypogeusic and 7 normogeusic patients, follow-up examination was performed after 3 months. It revealed improvement of gustatory sensitivity (P=0.0001) (Figure 1). In 4 of these patients the lateralized taste dysfunction resolved completely.
Controls and both normogeusic and hypogeusic patients had the least difficulties to identify “sweet,” whereas “sour” was the most difficult to recognize (P = 0.001) (Figure 2).

**Discussion**

Only patients with first-ever stroke were included in the present study; another inclusion criterion was that patients were conscious and medically stable, and that they had no history of pre-existing gustatory dysfunction. A certain selection bias may relate to the fact that patients with severe stroke were not included. Accordingly, it is possible that the prevalence of taste disorder in stroke may be higher than reported in this study.

The present study revealed an association between male gender, swallowing disorder, NIHSS and PACS stroke subtype, and taste dysfunction. Previous research indicates that the decline in taste sensitivity with age is more severe in men than in women.22 Overall, men are less sensitive to taste stimuli than women.23,24 Thus, with regard to the risk factor “male gender” as identified in the present study, we hypothesize that men are more vulnerable to taste dysfunction and/or have less capacity to compensate acute loss of gustatory function. This is supported by the fact that gustatory sensitivity declines more steeply with aging in men than in women.22

The correlation between taste disorder and PACS stroke subtype may be because of anatomical reasons. PACS often includes patients with lesions in the territory of the middle cerebral artery. From fMRI it is known that cortical areas involved in the processing of gustatory stimuli are found in the insula, the parietal and frontal opercula, and also in the orbitofrontal cortex areas, all of which belong to the anterior circulation.25–28 The current literature also includes a number of case series on taste disorders related to stroke in the insular region3,29–31—a region belonging to the anterior circulation system. Our additional finding that in hypogeusic patients the frontal lobe was most frequently affected goes along with this finding.

### Table 1. Baseline Characteristics of Stroke Patients According to Taste Capacity (Evaluated by Taste Strips Test)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Hypogeusics</th>
<th>Normogeusics</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Totals, %</td>
<td>31 (30.4)</td>
<td>71 (69.6)</td>
<td></td>
</tr>
<tr>
<td>Male (n=57)</td>
<td>24</td>
<td>33</td>
<td>0.004</td>
</tr>
<tr>
<td>Age ≥60 y (n=63)</td>
<td>19 (61.3%)</td>
<td>44 (62%)</td>
<td>0.948</td>
</tr>
<tr>
<td>Stroke type, ischemic (n=86)</td>
<td>26 (83.9%)</td>
<td>60 (84.5%)</td>
<td>0.935</td>
</tr>
<tr>
<td>NIHSS score on admission, mean (SD)</td>
<td>4.81 (3.23)</td>
<td>3.39 (2.36)</td>
<td>0.015</td>
</tr>
<tr>
<td>Diabetes</td>
<td>10 (32.3%)</td>
<td>18 (25.4%)</td>
<td>0.472</td>
</tr>
<tr>
<td>Smokers</td>
<td>15 (48.4%)</td>
<td>25 (35.2%)</td>
<td>0.329</td>
</tr>
<tr>
<td>Alcohol abuse (&gt;60 g/d)</td>
<td>6 (19.3%)</td>
<td>8 (11.3%)</td>
<td>0.275</td>
</tr>
<tr>
<td>Premorbid level of activity, mean (SD)</td>
<td>0.65 (0.76)</td>
<td>0.63 (0.66)</td>
<td>0.939</td>
</tr>
<tr>
<td>Taking drugs with potential influence on taste</td>
<td>13 (41.9%)</td>
<td>20 (28.2%)</td>
<td>0.172</td>
</tr>
<tr>
<td>MMST</td>
<td>28.9 (1.49)</td>
<td>28.4 (1.76)</td>
<td>0.691</td>
</tr>
<tr>
<td>Swallowing disorder, yes</td>
<td>8 (25.8%)</td>
<td>4 (5.6%)</td>
<td>0.004</td>
</tr>
<tr>
<td>Smell capacity, abnormal</td>
<td>6 of 21 (28.6%)</td>
<td>4 of 33 (12.1%)</td>
<td>0.039</td>
</tr>
</tbody>
</table>

MMST indicates Mini Mental State Test.

