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Olfactory dysfunction

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SUMMARY

Otolaryngologists, neurologists and other medical practitioners are often not well equipped for assessing olfactory (dys)function. They either use no or inadequate olfactory tests. This problem of inadequate olfactory testing was systematically attacked by American psychologists in the early 80's and led to the construction of odour identification tests which are easy to administer. Combining the advantages of two of these American tests we developed a Dutch odour identification test (GITU), consisting of two subsets of 18 natural odourants and applicable in two ways: one for use in the ENT clinic, the other for industrial purposes. The first results of this test indicate that the incidence of serious olfactory disorder among adults in the Netherlands may be conservatively estimated at about 1%. The GITU readily discriminates between patients and controls and is sensitive to variables known to affect olfaction (gender, age).

The recognition of olfactory dysfunction as a major problem has led in the U.S.A. to the establishment of clinical research centers for the study of human chemoreception. Evaluation results of four of those clinics together with data of three more case series - with a total number of patients of 4000 - show that two thirds of all patients fall into three etiological categories:

1. *Nasal disease and/or paranasal sinus disease*
2. *Viral infection of the upper respiratory pathway*
3. *Head trauma.*

For each of the three categories the literature is reviewed in order to arrive at a clearer picture of the olfactory patient with respect to age, gender, degree of olfactory deficit, spontaneous recovery, effectiveness of therapy and localization of the defect along the olfactory pathway. Finally an appeal is made to clinicians with interest in the subject to exchange more information with research scientists in olfaction. Such exchange is considered essential to making progress in this field.

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INTRODUCTION

Disorders of the sense of smell have received little clinical attention in contrast to disorders of other special senses like audition and vision. This minimal attention seems to be related to a variety of conditions: a relatively low awareness of the average lay person of his own olfactory impressions, poor understanding of the basic olfactory transduction and coding processes, lack of a widely accepted odour classification, absence of suitable instruments for measuring olfactory (dys)function and - maybe as a consequence of all this - a widespread lack of interest in this subject on the part of many medical practitioners.

The latter fact is mirrored by the outcome of a questionnaire about the use of clinical tests among the ENT clinics of 152 leading Japanese hospitals and institutes (Yoshida, 1984). From the 41 responding clinics only 22 gave information on number and kind of the odourants used. Thirteen of these clinics regarded three or less odourants sufficient for a clinical diagnosis of smell disturbance. Cain and Gent (1986), comparing the recommendations for testing the sense of smell clinically in seven neurology manuals, also found that using a small number of odourants, commonly three or four, is usually advised. In such test situations the subject is presented with the odourants and asked to name them. Unfortunately, there is a surprising inability of the average person to name odours with some precision. Sumner (1962) who tested this ability of odour identification by asking 200 people with an apparently normal sense of smell to name 12 odours, most of which were recommended by neurology manuals of the time, got quite disappointing results: average performance equalled 50%. He therefore considered such a test unacceptable for evaluating olfactory functioning. Another, quite different complaint about odourants in current neurological use was voiced by Pinching (1977). He pointed at the ubiquity of moderate to strong trigeminal stimulants provided for testing the first cranial nerve.

The problem of poor performance in odour identification was systematically attacked by the American psychologist Cain (1979), who advised to use commonly encountered substances whose odours have longstanding connections with their verbal labels and to give aid in recalling these labels. Combination of these recommendations would lead to ready and almost perfect odour identification. To avoid a preponderance of trigeminal stimulation the best strategy seemed to choose odourants of a reasonably wide qualitative range (Cain and Krause, 1979). The final version of Cain's identification test contains the following ten items: baby powder, chocolate, cinnamon, ground coffee, mothballs, peanut butter, Ivory soap, Vicks inhaler, wintergreen and ammonia (Cain et al., 1983). The last three items were included to establish the presence of normal trigeminal sensitivity. The substances are presented in opaque plastic jars with gauze placed over them, precluding visual identification. The subjects respond to each of these items by choosing an odour name from a list of twenty

descriptors. The total olfactory scores run from zero to seven. This identification test is said to grade olfactory functioning into three categories: normosmia, hyposmia and anosmia (Cain, 1982). A score that reflects the combined outcome of the identification test and a threshold test using various concentrations of n-Butanol is used to indicate five categories of functioning: the hyposmic category is split up into mild, moderate and severe (Cain et al., 1983). This additional claim of resolution power has been challenged, however (Heywood and Costanzo, 1986).

A second odour identification test developed by another American psychologist was presented in 1984: the University of Pennsylvania Smell Identification Test (UPSIT). This identification test uses far more items. It consists of 40 microencapsulated fragrances which are released by scratching odour-impregnated test booklets. Each page contains an odourant strip with four alternative responses (Doty et al., 1984a). A subject's total score can range from 0 to 40. Although the UPSIT was originally delivered to detect gross olfactory dysfunction, it has been demonstrated to detect a number of more subtle olfactory problems such as some cases of hyposmia and parosmia (Doty et al, 1984b). The UPSIT is not universally applicable but shows a certain cultural dependency (Zusho et al., 1983: Doty et al., 1985).

Odour identification tests have shown to be sensitive to variables known to affect olfaction such as gender and age (Doty et al., 1984c; Cain et al., 1988).

THE UTRECHT ODOUR IDENTIFICATION TEST (GITU)

A short description of the development of this dutch odour identification test is given below, an extensive description of the development will be published elsewhere.

