

GENERAL SPECIFICATION

Medical

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Specific risks: clinical and therapeutic elements

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1. Scope

The aim of this General Specification is to define the medical reference procedures to be followed in the event of acute and sub-acute poisoning and in response to risks associated with the local environment.

2. Reference documents

The reference documents listed below form an integral part of this General Specification.

External Documents

Unless otherwise stipulated, the applicable version of these documents, including relevant appendices and supplements, is the latest revision published at the effective date of this document.

Reference	Title
Report N° 398	Health aspects of work in extreme climates. A guide for oil and gas industry managers and supervisors, OGP, December 2008
ISBN 9781882417797	2008 Threshold Limit Values (TLVs) and Biological Exposure Indices (BEIs), American Conference of Governmental Industrial Hygienists

Total General Specifications

Unless otherwise stipulated, the applicable version of these documents, including relevant appendices and supplements, is the latest revision published in the applicable yearly collection.

Reference	Title
Not applicable	

3. Medical procedure for acute and sub-acute intoxication

3.1 Hydrogen Sulphide (H₂S)

3.1.1 Critical concentrations

The telltale odour of rotten eggs can serve as an alarm signal, but once the concentration has reached a level of 150 ppm, the olfactory nerve is paralysed.

A concentration of more than 1000 ppm causes acute neurological traumas after only a few seconds' exposure.

3.1.2 Toxic-physiopathology

Just like cyanide, H₂S acts as a cytochrome oxydase poison, blocking the final element in the electron transmission chain, thus inhibiting cellular respiration.

The symptoms which appear progressively are: cephalalgia (headaches), nausea, coughing,

dizziness, local irritation of mucous membranes and airways.

H₂S can cause bronchospasm and, at a higher concentrations, a pulmonary oedema with dyspnoea (shortness of breath) and respiratory distress, hypotension, disturbance of the cardiac rhythm, convulsions, coma and death. In the event of exposure to very high concentrations, death may be almost instantaneous.

3.1.3 Procedure to follow

- Transport patient in the open air and provide oxygen supply.
- Do not perform mouth to mouth respiration (ventilation using mask + Ambu).
- Endotracheal intubation and assisted ventilation if necessary.
- Controlling convulsions. Maintenance of haemodynamics. Fight against pulmonary oedema (lesional or combined) and cardiac rhythm disturbance.
- Nitrites (amyl nitrite) could theoretically be useful. However, this therapy is only effective if administered immediately at the site of the accident. A test for methemoglobinemia should be performed subsequent to nitrite use. Moreover, Nitrites are often “misused” as a euphoriant for self-intoxication, which means that they cannot be used in industrial practice.

3.1.4 Secondary effects

Neurological side effects in the form of memory problems, shaking, tiredness, balance problems, have been reported in patients who have been severely intoxicated.

3.2 Sulphur dioxide (SO₂)

3.2.1 Toxic-physiopathology

If inhaled: eye irritation, irritation of the mucous membranes, coughing, respiratory difficulties, inflammation and oedema of the respiratory tract (formation of sulphuric acid on mucus membranes), mental disorders.

If large quantities are absorbed: pulmonary oedema with respiratory distress, circulation and cardiac disorders.

3.2.2 Actions to be taken

In case of contact with the skin: wash off with plenty of water. Remove any contaminated clothing.

In case of contact with the eyes: rinse with plenty of water for at least ten minutes, keeping the eyelids wide open. Consult an ophthalmologist immediately.

In case of inhalation: transport patient in the open air or provide oxygen supply. Take preventive measures against bronchospasms (Beta mimetics).

In case the patient risks falling unconscious or is showing signs of respiratory distress: endotracheal intubation and assisted ventilation.

3.3 Smoke/ Fumes from fires

Fires cause three types of physical injuries:

- Thermal injuries – cutaneous burns or tracheobronchial burns.
- Traumatic injuries can be caused by an explosion (blast phenomenon, projections) or to a fall as the patient was fleeing the fire.
- Toxic injuries are the result of inhaling smoke/ fumes from the fire. Independently of the temperature of the gases, the smoke/ fumes cause damage due to their own toxic nature which depends on the type of materials which are burning.

