Nitrous Oxide (N2O)



Nitrous oxide (N2O) is used as a propellant in food preparation apparatus. Recreational use or 'nanging' can lead to permanent neurological sequelae.

Toxicity / Risk Assessment

- Recreational use is referred to as 'nanging'
- Nitrous oxide bulbs are referred to as 'nangs'

 ('whippets', 'whip-its' or 'hippy crack')
- Acute exposure: short-lived and reversible CNS effects
- Risk of neurological dysfunction appears to be higher with regular, high-volume chronic use

Investigations

- Total B_{12} and active B_{12} (holotranscobalamin): may be low-normal or normal
- MRI: may demonstrate demyelination in either the central or peripheral nervous system

Clinical features

- Initial features are often consistent with sub-acute degeneration of the spinal cord with sensory changes, gait disturbance and impaired joint position sense
- Progression, motor and bladder dysfunction may occur
- Psychiatry: depression
- Haematology: macrocytic anaemia (uncommon), bone marrow suppression

Management

Chronic exposure leads to inactivation of vitamin B_{12} and reduction of available methionine, leading to demyelination in the peripheral +/- central nervous system

Reduction in tetrahydrofolate may lead to bone marrow suppression

Chronic Toxicity (neurological dysfunction associated with chronic high-volume N₂O use):

Encourage cessation of N₂O use

Vitamin B_{12} and methionine should be administered even if B_{12} concentration is normal

Vitamin B₁₂ (Hydroxocobalamin):

- Administer 1mg daily IM for 2 weeks, weekly for four weeks then monthly until maximal recovery

Methionine:

- Administer 1g TDS orally for 2 weeks.

Folinic acid:

- Administer 30 mg of intravenous folinic acid if there is evidence of bone marrow suppression Consider the presence of other nutritional deficiencies

Disposition

- If unable to safely mobilise or other significant N_20 related toxicity is present, admit for treatment until clinical improvement
- Referral to alcohol and drug service should be considered

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