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CT of the Brain in Taste and Smell Dysfunction

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Three hundred fifty-four patients with taste and/or smell disorders were evaluated with computed tomography (CT). The largest group was characterized by head trauma (27%), followed by idiopathic causes (26%), postinfluenza-like hyposmia and hypogeusia (15%), and congenital etiologies (14%). Hyposmia and hypogeusia occurred concomitantly in 21%–45%, the percentage varying according to etiologic subgroup. CT abnormalities were found in 108 (31%) of the 354 patients. The most frequent pathologies were frontal encephalomalacia, subfrontal atrophy in the region of the olfactory bulbs, and anterior temporal lobe atrophy. These changes were found alone or in tandem. Some CT findings suggest common cerebral taste and smell centers and common neural pathways and association centers.

Although general information about neural pathways involving cranial nerves and, specifically, about their central nervous system pathways for taste and smell has been available for both humans and lower animals for some time, much detailed information is lacking, particularly for humans. The results of this study, which associates specific taste and smell disorders with observed pathology, may be useful in supplying additional information about these pathways.

It is estimated that taste and smell disorders affect well over one million patients annually in the United States [1]. Disease entities that are manifested as taste and smell disorders may involve the sensory receptors, the cranial nerves serving the system, or the brain itself. This paper focuses on cranial computed tomographic (CT) abnormalities seen in patients who were referred to the CT laboratory from the Georgetown University Center for Molecular Nutrition and Sensory Disorder. We shall elicit those CT changes that various taste and smell dysfunction categories share.

Materials and Methods

Three hundred fifty-four patients with taste and/or smell disorders were referred to the CT laboratory since 1978. Referral was made only for those patients in whom a CT-depictable structural change was either expected or had to be excluded on the basis of the history. Subdivided by etiology, the largest group was characterized by head trauma (27%), followed by idiopathic causes (26%), postinfluenza-like hyposmia and hypogeusia (15%), and congenital etiologies (14%). Other less frequent etiologies included allergic rhinitis, postoperative dysfunction, and cerebrovascular accidents. Hyposmia and hypogeusia often occur simultaneously. This was most common in the head trauma and the postoperative groups (45%), but less common in the remaining categories (21%). Taste and smell functions were evaluated by a standard three-stimulus forced-choice drop technique (for taste) and sniff technique (for smell) [2].

Most of the CT scans were obtained on a Philips 310 scanner. For efficiency, only contrast-enhanced scans were obtained. First, three contiguous slices were obtained through the floor of the anterior fossa at an angle 20° to the Reid base line. A series of equally angled axial scans was obtained, covering the entire brain, followed by coronal sections through the facial bones and anterior fossa. Slice thickness was 6 mm.

Results

Of the 354 patients evaluated, 108 (31%) had positive or abnormal CT findings, and 246 (69%) had negative CT findings. The largest percentage (53%) of positive scans or abnormal findings was found in the group characterized by head trauma. The various cerebral CT abnormalities are listed in table 1 according to the etiologic subgroups in which they occurred.

The most frequent pathologies, located in the frontal and/or temporal lobe regions, were unilateral or bilateral subfrontal atrophy and unilateral or bilateral temporal lobe encephalomalacia (fig. 1). While each of these could present as solitary abnormalities, combined defects were not infrequent. Subfrontal atrophy was either diffuse, involving the entire subfrontal region, or more localized in proximity to the cribriform plate, including the territory around the olfactory bulb with gyrus rectus (fig. 2) and the neighboring frontal-lobe white matter. The disease process sometimes extended posteriorly toward the posteromedial surface of the frontal lobe, known as the paraolfactory area. Some patients showed pathologic changes at the base of the brain, adjacent to the optic chiasm, in the region of the anterior perforated substance (fig. 3). This is an alleged relay station for olfactory stimuli.

Temporal lobe atrophy was usually anteriorly located, adjacent to the greater wing of the sphenoid, and occasionally included the anteromedial portion with uncus and hippocampal gyrus. Isolated temporal atrophy or encephalomalacia was infrequent and occurred more often in combination with frontal or subfrontal abnormalities.

The majority (53%) of the 97 patients who developed taste and/or smell dysfunction as an immediate or delayed result of head trauma had CT abnormalities. In this group, CT usually images the late effects of brain injury, namely focal atrophy with compensatory widening of the adjacent cerebrospinal fluid space. In 13 patients, (sub)frontal pathology was accompanied by temporal lobe atrophy or encephalomalacia. In only three cases did temporal lobe pathology exist alone.

