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Brain Damage Caused by Hydrogen Sulfide: A Follow-Up Study of Six Patients

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Hydrogen sulfide (H_2S) poisoning involves a risk of hypoxic brain damage. Six patients who lost consciousness due to H_2S poisoning are described. The symptoms varied from anosmia in the patient with the shortest but highest exposure to delayed neurological deterioration in the patient with the longest exposure. The two patients with the most serious symptoms developed pulmonary edema, which may have prolonged the hypoxia. The patients were reexamined 5 years or more after the poisoning. The five patients who had been unconscious in H_2S atmosphere for from 5 to 15–20 min showed persisting impairment at neurological and neuropsychological re-examination. Memory and motor function were most affected. One patient was seriously demented. Recent reports of large groups of H_2S-poisoned workers probably underestimate the risk of sequelae, due to the inclusion of cases with exposure of short duration and lack of follow-up.

Key words: amnesic syndrome, delayed encephalopathy, anoxia, neuropsychological tests, neurotoxin, hydrogen sulfide exposures, ammonia, animal waste products

INTRODUCTION

Many patients with signs of brain damage due to H_2S poisoning have been described [NIOSH, 1977; WHO, 1981]. Divergent reports of the risk of permanent damage have been published. According to Baader [1931], the prognosis is serious, whereas recent studies based on more than 200 cases of poisoning reported no or few long-term adverse effects [Burnett et al., 1977; Arnold et al., 1985].

One reason for the divergence is lack of follow-up after discharge from hospital. We describe six patients who have been followed for 5 years or more after H_2S poisoning. They have been examined using both neurological and neuropsychological protocols. We have studied the relation between the duration of H_2S exposure and the neurotoxic effects, since duration of exposure reports are sparse.

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TABLE I. Medical Findings in the Patients at Follow-Up, and Duration of Unconsciousness in the H_2S Atmosphere

Patient number	Age[a]	Years[b]	Main affected functions	EEG	CT PEG	Test[c]	H_2S exposed unconscious (min)
1	46	5	(Smell, vision, memory)[d]	−[e]	−	−	<1
2	31	8	Dementia, motor function, vision	−			5–10
3	59	7	Memory, motor function (vision)	+[f]	+	+	~15
4	53	6	Motor function (memory, vision)	(+)		+	~10
5	30	10	Visual abilities, memory	+	−	+	<15
6	31	6	Motor function, vision, memory	+	+	+	15–20

[a]Age at the time of the poisoning.
[b]Number of years between the poisoning and the last examination.
[c]Neuropsychological examination.
[d]Temporary and uncertain findings are given in parenthesis.
[e]Normal.
[f]Pathological finding.

MATERIAL AND METHODS

The material includes all patients with neurological symptoms after loss of consciousness caused by H_2S poisoning who have been registered at the National Institute of Occupational Health since 1983. They were poisoned and first examined between 1977 and 1984 and have been reexamined 5–10 years (mean 7 years) after the accident. One patient died before the planned reexamination.

Information on the exposure events and acute and chronic symptoms was recorded. A clinical neurological examination was performed. All patients were examined with EEG, two with cerebral computerized tomography (CT), one with air encephalography (PEG), and one with CT and cerebral magnetic resonance imaging (MRI). A neuropsychological examination [Lezak, 1983] included (Table II) six subtests from WAIS, Benton visual retention test, learning of ten words, learning of 15 word pairs and visual gestalts [Andersen, 1976], tests from the Halstead-Reitan battery [Reitan and Davison, 1974], and simple visual reaction time [Elsass and Hartelius, 1985].

RESULTS: CASE REPORTS

Table I shows the main medical findings at follow-up and the duration of exposure.

Patient 1

A 46-year-old man had worked at a sewage treatment plant for 7 years. He fainted when opening the lid of a tank to test the discharge water from a sludge centrifuge. He probably was exposed for less than 1 min, because he fell away from the gas, and he regained consciousness in the course of a few minutes.

In the evening, he was taken to the hospital due to respiratory obstruction and blurred vision. A neurological examination was normal, except for reduced memory and concentration and complete loss of the sense of smell. EEG was normal. He was discharged after 2 days, but his respiratory symptoms returned, and he spent one more

day in hospital. The skin peeled off his face during the days after the accident. He resumed his work after a few days.