3.3.1 Toxic-physiopathology

Carbon monoxide (CO) has a very high affinity for haemoglobin and myoglobin (resulting in an incapacitating effect). It also affects the mitochondrial respiratory chain.

Carbon dioxide (CO₂) is produced in very large quantities when something burns. It causes hypercapnia which in its turn causes hyperventilation, increasing the absorption of other toxic products.

Cyanhydric acid and cyanide derivatives block cellular oxidation. They provoke a state of shock and coma.

Other toxic derivatives are produced by the combustion of polymer-based industrial materials (PVC, etc.). They can cause pulmonary or systemic intoxication (hydrochloric and fluoric acids). The products of combustion of natural fibres, such as wool and silk or of synthetic fibres such as polyesters produces sulphurous anhydride and acid anhydrides. Aldehydes also induce pulmonary toxicity.

3.3.2 Actions to be taken

Intoxication by fire smoke/fumes often affects a number of victims simultaneously.

The toxic effects of fumes appear rapidly which means that the condition of the victims has to be rapidly assessed: number, circumstances of the accident (explosion, associated intoxication).

The medical response includes:

- Extracting the victims from the toxic environment.
- Maintaining the vital functions.
- Early oxygen therapy: freeing the respiratory tracts and administration of oxygen at a high flow rate (10 l/min using a high concentration mask) for all victims undergoing spontaneous ventilation.
- Confirmation of the diagnosis that fumes have been inhaled and prevention of delayed onset respiratory or toxic injuries. Dysphonia is a serious clinical symptom and could indicate a laryngeal oedema.
- If the patient shows signs of falling unconscious: endotracheal intubation and assisted ventilation (very broad indications).
- Blood samples should be taken as early as possible (at least two heparinised tubes to test carbon monoxide and cyanide levels).

- Search for signs of any change, even temporary, of the victims' state of consciousness (coma, confusion syndrome, aggressiveness).
- Haemodynamics: if an initial state of shock is detected (tachycardia associated with a collapsus) the patient should be examined for a post-traumatic hemorrhagic injury, which may require vascular filling and emergency surgery, or in the rarest cases, a myocardial failure (an attack of angina pectoris, myocardial infarction, or rhythmic disorder which may go as far as ventricular fibrillation). An ECG shall be performed and the patient monitored using an electrocardioscope.
- Injury report: cutaneous burns, traumatic injuries, blast, and ocular injuries – either burns or injuries caused by exposure to irritant gases.

3.3.3 Antidotes

The antidote to CO poisoning is oxygen.

The antidote for cyanide poisoning is hydroxocobalamin (commercialised in France as Cyanokit®). This product is sold freeze-dried in two phials of 2.5g.

Dosage of hydroxocobalamin: 5 g in a slow perfusion (15 minutes) to be repeated depending on the neurological and haemodynamic responses. In the event of cardiac arrest or persistent haemodynamic instability, repeat immediately after the first dose. The product tolerance is excellent. Cyanokit® shall be used at an early stage, on the disaster site itself.

3.4 Carbon monoxide (CO)

3.4.1 Origin

CO is an undesirable waste product of many thermal processes. Carbon monoxide is emitted whenever carbon or any of its composites is burned in an environment with a poor oxygen supply.

3.4.2 Toxic-physiopathology

The toxicity for humans and animals is associated with the fact that CO prevents the haemoglobin in red blood cells from being used. Red blood cells are responsible for transporting oxygen (CO affinity approx. 250 times greater than that of oxygen).

Absorption occurs solely by inhalation.

Carbon monoxide is invisible: it is odourless, colourless, and does not cause any skin irritations or pain.

Acute poisoning causes symptoms such as cephalgia (headaches) nausea, muscular atony, loss of consciousness, respiratory difficulties and may, depending on the concentration and length of exposure, cause death.

3.4.3 Actions to be taken

- Extracting the victims from the toxic environment.
- Maintaining the vital functions.

