CO Poisoning

Carbon monoxide intoxication

Bleecker, M. L. (2015). Carbon monoxide intoxication. *Handbook of clinical neurology*, *131*, 191-203.

Carbon monoxide (CO) is a colorless, odorless, nonirritant gas that accounts for numerous cases of CO poisoning every year from a variety of sources of incomplete combustion of hydrocarbons. These include poorly functioning heating systems, indoor propane-powered forklifts, indoor burning of charcoal burning briquettes, riding in the back of pick-up trucks, ice skating rinks using propane-powered resurfacing machines, and gasoline-powered generators that are not in correct locations. Once CO is inhaled it binds with hemoglobin to form carboxyhemoglobin (COHb) with an affinity 200 times greater than oxygen that leads to decreased oxygen-carrying capacity and decreased release of oxygen to tissues leading to tissue hypoxia. Ischemia occurs with CO poisoning when there is loss of consciousness that is accompanied by hypotension and ischemia in the arterial border zones of the brain. Besides binding to many heme-containing proteins, CO disrupts oxidative metabolism leading to the formation of free radicals. Once hypotension and unconsciousness occur with CO poisoning, lipid peroxidation and apoptosis follow. Because COHb has a short half-life, examination of other biomarkers of CO neurotoxicity that reflect inflammation or neuronal damage has not demonstrated consistent results. The initial symptoms with CO exposure when COHb is 15–30% are nonspecific, namely, headache, dizziness, nausea, fatigue, and impaired manual dexterity. However individuals with ischemic heart disease may experience chest pain and decreased exercise duration at COHb levels between 1% and 9%. COHb levels between 30% and 70% lead to loss of consciousness and eventually death. Following resolution of acute symptoms there may be a lucid interval of 2–40 days before the development of delayed neurologic sequelae (DNS), with diffuse demyelination in the brain accompanied by lethargy, behavior changes, forgetfulness, memory loss, and parkinsonian features. Seventy-five percent of patients with DNS recover within 1 year. Neuropsychologic abnormalities with chronic CO exposure are found even when magnetic resonance imaging (MRI) and magnetic resonance spectroscopy are normal. White-matter damage in the centrum semiovale and periventricular area and abnormalities in the globus pallidus are most commonly seen on MRI following CO exposure. Though not as common, toxic or ischemic peripheral neuropathies are associated with CO exposure in humans and animals. The cornerstone for treatment for CO poisoning is 100% oxygen using a tight-fitting mask for greater than 6 hours. The indications for treatment with hyperbaric oxygen to decrease the half-life of COHb remain controversial.

Indoor air pollution and prevalence of acute respiratory infection amoung children in rural area of Bangladesh

Azad, S. Y., Bahauddin, K. M., Uddin, M. H., & Parveen, S. (2014). Indoor air pollution and prevalence of acute respiratory infection among children in rural area of Bangladesh. *Indoor Air*, *4*(2).

This study was undertaken in the village Shalchura under Nalitabari Thana in Sherpur district where total of 145 study populations (Children) were selected by systematic random sampling. Prevalence of acute respiratory infection (ARI) was seen in 76.6% of study children during last one year most of whom were found to be in the age group 0-5 years. The difference in the prevalence of acute respiratory infection (ARI) in 0-5 years group compared to prevalence of 6-18 years group were found to be statistically significant. Having a poor or lower medium class socio-economic background, majority of the family used mixture of biomass fuel like wood, cropresidue, cow-dung, saw dust, leaves etc. for cooking purpose. Association with disease symptoms to poor ventilation condition of the house and kitchen were also found to be significant. Another important factor for acute respiratory infection (ARI) symptoms was exposure to biomass fuel at daily cooking time. The disease symptoms tend to increase in 0-5 years group and the children who spent more time in cooking were more exposed to indoor air pollution as they have been showed more incidences of acute respiratory infection (ARI). It was revealed that due to smoking habit of family members and did that inside the living room, increased the occurrence of acute respiratory infection (ARI) among children. It was also found that use of mosquito's coils and poor living space increased the incidences of acute respiratory infection (ARI) among children. It was revealed that among the children with exposed (ARI) 41(28.8%) were liberated gases enter into their living room during cooking compared to 8 (23.5%) children in the unexposed (non ARI) group.

Assessing exposure to household air pollution: A systematic review and pooled analysis of carbon monoxide as a surrogate measure of particulate matter

Carter, E., Norris, C., Dionisio, K. L., Balakrishnan, K., Checkley, W., Clark, M. L., ... & Baumgartner, J. (2017). Assessing exposure to household air pollution: a systematic review and pooled analysis of carbon monoxide as a surrogate measure of particulate matter. *Environmental health perspectives*, *125*(7), 076002.

Household air pollution from solid fuel burning is a leading contributor to disease burden globally. Fine particulate matter (PM 2.5) is thought to be responsible for many of these health impacts. A co-pollutant, carbon monoxide (CO) has been widely used as a surrogate measure of PM 2.5 in studies of household air pollution. The goal was to evaluate the validity of exposure to CO as a surrogate of exposure to PM 2.5 in studies of household air pollution and the consistency of the PM 2.5–CO relationship across different study settings and conditions. We conducted a systematic review of studies with exposure and/or cooking area PM 2.5 and CO measurements and assembled 2,048 PM 2.5and CO measurements from a subset of studies (18 cooking area studies and 9 personal exposure studies) retained in the systematic review. We conducted pooled multivariate analyses of PM 2.5–CO associations, evaluating fuels, urbanicity, season, study, and CO methods as covariates and effect modifiers. We retained 61 of 70 studies for review, representing 27

countries. Reported PM2.5–CO correlations (r) were lower for personal exposure (range: 0.22–0.97; median=0.57) than for cooking areas (range: 0.10–0.96; median=0.71). In the pooled analyses of personal exposure and cooking area concentrations, the variation in ln(CO) explained 13% and 48% of the variation in ln(PM 2.5), respectively. Our results suggest that exposure to CO is not a consistently valid surrogate measure of exposure to PM 2.5. Studies measuring CO exposure as a surrogate measure of PM exposure should conduct local validation studies for different stove/fuel types and seasons.