

Piratox sheet #2 "Hydrogen arsenide or arsine"

! Key points not to forget

- The 1st emergency measures are:
 - extraction of victims from the hazard area: respiratory protection of rescuers is essential;
 - emergency decontamination (first and foremost undressing) of victims, possibly completed by thorough decontamination depending on the context¹.
- Treatment is symptomatic and linked to the existence and extent of intravascular haemolysis.
- As a general rule, the shorter the symptom onset time, the more serious the intoxication and the more severe the symptoms.
- Asymptomatic patients shall be kept under observation for at least 6h due to the risk of haemolysis.
- Symptomatic patients shall be kept under observation for at least 48h to monitor the onset of acute effects. Schedule increased observation of patients presenting with significant symptoms.
- The mode of hydrogen arsenide intoxication is mainly by inhalation.
- Hydrogen arsenide can lead to delayed arsenic intoxication.
- For additional information concerning the risk, assistance with patient treatment and follow-up, we recommend contacting the poison control centers, military health service, or referring healthcare establishments.

1. Pharmaco-toxicological class of the toxic compound

Hydrogen arsenide is a powerful haemolytic poison that acts directly on red blood cells, causing massive intravascular haemolysis.
It should be noted that inorganic arsenic is carcinogenic in humans.

2. Physicochemical properties of arsine relevant to treatment

ARSINE (Hydrogen arsenide, AsH ₃) CAS number: 7784-42-1	Comments	Characteristics and values
Physical state of the product	Gas at ordinary temperature	BP* = - 63 °C; MP* = - 116 °C;
Vapour phase dispersion	Gas heavier than air	Vapour density: d= 2.7
Water-solubility	Water-soluble up to 200 mL for 1L of water.	
Transfer of contamination	Not expected	

*BP: boiling point = temperature of transition from liquid to vapour state.

**MP: melting point = temperature of transition from solid to liquid state.

¹Decontamination procedures (cf. french circular no. 700/SGDN/PSE/PPS of November 7th 2008 and introduction sheet).

3. Main intoxication characteristics

Arsine is present only in gaseous form (BP = -63°C). Arsine enters the body mainly via the respiratory route. Skin penetration has never been reported and remains negligible relative to the respiratory route. Highly liposoluble, arsine rapidly crosses alveolar-capillary and red blood cell membranes, causing intravascular haemolysis.

Specific route of exposure: respiratory route				
Toxic doses	AEGL: Acute Exposure Guideline Levels, limit AsH ₃ concentrations in air, in parts per million (ppm), beyond which health effects are likely to develop over a given exposure time.			
	Time of exposure to AsH₃	AEGL₁ discomfort	AEGL₂ chronic impairment	AEGL₃ death or risk of death
	30 minutes	Exposure not recommended	0.21 ppm	0.63 ppm
Main symptoms	<p>During the acute phase, the symptoms and onset time vary according to intoxication intensity.</p> <ul style="list-style-type: none"> ▪ <i>In the event of mild intoxications and/or prolonged exposure to low concentrations, the following is observed:</i> <ul style="list-style-type: none"> - asthenia, headaches, nausea, muscle weakness, aches, - appearance of "port wine" colored urine, - garlic-smelling breath. ▪ <i>In the event of pronounced acute intoxications, the following is observed:</i> <ul style="list-style-type: none"> - headaches, dizziness and shivering, digestive signs (nausea and vomiting), abdominal, lumbar and muscle pains, - massive intravascular haemolysis with its consequences= disseminated intravascular coagulation, hyperkalaemia, metabolic acidosis, state of shock, acute pulmonary oedema, acute anuric kidney failure. ▪ <i>In hyperacute forms:</i> <ul style="list-style-type: none"> - loss of consciousness/syncope/coma, - the situation rapidly progresses to death by acute circulatory failure and multiple organ failure. <p><u>Complications:</u></p> <ul style="list-style-type: none"> - death by cardiovascular collapse associated with the massive haemolysis and by direct myocardial toxicity, - oligo-anuric kidney failure associated with the haemolysis and direct tube impairment; - anaemia. <p>Following the acute phase, signs related to inorganic arsenic intoxication may appear.</p>			
Onset times after exposure	<ul style="list-style-type: none"> . 2 - 24 h: headaches, malaise, weakness, state of shock, dizziness, dyspnoea, thirst, abdominal pains, nausea, vomiting, pallor, jaundice, cyanosis. . 4 - 6 h: burgundy "haematuria" urine. . 24 - 48 h: jaundice. . In severe intoxications, the symptoms may appear as early as 30 to 60 minutes, but may be delayed up to 36 hours. 			

4. Antidotes (specific treatments):

No antidote for hydrogen arsenide-induced haemolysis has been approved to date.

The chelating agent dimercaprol (BAL®) has repeatedly been reported to be ineffective in preventing haemolysis, even when administered early. Subsequent monitoring of these patients should be organised due to the possible late complications associated with the presence of mineral arsenic. Indeed, there is a risk of secondary inorganic arsenic intoxication that could justify the administration of a chelating agent. In this case, initiate chelator treatment (DMSA® or BAL® have been suggested in the event of vomiting, see Piratome sheet no. 1, table A: treatment of arsenic) as rapidly as possible to prevent development of severe renal impairment.

5. Symptomatic treatments

Treatment is symptomatic and is dictated by the existence and severity of intravascular haemolysis:

- **Symptomatic treatment of acidosis and state of shock.**
- **Transfusions of packed red blood cells.**
- **Exchange transfusion.**
- **Dialysis**; renal impairment prevents the use of chelating agents.

Monitoring of acute effects:

- Asymptomatic patients: 6h observation for risk of haemolysis.
- Symptomatic patients: observation for 48h.

Special monitoring:

- Acute kidney failure.
- Delayed arsenic intoxication (e.g.: neurological disorders).