

SHORT REPORT

Long term effects on the olfactory system of exposure to hydrogen sulphide

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Abstract

Objective—To study chronic effects of hydrogen sulphide (H₂S) on cranial nerve I (nervi olfactorii), which have been only minimally described.

Methods—Chemosensations (smell and taste) were evaluated in eight men who complained of continuing dysfunction 2–3 years after the start of occupational exposure to H₂S. Various bilateral (both nostrils) and unilateral (one nostril at a time) odour threshold tests with standard odourants as well as the Chicago smell test, a three odour detection and identification test and the University of Pennsylvania smell identification test, a series of 40 scratch and sniff odour identification tests were administered.

Results—Six of the eight patients showed deficits of various degrees. Two had normal scores on objective tests, but thought that they continued to have problems. H₂S apparently can cause continuing, sometimes unrecognised olfactory deficits.

Conclusion—Further exploration into the extent of such problems among workers exposed to H₂S is warranted.

(Occup Environ Med 1999;56:284–287)

Keywords: hydrogen sulphide; smell; taste disorders

Hydrogen sulphide (H₂S), a colorless gas easily recognised at 0.025 ppm by its distinctive rotten egg odour, is well known to affect diffuse areas of the human nervous system including the cortex, subcortex, cranial nerves, and peripheral nervous system.^{1,2} Its effects on cranial nerves II, V, and VIII have been extensively documented, but its chronic effects on cranial nerve I (nervi olfactorii) have been minimally described.^{3,4}

An acute effect of H₂S on the olfactory nerve is temporary paralysis, or olfactory fatigue, which disables the sense of smell, rendering it a poor warning system for detecting the continued presence of the gas.^{3,5} Acute effects have generally been described as transient.

Chronic effects of H₂S on the olfactory system have been described in only one instance⁵: a 46 year old man working next to a sludge centrifuge at a sewage treatment plant

was overcome and rendered unconscious for less than a minute. After regaining consciousness, a neurological examination showed reduced memory, impaired concentration, and complete loss of the sense of smell. His electroencephalogram (EEG) was normal at that time. Ten months after the accident, the patient continued to complain of tiredness, impaired initiative, expressive aphasia, and episodes of trembling. His loss of the sense of smell persisted, except that he was able to detect ammonia. Examination at that time showed impaired visual accommodation and fusion, but neuropsychological test results were normal. Two years after the exposure, CT, and EEG were normal, as were neuropsychological test results, and his sense of smell had returned to normal. Whether his sense of smell was objectively assessed or his subjective evaluation reported, however, is not documented.

Our purpose in the present study was to evaluate chronic effects on olfaction after exposure to H₂S.

Methods

SUBJECTS

Eight men who came for medical evaluation of continuing problems with smell and taste had had accidental exposure to H₂S during their jobs 2–3 years previously.

Patients 1–4

On 8 January 1993, 900 pounds of H₂S were released into the environment over a 2.5 hour period at a construction site at a gas refinery in St Croix, Virgin Islands. Two years later we examined four of the workers, who had experienced various local irritating effects including lacrimation, eye irritation, nausea, vomiting, headache, sore throat, and skin irritation. They had also experienced chemosensory phenomena consistent with H₂S exposure, the smell of rotten eggs, and a strong metallic taste.⁶ None of the patients had lost consciousness during the accident. At the time of the accident, atmospheric H₂S concentrations were not obtained, but in previous months the patients had been exposed to similar low level releases, when the recorded concentrations of H₂S were as high as 243 ppm in the work area. The past exposures of these four patients is considered chronic.

