Respiratory and Circulatory Effects of Inhalation Exposure to Air Mixtures with Sublethal and Lethal Concentrations of Natural Hydrogen Sulfide-Containing Gas

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With the continuing development of new gas fields where hydrogen sulfide-containing gases are present, an ever-growing number of people are exposed to toxic gases during their recovery and processing, and cases of acute poisoning continue to occur, despite the introduction of automation and safety measures. Hence the interest of research into the mechanisms by which such natural gases cause damage to the human organism [4,6]. Although hydrogen sulfide has been described in the literature as generally an irritant gas, human beings may lose consciousness and die when exposed to its high concentrations [3,5]. The underlying mechanism of acute hydrogen sulfide poisoning is believed to be the primary impact of this gas on the central nervous system, including the respiratory and cardiovascular centers. Many investigators consider dysfunction of the respiratory and cardiovascular systems to be the major factor in poisoning [4]. It has been stated, in particular, that one cause of systemic changes in circulation in acute poisoning by a natural hydrogen sulfide-containing gas is the impairment of hemodynamics in the pulmonary circulation [2,6]. The natural gas from the Astrakhan condensed-gas deposit has several specific features, primarily high levels of hydrogen sulfide, carbon dioxide, and hydrocarbons which possess high chemical and biological activities. While the isolated effects of each of these gases on the respiratory function and cardiovascular system have been described in the literature, the impact of their combinations has been studied inadequately.

The purpose of this study was to examine hemodynamic and respiratory disturbances occurring in animals acutely poisoned with the hydrogen sulfide-containing natural gas from the Astrakhan condensed-gas deposit.

MATERIALS AND METHODS

The experimental procedure used is detailed in a following article [1]. In addition, in the study described here, where cats were exposed to air mixtures containing H_2S in sublethal or lethal concentrations, their survival times were recorded and, after their death, their lungs were dissected out, weighed, and studied both in the wet state and after a 2-week period of drying at 83°C.

RESULTS

In tests where cats were spontaneously inhaling a gaseous mixture containing 600 mg/m^3 of H_2S,