### Table 2. Distribution of Stroke Causes (TOAST Criteria) and Stroke Location (Oxfordshire Community Stroke Project Criteria and Neuroanatomical Location)

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Hypogeusics</th>
<th>Normogeusics</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>TOAST criteria</td>
<td></td>
<td></td>
<td>0.206</td>
</tr>
<tr>
<td>Cardioembolic</td>
<td>2 of 31 (7%)</td>
<td>7 of 71 (10%)</td>
<td></td>
</tr>
<tr>
<td>Macroangiopathy</td>
<td>4 (13%)</td>
<td>1 (1%)</td>
<td></td>
</tr>
<tr>
<td>Microangiopathy</td>
<td>10 (32%)</td>
<td>30 (42%)</td>
<td></td>
</tr>
<tr>
<td>Concurrent etiology</td>
<td>5 (16%)</td>
<td>8 (11%)</td>
<td></td>
</tr>
<tr>
<td>Nondeterminable</td>
<td>5 (16%)</td>
<td>14 (20%)</td>
<td></td>
</tr>
<tr>
<td>Hemorrhagic cases</td>
<td>5 (16%)</td>
<td>11 (16%)</td>
<td></td>
</tr>
<tr>
<td>OCPS criteria</td>
<td></td>
<td></td>
<td>0.079</td>
</tr>
<tr>
<td>PACS</td>
<td>14 (45%)</td>
<td>17 (24%)</td>
<td></td>
</tr>
<tr>
<td>POCs</td>
<td>8 (26%)</td>
<td>31 (44%)</td>
<td></td>
</tr>
<tr>
<td>TACS</td>
<td>...</td>
<td>...</td>
<td></td>
</tr>
<tr>
<td>LACS</td>
<td>9 (29%)</td>
<td>23 (32%)</td>
<td></td>
</tr>
<tr>
<td>Neuroanatomical location</td>
<td></td>
<td></td>
<td>0.009</td>
</tr>
<tr>
<td>Frontal lobe</td>
<td>16 of 31 (52%)</td>
<td>11 of 71 (15%)</td>
<td></td>
</tr>
<tr>
<td>Parietal lobe</td>
<td>1 (3%)</td>
<td>12 (17%)</td>
<td></td>
</tr>
<tr>
<td>Temporal lobe</td>
<td>1 (3%)</td>
<td>5 (7%)</td>
<td></td>
</tr>
<tr>
<td>Occipital lobe</td>
<td>1 (3%)</td>
<td>9 (13%)</td>
<td></td>
</tr>
<tr>
<td>Subcortical/thalamic</td>
<td>6 (19%)</td>
<td>16 (23%)</td>
<td></td>
</tr>
<tr>
<td>Brain stem</td>
<td>6 (19%)</td>
<td>18 (25%)</td>
<td></td>
</tr>
</tbody>
</table>

OCPS indicates Oxfordshire Community Stroke Project; POCs, posterior circulation syndrome; TACS, total anterior circulation syndrome; LACS, lacunar circulation syndrome.

### Table 3. Results of Logistic Regression Analyses

<table>
<thead>
<tr>
<th>Variable</th>
<th>Regression Coefficient</th>
<th>OR</th>
<th>95% CI</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>1.753</td>
<td>5.77</td>
<td>1.85–18.03</td>
<td>0.003</td>
</tr>
<tr>
<td>NIHSS at admission</td>
<td>0.252</td>
<td>1.29</td>
<td>1.06–1.56</td>
<td>0.009</td>
</tr>
<tr>
<td>Swallowing disorder</td>
<td>−1.607</td>
<td>0.20</td>
<td>0.05–0.82</td>
<td>0.026</td>
</tr>
<tr>
<td>PACS subtype</td>
<td>−1.685</td>
<td>0.19</td>
<td>0.05–0.65</td>
<td>0.008</td>
</tr>
</tbody>
</table>

