

# Olfactory Dysfunction: Course of Disease

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## Summary

Olfactory dysfunction can be secondary to a large array of causes. These can be peripheral (transmission hyposmia) or central, neurosensorial (head trauma, viral infection, neurologic disease, iatrogenic, exposure to neurotoxics, idiopathic). Finally psychiatric diseases and malingering with normal functioning olfactory system must be considered. Each causal situation has its specific course and treatment. Besides pathology, physiologic decay of the sense with age is well known (presbionmia). Once we exclude CNS and psychiatric problems, hyposmia can be due to damage of the receptor or air flow obstruction in the nose. Neurosensorial olfactory damage does not allow for any treatment. Early prescription of neurotrophic and neuroprotective drugs, as well as corticosteroids is done mostly for the tranquillity of the patient. In cases of acute neurosensorial damage, removal of the damaging agent in professional exposure and anti-inflammatory treatment in viral and posttraumatic hyposmia can be useful. The fact that the olfactory cells are the only fully differentiated neuronal cell that maintains throughout life its regrowing power can account for this. One can expect recovery up to 12/18 month after the acute damage. Also if many treatments have been proposed, none has unanimous approval based on clinical evidence. Once the neuronal damage is stabilised or the neuronal axon regrowth through the cribroid lamina of the ethmoid after damage due to head trauma is completed, no further therapy is of use. In case of transmission hyposmia thorough ORL assessment is mandatory and in most cases proper surgical treatment of the air flow obstruction can re-establish normal olfaction. Development of proper diagnostic methods and possibilities, especially recording of olfactory potentials, is the most

important frontier today in olfactology. This may open the door towards many exciting and clinically useful future options of treatment. Epidemiological data of causal prevalence in olfactory damage in 572 cases seen by the authors in the Milan General University Hospital is presented elsewhere in this meeting.

## **Introduction**

The chemical senses are among the most archaic human sensorial modalities. Olfactory information is already highly integrated in peripheral ganglionic structures and projects only indirectly to the limbic lobe. It's information reaches directly the subconscious integration areas, rather than the vigilant conscient ones. Often underrated in its importance, olfaction has for long time been considered as of secondary importance in neurophysiological and clinical studies. However, the discovery of its close connection with the emotive sphere and olfaction and its influence on the reproductive and social functions, accounts for the growing interest in olfaction. Lately interest is growing also in forensic medicine and compensation claims, as more reliable tests become available. Chemosensory Laboratories are being founded and research on olfaction is growing out a more cornered existence. Distribution of olfactory damage related to age and sex is also of rising interest. Olfaction contributes greatly to the so-called "enjoyment of life" and this is true not only in the younger population. Following the overall functional reduction of the sensorial input in the elderly, its importance is undeniable also in this age group. Major drawback remains the fact the neurosensorial olfactory damage, which accounts for the large majority of cases, has still no treatment, conducing to a more resignation-like behaviour of the physicians as well as the patients.

At the Milan Ospedale Maggiore an Olfactologic Centre is operating since 1991. In this time period close to 600 patients have been examined and we can now present epidemiologic study results pointed to the evaluation of incidence of olfactory pathologies among the population in general, subdivided on the basis of age and causality.

## **Materials and Methods**

A population of 572 subjects with dysfunction of olfactory sensibility examined at the olfactologic centre of Milan Ospedale Maggiore between 1992 and 1998 has been considered.

All subjects underwent full ENT examination with nasal endoscopy. In case of nasal obstruction, Computerised Tomography was done to complete the study.

Olfactory testing started with the base sniff-test (1-5). This was based on the administration of predetermined volumes of air saturated with odorants to each nasal cavity. The method is easy and of good attendibility in the collaborating patient. If the data showed incongruences or generally the patient did not collaborate perfectly, the sniff test was completed by Doty's UPSIT test (6-7) in order to identificate simulation attempts.

Hypo-anosmia was classified as follows: transmission, posttraumatic, postviral, neurological, congenital, iatrogenic, workplace toxic exposure and idiopathic (Table 1).