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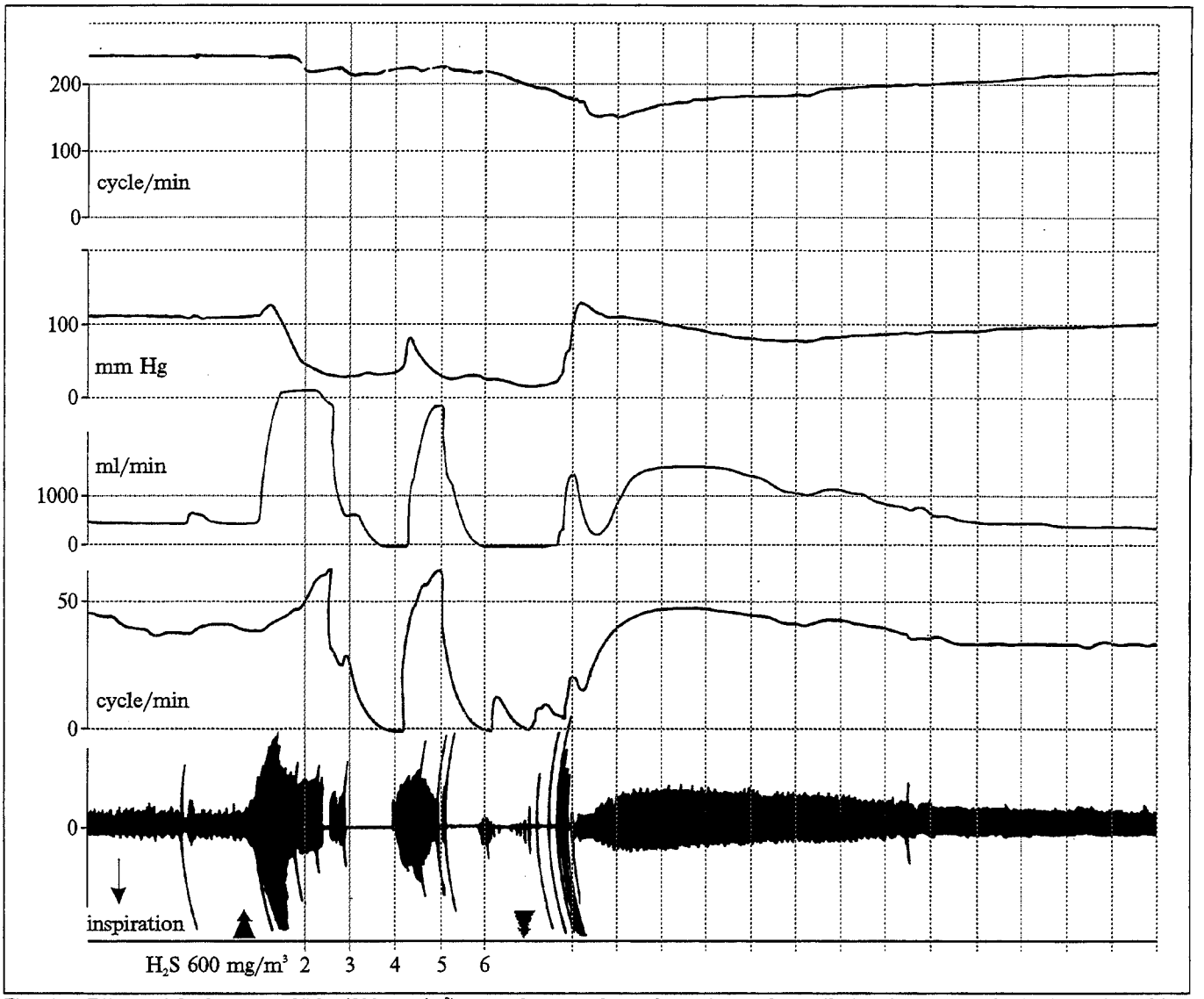

Fig. 1. Effect of hydrogen sulfide (600 mg/m³) on pulmonary hemodynamics and ventilation in an anesthetized cat breathing spontaneously. From the top down: heart rate, beats/min; systemic arterial pressure, mm Hg; minute volume of respiration, ml/min; breathing rate, breaths/min; pneumotachogram (inspiration); time marks (min). The two arrows indicate the beginning and end of gaseous mixture supply.

severe respiratory and circulatory disturbances were noted after 10-15 min of exposure in 30% of animals and within 20-25 min in all animals (Table 1; Figs. 1, 2). By minutes 20-25, the cats were exhibiting the terminal type of respiration (apneusis, gasping), accompanied by a sharp fall in the mean systemic arterial pressure (AP) and by decreases in minute volume and cardiac output, i.e., they were developing changes similar to those observed in peripheral collapse.

In the course of exposure to the gaseous mixture with an H_2S concentration of 600 mg/m³, the parameters of external respiration were undergoing periodic changes (Figs. 1 and 2) of various duration; however, after statistical treatment of the measurement results, these changes were smoothed out to be reflected just in increased errors of the arithmetic means, as can be seen in Table 1. Another feature of exposure to H_2S at 600 mg/m³ was that neither the breathing frequency nor minute volume was altered markedly during the first 10 min, i.e., these two parameters behaved in much the same way as they did in cats inhaling a gaseous mixture containing 250 mg/m³ H_2S (see the preceding article). Subsequently, between the 10th and 20th-25th min the values of the ventilatory parameters were rapidly decreasing and the terminal type of respiration often set in (Figs. 1 and 2). Whenever this life-threating situation occurred, the supply of the gaseous mixture was discontinued, after which the respiration returned to normal over a period comparable to that required

