two subjects reported light-headedness after 20 min of exposure, which was believed to be due to hyperventilation. After 1 h of exposure, both subjects were aware of a 10% increase in heart rate with the minimal exertion of walking to the blood port. After 90 min of exposure, the second subject noted the onset of mild frontal headache. During the second exposure to CO at 500 ppm for 2.3 h, the same subjects developed mild frontal headaches after 1 h of exposure. Minimal exertion caused a transient intensification of the pain. Both headaches remained mild during the first post-exposure hour; then they intensified into excruciatingly severe occipitofrontal headaches, reaching a pain peak 3.5 h after exposure, and persisted for 7 h. During the third exposure at 500 ppm, the occurrence of mild frontal headaches was noted after 1 h of exposure. Immediately after exposure, both subjects were placed in a hyperbaric chamber and administered oxygen and the mild headaches were gone within minutes. The mean COHb reached after 2.3 h of exposure at 500 ppm was about 25.5%; after 4 h of exposure at 200 ppm, about 16.0%; and after 8 h of exposure at 100 ppm, about 12.5%.

In another experiment (Kizakevich et al. 2000) evaluating cardiovascular responses of exercising individuals, 16 healthy young men performed a sequence of brief (5 min) multilevel treadmill and hand-crank exercises at <2% COHb and again after attaining 5%, 10%, 15%, or 20% COHb on different days. Noninvasive impedance cardiography was used to estimate cardiac output, stroke volume, heart rate, cardiac contractility, and time-to-peak ejection time. The electrocardiogram was used to assess myocardial irritability and ischemia and changes in cardiac rhythm. The results showed that compensatory cardiovascular responses to submaximal upper- and lower-body exercise (e.g., increased heart rate, cardiac contractility, cardiac output) occur after CO exposures. These changes were highly significant for exposures attaining 20% COHb. The authors concluded that healthy young men can perform submaximal exercise without overt impairment of cardiovascular function after CO exposures attaining 20% COHb.

Nielsen (1971) investigated the effect of CO exposure on thermoregulation. Experiments were performed repeatedly on two subjects. Subject JHB reached COHb concentrations of 25% (mean of eight experiments) and 33% (four experiments), and subject PJC reached 30% (four experiments). After reaching the desired COHb concentration, the subjects exercised on a chair-ergometer for 1 h at a medium-to-high workload (mean heart rate 120-170 beats per minute). The subjects were not exposed continuously to CO during exercise, but the COHb level was maintained by breathing a calculated volume of CO from an anesthesia bag for 1-1.5 min every 15 min during exercise. CO exposure led to an increase in the plateau level of the deep-body temperature during exercise of 0.3-0.5°C. The lactic acid concentration was not increased after exercise at air exposure (120 mg/L in JHB and 79 mg/L in PJC) but increased during CO exposures (309-660 mg/L in both subjects). The authors stated neither the absence nor the presence of any symptoms of CO exposure.