

4.3. Issues Related to Postmortem CO Determination in Humans

4.3.1. Potential Factors Influencing COHb Levels

Data on the postmortem decay of COHb are sparse. Rodat et al. (1987) reported on the stability of CO after death. A CO poisoning due to a running truck engine in a nonventilated area was discussed. The autopsy was performed 10 months after the death due to insurance claims. The body was decomposed, but some muscle tissue was recovered and tested for COHb levels, which were 26%. Muscle tissue as well as other human tissues, such as brain, lung, and kidney, may be used for diagnosing death due to lethal exposure to CO (Vreman et al. 2006). The report did not discuss measurements from blood samples, presumably due to the decomposition of blood. Following death, the report indicated that COHb levels disintegrate over time, releasing reduced hemoglobin. In addition, the report indicated that the formation of sulfur compounds in a putrefied cadaver makes it difficult to interpret the absorption spectra of the COHb measurements, a phenomenon that has been acknowledged by others (Winek and Prex 1981; Kojima et al. 1986). Rodat et al. (1987) and Kojima et al. (1986) also suggested that the endogenous formation of CO after death is very low.

People who die of CO poisoning often show sublethal COHb levels in their blood (R. Coburn, personal commun., April 8, 2008). The lungs rapidly absorb CO, which avidly combines with hemoglobin at 230 to 270 times greater than with oxygen (Ellenhorn 1997; Larsen 2006). Oxygen therapy increases oxygen delivery and pulmonary excretion of CO by displacing CO from the hemoglobin and decreasing the half-life of COHb (Roos 1994; Ellenhorn 1997), in turn explaining the sublethal COHb levels in blood samples from deceased people exposed to CO.

CO shifts from the blood into the muscle tissue have been reported in the literature (Luomanmaki and Coburn 1969; Bruce and Bruce 2006). In order for shifts to occur, blood must be flowing in capillaries. Presumably during the moving of corpses after death, blood could be pushed through capillaries to a small extent (R. Coburn, personal commun., April 8, 2008), leading to CO shifts, but no studies were found reporting this phenomenon in human cadavers.

Oxidation of CO to CO₂ has been reported in living animals and humans. The rate of oxidation in skeletal and cardiac muscle was found to be small but still measurable (Fenn and Cobb 1932; Clark 1950; Luomanmaki and Coburn 1969). It is unknown whether this oxidation occurs in cadavers and what its effects are on the CO decay rates after death.

Once blood is collected from a cadaver, the postmortem samples may be measured more than once, and results would depend on storage and treatment of samples. Levine et al. (1990) found a 19% decrease in measured COHb levels when blood was refrigerated for 3 months and then frozen for 3 months. The refrigerated blood samples were first tested by microdiffusion techniques (sensitivity of approximately 5%) within 1 month of being obtained from victims of a