

COHb: This value can be converted to the more conventional percentage saturation by $\% \text{COHb} = \text{COHb} / \text{OHb}_{\text{max}} \times 100$.

Tikusis et al. (1992) studied the rate of formation of COHb in healthy young males at a low (45 W) and moderate (90 W) exercise load. Ten nonsmoking subjects were exposed to CO on two separate occasions distinguished by the activity level. Each experiment began with an exposure to 3,000 ppm for 3 min during a rest period followed by three intermittent exposures ranging from 3,000 ppm for 1 min at low exercise to 667 ppm for 3 min at moderate exercise. The net increase in COHb after all exposures (about 10%) deviated by <1% between the values measured and the values predicted from the CFK model. Within this deviation, there was a general tendency of the CFK equation to underpredict the increase in COHb for the exposures at rest and the first exercise exposure and to overpredict levels for the latter two exposures at exercise.

Benignus et al. (1994) exposed 15 men to 7,652 mg/m³ (6683 ppm) CO for 3.1-6.7 min at rest. Except for the Haldane constant M, which was assumed to be 245, all other physiologic parameters of the CFK equation were measured for each individual from the very beginning of exposure. Arterial COHb was considerably higher than the venous COHb. The rate of increase in blood COHb and the arterial-venous COHb differences varied widely among individuals. The peak arterial COHb at the end of exposure ranged from 13.9% to 20.9%. The peak venous levels reached during the recovery period ranged from 12.4% to 18.1%. The arterial-venous difference ranged from 2.3% to 12.1% COHb. The CFK equation overestimated venous blood COHb, whereas arterial blood levels were significantly and consistently underestimated.

Hill et al. (1977) developed a mathematical model to predict values of blood COHb in mother and fetus for prolonged exposures to CO at 30-300 ppm. During CO exposure, fetal COHb lag behind maternal COHb by several hours. During prolonged uptake, fetal levels eventually overtake maternal levels and approach equilibrium values as much as 10% higher than the mother's due to the higher affinity of CO for fetal hemoglobin than for adult hemoglobin. During CO washout, the fetal levels again lag behind the mothers.

5. DATA ANALYSIS FOR AEGL-1

5.1. Human Data Relevant to AEGL-1

CO has no odor and does not cause irritative effects. A large number of studies investigated the effects of low CO exposure (COHb at <10%) on healthy individuals and high-risk groups. In these studies, effects on healthy persons, such as decreases in work capacity and decrements of neurobehavioral function, start at a COHb of 5% (WHO 1999a; EPA 2000).