The group characterized by idiopathic taste and smell disorders was quite diverse. Only 18% of the CT studies in these 90 patients

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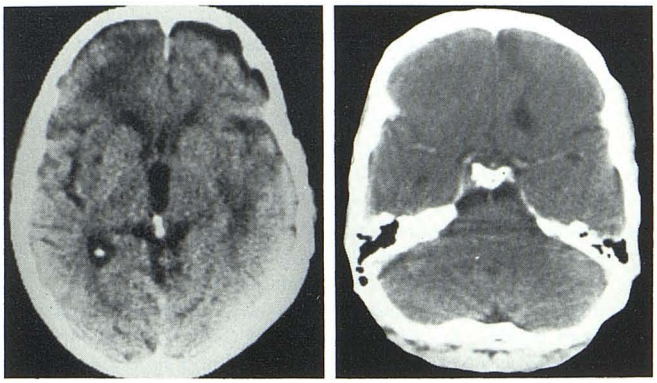
TABLE 1: **Cerebral CT Abnormalities in Patients with Taste/Smell Dysfunction**

CT Findings	Etiology of Dysfunction						Totals (n = 354)
	Head Trauma (n = 97)	Idiopathic (n = 90)	Postinfluenza (n = 52)	Congenital (n = 48)	Postoperative (n = 11)	Other (n = 56)	
(Sub)frontal or combined (sub)frontal and (sub)temporal pathology	37	8	4	1	4	5	59
Temporal pathology only	3	1	...	...	...	3	7
Temporal arteriovenous malformation	...	...	...	1	...	...	1
Thalamic infarct	...	1	...	...	1	...	2
Thalamic calcification	...	1	...	...	...	...	1
Diffuse frontal atrophy	...	1	...	...	...	...	1
Frontal calcification	...	...	...	1	...	...	1
Parietal encephalopathy	...	...	...	...	...	1	1
Scattered encephalomalacia	...	1	...	...	...	...	1
Generalized atrophy	2	1	...	2	...	1	6
Ventricular enlargement	...	...	1	...	...	...	1
Probable multiple sclerosis	...	...	...	1	...	...	1
Meningioma	...	1	...	...	...	...	1

Note.—Noncerebral CT abnormalities are not tabulated.



A B

Fig. 1.—Contrast-enhanced CT in two cases of posttraumatic encephalomalacia. **A,** Diffuse involvement in right frontal and left temporal lobe. **B,** Focal involvement of inferior left frontal lobe.

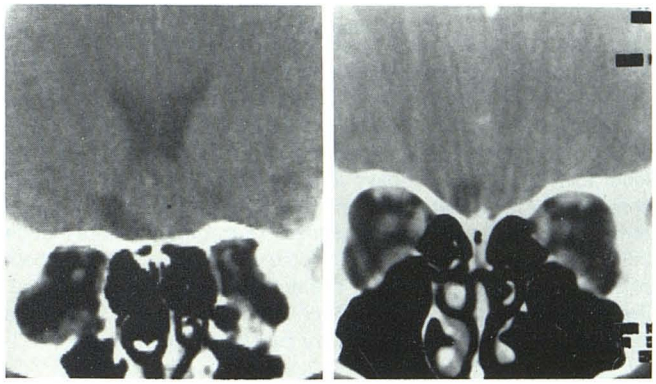


Fig. 2.—Contrast-enhanced CT in two cases with posttraumatic subfrontal atrophy around right olfactory bulb and encephalomalacia of adjacent frontal lobe.

were positive, and the CT findings were as wide-ranging as the clinical histories. The most frequent CT changes were seen in the frontal lobe and/or the vicinity of the olfactory bulb. Frontal lobe encephalomalacia and/or subfrontal atrophy occurred in eight patients in this group. Other abnormalities are listed in table 1. A 43-year-old man who had a 3 year history of gradual onset of hyposmia without any other complaints proved to have a huge olfactory groove meningioma (fig. 4). A thalamic infarct and a posterior thalamic calcification were also seen in this group.

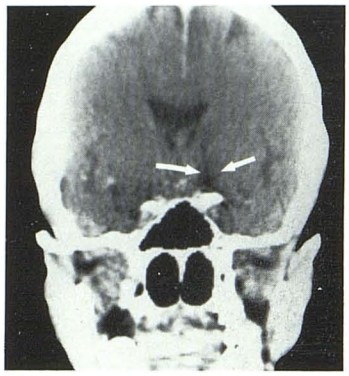
The low diagnostic efficiency of CT in the group characterized by congenital taste and smell dysfunction was somewhat surprising, as we expected to find a higher incidence of congenital cerebral abnormalities. In contrast, the group representing postinfluenza-like hyposmia and hypogeusia was not expected to (and in fact did not) yield a large number of cerebral pathologies.

