room air or CO (100 or 200 ppm) administered in a randomized double-blind fashion. COHb measurements were performed before exposure, 30 and 60 min into exposure, at the end of exposure, and before and after exercise using an IL-282 CO oximeter. Exposures were stopped when the target level of 4% or 6% COHb was reached. Exposure durations were 94.2 ± 4.2 (SE) min (range 40 to 170 min) for the 4% level and 82.3 ± 2.9 (SE) min (range 39 to 135 min) for the 6% level. On all three test days, the mean pre-exposure COHb was 1.8%. The post-exposure and post-exercise COHb measured were 1.46% and 1.36% for air exposure, 4.01% and 3.93% for the 4% group, and 5.91% and 5.02% for the 6% group. Comparisons of arrhythmia data were done at 1.41%, 3.71%, and 5.33% COHb, respectively.

During the exposure period, the mean number of single VPD/h on the room-air day was significantly higher than that on the 4% COHb day, but no significant difference in the mean number of VPD/h was noted between room-air and 6% COHb exposure. When the baseline level of VPD frequency was controlled for by calculating the difference between the VPD frequency during exposure and the VPD frequency before exposure, there was no significant difference between the room-air and the 4% COHb exposure.

During the exercise period, the frequency of single VPD/h was greater on the 6% exposure day than on the room-air day (167 ± 38 vs. 127 ± 28 VPD/h; $p = 0.03$). This effect was still significant when the baseline VPD level was controlled for (117 ± 34 vs. 74 ± 26, $p = 0.04$). For this analysis, data from subjects in the low, medium, and high VPD frequency groups were pooled. The difference remained significant when all subjects, including those categorized in the "no arrhythmia" group were included in the analysis. The VPD frequency was not significantly increased at 4% COHb.

The initial findings (essentially negative) of this study in 10 patients with ischemic heart disease and no ectopy during baseline monitoring were published separately (Hinderliter et al. 1989).

Dahms et al. (1993) studied 28 men and 5 women with documented coronary artery disease and a minimum of 30 ventricular ectopic beats per hour over a 20 h period. On three testing days, the subjects were exposed in a randomized double-blind fashion to either room air or sufficient CO to increase their COHb concentrations to 3% or 5% in 1 h. The mean exposure concentrations during this hour were 159 ± 25 ppm and 292 ± 31 ppm, respectively. This was followed by a maintenance exposure to mean concentrations of 19.3 and 31 ppm, respectively, for an additional 90 min, which included the exercise test (after 60 min of equilibrium exposure) and the immediate post-exercise phase. The subjects then left the laboratory and resumed their normal daily activity to determine changes in ventricular ectopic beats after CO exposure. To this end, continuous 20 h ambulatory electrocardiograms were obtained with the recorder placed on the patients 2 h before CO exposure. There was no significant change in the frequency of single ventricular ectopic beats at rest from 115 ± 28 (in room air) to 121 ± 31 at 3% COHb and 94 ± 23 at 5% COHb. Exercise increased the frequency of ven-