difference in measured COHb (mean 21% [range 13-32%] in the younger group vs. 22% [16-26%] in the latter group). A short-term followup (3 months after the poisoning) suggested that medium intoxications (reported COHb of 16-27%) did not produce manifest sequelae except for a momentary standstill in the child's progress of about 2 months.

Kizakevich et al. (2000) reported that healthy young men can perform submaximal exercise without overt impairment of cardiovascular function after CO exposures attaining 20% COHb. Stewart et al. (1970) found that a CO exposure of healthy subjects resulting in 12.5% to 25.5% COHb did not affect the results of several neurophysiologic tests. Nielsen (1971) did not report on severe effects in three subjects that were repeatedly exposed to CO resulting in concentrations of 25-33% COHb. In a poisoning incident at the workplace, severe headaches, dizziness, weakness, nausea, chest pain, shortness of breath, and other symptoms were reported for a COHb of about 35% (Ely et al. 1995).

6.2. Animal Data Relevant to AEGL-2

In a study in cynomolgus monkeys, Purser and Berrill (1983) reported that during exposure to CO at 900 ppm for a total of 30 min, no signs of intoxication occurred until 20-25 min (corresponding to COHb of about 16-21%). At 25 min into the exposure, the animals' performance in a behavioral test significantly decreased. At the end of the exposure period, the animals became less active, most of them were lying down, but did not collapse. At 1,000 ppm, no effects were observed during the first 16-20 min. At this time, the animals became less active and sat down for short periods. At about 25 min, the animals went into a state of severe intoxication within 1-2 min, in which animals were lying down with eyes closed, they sometimes vomited and were virtually unable to perform coordinated movements.

Significant memory impairment in behavioral tests were found in young rats after continuous CO exposure throughout gestation (mean maternal COHb was 15.6%) (Mactutus and Fechter 1985).

In monkeys, a COHb of 9.3% resulted in reduced threshold for electric-shock-induced ventricular fibrillation (DeBias et al. 1976). Aronow et al. (1979) reported that CO exposure increased the vulnerability of the heart to induced ventricular fibrillation in normal dogs breathing 100-ppm CO for 2 h (resulting COHb was 6.3-6.5%). The ventricular fibrillation was induced by an electrical stimulus applied to the myocardium. A COHb of 13-15% increased the severity and extent of ischemic injury and the magnitude of ST-segment elevation in a myocardial infarction model in dogs (Sekiya et al. 1983).

6.3. Derivation of AEGL-2

The derivation of AEGL-2 values was based on effects in patients with coronary artery disease. An estimated 62 million people in the United States