TABLE 1. Respiratory and Circulatory Parameters Measured in Anesthetized Cats Inhaling an Air Mixture Containing 600 mg/m³ Hydrogen Sulfide under Conditions of Spontaneous Respiration (*a*), and Artificial Respiration (*b*). The Values are Means±SEM

Respiration	Parameter	Time, min							
		0	1	3	5	10	15	20	*n*
a	MV, ml/min	1340±490	1760±630		1250±110	1070±240	1050±150	1000±170	4
	BR, breaths/min	28.2±1.9	29.1±3.4	27.1±3.9	23.4±1.6	26.2±4.6	24.5±3.2	35.0±3.6	8
	HR, beats/min	226±11	191±9*	202±11	193±13	198±10	194±15	196±31	9
	Mean systolic AP, mm Hg	85.3±8.2	68.6±7.3	61.4±10.7	66.9±10.7	89.0±5.3	66.3±11.4	77.3±8.9	9
b	CO, ml/min	179±18	181±21	201±7	221±15				4
	HR, beats/min	209±15	189±24	182±24	184±21	167±47			4
	Mean systemic AP, mm Hg	60.0±9.0	45.0±6.7	37.5±6.5	36.7±3.9	30.6±3.9*			4
	Mean pulmonary AP, mm Hg	15.4±3.9	12.8±1.7	10.3±1.1	14.9±1.7	9.6±1.8			6
	Blood flow in left lobar pulmonary artery, ml/min	64.5±6.8	54.7±7.5	48.6±8.2	49.4±16.1	62.5±15.6			5

Note. Asterisk denotes a significant difference at $p<0.05$; *n*: number of tests; MV: minute volume; BR: breathing rate; HR: heart rate; AP: arterial pressure; CO: cardiac output.

for respiratory disturbances to develop during exposure to an H_2S-containing gaseous mixture.

A similar pattern of variation was observed for the hemodynamic parameters: the initial minor changes in the heart rate and mean AP were followed by their large decreases (Figs. 1 and 2), although these occurred somewhat later than the decreases in the respiratory parameters. It may also be noted that the heart rate decreased significantly during the 1st minute of exposure to 600 mg/m³