For some time, decaying offal from a shrimp processing plant had caused problems with gas in the plant. Concentrations between 8 and 16 ppm H_2S were repeatedly measured during the months after the accident, but only about 1 ppm was present when there was less shrimp offal.

A neuropsychological examination 10 months after the accident showed mainly normal results, including results better than the average for his age on motor tests. However, this man reported that he quickly became tired, had less initiative, had difficulties with expressing himself, and had episodes of trembling. His sense of smell was still totally absent. He reported that he noticed ammonia, which is a trigeminal irritant. A visual examination showed reduced accommodation and fusion. At re-examination 2 years later, CT and EEG were normal. At a follow-up examination after 5 years, his symptoms had improved. The neuropsychological test results (Table II) were in accordance with the first examination. His sense of smell had returned 3 years after the accident.

Patient 2

A 31-year-old farmer lost consciousness during pumping of swine manure. His wife had observed him working 5–10 minutes before he was found lying unconscious with his head close to the pump. He was breathing irregularly and was cyanotic. On arrival at hospital 45 min later, he was deeply comatose. He was cyanotic, and blood gas status showed pH 7.12, pCO_2 8.0 kPa (PO_2 was not available). He was treated with oxygen and sedatives. He developed increasing signs of pulmonary edema and had several generalized seizures. EEG showed diffuse dysrhythmia with theta/delta activity. He was comatose for 2 weeks. During the next weeks, he was extremely agitated but spoke only a few words.

After 3 months, he repeatedly spoke his own name, birth date, and adress and the names of his wife and children. EEG improved gradually and was described as normal 1 year after the accident. Since then his condition remained unchanged, with signs of serious brain damage. He could say some stereotyped words and phrases, but neither the nursing staff nor his family could achieve rational contact with him. He was probably blind (he was uncooperative at examinations). He was mostly bedridden, due to uncontrolled movements but could sit up with support and ate when fed; he was incontinent of urine and feces. The deep tendon reflexes were brisk; the plantar reflexes were downward. He stayed at a nursing home until he died, 8 years after the accident, following a few days of high fever and renal symptoms. A neuropathological examination was not performed.

Patient 3

A 59-year-old man, who had been unloading fishing boats at a herring oil plant for 8 years, had entered a hold with spoiled fish and immediately lost consciousness when he hosed water down into the fish. A worker who tried to help him also fell unconscious (patient 4). Patient 3 was rescued after about 15 min. He had a probable tonic-clonic seizure in the ambulance. According to the hospital records, he was awake and without symptoms at arrival, except for headache, and was discharged in good health after 2 days.

When he resumed his work after 1 week, he felt dizzy and trembled easily. His

TABLE II. Neuropsychological Test Results*

	Patient				
	1	3	4	5	6
Age when examined (years)	51	66	58	40	36
Years since the poisoning	5	7	4 1/2	10	5
Test					
Similarities	11 (7)[e]	4 (6)	13 (8)	12 (7)	17 (8)
Digit span	9 (8)	9 (9)	7 (3)	7 (3)[h]	10 (9)
Vocabulary (split half)[a]	46 (8)[e]	24 (5)	24 (4)	34 (5)	38 (5)
Digit symbol	36 (8)[e]	21 (7)	21 (6)	32 (5)	39 (7)
Picture completion	16 (12)	8 (6)	7 (4)	5 (2)[g]	15 (10)
Block design	24 (7)	20 (7)	24 (7)	14 (2)[g]	13 (12)
Benton vis. ret. test, errors	4	7	8	6	3
Ten words learning, five trials	12[b,e]	7[g]	9	10[b]	12[b]
Ten words, retention 1 hr	11[b,e]	3[g]	7	9[b]	11[b]
Fifteen word pairs, learn. errors	19	67[c,g]	12	27	42[h]
Fifteen word pairs, ret. errors	9[h]	—[c,g]	6	11[g]	8[h]
Visual gestalts, learn. err.	4[e]	9	—	7[h]	7[h]
Visual gestalts, ret. errors	5[e]	11	—	13[g]	8[h]
Trail making, part A	37	50	42	41	40
Trail making, part B	129[h]	166[h]	170[h]	230[g]	73
Finger tapping, dominant hand	50	41	17[g]	49	36[g]
Finger tapping, non-d. hand	48	40	17[g]	47	35[g]
Pegboard, dominant hand	58	—[d]	185[g]	75	79
Pegboard, non-d. hand	70	150[g]	155[g]	83	83
Static steadiness, number d. h.	73[e]	235[g]	210[g]	150	131
Static steadiness, n. non.-d. h.	44[e]	222[g]	>210[g]	133	260[g]
Reaction time, 10th percentile[f]	21	20	—	20	19
Reaction time, median	23	22	—	24	22
Reaction time, 90th percentile	28	27	—	31	27

