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Chemical Emergency Medical Guideline

Information and recommendations for healthcare professionals

Nitrogen dioxide

CAS No.: 10102-44-0

GHS symbols:

GHS05
Corrosive

GHS06
Acute toxicity

Signal word: Danger

Hazard statements:

H314	Causes severe skin burns and serious eye damage.
H330	Fatal if inhaled.

Brief information

- A patient who is covered in nitrogen dioxide or whose clothing is covered in nitrogen dioxide may endanger other people through direct contact or through nitrogen dioxide gas emissions.
- Nitrogen dioxide and its vapors or fumes quickly cause burns on contact with tissues such as the eyes, skin and upper respiratory tract, causing symptoms such as irritation, burning, coughing, chest tightness and shortness of breath. Laryngospasm and toxic pulmonary oedema (shortness of breath, cyanosis, sputum, coughing) may occur.
- Ingestion of nitrogen dioxide can cause severe burns to the lips, mouth, throat, esophagus and stomach.
- There is no known specific antidote. Treatment depends on the extent of exposure and the symptoms.

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1. Information about the substance

Nitrogen dioxide (NO_2), CAS 10102-44-0
Synonym: Dinitrogen tetroxide (N_2O_4)
Up to 21°C, nitrogen dioxide is a colorless to yellow liquid. Above 21°C, it is a gas. The concentrated gas has a dark purple to black color. When diluted, it turns on a reddish-brown to yellow color. Nitrogen dioxide has a pungent, irritating odor at concentrations of 1 to 5ppm. Nitric acid is formed in contact with water. Nitrogen dioxide itself is not flammable, but it can increase the flammability of other materials or cause them to spontaneously combust. Nitrogen dioxide occurs naturally during the combustion of fossil fuels such as coal, oil or gas, as well as during the fermentation of grain in silos. It is also a component of smog. Nitrogen dioxide is released during the reaction between nitric acid and organic substances. It is also formed when nitric acid acts on metals, e.g. during electroplating, pickling and etching.

2. Exposition

2.1. Inhalation

The odor and irritant effect of nitrogen dioxide provide a clear warning of acutely dangerous concentrations.

2.2. Skin/eye contact

Direct contact of wet or damp skin with liquid nitrogen dioxide or concentrated vapors causes severe chemical burns. Nitrogen dioxide is hardly absorbed through the skin.

2.3. Ingestion

Ingestion of nitrogen dioxide can cause severe burns to the lips, mouth, throat, esophagus and stomach.

3. Acute health effects

3.1. Dose-response relationship

Nitrogen dioxide-concentrations		Effect/effects
1 – 5 ppm	-	Odor threshold (tolerance development possible)
5 – 10 ppm	-	Mild irritation of the mucous membranes
25 ppm	-	Immediate onset of chest pain, shortness of breath, coughing, bronchitis, usually completely reversible
50 – 150 ppm	-	Bronchiolitis, focal pneumonia, irreversible lung damage possible
> 100 ppm	-	Pulmonary oedema possible
1000 ppm	-	Fatal within a few minutes

3.2. Respiratory tract

Exposure to nitrogen dioxide usually causes dryness of the nose and throat and coughing. Inhalation of very high concentrations can lead to bronchospasm and ultimately to airway obstruction and death. The development of breathing difficulties with chest tightness and toxic pulmonary oedema (shortness of breath, cyanosis, sputum, coughing) can occur with a delay of more than 24 hours.

Exposure to nitrogen dioxide and nitrogen oxides causes acute and chronic changes in the pulmonary system, e.g. pulmonary oedema, pneumonia, bronchitis, bronchiolitis, emphysema, and possibly methemoglobinemia. Immediately after exposure, there are usually no symptoms other than a slight cough, fatigue or nausea. Nevertheless, even after initially minimal symptoms, the development of fatal toxic pulmonary oedema is possible.

Further acute effects may develop within 1-2 hours of exposure, e.g. tachypnoea, tachycardia, fine alveolar crackles, wheezing and cyanosis. Acute dyspnea and coughing may develop, for example, which slowly subside over two to three weeks.

A second phase may include sudden onset of fever and chills, more severe shortness of breath, cyanosis and pulmonary oedema. There is no correlation between the severity of the symptoms of the first phase and those of the second phase.

3.3. Skin contact

Deep chemical burns to the skin and mucous membranes can occur through contact with concentrated nitrogen dioxide; sometimes the skin turns yellow. Contact with less concentrated vapors or smoke can cause burning pain, redness and inflammation.

