flow transducer placed around it for measurement of coronary blood flow. A 1 mm diameter teflon catheter was introduced into a small branch of the anterior descending coronary artery for local injection of drugs. A left atrial catheter was used for adenosine infusions. In the first part of each experiment, it was essential to show that dipyridamole and lidoflazine enhanced adenosine induced coronary vasodilatation, while aminophylline had the opposite effect. This was usually accomplished using the following dosages: dipyridamole 0.05 mg, lidoflazine 0.4 mg (both infused directly into the coronary circulation), aminophylline 200 mg i.v. and 1.0 mg adenosine injected into the left atrium. Reactive hyperemia was produced by temporary 60 sec occlusions of the anterior descending artery. Following control studies, occlusions were repeated after intracoronary administration of either dipyridamole or lidoflazine. Aminophylline was given i.v. All drugs were given in the same dosage as for the adenosine experiments.

The results of these experiments are summarized in the Figure. Following the infusion of dipyridamole, enhancement of both reactive hyperemia and adenosine induced coronary vasodilatation occurred ($p < 0.001$). Lidoflazine enhanced the coronary response to infused adenosine ($p < 0.001$) but no significant change ($p > 0.5$) occurred in the reactive hyperemia response. Although able to diminish adenosine induced vasodilatation ($p < 0.001$), aminophylline failed to produce a change in myocardial reactive hyperemia ($p > 0.3$).

The mechanism by which these drugs influence coronary vasodilatation produced by adenosine is unknown. It has been suggested that dipyridamole and lidoflazine prevent the uptake by the myocardium of infused adenosine[8] and that dipyridamole inhibits the degradation of adenosine in myocardial tissue[9]. These actions might explain the observed effects of these two drugs. Caffeine has been shown to antagonize the cardiac effect of adenosine[10], a property which may well be shared by aminophylline and other xanthines.

The provisional conclusion drawn from these experiments is that myocardial reactive hyperemia in the dog is not solely mediated by adenosine[11].

Résumé. Le dipyridamol, la lidoflazine et l'aminophylline modifient la vasodilatation provoquée par l'adénosine. Le dipyridamol seul augmente l'hyperhémie réactionnelle et les résultats ne confirment pas l'hypothèse que l'adénosine peut être un médiateur de l'hyperhémie réactionnelle.

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Haloforms: Sweet Taste or Smell?

Haloforms are important compounds in elucidating the initial chemistry of sweet taste and 'smell'. Many subjective reports indicate that as a class these compounds taste sweet[1,2], but they also have been reported to have a 'sweet' odor. Thus, there may be some confusion as to whether haloforms taste sweet, smell sweet or both. In this sense haloforms may serve as a focal point for distinguishing the initial chemistry of gustatory and olfactory responses.

The purpose of this report is to separate the gustatory and olfactory responses to haloforms in the absence of one or the other of these sensory modalities. To do this, we have recorded the gustatory and olfactory responses to haloforms of patients with abnormalities of taste, smell or both of these sensory modalities. These patients have either anosmia[3], Type I hyposmia[4], Type II hyposmia with dysosmia and dysgeusia[5,6] or aglycogeusia[7].

The subjects of this study were 1 patient with anosmia (i.e., a patient who was unable to detect or recognize any vapor using primary or accessory areas of olfaction), 5 patients with Type I hyposmia (i.e., patients who were unable to detect or recognize vapors using their primary olfactory area but who had intact accessory areas), 2 patients with Type II hyposmia with dysosmia and dysgeusia (i.e., patients who had quantitatively decreased olfactory acuity at the primary olfactory area with hypogeusia and an associated abhorence toward various odorants and tastants) and 1 patient with aglycogeusia (i.e., a patient who was unable to recognize the taste of any sweet substance).

Detection and recognition thresholds were determined for representatives of each of four taste qualities (NaCl for salt, sucrose for sweet, HCl for sour and urea for bitter) and for chloroform, bromoform and iodoform by a modification of a forced choice, 3 stimulus drop tech-

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Responses to tastants and odorants in normal subjects and in patients with various gustatory and olfactory abnormalities

Subjects	Taste			Smell		
	Detection thresholds	Recognition thresholds	Haloform response	Detection thresholds	Recognition thresholds	Haloform response
Normal	normal[a]	normal	sweet[c]	normal[a]	normal	sweet[a]
Anosmia	normal	normal	sweet	absent	absent	no response
Type I hyposmia	normal	normal	sweet	increased[b]	increased or absent	no response
Type II hyposmia with dysosmia and dysgeusia	increased[b]	increased	sweet	increased	increased	unpleasant
Aglycogeusia	normal	normal (except absent sweet response)	bitter	normal	normal	unpleasant

