

**District III EMS
Quality & Improvement
“Case of the Month – March ‘05”**

This month’s case is more of an informational rather than a quality review. Most of you are aware of the Carbon Monoxide incident that occurred last month. There has been a great deal of interest in the subject so I thought a brief review of CO toxicology might be appreciated.

If you have any questions or comments, please feel free to contact me at Daniel.Wolfson@vtmednet.org

Chief Complaint: Carbon Monoxide Poisoning

Pre-hospital Course:

11:58 - An ALS crew arrives on scene of a carbon monoxide saturated apartment complex and received from the Fire Department a 20 yo female who was found in the apartment unresponsive. The patient was one of seven other individuals removed from the scene, with one patient being DOA. When the EMS crew assumed patient care she continued to be unresponsive with a respiratory rate of 4, fixed pupils with a rightward gaze, clenched jaw, and an irregular rhythm on the cardiac monitor. The crew appropriately provided assisted ventilations with 100% oxygen. Placement of an oral airway was attempted but unsuccessful due to the patient’s trismus (locked jaw). The patient was transported with continuous ventilatory support by bag mask valve to the Emergency Department.

Emergency Department Course: Pt arrives in the ED comatose with a GCS of 3 and assisted ventilation by bag mask. The patient was immediately intubated and ventilated with 100% oxygen. The Fire Department reported that the measured Carbon Monoxide level in the apartment where the patient was found was 350ppm. The patient’s carboxyhemoglobin level returned at 59.2. The patient had a volatile heart rate, alternating between tachy and bradycardic and a wide complex bigeminy. After continuous ventilatory support the patient’s heart rate stabilized to a narrow complex sinus tachycardia. The patient was transferred via life flight to a Hyperbaric chamber in Boston. Reports are that the patient is doing well.

Quality Improvement: The responding EMS services did an excellent job in the care of this patient. The prompt rescue and evacuation by the Fire Department, the rapid administration of oxygen by bag mask and transport by the ambulance crew, and the immediate intubation and additional oxygenation and ventilatory support in the ED undoubtedly pulled this patient back from the brink of death.

Carbon monoxide (CO) is a colorless, odorless gas produced by incomplete combustion of carbonaceous material (in this case it is thought that a ventilation pipe from the furnace become dislodged and exhaust fumes were pumped into the building). CO toxicity causes impaired oxygen delivery and utilization at the cellular level. CO affects several different sites within the body but has its most profound impact on the organs with the highest oxygen requirement (e.g., brain, heart).

Toxicity primarily results from cellular hypoxia caused by impedence of oxygen delivery. CO reversibly binds hemoglobin, resulting in relative anemia. Because it binds hemoglobin 230-270 times more avidly than oxygen, even small concentrations can result in significant levels of carboxyhemoglobin (HbCO).

An ambient CO level of 100 ppm produces an HbCO of 16% at equilibration, which is enough to produce clinical symptoms. Binding of CO to hemoglobin causes an increased binding of oxygen molecules at the 3 other oxygen binding sites, resulting in a leftward shift in the oxyhemoglobin dissociation curve and decreasing the availability of oxygen to the already hypoxic tissues.

CO binds to cardiac myoglobin with an even greater affinity than to hemoglobin; the resulting myocardial depression and hypotension exacerbates the tissue hypoxia.

HbCO levels often do not reflect the clinical picture, yet symptoms typically begin with headaches at levels around 10%. Levels of 50-70% may result in seizure, coma, and fatality. This pt had a level of 59.2 and the ambient CO level in the apartment was 350ppm, both well above the toxic level.

CO is eliminated through the lungs. Half-life of CO at room air temperature is 3-4 hours. One hundred percent oxygen reduces the half-life to 30-90 minutes; hyperbaric oxygen at 2.5 atm with 100% oxygen reduces it to 15-23 minutes. Thus, the most important action in the care of the Carbon Monoxide poisoned patient is the rapid administration of oxygen and ventilatory support if necessary.



Prehospital Care:

- Promptly remove from continued exposure and immediately institute oxygen therapy with a nonrebreather mask.
- Perform intubation for the comatose patient or, if necessary, for airway protection.
- Institute cardiac monitoring. Pulse oximetry, although not useful in detecting HbCO, is still important because a low saturation causes an even greater apprehension in this setting.
- Give notification for comatose or unstable patients, because rapid or direct transfer to a hyperbaric center may be indicated.
- If possible, obtain ambient CO measurements from fire department or utility company personnel, when present.
- Early blood samples may provide much more accurate correlation between HbCO and clinical status; however, do not delay oxygen administration to acquire them.
- Obtain an estimate of exposure time, if possible.
- Avoid exertion to limit tissue oxygen demand.