tricular ectopic beats (from a baseline of 116 to 206 during exercise and 375 during exercise recovery for the room-air exposure), but there was no additional effect from CO exposure. Analysis of the data based on grouping of the subjects by the severity of disease (ventricular ectopic beat frequency, ejection fraction, and presence of exercise-induced ischemia) indicated no proarrhythmic effect of CO.

2.2.1.2. Healthy Adults

Chiodi et al. (1941) exposed each of 4 male subjects (aged 21-33 years) repeatedly to CO concentrations of 0.15-0.35% (1,500-3,500 ppm) for 70 min or longer. During 1 h before exposure, basal oxygen consumption, ventilation, pulse rate and blood pressure were recorded, and arterial blood for pH determination was obtained. The subject, remaining in rest during exposure, then breathed CO-containing air from a 600-liter gasometer. The measurement of the above mentioned parameters was continued during exposure. In one set of experiments, the test subjects reached 3.4% to 10.4% COHb (eight experiments in total with the following COHb at the end of exposure: 4.6%, 6.3%, 7.2%, 9.2%, and 9.8% in one subject and 3.4%, 9.5%, and 10.4% in the other). In another set of experiments, three subjects reached 27% to 52% COHb at the end of exposure (in 11 of a total of 22 experiments a COHb of 40% to 52% was measured). The following COHb values were measured at the end of exposure: 0, 31, 32, 32, 33, 39, 41, 42, 43, 45 and 52% in subject H.C., 0, 27, 35, 41, 43 and 48% in subject F.C. and 0, 0, 41, 42 and 44% in subject S.H. No statement was made on whether any symptoms were observed. The cardiac output increased 20-50% at COHb >40%, while the changes were negligible at COHb of <30%. No effects on the other parameters measured were found.

Henderson et al. (1921) exposed volunteers in a 6.4-m^3 gas-tight, steel-walled exposure chamber. CO was generated by dripping formic acid into strong sulfuric acid. A defined volume of CO was led into the chamber and mixed with an electric fan. Analysis of the exposure concentration in the chamber was done using the iodine pentoxide method. Subjects (9 men and 1 woman; number of subjects at each concentration given in brackets) were exposed for 1 h at 200 ppm (2), 300 ppm (3), 400 ppm (11), 500 ppm (1), 600 ppm (9), 800 ppm (4), 900 ppm (1) or 1000 ppm (1) CO. Blood samples were taken before exposure, at 30 min into the exposure, at the end of the exposure (60 min) and once or twice during the next 3 h after exposure. The COHb was determined using the carmine method. Directly after leaving the exposure chamber, subjects breathed several times into a bladder bag and CO was determined in the exhaled air using the iodine pentoxide method. CO concentrations in alveolar air after 60 min were 130-136 ppm at an exposure concentration of 400 ppm, 120-230 ppm at 600 ppm and 140-230 ppm at 800 ppm. The COHb percentage ranged from 11-12% at 200 ppm, 10-14% at 300 ppm, 14-22% at 400 ppm, 16-26% at 600 ppm, 26-