Tab. 1: Classes of causes

Hypo-anosmia from causes	Class of diagnosis	Possible causes
Transmission	1	Nasal poliposis, allergic rhinitis, anatomical obstruction
Posttraumatic	2	Folowing head trauma
Postviral	3	Viral infections of the upper airways
Neurologic	4	Parkinson's disease, Alzheimer's disease, Multiple Sclerosis, Amiotrophic Lateral Sclerosis sclerosi multipla, schizophrenia, depression, tumours et.al.
Iatrogenic	5	Following polipectomy or septoplastic
Congenital	6	Hormonal, disgenetic, et.al. (f.e. Kallmann)
Tecnopathic	7	Following workplace exposure to olfactotoxic substances (f.e. organic solvents, soldering gasses, methyl bromide)
Idiopathic	8	Unknown cause
Not confirmed Hyposmia	9	Psicogenic, simulation

## Results

Postviral (154 cases, 26,9% - class 3) and transmission hyposmia (136 cases, 23,8% - class 1) resulted to be the main causes of olfactory damage. 116 cases (20,3%) did not relate to known causes (class 8). Surprisingly in a clinical situation not related to compensation claims or forensic medicine 5.1% of the patients showed a subjective hyposmia which did not correlate with real olfactory damage. Table 2 resumes distribution on the different cause classes. Table 3, 4 and 5 reports classes by age and sex. Age ranged from 5 to 90 years.

Tab. 2: Class distribution

Hypo-anosmia	Class of diagnosis	n° cases	%
Transmission	1	136	23.78
Posttraumatic	2	78	13.63
Postviral	3	154	26.92
Neurologic	4	14	2.45
Iatrogenic	5	15	2.62
Congenital	6	20	3.50
Tecnopathic	7	10	1.75
Idiopathic	8	116	20.28
Psychological - Simulation	9	29	5.07
<b>Total</b>		<b>572</b>	<b>100</b>

Tab. 3: Distribution by age and class

Cause	Age group									Total	
	0-10	11-20	21-30	31-40	41-50	51-60	61-70	71-80	81-90	N°	%
Transmission	0	11	28	18	23	28	21	3	4	136	23,8%
Posttraumatic	0	9	19	15	7	17	8	3	0	78	13,6%
Postviral	0	1	6	4	34	52	42	13	2	154	26,9%
Neurologic	0	0	0	0	1	1	5	5	2	14	2,4%
Iatrogenic	0	0	3	5	4	2	1	0	0	15	2,6%
Congenital	0	9	8	2	1	0	0	0	0	20	3,5%
Tecnopathic	0	0	0	2	1	6	1	0	0	10	1,7%
Idiopathic	0	1	6	11	16	31	39	10	2	116	20,3%
Psychological-Simulation	1	3	6	8	5	2	2	1	1	29	5,1%
<b>Total</b>	<b>1</b>	<b>34</b>	<b>76</b>	<b>65</b>	<b>92</b>	<b>139</b>	<b>119</b>	<b>35</b>	<b>11</b>	<b>572</b>	

Tab. 4: Distribution by age and class – males

Class	Age Group									Total	
	0-10	11-20	21-30	31-40	41-50	51-60	61-70	71-80	81-90	N°	%
Transmission	0	6	10	5	9	9	13	1	0	53	22,3%
Posttraumatic	0	5	12	5	4	6	3	2	0	37	15,5%
Postviral	0	0	2	2	11	23	13	1	0	52	21,8%
Neurologic	0	0	0	0	0	0	3	2	0	5	2,1%
Iatrogenic	0	0	2	4	3	0	1	0	0	10	4,2%
Congenital	0	7	6	1	0	0	0	0	0	14	5,9%
Tecnopathic	0	0	0	0	1	4	0	0	0	5	2,1%
Idiopathic	0	0	2	2	9	17	16	4	1	51	21,4%
Psychological-Simulator	1	1	2	3	2	2	0	0	0	11	4,6%
<b>Total</b>	<b>1</b>	<b>19</b>	<b>36</b>	<b>22</b>	<b>39</b>	<b>61</b>	<b>49</b>	<b>10</b>	<b>1</b>	<b>238</b>	

Tab. 5: Distribution by age and class – females

Diagnosis class	Age group									Total	
	0-10	11-20	21-30	31-40	41-50	51-60	61-70	71-80	81-90	N°	%
Transmission	0	5	18	13	14	19	8	2	4	83	24,9%
Posttraumatic	0	4	7	10	3	11	5	1	0	41	12,3%
Postviral	0	1	4	2	23	29	29	12	2	102	30,5%
Neurologic	0	0	0	0	1	1	2	3	2	9	2,7%
Iatrogenic	0	0	1	1	1	2	0	0	0	5	1,5%
Congenital	0	2	2	1	1	0	0	0	0	6	1,8%
Tecnopathic	0	0	0	2	0	2	1	0	0	5	1,5%
Idiopathic	0	1	4	9	7	14	23	6	1	65	19,5%
Psychological - Simulation	0	2	4	5	3	0	2	1	1	18	5,4%
<b>Total</b>	<b>0</b>	<b>15</b>	<b>40</b>	<b>43</b>	<b>53</b>	<b>78</b>	<b>70</b>	<b>25</b>	<b>10</b>	<b>334</b>	

Severe dysosmia was present in 67 patients with postviral hyposmia, 27 of whom males and 40 females. Dysosmia was also prevalent in the group of patients for which no cause could be identified.