*Patient 2 was too demented to be tested. Norwegian age-adjusted scaled score (median 10) for the WAIS tests in parentheses.

[a]The Vocabulary Norwegian scaled scores are probably too low.

[b]Twelve words instead of ten words, max. number of words correct in five trials.

[c]Stopped due to slow learning, and retention then could not be tested.

[d]Patient 3 had lost a finger in a working accident.

[e]Results at the first examination 1 year after the accident. Not retested.

[f]Simple visual reaction time 5 min.

[g]Test results that are definitely reduced compared with age and estimated premorbid function.

[h]Test results that are probably reduced.

hands were unsteady, his vision was reduced, and he was irritable and forgetful. One year later, he was awarded 100% disability pension.

Two years after the accident, thyrotoxicosis and atrial fibrillation were diagnosed. Cerebral CT showed widened ventricles temporally and occipitally; the visual fields were normal. At an examination 4 years after the accident, he had reduced sensibility in his hands and feet and slight tremor and ataxia.

A more comprehensive neurological examination 7 years after the accident showed several minor deficits. He had bilateral exophthalmus and a slight aphasia. There was ataxia in his hands and feet, and a positive Romberg's sign. The vibratory sensibility was reduced at the ankle level, the sense of temperature was reduced in a stocking-like pattern, and the Achilles tendon reflexes were weak. EEG demonstrated

a slight bitemporal theta dysrhythmia with left-side preponderance, and visual evoked responses (VER) showed a borderline value for P2 latency on the left side. Cerebral CT displayed some cortical atrophy and a localized widening of the third ventricle and of the posterior horns of the lateral ventricles. This was interpreted as a partial agenesis of the corpus callosum. Findings at electromyography (EMG) and neurography were compatible with a slight motor polyneuropathy in the legs.

Neuropsychological examination was performed 4 and 7 years after the accident. There was very good agreement between the results at the two examinations. The main finding was considerably reduced learning and retention (Table II). He learned maximally six of ten words in eight trials at the first examination, seven words at the second examination. After 1 hr, he remembered only one and three words, respectively. Learning of 15 word pairs was discontinued due to slow learning. Pegboard and static steadiness showed reduced motor function.

Thyrotoxicosis may explain some of his early symptoms, but the neurological findings cannot be explained by changes due to this or to age alone. The abnormalities at cerebral CT were read as a possible agenesis of the corpus callosum and cortical atrophy at the last examination. Such agenesis may be asymptomatic, and in that the patient apparently had functioned normally before the accident, it is unlikely that this anomaly alone explains his symptoms and clinical abnormalities.

Patient 4

A 53-year-old man, who had worked in the herring oil industry for 13 years, lost consciousness while trying to help patient 3. He was rescued after about 10 min and woke up in the ambulance. According to the hospital case records, he had headache at arrival and was discharged without symptoms after 2 days. A week later he resumed his work, but, due to nervous trouble, aversion to the smell of fish, and pain in the breast, he had to give up working and was awarded 80% disability pension 7 months after the accident.

He was examined 4.5 years later. He reported that after the accident his memory and vision were impaired and his movements were slower and more rigid. A clinical examination showed slight reduction of distal sensibility in his feet, slight tremor and ataxia, and positive Romberg's sign. The main finding at the neuropsychological examination was reduced motor function (Table II).

At a neurological examination 6 years after the accident, his symptoms were unchanged. EEG was marginally normal, with more theta activity than expected for his age.

