

07-EH-03

Committee: Environmental Health

Title: Updates to 1998 Case Definition for Acute Carbon Monoxide Poisoning Surveillance

Statement of the Problem:

The Carbon Monoxide Surveillance Workgroup (CO-SWG) was formed in 2005 under the national Environmental Public Health Tracking (EPHT) grant, though its members include non-grantee state epidemiologists as well as clinicians interested in improving the surveillance and recognition of carbon monoxide poisoning. In the process of developing and testing nationally consistent measures for carbon monoxide (CO) poisoning and exposure, the CO-SWG has evaluated the *Surveillance Case Definition for Acute Carbon Monoxide Poisoning* adopted by CSTE in 1998. Several proposed revisions were identified:

First, the COSWG proposes the addition of three ICD-9 external cause of injury E-codes to the suspected case definition for administrative data sets. These are for 1) accidental, 2) intentional, and 3) unknown cause of poisoning by unspecified gases or vapors (E.869.9, E952.9, and E962.9, respectively). Although these were not included in the 1998 position statement, they are consistent with the suspected case definition for administrative data, which calls for E-codes when CO exposure is plausible, though not exclusively mentioned.

Second, the 1998 clinical case definition for confirmed CO poisoning requires either 1) laboratory confirmation of an elevated carboxyhemoglobin (COHb) level *from a blood specimen* or 2) environmental monitoring data suggesting a source of CO exposure. Since 1998 there have been advances in biomedical instrumentation for pulse oximetry, allowing COHb levels to be measured quickly and non-invasively. While traditional pulse oximeters use two wavelengths of light to measure oxygen saturation, a new device approved by the FDA (Rad-57 Pulse CO-Oximeter, Masimo Inc) uses eight wavelengths to measure not only oxygen saturation, but also carboxyhemoglobin and methemoglobin levels. One validity study found that the Rad-57 pulse CO-oximeter measured COHb levels below 15% with strong precision (2.2% uncertainty) relative to laboratory CO-oximetry of blood specimens drawn concurrently (1). Another recent study documented the utility of pulse CO-oximetry in emergency room triage, where three unsuspected cases of CO toxicity were identified over twelve days using the Rad-57 and confirmed with blood samples (2). The CDC is presently funding a multi-state study to further test the sensitivity and specificity of pulse CO-oximetry in the emergency room setting (3). Given the favorable nature of these early findings, the CO-SWG recommends that pulse CO-oximetry be added to the clinical case definition as a validation of cases presenting with signs and symptoms of CO poisoning; until further study results are available, it is not recommended as sufficient criteria alone in the absence of signs and symptoms of CO poisoning.

Third, since the 1998 position statement was adopted, several states have evaluated and demonstrated the utility of monitoring local Poison Control Center (PCC) data in their surveillance of acute carbon monoxide poisoning. Nationally, the CDC and American Association of Poison Control Centers (AAPCC) conduct routine surveillance of local PCC data to detect potential public health emergencies such as biological or

chemical events (4). However, because identifying data and case notes are removed from local PCC data prior to submission to the national database, local PCC partnerships can provide increased case details for investigation and analysis. In New York City, the health code was updated in 2004 to specify CO poisoning as an immediately reportable condition, where the NYC PCC receives reports from healthcare providers and rapidly notifies the fire department for follow-up investigation in order to prevent secondary cases at the incident site (5). In Florida, the state Department of Health has partnered with their regional PCC since 2003 to monitor exposure reports during hurricane season, identifying CO and hydrocarbon fuel poisonings related to generator use, and using these data to tailor public health safety advisories before, during, and after hurricane landfall (6). More generally, PCC data provide the added value of identifying exposures and poisoning cases that may not otherwise be captured in emergency department or hospitalization datasets, such as when the case experienced a health effect but did not seek medical treatment. Two state-based evaluations (Connecticut (7), Wisconsin (8)) found minimal overlap between PCC cases and patients treated in emergency rooms or hospitalized for CO poisoning.