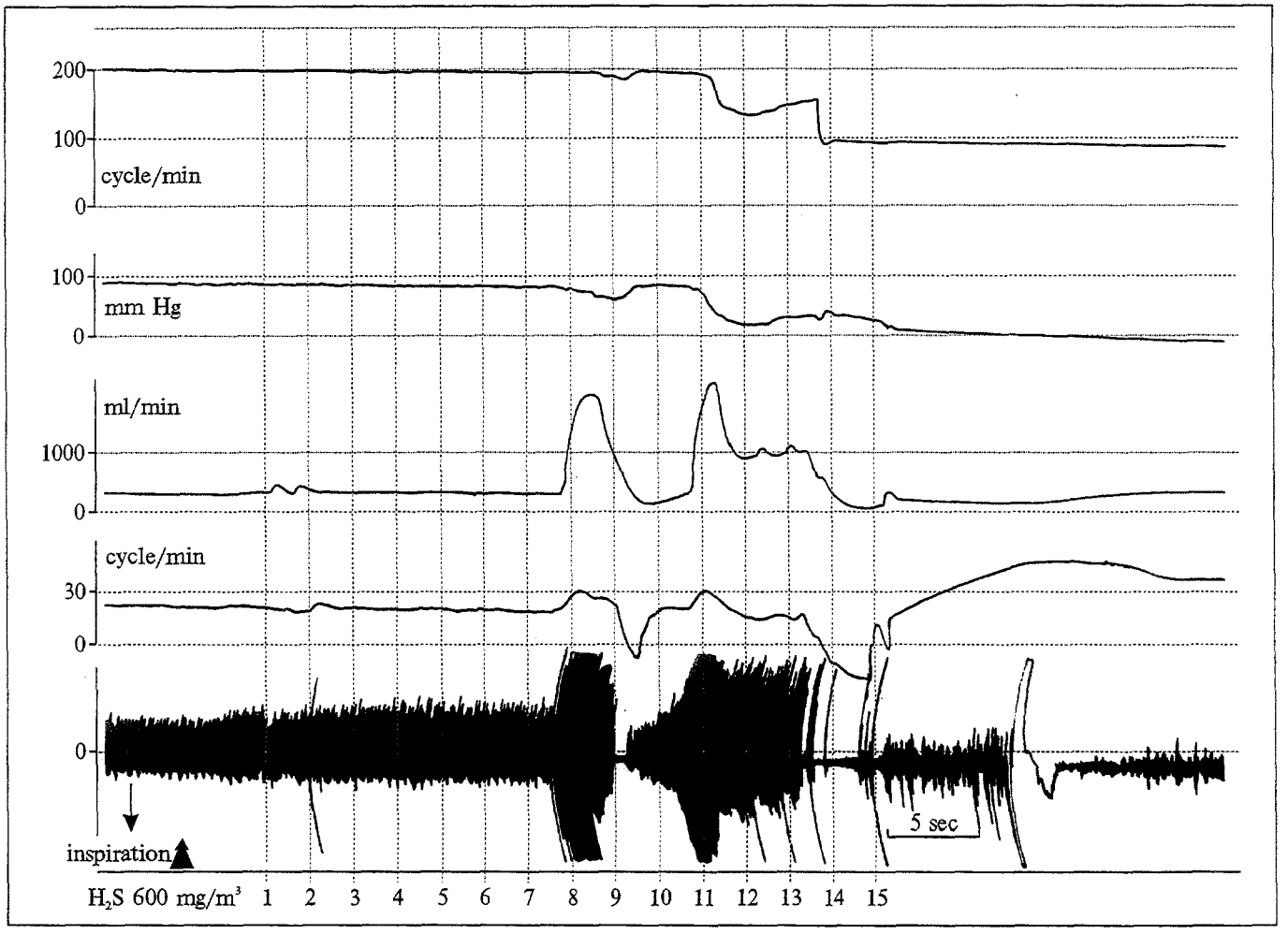

Fig. 2. Effect of hydrogen sulfide (600 mg/m³) on pulmonary hemodynamics and ventilation in another anesthetized cat breathing spontaneously. Same designations as in Fig. 1.