Patient 5

A 30-year-old man who worked in a tannery lost consciousness during work in a waste tank. He was found after maximally 15 min of unconsciousness. He was conscious on arrival at the hospital but had slight respiratory acidosis and a severe headache. He was discharged after 2 days. Three months later, EEG showed diffuse slow activity; air encephalography and analyses of cerebrospinal fluid were normal. He then returned to his work.

He was reexamined 8 years later, due to persistent headache. EEG showed slight generalized dysrhythmia. The clinical neurological examination was normal, except for failing short-term memory. A neuropsychological examination after 10 years showed generally reduced function, most pronounced for tests of visual abilities

and visual learning and retention. He was still at work, but worked outdoors, due to increased susceptibility to odors.

Patient 6

A 31-year-old man, who worked at a shipyard, overhauling an oil drilling platform, suddenly lost consciousness inside the platform, after fetid water had leaked out. A worker who tried to help him fainted for a short while. The patient was rescued after 15–20 min; 2.5 hr later, after fresh air had been blown in, 200 ppm of H_2S was measured.

On arrival at the hospital, he was deeply comatose. His lips and nails showed slight signs of cyanosis. Blood gas status showed pH 7.41, pO_2 5.2 kPa, and pCO_2 6.3 kPa, and he had signs of pulmonary edema. He was treated with a respirator, woke up after 2 days, and appeared quite well. Five days after the accident, he complained of gradually reduced vision and hearing and became psychotic. He was more or less comatose, with periods of extreme motor activity, for about 4 weeks. When he woke up, he was amnesic, nearly blind, and had reduced hearing, a moderate spastic tetraparesis, and ataxia. He was incontient of urine and feces.

After 2 months, he left the hospital, at which time an EEG showed moderate general dysrhythmia. A CT scan was considered normal. His vision was normalized, except for narrowed visual fields. A pure-tone audiogram showed a U-shaped sensorineural pattern, with maximum hearing loss at 2,000 Hz bilaterally, and a speech audiogram was comparably much more reduced, compatible with cortical damage. One month later, the audiograms were improved. Neuropsychological examination 4.5 months after the accident showed tremor and ataxia and slightly reduced memory.

When he was reexamined 5 and 6 years later, he had been awarded a 100% disability pension. His symptoms had not improved during the last 3–4 years. The clinical neurological examination showed slight pathology. EEG showed theta dysrhythmia with frontal preponderance; MRI showed slight cerebral atrophy. The neuropsychological examination (Table II) yielded mainly normal results. The exceptions were some of the tests of learning and retention and of motor function. Details concerning this patient and delayed sequelae will be reported separately.

DISCUSSION

Exposure

H_2S must be presumed to be the main cause of the hypoxia in all these cases. A high concentration of H_2S was measured shortly after the accident in patient 6. Relatively high concentrations were repeatedly measured at the working place of patient 1. In the latter case, mercaptans, which often occur together with H_2S in sewage treatment plants, may have contributed to the hypoxia. Most often the concentrations of both are below 1 ppm in such plants [Kangas et al., 1986].

In addition to H_2S, high concentrations of CO_2 and NH_3 and low concentrations of O_2 have been measured in fishing holds [Dalgaard et al., 1972]. However, patient 3 immediately lost consciousness when he started stirring the fish, and this indicates that H_2S produced by anaerobic bacteria in the decaying fish was the main cause.

It is well known that pumping of manure, especially from swine, involves risk of H_2S poisoning [NIOSH, 1977]. Cases of poisoning have also been reported among tannery workers [Smith and Gosselin, 1979].

Level and Duration of H_2S Exposure

Patient 1 had the shortest but probably the highest exposure. He had mainly symptoms of irritation of mucous membranes. The respiratory and visual symptoms, the skin peeling off in the face, and the complete loss of smell for 3 years indicated very high exposure. The olfactory nerve cells have the ability to regenerate, in contrast to other central nervous system (CNS) cells [Graziadei and Graziadei, 1979]. He probably did not inhale large amounts of H_2S and avoided significant brain damage, because he fell away from the gas.

Patient 2 was cyanotic and suffered severe brain damage, in spite of only 5–10 min of exposure. This suggests that the exposure had caused respiratory failure, and accordingly was higher than 1,000 ppm [NIOSH, 1977; WHO, 1981]. Animal experiments [Haggard, 1925] have shown that exposure above 1,500–2,000 ppm causes an almost immediate paralysis of respiration, which is not reestablished spontaneously. Pulmonary edema, aspiration, and seizures may have contributed to and prolonged his hypoxic condition.

