Annex I 103.01

Carbon monoxide

Definition of causal agent

Carbon monoxide (CO) is, at ambient pressure and temperature, a colourless, odourless and non-irritant gas generated by incomplete combustion of organic material (coal, paper, wood, oil, gasoline, gas). It has a > 200-fold greater affinity for haemoglobin than oxygen.

Main occupational uses and sources of exposure:
The largest sources are motor vehicle exhaust, heating facilities, incineration and industrial processes. Occupations with potential exposure are numerous: garage personnel, fire-fighters, tunnel workers; petroleum, metallurgical, gas and chemical industries workers. Direct and/or indirect exposure to cigarette smoke also contributes to carbon monoxide exposure. Methylene chloride (used as paint stripper) is also metabolised to carbon monoxide, resulting in increased carboxyhaemoglobin levels (see section on ***Methylene chloride*** in Annex entry nr. 117).

Toxic effects

The principal cause of carbon monoxide toxicity is tissue hypoxia due to carbon monoxide binding to haemoglobin.

1. Acute and subacute effects

10%-30% Carboxyhaemoglobin (HbCO):

Headache, dizziness, weakness, nausea, confusion, disorientation and visual disturbances

30-50% HbCO:

Exertional dyspnoea, increases in pulse and respiratory rate, severe headache and syncope >50% HbCO:

Convulsion, coma, cardiopulmonary arrest. Complications occur frequently in carbon monoxide poisoning: immediate death, myocardial impairment, hypotension, arrhythmias, pulmonary oedema. Delayed development of neuropsychiatric impairment may occur within 1-3 weeks. Carbon monoxide poisoning during pregnancy may cause foetal death, developmental disorders and cerebral anoxic lesions in the foetus

☐ Exacerbation of ischaemic heart disease:

Prolonged exposure to carbon monoxide which gives rise to levels of carboxyhaemoglobinaemia in excess of 5 % can exacerbate a pre-existing heart disease, for example aggravation of angina pectoris and arrhythmia.

Exposure criteria:

Minimum intensity of exposure: Occupational exposure confirmed, if possible assessed by:

- History and analysis of the working conditions revealing a significant exposure to carbon monoxide,
- and, if available:
  - workplace air monitoring
  - biological monitoring:
  - carboxyhaemoglobin concentration in blood (sample taken at the time of removal from exposure before any treatment) or increase of carbon monoxide in exhaled breath.
  - The appearance of symptoms depends on the concentration of CO in the air, the duration of exposure, the degree of exertion, individual susceptibility, pre-existing cardiovascular or neurological diseases etc. (Note that the carboxyhaemoglobin concentration in heavy smokers can be as high as 10%)

Minimum duration of exposure: A few minutes to a few hours depending on the intensity in case of acute exposure, two weeks in case of sub acute exposure.

Maximum latent period:
For acute effects: 24 hours
For cardiovascular or neurological effects: 1 month

2. Chronic effects

Not well defined, although, prolonged exposure to carbon monoxide which gives rise to carboxyhaemoglobinaemia in excess of 20%, or following severe acute carbon monoxide poisoning can cause chronic detriment in neurobehavioural functioning.