Test development took place in three stages. In stage one a great number of current products from every day's life was selected as odorous substances. These natural odourants were preferred to the more artificial microfragrances because it has been shown that they are easier to identify (Cain and Krause, 1979). As subjects served 15 male (mean age 23.1 yr; sd 2.0; range 20–26) and 15 female (mean age 23.7 yr; sd 3.3; range 18–30) students. The substances were presented to the subjects in black plastic jars with a covering gauze preventing visual inspection. For each substance they were asked to smell it attentively, rate it for familiarity and find the right name for it. On the basis of the ratings for familiarity and percentages of correct identification 36 substances were selected for further experimentation. These are listed in Table 1.

In the second stage two experiments were carried out (conditions I and II). For both conditions the selected 36 odourants were divided into two groups of eighteen. The rationale behind this was to create two equivalent subtests that could serve as pre- and posttreatment tests. In condition I for each subtest a list of

Table 1. Percentages of correct response to two subtests (A and B) of 18 stimuli presented under two conditions (I and II).
Condition I: list of odour descriptors (1 out of 24), Condition II: multiple choice items (1 out of 4).
Numbers in parentheses are percentages corrected for chance.

			I		II	
A	1	curry powder	68	(67)	97	(96)
	2	maggi seasoner	74	(73)	97	(96)
	3	nutmeg	73	(72)	97	(96)
	4	booth polish	79	(78)	97	(96)
	5	lilac	53	(51)	97	(96)
	6	soft soap	73	(72)	96	(95)
	7	coffee	95	(95)	96	(95)
	8	buttermilk soap	45	(43)	95	(93)
	9	peppermint	81	(80)	95	(93)
	10	tar	32	(29)	93	(90)
	11	garlic powder	53	(51)	91	(88)
	12	honey	68	(67)	89	(85)
	13	cod liver oil	67	(66)	89	(85)
	14	air refreshener	55	(53)	89	(85)
	15	almond	29	(26)	87	(83)
	16	licorice	81	(80)	87	(83)
	17	cinnamon	64	(62)	87	(83)
	18	pine	37	(34)	72	(63)
B	19	motor oil	77	(76)	100	(100)
	20	toilet soap	62	(60)	99	(99)
	21	coconut	79	(78)	97	(96)
	22	onion powder	67	(66)	97	(96)
	23	anise	89	(89)	96	(95)
	24	baby powder	64	(62)	96	(95)
	25	cloves	78	(77)	96	(95)
	26	mustard	73	(72)	96	(95)
	27	peanut butter	95	(95)	96	(95)
	28	green Swiss cheese	88	(87)	96	(95)
	29	fruit flavoured chewing gum	86	(85)	95	(93)
	30	moth balls	32	(29)	95	(93)
	31	beeswax	77	(76)	92	(89)
	32	chocolate	82	(81)	91	(88)
	33	lemon	78	(77)	88	(84)
	34	lavender	37	(34)	84	(79)
	35	ginger	33	(30)	74	(65)
	36	cumin	30	(27)	39	(19)

24 odour descriptors was used. These descriptors included the 18 odour names of that subtest plus six other familiar odour names. The subjects were asked after smelling an odourant to find the correct name by choosing one of the 24 odour descriptors.

In condition II 36 sets of four odour descriptors were created. For the odourant

baby powder for instance the four descriptors are a) strawberry, b) baby powder, c) garlic powder, d) cod-liver oil. Here the subjects were asked after smelling the odourant baby powder to find the right name by selecting one of the four descriptors. In condition I testing was performed on 24 males (mean age 35.0; sd 17.0; range 15-65) and 49 females (mean age 39.4; sd 14.4; range 15-64) and in condition II on 32 males (mean age 33.3; sd 15.1, range 17-68) and 44 females (mean age 36.0; sd 13.0; range 16-60). All the subjects reflected on an advertisement and were paid for their cooperation. Results of both experiments are presented in Table 1, Table 2 and Figure 1.

Table 2. Influence of age, sex and smoking behaviour on odour identification performance.
Per subject scores on both subtests have been added. For the variable age the subjects were divided into those under and those over 40 years.
Condition I: list of odour descriptors (1 out of 24), Condition II: multiple choice items (1 out of 4).

	condition	means		*t*-value	significance $p<0.05$
AGE	I	under 40	: 25.44	2.34	yes
		over 40	: 21.81		
	II	under 40	: 33.67	2.24	yes
		over 40	: 31.12		
SEX	I	males	: 20.54	3.08	yes
		females	: 25.47		
	II	males	: 31.25	2.98	yes
		females	: 33.98		
SMOKING	I	yes	: 23.69	0.35	no
		no	: 24.32		
	II	yes	: 32.86	0.08	no
		no	: 32.79		

In Table 1 percentages of correct response to the 36 odourants under conditions I and II are given. Overall performance in condition I is 64% correct identification for subtest A and 68% for subtest B. For condition II these figures are 92% and 89% respectively. Furthermore, the results for condition I show that women perform better on both subtests (70% and 72% correct identification) than men (53% and 61%). Figure 1 (top) shows that this form of the test differentiates well between subjects. Therefore this form is meant to be used as a selection test, for sensory panel work in industrial settings. The correlation between the two