The other patients were probably unconscious in the H_2S gaseous atmosphere for 10 min or longer. Only patient 6 was reported to have had a slight cyanosis. The other patients had less serious acute symptoms. This indicates that the level of H_2S had been on the order of 1,000 ppm or lower. On the other hand, the rapid loss of consciousness following the gas exposure indicates a concentration of at least 500 ppm [NIOSH, 1977; WHO, 1981]. In cases of H_2S exposure without paralysis of respiration, the direct effect of H_2S on nerve cells is considered the main mechanism of toxic effect. H_2S prevents the cellular utilization of oxygen by combining to Fe^{3+} in the cytochrome oxidase complex.

Patient 6 developed delayed neurological deterioration, which started 5 days after the poisoning. He was probably exposed for a longer time than the other patients, 15–20 min (Table I). Pulmonary edema and aspiration may have prolonged the hypoxic condition. Delayed sequelae have most often been described after hypoxia caused by CO poisoning [Plum et al., 1962] but very seldom after H_2S poisoning. We are aware of only one published case [Ahlborg, 1951] of delayed sequelae after H_2S poisoning. The uncommon occurrence may reflect "the improbability of achieving the precise conjunction of circumstances necessary to produce the syndrome—a period of prolonged yet sublethal hypoxemia" [Ginsberg, 1979]. Serious H_2S poisonings most often occur in tanks or other closed spaces, and without prompt rescue there is a high risk of death.

The five patients with permanent brain damage had been unconscious for 5 min or longer in H_2S gas, whereas patient 1, who had mainly reversible symptoms, was exposed for less than 1 min (Table I). This is in accordance with the critical limit of 4–5 min for brain damage caused by hypoxia and confirms the importance of the duration of exposure.

In other reports of permanent brain damage due to H_2S poisoning, the duration of exposure has varied from 5–10 min [Arnold et al., 1985] to 20 min [Hoidal et al., 1986] or 30 min [Hurwitz and Taylor, 1954] or longer. In most published cases, the duration of exposure has not been reported.

On the other hand, many reports confirm that loss of consciousness caused by very short duration of H_2S exposure is generally followed by complete recovery. In another study, we registered eight farmers who had lost consciousness due to manure

gas, which consists mainly of H_2S [Skjelhaugen, 1986]. Six of the farmers had been unconscious for less than 1 min in the gas atmosphere. All of them had regained consciousness within about 3 min. None of them reported permanent symptoms.

Cases of brain damage caused by H_2S exposure without unconsciousness confirms the importance of the duration of exposure. One case of permanent and serious encephalopathy and polyneuropathy after 9 hr of exposure, but without unconsciousness, has been reported by Zeyer [1955] and Baader [1960]. Ahlborg [1951] has described 15 cases from the Swedish shale oil industry with long-lasting symptoms following poisonings without loss of consciousness. The majority had suffered repeated episodes of poisoning. This indicates subclinical damages, which are aggravated by repeated poisonings. Animal studies seem to confirm this [Savolainen et al., 1980].

Medical Findings

The symptoms and medical findings in our patients varied but there were also many similarities (Table I). The neuropsychological test results clearly demonstrate both the differences and the similarities (Table II).

Patient 2 was not oriented for time and place, which is typical of an amnesic syndrome. Patient 6 had a serious but transitory amnesic syndrome. Patient 3 had a moderate permanent amnesic syndrome. The other patients reported slight memory problems. However, patients 1 and 4 had normal results on tests of learning and retention (Table II). The high occurrence of memory problems is in accordance with the clinical experience that reduced memory is the most common symptom in hypoxic brain damage [Spinnler et al., 1980] and that the hippocampus is particularly vulnerable to hypoxia [Kirino and Sano, 1984; Pepito et al., 1987].

Motor symptoms, such as ataxia, tremor, and rigidity, were relatively pronounced in four patients. Contrary to the resting tremor in Parkinsonism, we found position/intention tremor, which may be difficult to differentiate from essential tremor. The deficits were most clearly demonstrated in pegboard and static steadiness tests (Table II). Damage to the basal ganglia is considered the main cause of the motor symptoms [Carconna, 1979].