- Early oxygen therapy (oxygen is the antidote for CO poisoning), endotracheal intubation and assisted ventilation if the patient shows signs of falling unconscious.
- Confirmation of the diagnosis of CO poisoning from blood samples, which shall be taken as soon as possible (heparinised tubes).
- If the clinical examination, the results of blood tests and the ECG all confirm benign CO poisoning, the patient can be discharged after approximately 4 hours of observation. Otherwise, oxygen therapy shall be maintained and the patient kept under observation, in hospital for 24 to 48 hours.
- Hyperbaric oxygen therapy is the best treatment for severe carbon monoxide poisoning (objective neurological symptoms, myocardial ischemia). Call a hyperbaric specialist if necessary, for an expert opinion. In all cases, the effectiveness of the treatment depends on how quickly it is administered. Hyperbaric oxygen therapy is not contra-indicated for pregnant women in the event of severe poisoning.

3.5 Chlorine

3.5.1 Exploration & Production context

Chlorine is used to disinfect water. Another important chlorine derivative is bleach, which may give off chlorine vapours if mixed with an acid-based product.

3.5.2 Toxic-physiopathology

Chlorine has a highly caustic effect on the skin (chemical burn).

The symptoms of poisoning after inhalation are:

- Eye irritation and irritation of the mucus membranes in the airways.
- Painful cough accompanied by dyspnea (shortness of breath) cephalia (headaches) and vomiting.
- At a degree of severity one level higher: bronchospasms and acute lesional pulmonary oedema. This phenomenon may develop and lead to death if symptoms of acute respiratory distress continue.

The inhalation of chlorine vapours may be responsible for delayed onset respiratory disorders (up to 48 hours after exposure).

3.5.3 Actions to be taken

- Evacuate the patient from the contaminated environment, put them in the recovery position
- Administer oxygen by inhalation using a mask and bronchodilators (Salbutamol +/- atropinics) by nebulisation.
- If necessary: administer beta mimetics (Salbutamol, Adrenalin) by continuous injection using an electrical syringe and respiratory resuscitation by intubation and assisted ventilation.
- Clinical and radiological observation in a hospital environment.

3.6 Chlorinated solvents

3.6.1 Toxic-physiopathology

The inhalation of large quantities of chlorinated solvents causes a narcotic effect with depression of the central nervous system, preceded by a phase of excitation and ebriety.

Some of these agents, such as chloroform and trichloroethylene increase the heart's sensitivity to beta mimetics and can cause ventricular fibrillation.

Methylene chloride is partially metabolised as carbon monoxide with the formation of carboxyhaemoglobin up to a concentration of approximately 10%. In the event of poisoning, an acute coronary syndrome can occur in patients with coronary deficiencies.

Carbon tetrachloride causes the delayed onset (24 to 48 hrs) of hepatic damage (cytolysis which develops into hepatic failure) and acute anuric renal failure.

In the event of external contact, these agents can cause skin and eye irritation.

3.6.2 Actions to be taken

- Wash with plenty of water in the event of contact with the eyes or skin
- If ingested:
 - Gastric lavage is only possible after endotracheal intubation and assisted ventilation in a hospital environment.
 - Haemodynamic monitoring in case of chloroform or trichlorethylene poisoning. Adrenergic drugs are not recommended. Use of beta-blockers by continuous perfusion (Propranolol, Atenolol) in the event of cardiac rhythm disorder with myocardial hyperexcitability.

3.7 Hydrocarbons

3.7.1 Industrial context

Three types of hydrocarbons are discussed here; aromatic hydrocarbons (Benzene, Toluene, etc.), aliphatic hydrocarbons (Hexane) and alicyclic hydrocarbons (Cyclohexane).

3.7.2 Toxic-physiopathology

These are central nervous system depressants.

The inhalation of hydrocarbon vapours causes a state of euphoria with a feeling of ebriety followed by drowsiness, cephalia, vertigo and nausea followed by coma.

The ingestion of large quantities of hydrocarbons is followed by digestive disorders: nausea, vomiting with a sensation of buccopharyngeal, retrosternal and epigastric burning, diarrhoea. Frequently, an inhalation pneumopathy can also occur.

Skin and eye irritations may also occur in the event of prolonged contact with the product.

Moreover, they are substances which produce chronic toxicity requiring medical observation (occupational medicine). Animal testing has shown that certain substances belonging to the hydrocarbon group are carcinogenic and that some of them may even have a mutagenic effect.