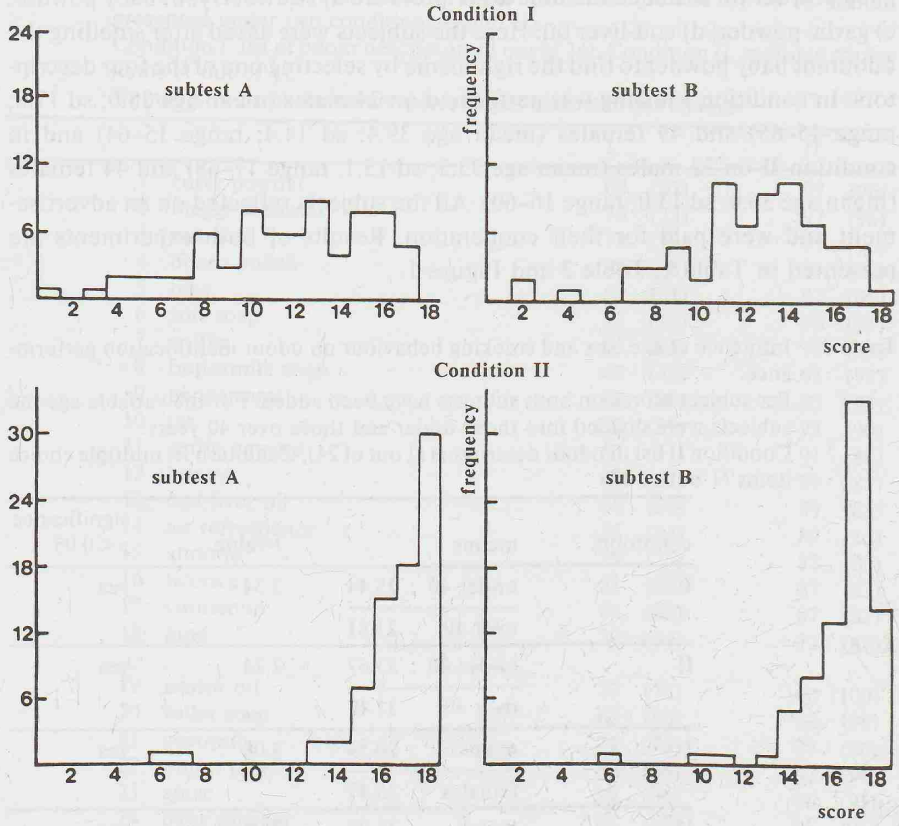

Figure 1. Frequency distributions of the individual scores on the two subtests (A and B) under two conditions.
Condition I: list of odour descriptors (1 out of 24). Condition II: multiple choice items (1 out of 4).

subtests is satisfactory (r = .77). In condition II the percentages correct identification are very high (Table 1). Again women do better on both subtests (96% and 93%) than men (87% and 86%). Due to a ceiling effect (see Figure 1, bottom) and consequently a lower variability of the identification scores the correlation between the two subtests is somewhat lower (r = .68). It is concluded that this form of the test is well suited for detection of odour deficiencies in the otorhinolaryngological clinic.

Inspection of Table 1 reveals that in condition II only one odourant has a raw identification score of less than 72%. Further analysis showed that this item (Cumin) was often found to be very weak and sometimes not detected. It was replaced by a cumin product with a stronger smell. In subsequent experiments its

identification score rose to 83%. In Table 2 the influence of age, sex and smoking behaviour on odour identification performance is analyzed.
The third and last stage of the test development is still running. For the two conditions data are collected on subjects of both sexes, varying ages and with no olfactory complaints. So far 221 subjects (74 males, mean age 35.5, sd 16.5, range 16–86 and 147 females, mean age 38.1; sd 14.1; range 16–87) have done both sub-

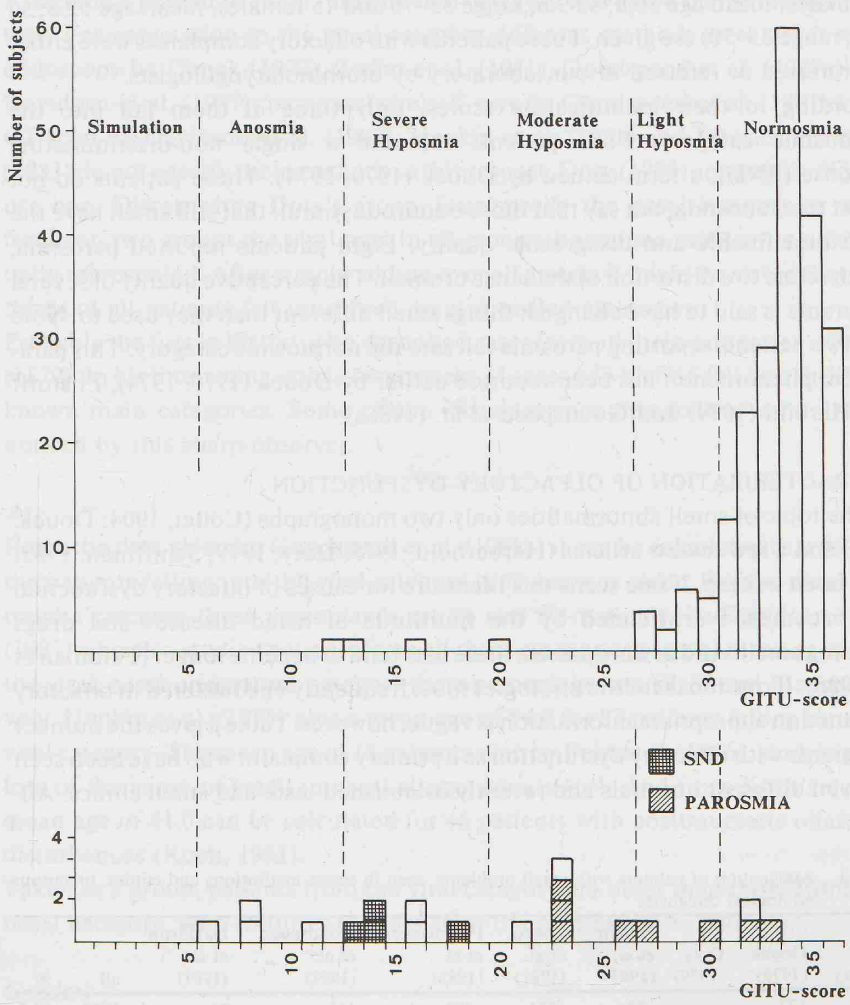

Figure 2. Distribution of GITU-scores for 221 normal controls (top) and 21 patients (bottom). The test is used to diagnose into six categories: normosmia, light hyposmia, moderate hyposmia, severe hyposmia, anosmia and simulation. A score in the latter category may indicate that the subject is pretending to be anosmic, where in fact he is not (Doty et al., 1984).