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Accepted 23 October 1998

Table 1 Clinical data 3 years after exposure to H₂S

	At refinery construction site (chronic)				At petroleum plant gas leak (acute)			
Patient No	1	2	3	4	5	6	7	8
Age (y)	45	39	44	41	52	69	40	61
Loss of consciousness with exposure	No	No	No	No	Yes	Yes	Yes	Yes
Rate of exposure	Chronic	Chronic	Chronic	Chronic	Acute	Acute	Acute	Acute
Onset of taste problem	3 Months after	5–10 Months after	Immediate	2 Years after	Unknown	1–1.5 Years after	Unknown	Immediate
Onset of smell problem	1 Month after	5–10 Months after	Immediate	Unknown	Immediate increase	1–1.5 Years after	Immediate	Immediate
Rate of deficit	Increased then no change	Increased then no change	Increased then no change	Increased then no change	No change	No change	No change	No change
Duration of deficit	To present	To present	To present	To present	To present	To present	To present	To present
Phantosmia?	No	Yes	No	No	No	No	No	No
Encephalopathy?	Subclinical	Clinical	Clinical	Clinical	Clinical	Clinical	Clinical	Clinical
Smell tests:								
Amoore's bilateral (25 ds):								
IV acid	Abnormal	Abnormal	Normal	Normal		Abnormal	Normal	Normal
Diacetyl	Abnormal	Abnormal	Normal	Abnormal		Normal	Normal	Normal
l-Carvone	Normal	Normal	Normal	Normal		Normal	Normal	Normal
PE-alcohol	Normal	Abnormal	Normal	Normal		Normal	Normal	Normal
Thiophane	Normal	Normal	Normal	Normal		Abnormal	Normal	Abnormal
Chicago smell test (CST)	4 of 6	4 of 6	4 of 6	4 of 6	6 of 6	3 of 6	3 of 6	4 of 6
UPSIT smell ID	30 of 40	35 of 40	35 of 40	38 of 40		29 of 40	17 of 40	23 of 40
	Mild hyposmia	Normosmia	Normosmia	Normosmia		Moderate hyposmia	Anosmia	Severe hyposmia
Amoore's unilateral thresholds:								
PM carbinol	Normal	Normal	Normal	Normal		Abnormal		Abnormal
PE phenol	Normal	Normal	Normal	Normal		Normal		Normal
Naphthalene	Normal	Normal	Normal	Normal		Normal		Normal
Pyridine	Normal	Normal	Normal	Normal		Normal		Normal
Cineole	Normal	Normal	Normal	Normal		Abnormal		Normal
ISOB-ISOB	Normal	Normal	Normal	Normal		Normal		Normal
IA acetate	Normal	Abnormal	Normal	Normal		Normal		Normal
PD lactone	Normal	Abnormal	Normal	Normal		Abnormal		Normal
CA phenone	Normal	Normal	Normal	Normal		Normal		Normal
Thiophane (ds)	15/15 Normal	15/25 Normal	15/15 Normal	20/15 Normal		40/20 Hyposmia		35/25 Hyposmia

Patients 5–8

On 14 May 1993, four workers were sent as part of a crew to fix a gas leak at a petroleum plant in Texas. On their arrival at the site of the leak, the workers noted fumes coming out from underground. They also noted that in the surrounding area, the odour of rotten eggs was strong, the grass was brown, and birds lay dead. The workers descended into a trenching system 8 feet deep, 7 feet wide, and 12 feet long. None wore protective garb. While attempting to fix the broken pipe, all were overcome by the gas and were unconscious for several minutes before being discovered. They were treated for gas intoxication, were admitted to the intensive care unit, and spent at least a week in hospital. At the time of exposure, atmospheric H₂S concentrations were not obtained, but 3 days after the accident, the concentration of H₂S in the gas leaking out of the pipe into the trench was 2873 ppm as measured by the United States Department of Labor and the Occupational Safety and Health Administration (OSHA). When we examined the four patients, 2.5 years had elapsed since their acute exposure. There were no known past or subsequent exposures to H₂S in these subjects.

Clinical evaluation

All patients were asked their subjective experience of anosmia, hyposmia (decreased odour sensitivity), hyperosmia (increased odour sensitivity), phantosmia (phantom smells), dysosmia (deranged sense of smell), hypogeusia (decreased taste sensitivity), phantogeusia (the taste equivalent of phantosmia), and dysgeusia (the taste equivalent of dysosmia). All were asked to follow the instructions in the appendix

in preparation for objective tests. They then underwent bilateral (with both nostrils simultaneously) odour detection tests according to the methods of Amoore and Ollman⁷ with the following standard odorants at concentrations of 25 decismels (ds), a normally detectable level (the ds scale of odour levels is modelled after the decibel scale of sound levels): phenyl ethyl methylethyl carbinol (pm carbinol), isovaleric acid (iv-acid), 2,3 butanedione (diacetyl), pentadecalactone (pd-lactone), 1-carvone, phenylethyl alcohol (pe-alcohol), and tetrahydrothiophene (thiophane). They also underwent the Chicago smell test, consisting of three forced choice questions pertaining to odour and three open ended questions pertaining to odour identification.^{8,9} Most also underwent other olfactory tests: the University of Pennsylvania smell identification test (UPSIT), a series of 40 scratch and sniff forced choice odour identification questions scored according to age and sex as described in the published guidelines for the UPSIT^{10,11}; and the unilateral (one nostril at a time) threshold tests of Amoore and Ollman for pm carbinol, para ethyl phenol (pe-phenol), naphthalene, pyridine, 1,8 cineole (cineole), isobutyl isobutyrate (isob-isob), isoamyl acetate (ia-acetate), pd-lactone, α -chloroacetophenone (ca-phenone), and thiophane. (Unilateral threshold tests are scored as normal when either the right or left nostril is normal, as described by Amoore and Ollman.)⁷