The CO-SWG proposes the addition of a Probable case classification for PCC data, for cases recorded as having exposure to CO that involved a health effect of at least a minor nature. By definition, the coding practices set forth by the AAPCC specify that the selection of a health effect implies that the case's symptoms were most likely attributable to his/her exposure, in the judgment of the PCC specialist based on the history provided by the case or the caller. Some case records may have additional details in the notes indicating a confirmed environmental sample for CO or an elevated carboxyhemoglobin level. These cases should be elevated to Confirmed status as delineated in the recommended actions below.

References:

1. Barker S, Curry J, Redford D, Morgan S. 2006. Measurement of carboxyhemoglobin and methemoglobin by pulse oximetry. *Anesthesiology* 105:892-7.
2. Chee K, Suner S, Patridge R, Sucov A, Jay G. 2006. Noninvasive carboxyhemoglobin monitoring: screening emergency department patients for carbon monoxide exposure. *Acad Emerg Med* 13(Suppl): 179.
3. Stock, A. Verbal communication, May 11, 2007.
4. Watson W, Litovitz T, Rubin C, Kilbourne E, Belson M, Patel M, Schier J, Funk A. 2004. Toxic exposure surveillance system. *MMWR* 53(Suppl):262.
5. New York City. Rule 63: Amends the rules of the health and mental hygiene department in relation to carbon monoxide poisoning. *Rules of the City of New York*. October 21, 2004.
6. CDC. 2006. Monitoring Poison Control Center Data to Detect Health Hazards During Hurricane Season --- Florida, 2003—2005. *MMWR* 55(15):426-428.
7. Toal B. Comparison of three CO databases in Connecticut. EPHT Web Seminar, June 26, 2006 .
8. Bekkedal M, Sipsma K, Stremski ES, Malecki KC, Anderson HA. 2006. Evaluation of five data sources for inclusion in a statewide tracking system for accidental carbon monoxide poisonings. *Wisconsin Medical Journal* 105(2):36-40.

Statement of the desired action(s) to be taken:

The COSWG proposes the following changes to the 1998 case definition for acute CO poisoning (changes in bold, with additions underlined and deletions in strikethrough):

Surveillance Case Definition for Acute Carbon Monoxide Poisoning

Type of surveillance: Outcome

Clinical Description

There is no consistent constellation of signs and symptoms resulting from acute carbon monoxide poisoning, nor are there any pathognomonic clinical signs or symptoms which would unequivocally indicate a case of acute carbon monoxide poisoning. The clinical presentation of acute carbon monoxide (CO) poisoning varies not only with the duration and magnitude of exposure, but also between individuals with the same degree of exposure and/or same venous carboxyhemoglobin (COHb) level.

Clinical signs and symptoms of acute carbon monoxide poisoning that are commonly reported to health care professionals include, but are not limited to: headache, nausea, lethargy (or fatigue), weakness, abdominal discomfort/pain, confusion, and dizziness.

Other signs and symptoms reported include: visual disturbances including blurred vision, numbness and tingling, ataxia, irritability, agitation, chest pain, dyspnea (shortness of breath) on exertion, palpitations, seizures, and loss of consciousness.

Clinical Case Definition

There is no clinical case definition for acute carbon monoxide poisoning. There are no pathognomonic clinical signs or symptoms that would unequivocally indicate a case of acute carbon monoxide poisoning (without ~~laboratory~~ confirmation **of an elevated carboxyhemoglobin level**). See Clinical Description and Laboratory Criteria for Diagnosis.

Laboratory Criteria for Diagnosis

A blood specimen with an elevated carboxyhemoglobin (COHb) concentration, as determined by a validated method (e.g., photometric, gas chromatography). Elevated levels of carboxyhemoglobin should be interpreted in light of endogenous production, patient smoking status, and exposures to second hand smoke (1).

Case Classification

Confirmed:

Notification Systems:

Clinicians/Medical Examiners/Coroners: (1) A report of a patient with signs and symptoms consistent with acute carbon monoxide poisoning and a confirmed elevated COHb level, as determined by either a blood specimen (See Laboratory Criteria for Diagnosis) **or pulse CO-oximetry**, OR; (2) a report of a patient with signs and symptoms consistent with acute carbon monoxide poisoning (in the absence of clinical **or** laboratory **confirmation of an elevated COHb level**), with supplementary evidence in the form of environmental monitoring data suggesting exposure from a specific poisoning source.