tests under condition I. Their total identification scores are given in Figure 2 (top). The tests have been termed Geur Identificatie Test Utrecht (GITU). As can be calculated from the score-distribution of normal controls in Figure 2 (top) the proportion of subjects having an identification score of 80% or more is .955. Comparable figures are: for Sumner's test about .06, for Cain's test about .89 (calculated from Cain et al. (1983), by means of interpolation) and for the UPSIT .88 (Mair and McEntee, 1986). At the bottom of Figure 2 the scores for 21 patients (six males, mean age 50.8; sd 9.6; range 35–70 and 15 females, mean age 52.3; sd 11.3; range 35–70) are given. These patients with olfactory complaints were either self-referred or referred to our laboratory by otorhinolaryngologists.
According to their identification scores, only three of them fall into the normosmic category. Four patients reported a single non-discriminating response (SND), a term coined by Douek (1970, 1974). These patients do not report total anosmia, but say that those odourous stimuli that still smell have the same undefinable and disagreable quality. Eight patients reported parosmia, which refers to a distortion of the sense of smell. The perceptive quality of several odourants is said to have changed: things smell different than they used to. Note that two patients reporting parosmia fall into the normosmic category. This paradoxical phenomenon has been reported earlier by Douek (1970, 1974), Zilstorff and Herbild (1979) and Goodspeed et al. (1986a).

CHARACTERIZATION OF OLFACTORY DYSFUNCTION

On the topic of smell abnormalities only two monographs (Collet, 1904; Douek, 1974) and a few review articles (Herberhold, 1975; Doty, 1979; Schiffman, 1983) have been written. If one scans this literature for causes of olfactory dysfunction one becomes overwhelmed by the multitude of listed diseases and drugs affecting smell. And as time passes, these lists tend to become longer (Feldman et al., 1986). If one looks for the etiologies most frequently encountered in olfactory dysfunction appropriate information is vague, however. Table 3 gives the number of patients with olfactory dysfunction as a primary complaint who have been seen in seven different hospitals and recently established taste and smell clinics. All

Table 3. Assignation of patients with smell problems, seen in seven institutions and clinics, to various etiological categories.

category	Douek (1970)	Doty (1979)	Henkin et al. (1981)	Zusho et al. (1981)	Fikentscher et al. (1983)	Goodspeed et al. (1987)	Davidson et al. (1987)	all	%
nasal	177	–	72	731	185	133	21	1319	33.2
viral	32	23	171	274	265	82	20	867	21.8
unknown	4	15	131	280	227	114	0	771	19.4
trauma	15	19	106	43	197	38	6	424	10.7
other	3	–	210	48	245	74	16	596	15.0
total	231	57	690	1376	1119	441	63	3977	100.0

these patients received some kind of olfactory testing and medical examinations in order to specify a probable etiological cause. In these seven patient groups two specified etiological categories are always recognized: a category of viral or viral-like diseases (influenza, common cold) and a category of head trauma. In all groups but one, a category of nasal and paranasal sinus diseases (chronic rhinitis, nasal polyposis, sinusitis) is mentioned. In Table 3 these three etiological categories are indicated as VIRAL, TRAUMA and NASAL.

Assigning a patient to the trauma or viral category was based on his medical history. For assignation to the nasal category different methods were used: nasal endoscopy by Douek (1970), Zusho et al. (1981), Goodspeed et al. (1987a) and Davidson et al. (1987); paranasal sinus X-rays by Goodspeed et al. (1987b) and CT-scan by Davidson et al. (1987). Henkin et al. (1981) and Fikentscher et al. (1983) do not specify their methods in this respect. Doty (1979) apparently did not use one. Disregarding Doty's group, four specify the nasal category as most frequent, two groups the viral one. In all groups the trauma category is substantially represented. After simply adding over all groups it might be stated that two thirds of all patients fall into three main specified categories.

Probably the first in history who described cases from all three categories is Notta (1870). In his interesting article he presents 24 cases of which 15 fall into the three known main categories. Some of the characteristics that follow were already noticed by this sharp observer.

Age

From the data given by Goodspeed et al. (1986a) it can be calculated that 95% of the patients falling into the viral category is 40 years or older. For the nasal and trauma category these percentages are 66 and 50 respectively. Davidson et al. (1987) who also studied patients from all three categories present mean ages. For the viral, nasal, and trauma category these percentages are 60, 51 and 33 respectively. Henkin et al. (1975) give a mean age of 54.0 for 87 patients falling into the viral category. The mean age of 18 patients seen by Fein et al. (1966), studying the loss of the sense of smell in nasal allergy equals 46.4. And from Koch's data a mean age of 41.0 can be calculated for 46 patients with posttraumatic olfactory disturbances (Koch, 1981).

Taken as a group, patients from the viral category are older than those from the nasal category, who in turn are older than the posttraumatic patients.

Gender

As a consequence of the fact that men experience more head injuries than women, post-traumatic olfactory disturbances are much more often found in males. From the data given by Pruszewicz et al. (1969), Kleinschmidt (1978), Koch (1981), Zusho (1982), Fikentscher and Müller (1985) and Davidson et al.

(1987), totally representing 623 cases, a mean sex ratio F : M of 1 : 4.5 can be calculated.

Men also outnumber women in the nasal category, but here appropriate information is less abundant. Goodspeed et al. (1987a) assigned 35 females and 65 males to this category and Davidson et al. (1987) report a sex ratio of 1(F) : 2.0(M) for 21 patients.

