The mean COHb measured at the end of the exposure was 32.9% (range 31.7-34.8%). CO_2 production, indicating the metabolism in the animals, decreased gradually throughout the exposure (statistically significant at 25 and 30 min of exposure) and then increased gradually toward pre-exposure levels during the recovery period (significantly lower until 15 min into the recovery period).

From earlier experiments, the authors estimated COHb of 16-21% for the period of 15-20 min when deficits in behavioral task performance were started during the exposure period. In the state of severe intoxication, the animals were capable of performing some coordinated behavioral actions when they were sufficiently stimulated (e.g., by loud noise or removing them from the chamber). The authors report that in unpublished experiments using higher CO concentrations, the animals passed rapidly from this stage to one of deep coma.

DeBias et al. (1976) reported that CO exposure (100 ppm for 6 h; resulting in a COHb of 9.3%) reduced the threshold for ventricular fibrillation induced by an electrical shock applied to the myocardium of monkeys during the final stage of ventricular repolarization. The voltage required to induce fibrillation was highest in normal animals breathing air and lowest in infarcted animals breathing CO. Additivity was found for the effects of infarction alone and CO exposure alone, each of which required significantly less voltage for fibrillation.

3.2.2. Dogs

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.

Sekiya et al. (1983) reported that exposure to CO concentrations of 3,000 ppm for 15 min followed by 130 ppm for 1 h (resulting COHb was 13-15%) increased the severity and extent of ischemic injury and the magnitude of ST-segment elevation, which was induced in anaesthetized dogs more by coronary artery ligation than by ligation alone.

3.3. Developmental and Reproductive Toxicity

3.3.1. Pigs

Dominick and Carson (1983) exposed pregnant sows to CO concentrations between 150 and 400 ppm for 48-96 h between gestational days 108-110 (average gestation was 114 days). They showed a significant linear increase in the number of stillbirths as a function of increasing CO concentration. Stillbirths were significantly elevated above control levels when the maternal COHb exceeded 23% saturation. These saturation levels were obtained at approximately 250 ppm.