Interestingly, simple visual reaction time was normal in the four patients tested. Reaction time has been proposed as a screening test for the presence of cerebral dysfunction [Elsass and Hartelius, 1985]. However, as also stated by these authors, reaction time is most sensitive to progressive cerebral disorders. Our results indicate that screening with one or a few tests cannot be recommended and that a relatively comprehensive battery of tests is necessary to show dysfunction due to H_2S.

Increased susceptibility to strong odors was common among our patients. This has been reported as typical of H_2S poisoning [Ahlborg, 1951]. The visual symptoms reported by most of our patients may be explained by effects on the parietooccipital cortex, which is particularly vulnerable to anoxia [Lund and Wieland, 1966; Caronna, 1979]. Patient 6 had transitorily reduced hearing, with maximum loss at 2,000 Hz, and a comparably much more reduced speech audiogram. This pattern is uncommon but has been described as typical of hearing loss following CO poisoning [Baker and Lilly, 1977].

The symptoms of our patients agree with other reports of H_2S poisoning. Both clinical experience and animal experiments indicate that brain damage due to H_2S

poisoning is identical to hypoxic brain damage from other causes [Lund and Wieland, 1966].

EEG was pathological in four of our patients but was described as normal in patient 2 who had the most serious brain damage. PEG, CT, or MRI was performed in four patients, with pathological findings in two. Other published case descriptions confirm that normal EEG and CT do not exclude permanent hypoxic brain damage [Min et al., 1986; Wasch et al., 1989].

Prognosis of Acute Poisonings

According to Baader [1931], the prognosis following H_2S exposure is serious, although two recent studies concerning Canadian oil and gas production reported no or few long-term adverse effects. Burnett et al. [1977] reported 221 cases of poisoning. Their only information of the exposure was that "most exposures necessitating hospitalization occurred in enclosed spaces, whereas exposure in the open air produced problems of lesser magnitude." Six percent of the patients died, but "no long-term adverse effects were apparent in the survivors." However, systematic neurological or neuropsychological examinations were not reported. Upon request, we received additional information from one of the authors. There was little evidence of objective neurological deficit in survivors. Soft data, however, included complaints of hazy memory, inability to concentrate, and emotional lability (King, 1984; personal communication).

Arnold et al. [1985] reported 250 cases of H_2S poisoning from the same industry; seven (2.8%) of the patients died. The only information about the exposure concerned a worker with permanent damage, "known to have had a relatively long period of hypoxia (probably 5–10 min), as well as a very high level (>200,000 ppm) of hydrogen sulfide exposure." This suggests that most patients had been exposed for a short time.

A report of numerous cases from a U.S. heavy water plant [Poda, 1966] seems to have covered mainly slight poisonings.

We may conclude that these large studies had scanty information concerning exposure and that most cases probably had been short-time exposed. Another explanation of the low number of reported sequelae is probably the lack of follow-up. The connection between the symptoms of our patients 3 and 4 and the H_2S poisoning was not established until our examination 4 years after the accident. According to Burnett et al. [1977], "no follow-up tracings recorded after discharge from hospital were available for evaluation." The same was the case in the study by Arnold et al. [1985]. A comparison with follow-up studies of CO poisoned patients is of interest. Studies with personal examinations at follow-up found that only a minority of the cases with sequelae had been registered when they left hospital [Smith and Brandon, 1973].

CONCLUSIONS

As illustrated by our patients, the outcome of acute H_2S poisonings is quite variable. Differences in duration and level of exposure may partly explain this. Recent reports of large groups of poisoned workers have stated a low risk of permanent impairment of cerebral functions. Lack of information about the duration and

level of exposure makes the results difficult to compare with reports of permanent impairment. Our patients with permanent impairment had been unconscious in H_2S atmosphere for more than 5 min. Inclusion of many cases with shorter duration of exposure in the former reports may explain the discrepancy. The lack of follow-up is another explanation. Follow-up may be necessary to exclude permanent impairment of CNS functions even in cases with seemingly transient symptoms. In addition, delayed neuropsychiatric sequelae may not be present at the time of discharge.

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