A reversed ratio is found for patients suffering a smell disturbance after a viral-like infection of the upper respiratory pathway. Adding together 216 cases from 16 different sources yields a mean sex ratio of 2.2(F) : 1(M), (Notta, 1870; Zwaardemaker, 1891; Reuter, 1899; Beyer, 1904; Kobrak, 1908; Stiefler, 1924; Hofmann, 1926; Nadoleczny, 1933; Hesse, 1956; Jefferson, 1961; Szmeja and Obrebowski, 1969; Hansen, 1970; Schaupp, 1971; Wentges, 1977; Davidson et al., 1987; Goodspeed et al., 1987a).

Degree of olfactory deficit

In the older literature reliable information on the degree of olfactory loss can hardly be found for the three etiologies. Fortunately, the establishment of clinical research centers for the study of human chemoreception in the USA has stimulated research on this matter. Recently, Cain et al. (1988) published olfactory scores as measured by their test (Cain et al., 1983) for patients from the three etiological categories. The distribution of olfactory scores of patients falling in the viral category differs from that of patients with nasal disease and head trauma. In the latter two categories most patients are found to be anosmic, whereas a majority of patients from the viral category have scores indicating hyposmia. That the loss of smell after head trauma tends to be complete is also reported by Fikentscher and Müller (1985) who classified 77 of 122 patients as anosmic. Using Cain's test, Davidson et al. (1987) could not confirm the finding that patients from the viral category differ from those from the nasal category with respect to their olfactory scores. However, these authors have only tested rather small groups of patients.

The incidence of parosmia

Throughout the literature, a bewildering array of different terms to denotate qualitative dysfunctions of the sense of smell has been used. Parosmia is the most commonly encountered term. Here we use parosmia to denotate all qualitative smell dysfunctions. Because appropriate testing for parosmia is elusive, the investigation is completely dependent on what the patient tells about this symptom.

For the nasal category information about the occurrence of parosmia is scarce. Only Douek (1970) and Goodspeed et al. (1986a) provide information. In their combined data, 9.2% of 185 patients reported a qualitative dysfunction.

For the trauma category the incidence of parosmia given by six authors ranges from 6.4% to 21.3% (Leigh, 1943; Sumner, 1964; Pruszewicz et al., 1969; Koch, 1981; Fikentscher and Müller, 1985; Goodspeed et al., 1986a). Together they present 480 patients 55 of which report parosmia (11.5%). For the viral category the following figures can be found: Schaupp (1967): five reports of parosmia in 10 cases; Hansen (1970): six in 14 cases; Douek (1970): eight in 32, Goodspeed et al. (1986a): two in 22 and in our laboratory 12 from 13 patients presumably falling into this category reported a qualitative dysfunction. Together 33 of 91 patients reported a parosmia which yields a percentage of 36.3.
Schaupp (1971) who saw 70 patients with post-influenzal olfactory disturbance does not provide quantitative information. Instead he writes: "Post-influenzal hyposmia is nearly always combined with parosmia, just like post-influenzal anosmia is often accompanied by cacosmia".
The conclusion seems justified that the symptom of parosmia is more a characteristic of patients with a postviral smell disorder than of patients from the other two categories.

Posttraumatic olfactory disturbance
An summary of what has been published on the incidence of olfactory disturbance after head injury is given in Table 4. The differences in percentages are very big, but might be explained by the following considerations. First, mention must be made of the fact that most investigators do no specify their means of establishing olfactory disturbance. Secondly, the percentages for the larger series (1000 cases or more) range from 4.3 till 10.5, while those for the smaller ones range from 2.1 till 65.4. So sampling variability must play a role. Thirdly, the insurance series yield on the average low percentages. These might represent more permanent cases of olfactory disturbance as compared with the others. Fourthly, series for which the severity of the injuries is explicitly stated to be high yield the higher percentages, ranging from 16.6 til 65.4. That the incidence of olfactory disturbance is related to the severity of head trauma has been shown by several investigators using different criteria to define severity (Klingler and Jost, 1963; Sumner, 1964; Rauh, 1967; Rous and Moravec, 1967; Costanzo and Becker, 1986). However, this relationship does not mean that trivial blows on the head are entirely innocent. Such blows leading to permanent anosmia have been described by authors like De Morsier (1938), Sumner (1964) and Douek (1974).
A number of authors have discussed the occurrence of olfactory disturbance as related to the site of the impact but the discussion remained obscure until Sumner's work on post-traumatic anosmia. He showed that although anosmias due to a frontal blow occur most frequently, an occipital blow is five times more likely to cause anosmia. The same conclusion can be drawn from Table 5.

Table 4. 27 case series in which the incidence of olfactory dysfunction in head trauma has been established.
In the 4th column the number of patients with head injuries is given. In the 5th column the number of patients for whom an olfactory disturbance is reported. This number is expressed as a percentage of all head injuries in the 6th column. Insurance means that the patients came for a follow-up evaluation in order to complete accident insurance claims.
Rel. unselected means that these series appeared to the present author as relatively unselected cases.
Data preceded by a dash (-) are not directly taken from the original authors.