[a] Detection and recognition of the taste of solutions of NaCl and sucrose (3–60 mM/L), HCl (0.5–6 mM/L) and urea (60–150 mM/L) and the vapor of solutions of pyridine in water (10^{-3}–10^{-8} M/L), nitrobenzene in mineral oil (10^{-2}–10^{-7} M/L) and thiophene in mineral oil (10^{-3} to 10^{-7} M/L) are normal. [b] Inability to detect or recognize NaCl or sucrose at or below 60 mM/L, HCl, at or below 6 mM/L, or urea, at or below 150 mM/L represents an increased taste threshold; inability to detect or recognize pyridine or thiophene at or below 10^{-3} M/L or nitrobenzene, at or below 10^{-2} M/L represents an increased smell threshold. [c] Placement of a drop of absolute chloroform, bromoform or crystals of iodoform in the oral cavity or inhalation of these vapors normally produces a sensation of sweetness.

niques previously described[8,9]. Detection and recognition thresholds were also determined for the vapors of pyridine, nitrobenzene, thiophene, chloroform, and bromoform and iodoform by a modification of a forced choice, 3 stimulus sniff techniques previously described[10].

Detection and recognition thresholds for each of the taste qualities tested in the patients with anosmia or with Type I hyposmia were within normal limits. Chloroform, bromoform and iodoform tasted sweet to each patient (Table). None of these patients were able to detect or recognize any of the vapors presented as was observed in most of the patients with this abnormality studied previously[4,5]. Even absolute solutions of chloroform, bromoform and iodoform were not appreciated as having any distinct, describable odor.

Detection and recognition thresholds for each of the 4 taste qualities tested were elevated above normal in the patients with Type II hyposmia with dysosmia and dysgeusia, as were their thresholds for the vapors of pyridine, nitrobenzene and thiophene. However, they stated that absolute solutions of chloroform, bromoform and iodoform each smelled unpleasant, similar to that of isopropyl rubbing alcohol[3].

Detection thresholds for each of the 4 taste qualities tested were within normal limits in the patient with aglycogeusia[7]. Recognition thresholds for 3 taste qualities, except sucrose and all other sweet tastants, were also within normal limits[7]. Absolute solutions of chloroform, bromoform and iodoform were described as tasting bitter. Detection and recognition thresholds for pyridine, nitrobenzene and thiophene were within normal limits in this patient; however, he described the vapors of chloroform, bromoform and iodoform as unpleasant, similar to that of isopropyl rubbing alcohol.

These results indicate that haloforms elicit a sweet taste in the absence of olfactory responsiveness. In the absence of a gustatory system capable of responding to sweet tastants they elicit a bitter taste and an unpleasant smell; i.e., patients with aglycogeusia cannot obtain information about sweetness by either gustation or olfaction. These data indicate that haloforms can elicit both a sweet taste and a sweet smell but these responses can occur independently.

These results also suggest that the sweet taste elicited by absolute solutions of haloforms introduced into the mouth is a very penetrating sensation for in patients who exhibit distorted responses for many tastants and odorants these compounds were appreciated appropriately as having a sweet taste. In spite of this, these patients state that haloforms had an unpleasant smell. Treatment of these patients with this disorder with various metal salts eliminated this distortion of smell and the vapor of each haloform was then described as sweet[6].

One interpretation of these gustatory and olfactory responses to haloforms as sweet suggests the possible presence of a two-site olfactory receptor which allows for the recognition of olfactory specificity in a manner similar to that proposed for the gustatory system[2,7]. Presumably, the sweet taste of each haloform arises from the interaction of its rigid AH, B system with a complementary receptor site in the taste bud. By analogy, interaction of the haloforms with some complementary receptor site in the olfactory system could possibly be involved with the perception of their characteristic odor. Since there is such a consistent similarity in responsiveness to the haloforms with respect to their taste and smell it is tempting to speculate that there may be some similarity in the chemical properties eliciting these responses[10].

Zusammenfassung. An Patienten mit Anosmia, Hyposmia, Hypogeusia oder Agylcogeusia, resp. mit klinischen Ausfällen im Bereich des Geruchs- und Geschmacksinnes, wird die Perzeption der chemischen Stoffgruppe der Haloforme untersucht und gezeigt, dass Chloroform, Bromoform und Jodoform unabhängig voneinander süssen Geschmack und Geruch hervorrufen können.

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