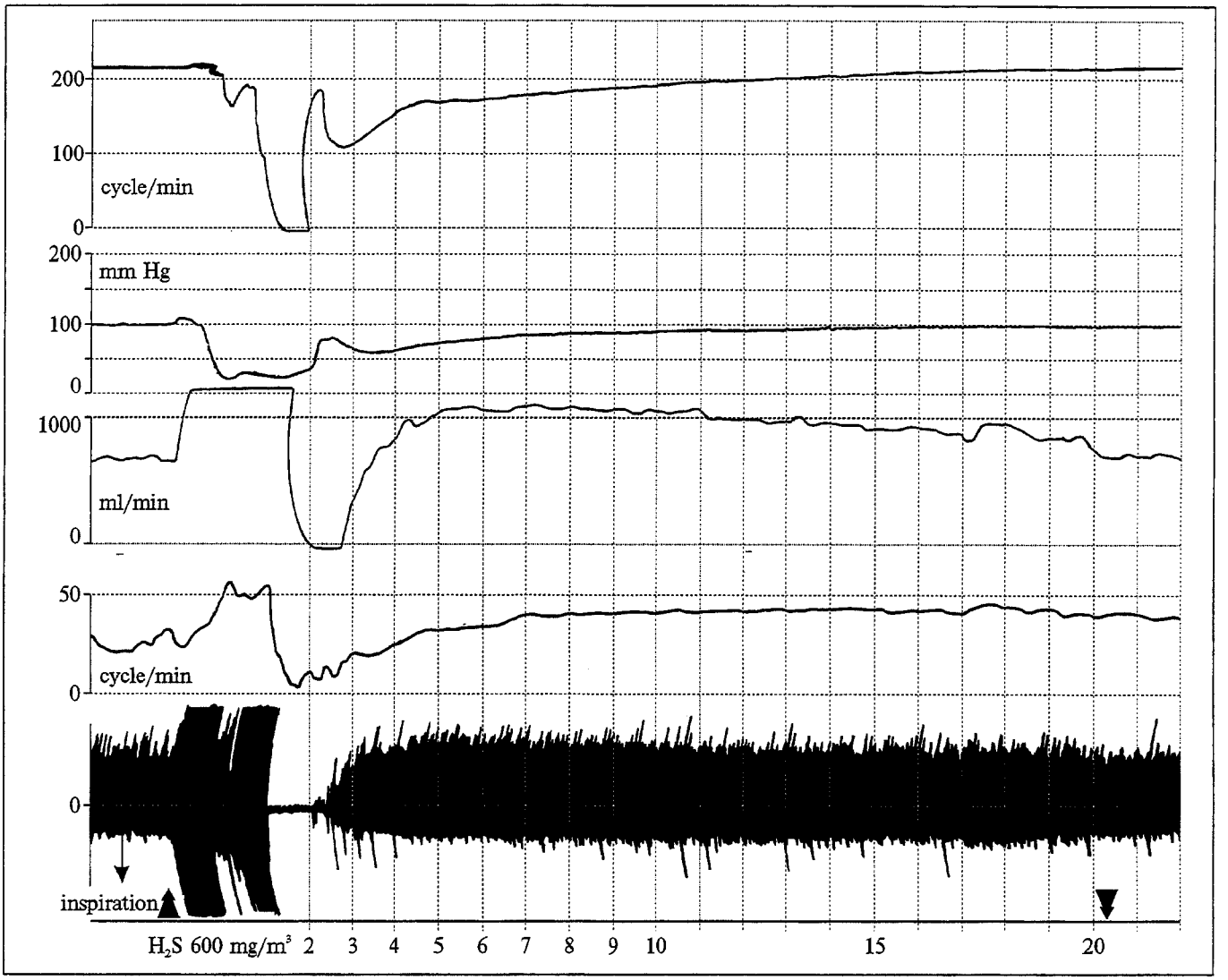

Fig. 3. Effect of hydrogen sulfide (600 mg/m^3) on systemic and pulmonary hemodynamics and on ventilation in an anesthetized and artificially ventilated cat with pneumothorax. Same designations as in Fig. 1.

H_2S and then rose, without, however, ever reaching its initial mean value.

In artificially ventilated cats inhaling the gaseous mixture containing 600 mg/m^3 H_2S, the parameters of pulmonary and systemic hemodynamics did not change in a consistent manner (Table 1). For example, whereas the cardiac output and pulmonary blood flow were maintained at a more or less stable level, the systemic and pulmonary arterial pressures progressively fell, possibly because of reductions in vascular resistance. These changes occurred in parallel with decreases in the heart rate. In general, however, the alterations in hemodynamic parameters were similar to those in the spontaneously breathing cats but developed more rapidly (Fig. 3). It should be noted that in the spontaneously breathing cats exposed to the high H_2S concentration (1000 mg/m^3), unlike in those exposed to 600 mg/m^3, the respiratory and hemodynamic parameters were not maintained by compensatory mechanisms for any length of time: in these cats, severe disturbances of the respiratory rhythm (periodic breathing, followed by apneusis and gasping) and a rapid and large fall in the mean systemic AP were observed together with a markedly progressive decrease in the heart rate.

Similar changes in heart rate and systemic AP were recorded in the artificially ventilated cats exposed to the gaseous mixture containing 1000 mg/m^3 H_2S. In these cats, however, decreases in pulmonary AP and in the volume blood flow rate in the lower lobar artery of the left lung were recorded as early as 3 min after the start of exposure. The high toxicity of H_2S at this concentration prevented the development of any compensatory reactions by the respiratory and cardiovascular systems.