			N(hi)	N(od)	%	
1931	Laemmle	GER	26	17	65.4	very serious injuries
1933	Helsmoortel et al.	BEL	37	15	40.5	serious injuries
1937	Goland	USA	38	6	15.8	
-	Müller	GER	128	20	15.6	
-	Anhalt	GER	137	23	16.8	
-	Symonds	-	99	5	5.0	
-	Barth	GER	400	28	7.0	
1943	Leigh	ENG	1000	72	7.2	rel. unselected
1947	Bay	GER	3215	300	9.3	
-1949	Piacentini	ITA	155	15	9.7	
1954	Lewin	ENG	930	50	5,4	rel. unselected
1958	Libersa and Decroix	FRA	898	22	2.4	insurance
1960	Miani et al.	ITA	348	12	3.4	insurance
-1961	Kondo	JAP	135	51	37.7	
1963	Klingler and Jost	GER	155	26	16.8	very serious injuries
1964	Hughes	ENG	1800	189	10.5	rel. unselected
1964	Sumner	ENG	1167	87	7.5	rel. unselected
1964	Mifka	AUS	1000	64	6.4	insurance
1966	Rebattu et al.	FRA	1000	80	8.0	insurance
1966	Appaix et al.	FRA	48	6	12.5	
1967	Rous and Moravec	CHE	120	24	20.0	very serious injuries
-1968	Azuma	JAP	435	9	2.1	
-1968	Umeda	JAP	87	11	12.6	
1969	Rauh	GDR	115	18	15.6	rel. unselected
1969	Pruszewicz et al.	POL	90	39	43.3	
1982	Zusho	JAP	5000	212	4.3	insurance
1986	Costanzo and Becker	USA	348	77	22.1	serious injuries
	27 authors		18911	1478	7.8	all series
	5 authors		5012	416	8.3	rel. unselected

Recovery from posttraumatic olfactory disturbance

1. Recovery rate:

Two authors have reported about recovery from posttraumatic olfactory disturbance for groups of more than 100 patients. Sumner (1964) reported that 72 out of 188 posttraumatic cases of smell impairment that were followed up at least one year showed recovery of smell (i.e. 38.3%). Kleinschmidt (1978) encountered 94 subjects (64.4%) in his investigations of head injuries who showed improve-

Table 5. The incidence of olfactory disturbance as related to the site of the impact. In the top of the table the number of patients with olfactory disturbance due to an occipital, frontal or other blow are given, as found by 40 different authors. The ratio occipital blows: frontal blows (for blows causing anosmia) is about 3:5. At the bottom of the table the distribution of the three types of blows is given for all kinds of accidents (Sumner, 1964) and for car accidents (Büttner, 1959). Averaging these percentages gives a ratio occipital blows: frontal blows (for all blows, whether or not causing anosmia) of about 1:8.
So an occipital blow may be estimated as $8/1 \times 3/5 = 4.8$ times as dangerous for alfactory functioning as a frontal blow.

	site of the blow			
series	occipital	frontal	other	N
1870–1943 22 authors	52	51	25	128
1949–1964 15 authors	97	190	76	363
1966–1982 3 authors	102	153	57	312
1870–1982 40 authors	251	394	158	803
	31.3%	49.1%	19.7%	
1964 Sumner (all types of accidents)	7%	78%	15%	584
1959 Büttner (car accidents)	11%	67%	22%	
mean	9%	72.5%	18.5%	
	occipital	frontal	other	N

ment after posttraumatic anosmia or hyposmia. For the smaller groups Zusho (1982) found eight out of 56 patients (14.3%) to improve and Costanzo and Becker (1986) 25 out of 77 patients (32.5%). This latter percentage might be too low for the investigators did not include light cases of head trauma in their series. Sumner (1964) who did include such cases showed that recovery rate is related to the severity of the injury. Zusho's figure certainly is too low because his subjects have not been studied and followed directly after their head injuries, being just that period of time in which most recovery takes place as is shown below.
The present author has taken together 75 cases of posttraumatic smell disturbance, in which there was mention of some follow-up, described by 25 different authors, ranging in time from Notta (1870) till Leigh (1943) and found 33 reports of smell recovery. This recovery rate of 44.0% is close to Sumner's. It can be concluded that, provided that also light cases of head trauma are included and follow-up starts as soon as possible after trauma, about four out of 10 cases of posttraumatic smell impairment will eventually recover. Furthermore, from Sumner's data it can be inferred that in cases in which posttraumatic amnesia has lasted less than 24 hours about 50% recover while in cases in which the amnesia has lasted longer about 10% do so.

2. Recovery time:
Figure 2 depicts two curves (circles) that show the relationship between time elapsed since the head injury and the rate of recovery from posttraumatic smell disturbance. One curve is based on Sumner's data, the other on data spread all over the literature. Similar types of curves are found by Kleinschmidt (1978) and by Costanzo and Becker (1986). These recovery curves typically consist of a steep initial part in which recovery takes place rapidly and a flat second part in which recovery is slow, implying that two different mechanisms are at work. Combining these curves with the fact that in 40% of the cases recovery ultimately takes place, curves can be constructed from which prognosis may be estimated. From the two curves in Figure 2 for instance, it can be inferred that after three months the recovery probability in an average head trauma with smell disturbance is about $0.40 \times 0.25 = 0.10$.
Interestingly enough, Sumner observed a relation between the severity of the injury and recovery rate but did not find such a relationship between severity and recovery time.

