Organophosphorous esters

Definition of causal agent

Organophosphorous insecticides (OP) are derivative of esters, amides or thiols of phosphoric, phosphonic, phosphorothionic or phophonothioic acids. They act through an inhibition of acetylcholinesterase (AchE). This effect is not usually present in organophosphorous herbicides.

Main occupational uses and sources of exposure:
Use in agriculture and public health as insecticides and herbicides. Occupational exposure may occur also during the production and formulation of these compounds.

Toxic effects

1. Acute systemic effects

The time of onset and sequence of symptoms and signs are function of the type of OP compound, the entity of the absorbed dose and the absorption and metabolic pathways.

The clinical picture of acute organophosphorous insecticide poisoning is attributable to the inhibition of AchE activity in the nervous system and to the consequent acethylcholine accumulation in the nerve synapses and neuro muscle junctions (muscarinic and nicotinic systems).

Organophosphorous herbicides are not characterized by a significant acute toxicity.

Miscellaneous effects:
Profuse sweating, lacrimation, disturbed vision, muscular fasciculation, asthenia.

Effects on the digestive apparatus
Increases salivation, nausea, vomiting, diarrhoea, abdominal cramps.

Effect on the respiratory apparatus
Bronchial hypersecretion, tightness of chest, bronchioconstriction, dyspnoea, pulmonary oedema.

Effect on the cardiocirculatory system
Arrhythmia, hypotension, shock

Effect on the nervous system
Headache, dizziness, agitation, anxiety, mental confusion, tremors, convulsions, coma.

Exposure criteria:

Minimum Intensity of Exposure: Occupational Exposure confirmed, if possible assessed by:

- Anamnesis and study of working conditions showing a significant exposure. Also the possibility of skin absorption has to be taken into account.
- Significant reduction of AchE activity (at least 30%, but usually 50% as compared with baseline levels).
- When possible, it is recommended the determination of the compound or its metabolites in biological fluids.
- Environmental monitoring

Minimum duration of exposure: from a few minutes to several hours, depending on the intensity of exposure and the compound cause of poisoning.

Maximum latent period: three days.

2. Delayed effects

Intermediate syndrome

Onset: from 1 to 4 days after exposure.
Duration: 5-18 days.
Symptoms and signs: onset of proximal muscle weakness immediately after the acute cholinergic crisis, which might evolve into respiratory failure.

Exposure criteria:

Minimum Intensity of Exposure: Evidence of a previous severe acute OP poisoning (see ***exposure criteria for acute effects***).

Maximum latent period: from 1 to 4 days after exposure.

Peripheral neuropathy

Certain OP compounds may cause a peripheral neuropathy, usually involving motor nerves of the lower limbs. Exceptions are possible.

Symptoms and signs: typical picture of the second motor neuron impairment. In some cases, after the recovery of the flaccid paralysis, a spastic paralysis appears, consequent to an impairment of the first motor nerve at the level of the spinal cord.

Exposure criteria:

Minimum Intensity of Exposure: Evidence of a previous severe acute OP poisoning (see "exposure criteria for acute effects").

Maximum latent period: from 7 to 20-25 days after exposure and cholinergic crisis.

Neurobehavioral effects

Severe acute OP poisoning may lead to neurobehavioral changes with some features similar to those described in Annex I entry nr. 135 on ***Encephalopathies due to organic solvents which do not come under other headings.***

Exposure criteria:

Minimum Intensity of Exposure: Evidence of a previous severe acute OP poisoning (see "exposure criteria for acute effects").

Maximum latent period: Unknown

NOTE: Information about Carbamates are included in entry 122 (Organophosphorous Esters) because, although they are not organophosphorous compounds, acute overexposures cause clinical effects very similar to those of organophosphorous poisoning.

Carbamates

Definition of causal agent

Carbamates are nitrogen substituted urethanes. The salts and esters of substituted carbamic acid are more stable than carbamic acid itself. This stability is at the basis for the synthesis of many derivatives that are used as pesticides. Carbamate pesticides can be subdivided into three main classes: the substituted methyl substituted insecticides, the aromatic hydrocarbons substituted herbicides and the benximidazole substituted fungicides.

Main occupational uses and sources of exposure:
Use in agriculture and public health as insecticides, fungicides and herbicides. Occupational exposure may occur also during production and formulation of pesticides.

Toxic effects

1. Acute systemic effects

The clinical picture of acute carbamate poisoning is attributable to the inhibition of AChE (acetylcholinesterase) activity in the nervous system and to the consequent acetylcholine accumulation in the nerve synapses and neuro muscle junctions (muscarinic and nicotinic systems). Since, compared to Organophosphorous compounds, carbamates are weaker AChE inhibitors, the duration of AChE inhibition is shorter and the severity of the signs and symptoms of poisoning is usually lower.

Miscellaneous effects:

Profuse sweating, lacrimation, disturbed vision, muscular fasciculation, asthenia.

Effects on the digestive apparatus:

Increases salivation, nausea, vomiting, diarrhoea, abdominal cramps.

Effect on the respiratory apparatus:

Bronchial hypersecretion, tightness of chest, bronchioconstriction, dyspnoea, pulmonary oedema.

Effect on the cardiocirculatory system:

Arrhythmia, hypotension, shock

Effect on the nervous system
Headache, dizziness, agitation, anxiety, mental confusion, tremors, convulsions, coma.

Exposure criteria:

Minimum Intensity of Exposure: Occupational Exposure confirmed, if possible assessed by:

- History and study of working conditions showing a significant exposure. Also the possibility of skin absorption has to be taken into account.
- Significant reduction of AChE activity (at least 30%, but usually 50% as compared with baseline levels).
- When possible, the determination of the compound or its metabolites in biological fluids is recommended.
  Environmental monitoring

Minimum duration of exposure: From a few minutes to several hours, depending on the intensity of exposure and the compound cause of poisoning.

Maximum latent period: 24 hours.