Results

At the time of our evaluations, all patients had subjective complaints of chemosensory dysfunctions, which consisted of hyposmia (eight

patients), dysosmia (one patient), hyperosmia (one patient), phantosmia (one patient), hypogeusia (eight patients), and phantogeusia (one patient). Problems began immediately for three of the four patients who had acute exposure at the site of the gas leak, but for only one of the four patients exposed at the construction site. The onset of their problems ranged from immediately to 2 years after exposure (table 1).

Objective test results are summarised in table 1. Patients Nos 3 and 5 had normal scores on objective tests, but still considered subjectively that their chemical senses were deficient.

All three from the acutely exposed group who took the UPSIT odour identification test had poor scores, whereas three of the four in the chronic group had normal scores and the fourth was only mildly hyposmic.

Discussion

An impaired ability to identify odours along with a normal ability to detect them as shown by our patient No 7 has been described to occur with head trauma and may indicate the existence of a lesion of the central nervous system rather than a peripheral lesion in the olfactory nerves.¹² All of our patients with acute exposure were deficient in their ability to identify odour. Such olfactory deficit could be due to the loss of consciousness and head trauma associated with falling.

Patient No 1, however, was mildly hyposmic on the UPSIT without having lost consciousness and without head trauma. This deficit in the case of our subjects, could be due to the neurotoxic effect of the H₂S on the olfactory receptor site or through the retrograde degeneration of the olfactory nerves as they project through the olfactory bulb and tract to the olfactory cortex.

As some of our construction worker patients had histories of considerable toxic exposures including use of alcohol which we have shown can induce olfactory deficit,¹³ it seems possible that our findings of olfactory deficits are unrelated to exposure to H₂S, and instead are associated with confounding factors—such as collateral toxic exposures.

Conclusions

As long as three years after their exposure to H₂S, a high percentage if not all of our patients continue to have olfactory deficits. The long duration of this deficit indicates that it may be permanent.

Patients Nos 3 and 5 who complained of deficits yet were normosmic on tests may have originally been more sensitive than the average, and thus experience real losses.

Patients who have been exposed to H₂S should be routinely screened for olfactory deficits and if any are found, vigilant monitoring for H₂S must be undertaken at the work site. Also, these patients must be advised to take precautions at home, including the use of smoke detectors, gas detectors, and food tasters.

Poorly documented effects on the chemical senses have also been seen after exposures to various other neurotoxins including nitrogen tetroxide¹⁴ and benzene.¹⁵

In our industrial society, chemosensory dysfunction may well be a hidden epidemic among workers exposed to various toxic chemicals. The extent of such problems among workers exposed to H₂S and other industrial effluvia is a subject deserving of further careful exploration.

Appendix: Questionnaire

As we are testing abnormalities of smell and taste, the following precautions must be taken:

- You may eat or drink no caffeine for at least 48 hours before your visit (see list below of foods and beverages containing caffeine)*
- You may drink no alcohol for at least 4 days before your visit
- You may eat no pastry items for 24 hours before your visit
- From the midnight before your visit, you may have none of the following; food, gum, cigarettes, or any drink other than water

IT IS IMPORTANT THAT YOU FOLLOW THESE INSTRUCTIONS TO INSURE THE VALIDITY OF THESE TESTS

- From the midnight before your test day, you should use no scented soap, cosmetics, deodorants, shaving cream, aftershave, perfumes, or lipsticks. The only underarm deodorant you may use is Gillette roll on
- From the midnight before your visit, you may use only shampoo with no or minimal smell—that is, ivory shampoo
- You may brush your teeth, but we ask you not to use toothpaste
- All your usual medications should be taken and if you have diabetes you should eat.

*Foods and beverages containing caffeine that you should try to avoid:

Chocolate
Coffee
Cola
Tea
Cocoa
NoDoz
Other over the counter products that are “stimulants”

be sure to read your labels

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