Postviral olfactory disturbance
1. Recovery rate:
Numerous treatments have been applied in cases of enduring olfactory disturbance following viral infections. Reuter (1899) used strychnine, Hesse (1956) a strychnine derivative. Duncan and Briggs (1962) injected large doses of vitamin A in the buttocks of their patients. Von Rossberg (1966), Szmeja and Obrebowski (1969), Hansen (1970), Schaupp (1967, 1971), Goodspeed et al. (1986b) and Davidson et al. (1987) all gave their patients some kind of corticosteroid and some of them did so in combination with vitamins, strychnine or both. Henkin et al. (1981) used zinc and aminophylline. Some of these authors show good results, others report only temporary effects.
In general, the percentage complete and lasting recovery from postviral smell impairment seems not high: Schaupp (1971), treating the largest of all patient groups mentioned above, found only three out of 34 patients who experienced such recovery. In all these studies there were no untreated control groups and therefore the level of spontaneous recovery can not be estimated. The only exception is Fikentscher et al. (1983) who found more improvement in patients treated with vitamins and/or prednisone as compared to the untreated ones. Unfortunately, they had to conclude that this improvement was not specifically induced by their drugs, but had to be regarded as a general treatment effect.
An attempt has been made to estimate the percentage of spontaneous recovery. Eleven authors together describing 26 untreated patients of postviral smell disturbance, report nine cases of complete recovery (34.6%) (Notta, 1870; Zwaardemaker, 1891; Reuter, 1899; Kobrak, 1908; Stiefler, 1924; Bednár, 1930;

Nadoleczny, 1933; Jefferson, 1961; Duncan and Briggs, 1962; Von Rossberg, 1966; Wentges, 1977). A comparison with treated patients is hardly possible, because authors describing improvements in these patients use rather indefinite categories as some, partial or perceptible improvement. Nevertheless an attempt has been made by picking out those cases where full recovery is explicitly reported. Out of 55 treated patients nineteen showed such recovery (34.5%) (Reuter, 1899; Szmeja and Obrebowski, 1969; Hansen, 1970; Schaupp, 1971). In conclusion it might be said that the scarce information available leaves the impression that there is no difference in real improvement between treated and untreated patients.

2. Recovery time:
In absence of any systematic group measures of recovery times for patients with a postviral smell impairment, the only thing that can be done is collecting these times as reported for individual cases. Such data exist, but are extremely scarce and sometimes imprecise. The curve with filled squares in Figure 3 shows the result. This leads to the hypothesis that the process of spontaneous smell recovery in postviral disturbance is slower than that in posttraumatic impairment.

Prednisone
Although most authors in the older medical literature describe a temporary reversal of olfactory dysfunction via steroid therapy in patients from the viral category, there is also an old report of this effect in cases of nasal polyposis (Hotchkiss, 1956). However, in recent studies in which patients from both categories were given prednisone, an effect of this drug was noticed in patients from the nasal category only. Goodspeed et al. (1986b) report a significant association between the presence of nasal or paranasal sinus disease (but not polyposis) and a positive response to prednisone. Cain et al. (1988) report the olfactory test scores for 14 patients from the nasal and for five patients from the viral category, measured just before, during and at the end of a 7-day course of prednisone administration. As a group only the nasal/sinus disease patients showed reliable improvement. Similar findings are reported by Davidson et al. (1987). The reason for this discrepancy between the older and more recent findings is unclear. One obvious possibility is the uncertainty in the etiological assignation of olfactory patients.

Localization of the smell deficit
An interesting finding has recently been reported by Davidson et al. (1987). These investigators were able to inspect olfactory epithelia visually by means of endoscopic techniques in patients from all three categories. In most patients

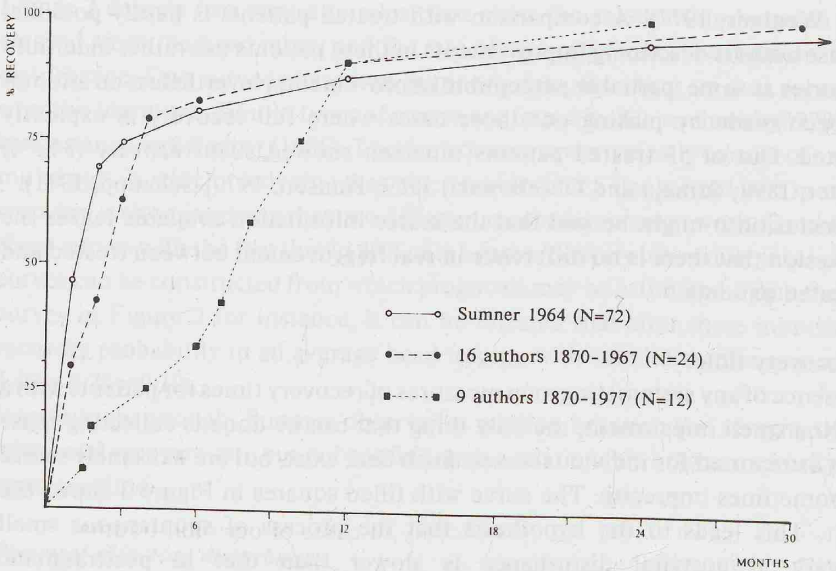

Figure 3. Percentages of persons recovered at varying times from posttraumatic olfactory disturbance (circles) and postviral olfactory disturbance (squares).
Open circles represent data from Sumner (1964).
The curve with filled circles summarizes the recovery times reported by Notta (1870), Jacob (1882), Sterling (1919), Collet (1933), Helsmoortel et al. (1933, 1936), De Morsier (1938), Leigh (1943), Hagan (1967) plus the recovery times from two other authors quoted by Helsmoortel et al. (1933) and five other authors quoted by Sterling (1919).
The curve with filled squares shows recovery from olfactory disturbance after viral infection of the upper respiratory pathway. This curve is composed of recovery times given by Notta (1870), Zwaardemaker (1891), Reuter (1899), Kobrak (1908), Stiefler (1924), Bednár (1930), Jefferson (1961), Von Rossberg (1966) and Wentges (1977).
For each curve the total number of persons recovered is set at 100%.

from the nasal category polyposis and other inflammatory changes prohibited observation of the olfactory epithelium, but in some of the cases in which observation was possible no epithelial abnormalities were perceived. In patients from the trauma category the dominant observation was that the olfactory epithelium appeared to be absent by visual inspection and most olfactory epithelia of patients from the viral category were found to be replaced with white strips of tissue that was interpreted as being scar tissue.
The latter observation resembles one of Douek et al. (1975), who took a biopsy from the septal surface of the olfactory epithelium in a case of post-influenzal anosmia. This biopsy showed signs of extensive scarring of subepithelial tissue and replacement of olfactory epithelium with respiratory epithelium. In the basal region of the epithelium, the investigators found cells resembling basal cells, normally capable of given rise to new receptor cells, but axons were not detected.