Dependent variable: taste disorder measured by taste strips.
point, because the insular, frontal opercular, and orbitofrontal cortices are part of the frontal lobe. Additionally, this is supported by the present finding that gustatory loss was associated with higher scores in the NIHSS. Higher scores in the NIHSS are most often the consequence of supratentorial lesions. This is particularly so if the motor cortex is affected, because motor function strongly influences the NIHSS scoring.14

In 2 patients with supratentorial lesion, hypogeusia was contralateral to the lesion, and in 3 patients with supratentorial lesion hypogeusia was ipsilateral to the stroke. Considering the role of the hemispheres in gustatory processing, the current literature indicates that gustatory function has a bilateral representation.4,11,27 Although this concept makes it difficult to understand how a unilateral lesion can produce general taste loss, examples of this have been reported previously.31 Thus, apart from the bilaterally ascending pathways, interhemispheric transfer of gustatory function has to be considered, which may contribute significantly to overall gustatory function. This idea if supported by the present data, which also suggest that, as a rule, unilateral lesions result in a significant change in overall gustatory function, whereas only in a small number of patients could lateralized differences in taste function be found.

Swallowing disorders exhibited a significant association with hypogeusia. In fact, 23% of hypogeusic patients had a swallowing disorder, whereas this was found in only 6% of normogeusic patients; 67% of patients with swallowing disorder also had a taste disorder. We offer 2 possible explanations for this association. First, although the swallowing pathway and the gustatory pathway are anatomically separate, their function is integrated. Gustatory stimuli evoke a complex, multiple-level recruitment in pharyngeal swallowing. The impairment of swallowing and taste may be a consequence of the disturbance of such an oropharyngeal reflex.52 A second point may relate to the idea that the swallowing disorder impairs the self-cleaning of the oral cavity, although not visible to the investigators. This might either impair the contact between tastants and taste receptors or produce desensitization of gustatory receptors.

The finding of an association between taste disorder and olfactory dysfunction may be explained through the role of the frontal cortex in olfaction. In fact, all 3 patients with frontal lobe lesion with olfactory dysfunction also exhibited a coexisting taste disorder. Numerous reports suggest that the orbitofrontal cortex is a tertiary center for the integration of smell and taste, so that it is easily understandable that lesions may affect both chemosensory systems.26

The clinical impact of taste disorders concerns the intake of food. In acute situations, if taste capacity is impaired, spoiled and potentially toxic foods are not easily recognized and may be ingested. In the long-run, taste impairment can lead to both abnormal weight loss7-3 and obesity because of an increased intake of foods rich in calories. It has been reported that individuals with chemosensory deficits are often more obese than individuals with normal chemosensation.33 A mechanism behind this may relate to an altered preference for sweets (with consequently higher caloric intake) and a reduced intake of food with sour or bitter taste such as found in fruits, vegetables, and herbs.39 This is supported by studies indicating that the perception of “sweet” remains relatively stable with aging compared with other taste qualities, particularly “bitter.”723,24,34 Although the preservation of “sweet” during aging may relate to its significance as an indicator of foods with high nutritional values, gustatory alterations can
lead to a change in food preference and, consequently, to the impairment of eating-related activities such as cooking, visiting a restaurant, or considering eating as a social act.

Extensive experiments in healthy subjects indicate that the taste test used produces no significantly different results when applied repeatedly. Thus, it is unexpected that normogeusic patients exhibited an increase in taste scores on retesting. However, given the fact that gustatory test scores of the patients with normogeusia diagnosed were, on average, in a relatively low range, it may be considered that the stroke per se produced a general, nonspecific effect on taste function that resolved during recovery from the stroke. Such a nonspecific effect can, for example, be caused by certain depressive symptoms that are present in approximately half of the stroke patients but were not addressed specifically in our study. These effects appeared to be on a comparable scale in patients with initial normogeusia and hypogeusia, respectively.

To conclude, results from the present study suggest that taste disorders in acute stroke are frequent. Taste disorders should be considered in relation to nutrition after stroke.

Acknowledgment

We thank Dr Christian Mueller for his help with the “taste strips.”

References

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Stroke. 2005;36:1690-1694; originally published online July 7, 2005;
doi: 10.1161/01.STR.0000173174.79773.d3

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

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