

Case Report

Nitrous Oxide

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SHORT NOTE

This inhalational agent has been in use since 1844 [1]. Of late there have been numerous debates on whether Nitrous Oxide should be used at all in the clinical practice of Anaesthesia. As it appears to be gradually phasing out of anaesthetic practice, here is a quick review on the properties of this gas, its clinical advantages and disadvantages.

HISTORY

Prepared by Priestly in 1772, its use as an analgesic was first suggested by Sir Humphrey Davy in 1779. It was first used for Dental extraction in 1844 by Colton and Horace Wells [1].

PHYSICAL PROPERTIES

It is the only inorganic anaesthetic gas in clinical use. It is a colourless gas and is one and a half times heavier than air. It has a sweetish smell and is non-irritating. It has a molecular weight of 44, is neither flammable nor explosive but, supports combustion. Its boiling point is -88°C, blood/gas solubility 0.47, oil/water solubility 3.2, critical temperature 36.5°C, critical pressure 72.6 bar and MAC 105. It is relatively inexpensive but, concerns regarding its safety have led to continued interest in other alternatives such as xenon [1,2].

Chief impurities are

- Nitrogen.
- Nitric oxide.
- Nitrogen dioxide and higher oxides which are toxic and may produce methaemoglobinaemia and pulmonary oedema.
- Ammonia.

It is manufactured by heating ammonium nitrate to 240°-270°C. The liberated gas is then collected, purified and compressed into cylinders at 51 atm. During continuous use, the cylinder cools down due to latent heat of vaporization.

MECHANISM OF ACTION

It acts by modulation of enkephalins and endorphins within the CNS [2].

METABOLISM

It is rapidly eliminated from the lungs. It is not metabolised by enzymes in human tissue. Less than 0.01% undergoes metabolism by anaerobic bacteria in the gut. A small amount

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diffuses out through the skin [2].

Systemic Effects [3]

CNS: CNS depressant, Causes mild elevation of ICP due to increase in CBF and CMRO₂.

CVS: Negative inotropic and chronotropic effects & α adrenergic stimulation of peripheral circulation. It may cause direct myocardial depression. Combined effect results in modest increase in CO and HR when nitrous oxide is used in 50-70% concentration. It increases pulmonary vascular resistance particularly in people with pulmonary hypertension.

Resp: Increases respiratory rate, but decreases tidal volume. Hypoxic drive is depressed.

Neuromuscular: Minimal muscle relaxation. High concentrations may produce muscular rigidity. Does not trigger malignant hyperthermia.

Renal: Might cause a decrease in RBF & thus decrease GFR.

Advantages [1-7]

1. It is less soluble in blood, because of which, induction and recovery of anaesthesia are faster.
2. Low fat solubility makes the gas of low anaesthetic potency. It reduces the MAC of the volatile anaesthetics by about 50% when 70% of nitrous oxide is administered with oxygen.
3. Second gas effect- it results in alveolar concentration of volatile agent rising rapidly.
4. It is very stable & is not affected by soda lime.
5. It is a good analgesic.
6. Sedation for outdoor procedures.

Disadvantages [1-3,9]

1. Subacute combined degeneration of the spinal cord.
2. Polyneuropathy.
3. Long continued therapy may cause bone marrow

depression & agranulocytosis.

4. Prolonged nitrous oxide anaesthesia may cause diffusion of the gas into closed body cavities.
 5. Reacts *invitro* with Vit B12 & may impair DNA synthesis.
 6. It oxidises Vit B12 and results in megaloblastic anaemia in chronically exposed patients.
 7. Some reports suggest teratogenicity.
 8. It may alter immunological response to infection by affecting chemotaxis and motility of polymorphs. .
 9. Fink effect during recovery.
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