They carefully concluded that possible attempts of basal cells at regeneration were not effective, perhaps because of the pathological changes at the surrounding tissue. For cases of postviral olfactory disturbance, localization of the defect at or just near the olfactory epithelium seems likely.

Biopsies from olfactory epithelium in cases of posttraumatic anosmia have been taken in a few instances (Moran et al., 1985; Hasegawa et al., 1986). As compared to normal human olfactory epithelium (Polyzonis et al., 1979; Moran et al., 1982) the entire olfactory epithelium in these biopsies appears shrunk and fully disorganized. The morphology of olfactory receptors is abnormal, involving degenerated olfactory vesicles with no or few cilia. In one instance in which the biopsy was taken as early as fourteen days after an occipital trauma, producing immediate anosmia, only few signs of degeneration were observed (Hasegawa et al., 1986). Together these findings suggest a retrograde degeneration of the olfactory epithelium after head trauma producing anosmia. The initial damage involves not the epithelium, but must be localized in more central parts of the olfactory pathway. Most authors on posttraumatic anosmia prefer to hypothesize a rupture of the olfactory nerve, especially in cases of occipital trauma.

But for cases in which anosmia persists, there are some objections to this explanation. First, sectioning the olfactory nerve in monkeys has been shown to lead to degeneration and a subsequent regeneration of the olfactory epithelium (Graziadei et al., 1980). Secondly, for two cases of posttraumatic anosmia a direct inspection of the more central parts of the olfactory pathway did show other lesions. In one case, a year after an occipital trauma, producing bilateral anosmia, a ruptured olfactory tract was observed during an operation (Guiot and Messimy, 1949). In the other case, 16 months after an occipital trauma, producing anosmia with fluctuating parosmic symptoms, tiny bone splinters in the olfactory tract and considerable distortion of the bulbar structure were seen during autopsy (Schmid, 1961). A safe conclusion seems that the initial damage to the olfactory system in patients with posttraumatic olfactory disturbance must be localized somewhere between the olfactory epithelium and the entorhinal area. For some cases of frontal injury the epithelium might be included.

Jafek et al. (1987) have recently taken olfactory biopsy specimens from two patients with nasal allergy, nasal polyposis and pansinusitis. Both biopsies showed electron-optically normal olfactory receptors with respect to number and fine structure. Much earlier a similar finding was reported by Douek et al. (1975) for an anosmic patient, suffering from allergic rhinitis. However, a biopsy taken by the same investigators from an anosmic patient with chronic infective rhinitis showed no true olfactory epithelium. As in the case of postinfluenzal anosmia, scar tissue in the lamina propria was observed also. Although the value of olfactory biopsies has been questioned (Nakashima et al., 1984) the latter observation indicates that more biopsies are needed from olfactory patients with nasal/

sinus disease. A systematic difference in findings may eventually lead to a subdivision of the nasal category into patients with allergic and non-allergic symptoms. It seems clear that the anosmia in at least some patients suffering from nasal allergy is potentially reversable and in fact this potential has been demonstrated with a combination of surgery and prednisone (Jafek et al., 1987).
From the information above, it can be inferred that for at least some patients from the nasal category, a simple nasal airway obstruction might explain their olfactory problems. This obstruction is presumably located in the region of the olfactory cleft. However, such an obstruction can not be a valid explanation for all patients from the nasal category, for it is for instance hard to see how it can produce parosmia. Jafek et al. (1987) mention the possibility of a biochemical block located at the epithelial level but do not elaborate this idea. Zusho et al. (1981) divide their patients with respect to the localization of the defect into four categories of disturbance: respiratory, epithelial, combined (respiratory and epithelial) and central. On the basis of their diagnoses only 1.9% of the 1376 patients were assigned to the category of respiratory disturbances, but from the information given in their article it is unclear how this was achieved.
A prudent conclusion is that for patients from the nasal category the deficit can be localized from any point of the upper nasal airway up to the lamina propria, including the latter.

This brief review of the literature on the three main known etiologies of olfactory dysfunction is summarized in Table 6.
118 years ago the Frenchman Notta ended his article like this: "Although some patients suffer greatly from the loss of their sense of smell, many hardly care for it and only accidentally do they inform a physician about it. This should not be regretted too much, because the therapeutic means for this inconvenience are absolutely nil. One finds nothing about it in the literature. My own therapeutic treatments have been completely unsuccessful and when patients regain their sense of smell it is solely to the efforts of nature that their healing is due."
The situation certainly has changed but is still far from satisfactory. The information examined in this review indicates that considerable basic research is needed for making progress in this field. This progress can not be achieved without the cooperation of medical practitioners with interest in the subject of olfactory dysfunction.

Table 6. Characterization of olfactory dysfunction.

	etiological category		
	TRAUMA	VIRAL	NASAL
incidence	11%	22%	33%
age	younger	older	middle-aged
sex ratio (F/M)	1 : 5	2 : 1	1 : 2
smell diagnosis	anosmia	hyposmia	anosmia
parosmias	few	many	few
spontaneous recovery	±40%	±35%	?
recovery rate	biphasic, "fast"	slower?	?
olfactory epithelium	degenerated	scarred	normal/abnormal/ invisible
localization	epithelium - entorhinal area	epithelium - lamina propria	airway - lamina propria
therapy	none	diverse drugs with dubious results	temporary response to prednisone/ surgery

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