3.4. Eye contact

Severe eye burns with clouding of the surface of the eye and even penetration of the eyeball with subsequent blindness can result from exposure to liquid nitrogen dioxide. Low concentrations of vapor or smoke cause painful discomfort, spasmodic blinking or involuntary closing of the eyelids, redness and tearing.

3.5. Blood

Methemoglobinemia may occur only after very high exposure, but usually only to an extent that does not require specific treatment. ***For further information on the treatment of methemoglobinemia, see BASF CEMG Professional for Aniline.***

3.6. Gastrointestinal tract

Abdominal pain, nausea and vomiting may occur. If swallowed, diffusing chemical burns of the mucous membrane may affect the entire gastrointestinal tract.

3.7. Kidney

Acid-base balance disorders and acute renal failure may occur.

3.8. Possible consequences

Damage to the skin, eyes and mucous membranes caused by chemical burns may be irreversible, e.g. gangrene, blindness or esophageal stenosis. Complete recovery usually occurs after inhalation; in individual cases, symptoms and restrictions of lung function may persist. e restrictive and obstructive lung diseases may remain after bronchiolar damage. Destruction of lung tissue or scarring can lead to chronic e dilation of the bronchi and increased susceptibility to infections. Chronic or prolonged exposure may result in an increased risk of yellowing or erosion of the teeth.

4. Measures

4.1. Self-protection of first aiders

If there is a suspicion that the area the helper must enter may contain dangerous concentrations of nitrogen dioxide (see above), a self-contained breathing apparatus and a chemical protection suit must be worn. Contaminated equipment must not be used.
A patient who is wet with nitrogen dioxide or whose clothing is wet with nitrogen dioxide may endanger other people through direct contact or through nitrogen dioxide gas emissions.

4.2. Rescue

Patients should be removed from the danger zone immediately. If they are unable to walk unaided, they should be removed from the danger zone quickly using appropriate means, taking care to protect themselves. The "A, B, C procedure" then has absolute priority.

A) Clear the airways (check for blockages caused by the tongue or foreign objects)
B) Ventilation (check the patient's breathing; if necessary, begin ventilation with adequate self-protection, e.g. breathing mask)
C) Circulation (begin resuscitation for any person who does not respond to verbal commands and is not breathing normally)

4.3. Cleaning

Patients who have only been exposed to nitrogen dioxide vapors and show no signs of skin or eye irritation do not require any special cleaning measures, unlike all others. If possible, patients should assist with their own cleaning. If liquid nitrogen dioxide has been exposed and clothing is contaminated, it must be removed and securely wrapped.

If the eyes have been exposed to nitrogen dioxide or if there is eye irritation, they must be rinsed with water or a neutral saline solution for at least 15 minutes. Any contact lenses must be removed, provided this can be done without additional risk to the eye. Other important first aid measures must be continued during this time.

Rinse affected skin and hair with water for at least 15 minutes. Continue other important first aid measures during this time. Protect eyes while rinsing.

4.4. Initial treatment (preclinical or clinical)

The following measures are recommended if the exposure concentration is 10ppm or more, or if symptoms such as eye irritation or pulmonary symptoms are present, or if no exposure concentration can be estimated but relevant exposure is likely to have occurred:

- Oxygen administration
- Administration of 8 sprays of beclomethasone (800µg beclomethasone dipropionate) from a metered dose inhaler.

If there are signs of airway constriction (e.g. bronchospasm or stridor)

- Nebulization of adrenalin (epinephrine): mix 2mg adrenalin (2ml) with 3ml NaCl 0.9% and administer via a nebulizer mask
- Administration of a ß2-selective adrenoceptor agonist, e.g. four puffs of terbutaline or salbutamol or fenoterol (one puff usually contains 0.25 mg terbutaline sulphate; or 0.1mg salbutamol; or 0.2mg fenoterol); this can be repeated once after 10 minutes.

Alternatively, 2.5mg salbutamol and 0.5mg ipratropium bromide can be administered via nebulizer mask.
If inhalation is not possible, administer terbutaline sulphate (0.25mg to 0.5mg) subcutaneously or salbutamol (0.2mg to 0.4mg over 15 minutes) intravenously.
Intravenous administration of 250mg methylprednisolone (or an equivalent steroid dose).