The group representing other (miscellaneous) etiologies of taste and smell dysfunction included four patients with previous cerebrovascular accident. Three of these had positive CT scans, in each case showing an old temporal lobe infarct located in the region of the sensory cortex, close to the central fissure. Two patients with central nervous system sarcoidosis failed to demonstrate significant CT changes [3]. One, a 42-year-old woman, was diagnosed as having sarcoid arachnoiditis. The second patient, a 30-year-old woman who had hyposmia and dysgeusia, was found on autopsy to have granulomatous basal meningitis with granulomas infiltrating the olfactory bulbs and tracts. A 30-year-old woman had hyposmia after a spontaneous gyrus rectus hemorrhage (fig. 5), presumably secondary to a cryptic arteriovenous malformation.

Discussion

Our study confirms some of the known taste and smell centers [4–6]. Pathologies at and around the olfactory bulb, including the gyrus rectus, were among the most common in this series. The posteromedial portion of the frontal lobe with its medial olfactory areas was found to be abnormal, as was an area of the brain containing the perforated substance. Presumed olfactory terminals such as the uncus with amygdaloid nucleus and hippocampal gyrus were found to be involved in the disease processes as suggested by the CT appearance of corresponding anatomic regions. The posterior thalamus, a relay station for gustatory stimuli, showed abnormal calcification in one case of hypogeusia (fig. 6).

Other findings in this series suggest that current views on taste and smell are limited and subject to review. For example, the frequent involvement of the frontal lobe without associated changes in the olfactory region implies that it plays a more prominent role in



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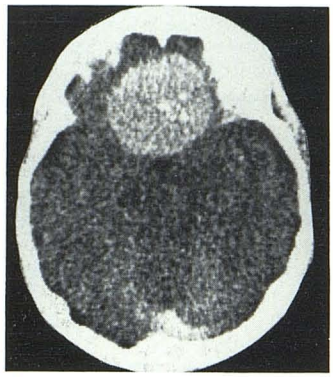

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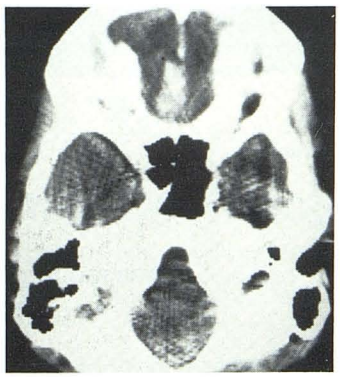
Fig. 3.—Idiopathic taste and smell dysfunction. Contrast-enhanced CT. Vertically aligned, linear encephalomalacia extends toward anterior clinoid, presumably the region of anterior perforated substance (*arrows*).

Fig. 4.—Contrast-enhanced CT in case of hyposmia and hypogeusia attributable to olfactory bulb meningioma.



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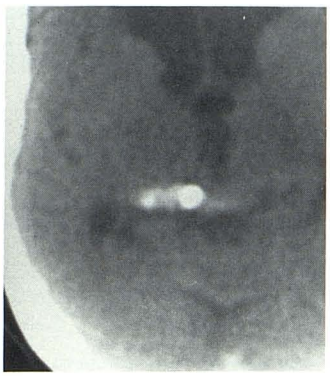

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Fig. 5.—Contrast-enhanced CT in case of hyposmia secondary to spontaneous gyrus rectus hematoma.

Fig. 6.—Idiopathic hypogeusia. Contrast-enhanced CT shows calcification of posterior right thalamus.

olfactation than previously postulated. Combined taste and smell loss in a number of cases with frontal encephalomalacia leads us to conclude that the frontal lobe also participates in the processing of gustatory stimuli. This conflicts with the traditional concept, which locates taste perception in the parietal operculum. Recent temporal-lobe ablation studies [2] have already challenged that theory by showing that the anterior temporal lobe is involved in discrimination of both olfactory and gustatory stimuli. Damage to this area alone could explain dual sensory loss in some cases. Finally, the concept of a combined taste and smell center was previously entertained, but later abandoned [5]. Some of our findings would seem to indicate that such a unitarian theory may be viable after all.

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