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Special Advisor to Governor Joe Manchin, III
Sago Mine Disaster Investigation

Dear Davitt,

I am writing to give you a few thoughts related to carbon monoxide (CO) intoxication as it relates to the Sago Mine Disaster.

**Can CO exposure affect judgement and cognition?**

1) Cognitive Impairment: It is well known and described in the medical literature that CO intoxication causes significant neurologic symptoms. These symptoms are more severe with higher COHb levels. Patients report and include headache, dizziness, weakness, nausea, vomiting, confusion, disorientation, and visual disturbances. 1 Confusion is the most important part of an altered mental status which applies to the miners involved in the Sago disaster. The neurologic impairment can manifest itself as decreased ability to make the correct critical decisions and impaired judgment. These symptoms are early and would occur soon after exposure since the brain is quite sensitive to the effects of CO. 2 It is therefore quite likely that the miners trapped in the Sago disaster had some degree of neurologic impairment related to their CO exposure which may have affected their judgment as it related to their ability to find escape routes. 3 The only way to prevent this is for miners to immediately don their SCSR’s and wear them continuously after an emergency where there is likely to be elevated levels of CO.

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General Points on CO Poisoning and Management

A few points about CO poisoning: The majority of miners killed in recent disasters died from CO poisoning, not trauma or burns.

Background Information:
There are 5000-6000 deaths from CO in US/year. CO in Environment is .001% or 10 ppm. The amount of CO absorbed depends on minute ventilation. If a miner is engaged in heavy physical exertion, there will be greater absorption of CO. The CO, and O2 concentration in the atmosphere are important determinants as well as the duration of exposure.

We can measure the CO levels in the atmosphere as well as measuring the levels of CO combined with hemoglobin in the blood, or CO-Hgb. This blood level is useful and tells us a little about the acute exposure, however the levels fall after the exposure to CO ceases and falls quickly if the miner is given supplemental oxygen. Normal CO-Hgb is generally less than 2% and can go as high as 10% in heavy smokers. Levels of 15-20% are considered mild CO poisoning. 20-60% moderate, and > 60% severe. I would like to emphasize that these levels may not correlate well with symptoms or with end organ damage. This is because the duration of exposure is very important, not just the concentration. CO causes damage at a cellular level over a longer period of time which may not be measured by CO-Hgb. It damages enzymes such as Cytochromes and Guanyl Cyclase and damages protein such as myoglobin. CO binds to hemoglobin in the blood 200 times tighter than O2. This is one mechanism of damage since it displaces oxygen. There is also a poor correlation between CO-Hgb and prognosis.

To give some examples of CO levels in different settings:

- Cooking indoors – up to 100 ppm
- Smoking cigarettes – 400-500 ppm while smoking
- Auto exhaust (breathing directly off the tail pipe) – 100,000 ppm
- Breathing 70 ppm for 4 hours = CO-Hgb of 10%
- Breathing 350 ppm for 4 hours = CO-Hgb of 40%
- OSHA limit – 50 ppm for 8 hour work day
- Sago as high as 2600 ppm (426 ppm where miners were found)

Diagnosis:
Diagnosis of carbon monoxide poisoning is made by a history of exposure. It is very useful to have data on the concentration of CO in the atmosphere where there exposure took place. CO-Hgb levels are very useful, however as noted before they may not correlate well with toxicity. Brief high exposures would result in a high level but less toxicity than exposure to lower levels over a longer period of time. The CO-Hgb level on admission to a hospital may not reflect the highest levels at the time of exposure since those levels decline with administration of oxygen. Blood should be drawn for CO-Hgb levels as soon as possible to give the best estimate of exposure.
Treatment:
Treatment for miners exposed to CO must include immediate management of their airway and circulation according to usual Advanced Cardiac Life Support protocols. Miners should then be given 100% oxygen and transported to a center with experience in managing severe carbon monoxide poisoning. This is especially important if they have any of the following:

1) Neurologic Findings:
   a. Altered Mental Status
   b. Coma
   c. Focal Neurologic Defects
   d. Seizures

2) History of loss of consciousness

Treatment with Hyperbaric Oxygen Therapy (HBOT)
While there is still controversy about using HBOT for CO poisoning, the general consensus is that strong consideration should be given to transferring any miner with the above symptoms to a facility which can provide Hyperbaric Oxygen Therapy (HBOT). A recent review by Kao and Nanagas summarizes the controversial issues. The CO levels can fall more or less quickly depending on how much oxygen the miner is given. This is measured in terms of CO-Hgb half life: The half life is 240-320 minutes for a miner breathing room air. It is 40-80 minutes when someone is breathing 100% Oxygen. It can be shortened to as fast as 20 minutes if a miner is placed in a hyperbaric chamber and given 100% oxygen at 2.5 to 3.0 atmospheres. HBOT also provides treatment for the toxic effect of CO on cellular enzymes and proteins and may prevent, or improve outcomes from long term sequelae. Studies have shown that HBOT given at 2.5 – 3.0 atmospheres may also reduce the incidence delayed neurologic syndrome (a syndrome that may occur some time after the event). It may also reduce overall mortality, 4/6 randomized controlled trials showed benefit for HBOT.

The data is less strong for other indications, however miners who have any of the following should be considered for possible transfer to a facility which can provide Hyperbaric Oxygen Therapy (HBOT).

1) Cardiovascular compromise  
   a. Myocardial ischemia, infarction, arrhythmias  
2) Metabolic acidosis  
3) Older Miners > 50  
4) Miners with persistent symptoms after treatment with 100% Oxygen.

Conclusions:
1) CO intoxication is an important cause of death, impairment, and disability in mining accidents.  
2) CO intoxication may also contribute to morbidity and mortality by causing impairment of judgment and making it less likely that miners will find ways to escape.  
3) Mine rescue teams should be trained in airway management and in protocols for managing CO intoxication.  
4) Consideration should be given to the utilization of Special Medical Response Teams such as the team in Western Pennsylvania, who have experience in rescue and treatment of CO intoxication in Mine Rescue.  
5) Miners with severe CO intoxication should be considered for transfer to specialized medical centers that have the capability for HBOT as soon as they are medically stable for transport.