Pulmonary edemas of varying degree were found in only 5 cats, probably because many of

the cats exposed to H_2S at 1000 mg/m³ died before edema could develop. In those five cats, the net gain in edematous fluid was less than that in blood, whereas in all other cats the reverse was true.

It is therefore very likely that by acting on cell membranes, H_2S makes vessel walls more permeable, thereby promoting the exit of blood into the interstitial space and increasing the hydration of the lungs.

In several tests, parameters of the oxygen regime were also recorded for arterial blood along with those of external respiration and hemodynamics. A quantitative analysis of these experimental data showed marked changes of the arterial blood reaction (pH) toward acidosis in artificially ventilated cats inhaling gaseous mixtures with various H_2S concentrations. It is noteworthy that the rate of blood acidification (i.e., that of the decrease in blood pH) was directly proportional to the H_2S concentration in the inhaled gaseous mixture. This is not surprising given that H_2S gas converts to a weak acid by dissolving in water, which results in an elevated hydrogen ion concentration in the solution.

In the spontaneously breathing cats, not only the acidification rate in arterial blood but also the direction in which its reaction was changed depended on how much H_2S the inhaled gaseous mixture contained: at low concentrations (up to 250 mg/m³), a shift toward alkalosis was usually observed, whereas at much higher concentrations (600 and 1000 mg/m³), the reaction shifted toward acidosis. The development of alkalosis in cats exposed to low H_2S concentrations appears to have been the result of an active respiratory response to the irritating action of H_2S; the reactions to high H_2S concentrations in the inhaled gaseous mixture are self-explanatory, but it should be noted that the exposure to 1000 mg/m³ H_2S resulted in a very rapid development of acidosis to life-threatening low values (2.7 units of pH/h). This provides further evidence for the extremely high toxicity of H_2S at this concentration.

In cats breathing spontaneously, oxygen tension was increased only when the inhaled gaseous mixture had a high H_2S concentration (600 or 1000 mg/m³). The failure of H_2S to alter oxygen tension at lower concentrations can probably be explained, on the one hand, by the weaker respiratory response to this gas and, on the other, by the alkalinization of the blood that occurred in the cats exposed to such concentrations.

Another indicator of the oxygen regime in arterial blood was the percentage content of oxyhemoglobin. In our experiments, it decreased from 94-96% to 88% or less, apparently as a result of the acidosis developing under the action of H_2S, although the possibility of H_2S directly altering hemoglobin affinity for oxygen cannot be ruled out.

The findings presented above clearly show that the inhalation of H_2S-containing gaseous mixtures adversely affects the oxygen regime of arterial blood.

In summary, this study, in which several major parameters characterizing the functioning of vital systems (respiratory and circulatory) were recorded simultaneously in anesthetized cats breathing spontaneously or ventilated artificially, has enabled us to detect signs of impaired activity of these systems caused by the inhalation of H_2S-containing mixtures. Disturbances of the regulation of respiratory and cardiovascular functions appear to have occurred even in some of the cats acutely exposed (up to 100 min) to low H_2S concentrations (200-250 mg/m³). Some such disturbances were present in a latent, compensated form, but compensatory reactions only occurred in animals inhaling low to moderate H_2S concentrations and, moreover, were incomplete because of the acidosis developing in arterial blood.

In cats inhaling gaseous mixtures with high H_2S concentrations (600-1000 mg/m³), the respiratory rhythm was disrupted and a retrograde blood flow was observed in the pulmonary arteries during certain periods. As indicated by the postmortem examination of animals that had been inhaling a gaseous mixture high in H_2S, this gas, by making the walls of pulmonary vessels more permeable, favors hydration of the lungs and increases the probability of pulmonary edema.

The results of the above experiments strongly suggest that even a short-term (for several minutes) inhalation of a gaseous mixture with a high hydrogen sulfide content is extremely dangerous and may have catastrophic consequences.

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