If there are signs of toxic pulmonary oedema (e.g. frothy sputum, moist rales)

- CPAP therapy
- Intravenous administration of 1000 mg methylprednisolone (or an equivalent steroid dose)
  In case of (increasing) respiratory insufficiency, advanced airway management, e.g. endotracheal intubation or, if necessary, coniotomy.

Note: The efficacy of corticosteroid administration has not yet been proven in controlled clinical trials.

Skin contact with nitrogen dioxide can result in severe damage; this should be treated as burns: adequate fluid administration, analgesic therapy, maintenance of body temperature, covering the affected skin area with sterile dressing or a clean cloth.
Severe damage may also result from exposure to the eyes; this should also be treated as a burn. Consult an ophthalmologist immediately.

Note: Any exposure to liquid nitrogen dioxide in the facial area can have serious consequences.

If nitrogen dioxide is swallowed, do not induce vomiting under any circumstances.
If there are signs or symptoms of esophageal irritation or burns, the patient should be referred to an endoscopy center as soon as possible. An endoscopy should be considered to determine the extent of the damage (especially if gastrointestinal necrosis or perforation is suspected).
Only if a significant dose was swallowed less than 30 minutes before the patient's endoscopic examination and perforation can be ruled out should immediate gastric lavage be considered.

Patients who have been exposed to a concentration of 10ppm or more or who have ingested nitrogen dioxide, as well as patients without exposure measurements but with suspected relevant exposure, should be transported immediately to a hospital with intensive care facilities.

4.6. Further procedure and treatment

In addition to medical history, physical examination and vital signs, pulse oximetry, a p.a. chest X-ray and spirometry should be performed. Routine laboratory tests should include complete blood count, glucose and electrolytes. Arterial blood gases and methemoglobin concentrations should be determined in symptomatic patients to assess possible acidosis or methemoglobinemia.

Radiological signs of pulmonary oedema – enlargement of the hila, typical, centrally accentuated, patchy shadows on the chest X-ray – are late signs that often cannot be detected until 24 hours after exposure. The X-ray is typically normal on initial presentation at the hospital, even after inhalation of a relevant dose. Patients with possible exposure or with significant complaints or symptoms should be monitored for an appropriate period and examined repeatedly before any consequential damage to health can be ruled out.
Delayed effects are unlikely in patients with only mild, rapidly subsiding upper respiratory symptoms (mild burning or coughing).

If oxygen saturation falls below 90%, arterial blood gas concentrations must be checked immediately and the chest X-ray repeated. If blood gas concentrations deteriorate and/or the chest X-ray shows signs of toxic pulmonary oedema, oxygen should be administered via a mask. If deterioration manifests (especially in the case of tachypnoea (>30/min) and a simultaneous decrease in carbon dioxide partial pressure), CPAP therapy should be started within the first 24 hours after exposure.

In the event of pulmonary oedema developing, fluid intake and excretion as well as electrolytes should be closely monitored. A positive balance should be avoided. To optimize fluid management, the insertion of a central venous catheter should be considered.

If signs of pulmonary oedema persist, intravenous administration of methylprednisolone (or an equivalent steroid) should be continued at intervals of 8 to 12 hours.

Prophylactic antibiotic administration is not routinely recommended but may be considered based on the results of sputum cultures. Pneumonia may occur as a complication of severe pulmonary edema.

4.7. Discharge of the patient / instructions for further rules of conduct

Clinically asymptomatic patients who have been exposed to nitrogen dioxide concentrations of less than 10ppm (depending on the duration of exposure) and who show no abnormal clinical findings and no signs of toxic effects after an appropriate follow-up period may be discharged under the following circumstances:

- Information and recommendations for patients with instructions for further action were provided verbally and in writing. The patient was advised to seek immediate medical attention if any health problems arise.
- The patient is aware of and understands the toxic effects of nitrogen dioxide.
- The attending physician has been informed that regular contact between the patient and the physician is possible in the following 24 hours.
- Heavy physical work should not be carried out in the following 24 hours.
- Do not smoke or be exposed to cigarette smoke for at least 72 hours; smoke can impair lung function.
- Patients with serious skin or eye injuries should be re-examined after 24 hours.
- Spirometry should be repeated at regular intervals after discharge until the values have returned to the patient's baseline values prior to exposure.

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Administrative Information

Document Type	Chemical Emergency Medical Guideline
Number of Version	DE.1.0.0
Initial Publication	01.01.2026
Next Revision	2029
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