In Kallman syndrome the anosmia was always confirmed through UPSIT tests and Nuclear Magnetic Resonance showed absence of olfactory bulbs and/or olfactory tracts (9, 10, 11).

Iatrogenic damages was generally secondary to surgery (class 5) on the nasal structures or the central nervous system.

## Conclusions

Olfactologic examination has still to become a routine clinical investigation. Also in presence of a rising interest, only few large series of investigative results are available and old suppositions – as: “no good tests are available”, “we cannot do anything about it anyway” and so on – are hard to die. Presbionmia, which is the physiologic decrease of olfactory capacity in the elderly is a reality, but is not of such large an extend as generally thought of (6).

We can see from this rather larger number of cases that the pathology of hyposmia consists for three quarters in the three classes of postviral, posttraumatic and idiopathic damage. Distribution according to age shows a bimodal course, with one peak at 21-30 years and the second at age 51-60. This fact corresponds to the different causes distribution, since in the period 21-30 prevail the transmissive and posttraumatic causes, while in the period 51-60 a prevalence of the postviral and idiopathic damages can be seen. Idiopathic cases include spontaneous degenerative manifestations. Only few persons over 70 asked for olfactory examinations, probably due to less consideration, also by general practitioners, for this problems. The age sex distribution in the analyzed cases shows a small overall prevalence of females, larger sex differences emerge in the data broken down in its causal classes.

The transmissive and postviral causes prevail in the females, and iatrogenous and congenital pathology in the males. A special attention is to be paid in analyzing the results of congenital pathology, because our sample contains a significative component of Kallman's syndrom cases, due to the fact that our structure is a reference center for this disease (9-11).

At an analysis of the distribution by age and sex for the separate pathologies our experience shows a specificity for age groups of the several causes of hyposmia, as well as for the different sex distribution in the age groups.

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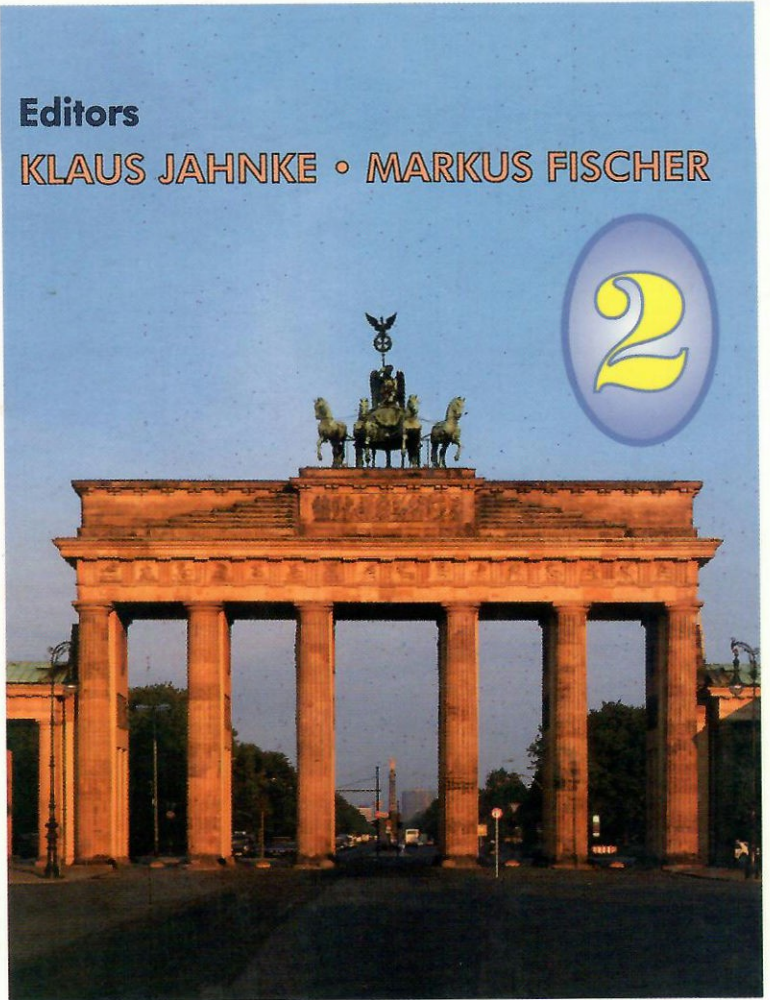
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2

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