- OR -

Hyperbaric Treatment Facilities: A report of a patient who has received hyperbaric treatment for acute carbon monoxide poisoning, regardless of carboxyhemoglobin concentration reported.

- OR -

Laboratories: A report of a blood specimen (in the absence of clinical and environmental laboratory data) with a carboxyhemoglobin level that is equal to or greater than a volume fraction of 0.12, ie. 12%. [Note: This level was selected to identify those people whose COHb levels are likely to cause clinically apparent adverse health effects, while attempting to minimize the number of chronic smokers reported as acutely poisoned (1) (see comment section).]

Administrative Data:

ICD-9 Coded Data: (1) A record in which the Nature of Injury code N-986 "Toxic effect of CO" is listed, OR; (2) a record in which an External Cause of Injury code (E-code), indicating exposure to carbon monoxide (exclusively) is listed, ie. E868.3, E868.8, E868.9, E952.1, or E982.1.

-OR-

ICD-10 Coded Data: A record in which T58, Toxic Effect of Carbon Monoxide, is listed

-OR-

ICD-10-CM (under development) coded data: A record in which T58, Toxic Effect of Carbon Monoxide, is listed

-OR-

Poison Control Center (PCC) Data: A record of a case with "exposure" recorded as the type of call, when the exposure substance was carbon monoxide (substance generic code = 0106000), AND a minor, moderate, or major medical effect or death was reported AND an elevated carboxyhemoglobin level or positive environmental sample for CO was indicated in the case notes [See comment section on PCC data].

Probable Case:

Notification Systems:

Clinicians/Medical Examiners/Coroners: In the absence of clinical and environmental monitoring data: (1) A report of a patient with signs and symptoms consistent with acute carbon monoxide poisoning and the same history of environmental exposure as that of a confirmed case, OR, (2) a report of a patient with smoke inhalation secondary to conflagration.

- OR -

Laboratories: A report of a blood specimen with a carboxyhemoglobin level that is equal to or greater than a volume fraction of 0.09, i.e. 9% and less than a volume fraction of 0.12, i.e. 12% ($9 < \text{COHb\%} < 12$).

Administrative Data:

ICD-9 Coded Data: A record in which an E-code indicating acute carbon monoxide poisoning inferred from motor vehicle exhaust gas exposure is listed, ie. E868.2, E952.0, or E982.0.

-OR-

Poison Control Center (PCC) Data: A record of a case with “exposure” recorded as the type of call, when the exposure substance was carbon monoxide (substance generic code = 0106000), AND a minor, moderate, or major medical outcome or death was reported [See comment section on PCC data].

Suspected Case:

Notification Systems:

Clinicians/Medical Examiners/Coroners: A report of a patient with signs and symptoms consistent with acute carbon monoxide poisoning and a history of present illness consistent with exposure to carbon monoxide.

Administrative Data:

ICD-9 Coded Data: In the absence of an N-986 code: (1) a record in which an E-code that mentions CO exposure is listed (E818.0-.9, E825.0-.9, E844.0-.9, E867, E868.0, E868.1, E890.2, E891.2), (2) a record in which an E-Code where carbon monoxide exposure is plausible is listed (E838.0-.9, **E869.9**, E951.0, E951.1, E951.8, **E952.9**, E962.2, **E962.9**, E968.0, E981.0, E981.1, E981.8, E988.1).

- OR -

ICD-10 Coded Data: In the absence of T58 code, a record in which a code that mentions CO exposure, is listed (X47, X67, Y17). [Note: The December 1997 draft of ICD-10-CM omits these codes.]

- OR -

Worker's Compensation Data: A report of a person with carbon monoxide poisoning documented in the record.

Comment:

The descriptions provided for identifying cases of acute carbon monoxide poisoning utilize a variety of sources of data that one may use or integrate into a surveillance system. These data elements are from commonly available data that may be consistently utilized by all states. Additional state specific databases may have greater detail and professional discretion is encouraged when using alternative or supplemental databases. These definitions should be updated as the clinical, diagnostic, and epidemiologic state-of-the-art evolves and as new data sources become available.

