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التوثيق الالكتروني والميكروفيلم

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تحفظ هذه الأفلام بعيدا عن الغبار

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To be Kept away from Dust in Dry Cool place of 15-25- c and relative humidity 20-40%

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بالرسالة صفحات لم ترد بالاصل

STUDY OF SOME ENZYMES AS PREDICTORS FOR THE SEVERITY OF ACUTE CARBON MONOXIDE INTOXICATION

THESIS

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Historical Review

Carbon monoxide (CO) is a colourless, odourless, tasteless, and non irritant gas.[1] Because of these physical properties, CO gives no warning of its presence, hence its description as a silent killer.[2]

It normally appears in atmospheric concentration of 0.001% (10 ppm). However, The normal rate of endogenous CO production in man is 0.42 ml/hr as a result of normal hemoglobin (Hb) catabolic process.[3]

CO was first discovered with the introduction of hydrocarbon fuels which undergo incomplete combustion.[4]

Napoleon's surgeon; saw soldiers with CO-induced myonecrosis when billeted in huts heated by wood burning stoves.[5]

Incidence

True incidence of CO poisoning is not known, since non lethal exposures may go untreated or it may has been confused with other diseases as flu–like viral illness and gastroenteritis.[6,7] It has been estimated that one-third of all cases of CO poisoning are undiagnosed.[8]

CO poisoning is the most common cause of toxin induced deaths in the U.S. and Great Britain and may account for as many as 5.000 deaths each year in the U.S., with an additional 10.000 patients seeking medical attention for toxic exposure.[9,10] Mortality rates as high as 31% have been reported in United States (U.S).[11]

CO poisoning may be deliberate or accidental, in the later case, it may be : acute, subacute, or chronic.[12,13,14]

Sources

I-Endogenous:

i. Hemoglobin catabolism:

Endogenous production of CO results from metabolism of the methane carbon atom in the protoporphyrin ring during hemoglobin catabolism and produces a COHb% of 0.4 – 0.7 %.[3]

Degradation of heme to biliverdine and CO appears to be the dominating source in most species.[15,16] The enzyme heme oxygenase (HO), with 2 isoenzymes (HO-1 & HO-2), seems to be the rate limiting factor. HO-1 is reported to be irreducible, whereas HO-2 constitutively expressed[12] as HO-2-like immuno reactivity is seen in local parasympathetic ganglia of human trachea and bronchi. These findings suggest that CO serves as a modulator of synaptic neurotransmission in the lung.[13] The intensity of HO-1-like immuno-staining in the lung increases as the result of hypoxic challenge.[14]

Induction of HO-1 by reactive oxygen species followed by increased CO production has been put forward as a general cytoprotective mechanism against oxidative stress and therefore it is clinically useful in the detection and management of inflammatory lung disorders.[15] This assumption is supported by increased levels of CO in which the exhaled air of asthmatic patients, during periods of non–steroidal treatment, during asthma exacerbation, and as a result of allergen–challenge.[16,17]

Hemolytic anemia rises endogenous CO production and may increase COHb to 4-8 %.[17] Effects of anemia and carboxyhemoglobin on oxyhemoglobin dissociation curve are illustrated in figure 1.[17]