The case descriptions provided are designed to ascertain cases of acute carbon monoxide poisoning regardless of the source of the exposure. Information on the history of the poisoning incident and the suspected exposure sources should be reviewed for targeting prevention efforts/interventions (see comments on persons with occupational exposure).

The surveillance case definitions for acute carbon monoxide poisoning have been formulated to address the general population. There are several sub-populations which may be more or less susceptible to the adverse health effects of carbon monoxide intoxication, due to pre-disposing environmental and physiologic conditions (see below). Separate surveillance case definitions for sub-populations have not been formulated at this time as it is unclear from the published peer-reviewed literature whether these groups suffer more extreme outcomes from poisoning at lower levels of exposure and/or lower levels of carboxyhemoglobin saturation. In addition it is unclear whether the reporting sources would be able to consistently differentiate these sub-populations based on the information currently available in their databases.

The following sub-populations have been identified in the literature as having special concerns with respect to acute carbon monoxide poisoning:

1. Persons Exposed to Tobacco Smoke: Active and passive exposure to tobacco smoke elevates carboxyhemoglobin levels as a result of inhalation of combustion by-products. Data from the National Health and Nutrition Evaluation Survey, II , 1976-80, revealed that self reported current smokers showed larger variability in COHb levels along with a relative insensitivity of this group to incremental changes in the environmental burden of CO when compared with the never

smoking group. NHANES, II also reported that approximately 95.9% of the current smokers had a concentration of COHb less than or equal to 9% (1).

2. Children: It is known that children have a higher minute ventilation per unit body weight than adults and that children have been reported to be more susceptible to the acute adverse health effects from exposure to CO. It is acknowledged that while children may accumulate COHb faster than adults, it is unclear whether they experience more severe outcomes at lower levels of COHb saturation, therefore a unique surveillance case definition has not been formulated at this time.

3. Fetal Exposure/Pregnancy: It is known that fetal blood has a higher affinity for CO than does adult hemoglobin and this is an important distinction considered by the clinician when treating the pregnant patient. It is unclear, at this time whether a unique surveillance case definition for this subgroup based on maternal COHb levels is necessary for reporting purposes.

4. Persons Living at Altitude: It is known that persons living at altitude experience faster loading of COHb as result of a leftward shift of the oxygen-hemoglobin dissociation curve. It is unclear whether persons at altitude experience more severe outcomes at lower levels of COHb saturation, therefore, a unique surveillance case definition for this sub-group has not been formulated at this time

5. Persons with Pre-morbid Conditions: It is known that sub-populations with conditions of low oxygen saturation (e.g.; chronic obstructive pulmonary disease) and conditions with decreased oxygen delivery to the tissues (e.g.; ischemic heart disease) experience adverse health effects with increased COHb levels. It is unclear whether these sub-groups experience more severe outcomes at lower levels of COHb saturation, therefore, unique surveillance case definitions for these sub-populations have not been formulated at this time.

6. Persons with Occupational Exposure: Occupational exposure to carbon monoxide is not uncommon. The outcomes from acute CO poisoning at work are no different than the consequences of other sources of exposure. It is important to gather information on potential occupational exposures as this information is important for preventing future poisonings.

References:

1. National Center for Health Statistics, EP Radford and TA Drizd: Blood carbon monoxide levels on Persons 3-74 Years of Age: United States, 1976-80. Advance Data From Vital and Health Statistics, No. 76. DHHS Pub. No. (PHS) 82-1250. Public Health Service, Hyattsville, Md. March 17, 1982.

Public Health Impact:

It is anticipated that these revisions will increase the sensitivity of the CSTE case definition for acute carbon monoxide poisoning surveillance. No change in specificity is expected. Ultimately, the public health impact should be increased recognition of the full spectrum and burden of CO poisoning, to the extent that pulse CO-oximetry becomes more widely adopted as a standard of care in emergency room triage. Likewise, the impact of these changes will increase as more states and territories adopt and maintain CO poisoning as a reportable condition.

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