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***Incidence and Cost
of
Carbon Monoxide Poisoning for All Ages,
Pool and Spa Submersions for Ages 0–14,
and
Lead Poisoning for Ages 0–4***

Final Report

Task Order 1, CPSC Contract D-09-0003

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March 28, 2013

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Introduction

The U.S. Consumer Product Safety Commission's (CPSC's) major tool for estimating the benefits from prevention of injuries related to consumer products is its Injury Cost Model (ICM). CPSC developed the ICM in the late 1970s and continues to refine it. The ICM provides a common basis for measuring the dollar burden of a wide range of product-related injuries. Used in conjunction with injury and fatality data, the ICM permits the CPSC to compare alternative policies designed to reduce consumer product injuries and evaluate the relationships of their costs and benefits.

The CPSC periodically investigates issues related to swimming pool and spa submersion, lead poisoning, and carbon monoxide (CO) poisoning because of their frequency and severity. These injuries are primarily brain injuries with rare, but potentially serious long-term neurological sequelae. Lead poisoning also adversely affects other body systems. Unlike traumatic brain injuries (TBIs) (*e.g.*, from bicycle crashes or sports), the ICM is not designed to capture these injuries. Thus, data on incidence and costs of health problems resulting from these injuries are less complete and less reliable than for most injuries associated with consumer products. The purpose of this task order was to improve knowledge of their incidence and costs. Improved estimates will enhance decision making about relevant exposures.

CPSC's task order request focused on unintentional nonoccupational and nonfire carbon monoxide poisonings of victims at all ages, swimming pool and spa submersions of victims younger than 15 years of age, and lead poisonings of victims younger than 5 years of age. The task order called for analyses of incidence of medically treated cases, associated work losses, and quality-of-life losses, and acute, but (due to budget constraints) not long-term, medical care costs for cases associated with products under CPSC's jurisdiction. Lead poisoning has other sequelae, including increased need for special education and elevated risk of occurrence of attention deficit hyperactivity disorder (ADHD) and criminal behavior. The appendix examines the rates and costs of those events.

Data Sets and Diagnosis/Cause Codes Used

We primarily used Healthcare Cost and Utilization Project (HCUP) and National Electronic Injury Surveillance System (NEISS) data. HCUP is a family of health care databases

and related software tools and products developed through a federal-state-industry partnership and sponsored by the Agency for Healthcare Research and Quality (AHRQ). We analyzed 2006 HCUP-KID (kid's inpatient database), 2007 HCUP NIS (nationwide inpatient sample), and 2007 HCUP NEDS (nationwide emergency department (ED) sample).¹ NEISS is the CPSC's ED-based injury surveillance system. It draws data from a much smaller sample of EDs than NEDS, but it investigates cases in far greater detail. Unlike the HCUP data sets, NEISS contains narratives that can be used to differentiate swimming pool and spa submersions from other submersions. For submersions, therefore, we also used 2008 NEISS consumer product injury and all-injury profile (AIP) data.

We obtained and incorporated data from the Undersea and Hyperbaric Medical Society (UHMS) hyperbaric oxygen treatment database (a joint project with the Centers for Disease Control and Prevention (CDC)) for treatment of CO poisoning cases. We also examined CO poisoning incidence reported in the National Poisoning Data System (NPDS) operated by the American Association of Poison Control Centers.

In the United States, mortality data are coded by diagnoses, intent, and cause/mechanism of injury/poisoning using International Classification of Diseases, 10th Edition (ICD10). Hospital inpatient and emergency department discharge data are coded with this information using the older International Classification of Diseases, 9th Edition, Clinical Modification (ICD9-CM). Coding of hospital data will move to ICD10-CM on October 1, 2014. Appendix B lists ICD codes used in this project.

We drew fatal and ED-treated submersion incidence and location data from published sources: the CPSC's analysis of 2006–2008 National Vital Statistics System (NVSS) mortality data; 2008–2010 NEISS consumer product injury data and supplemental NEISS investigations (Gipson 2011a, 2011b); and the CDC's analysis of 2005–2009 NVSS and NEISS-AIP data (Laosee et al. 2012). All three studies searched text narratives to determine place of submersion. Unintentional, non-transport, nonfatal submersion injuries in NEISS and NEISS-AIP cases were defined as cases with NEISS injury code 64. CPSC and CDC identified fatal submersions as

¹ Adjustments were required for the state of Texas, which provided age categories rather than actual ages. For lead poisoning at ages 0–4, we used the Texas count of lead poisoning patients in age categories 1 and 2 (ages 0–17) times percentage of patients ages 0–4 among those ages 0–17 in all other states. We used a parallel computation for submersions.

ICD10 codes W16, W29, W65, W66, W73, W74, or Y21 and also searched narrative fields for the terms “drown” or “submer.” CPSC and CDC used NEISS product codes and NEISS and NVSS narratives to identify pool and spa submersions. (See Appendix B for ICD and NEISS codes descriptors.)

We tabulated lead poisoning fatalities (ICD10 code T56.0) with 2009 NVSS data. For CO poisoning, we used a CPSC CO fatality count restricted to nonfire deaths associated with the use of consumer products under CPSC’s jurisdiction (Hnatov 2012). This count started from all cases with diagnosis code X47 or Y17. CPSC searched accompanying death certificate narratives to identify CO cases and determine their etiology. We also give a broader CO fatality count for ICD10 diagnosis code T58, modified by external cause codes X47 or Y17, and excluding uncontrolled fire deaths and deaths in motor vehicles. This count includes all remaining unintentional CO deaths and an allocated portion of deaths of undetermined intent.

In analyzing hospital and ED discharges using HCUP, we excluded cases paid by Worker’s Compensation. Among nonoccupational cases, unintentional, non-transport, nonfire CO poisoning cases were defined as cases with an ICD9-CM external cause code between E868.2 and E868.9 or with diagnosis code 986, as well as an external cause code of E868.1 or E868.2. Lead poisoning was defined to be cases with diagnosis 984 and/or external cause code E861.5 or E866.0. Submersion was defined as cases with diagnosis 994.1 and/or external cause code E830, E832, or E910. ICD9-CM does not distinguish swimming pool and spa submersions from bathroom, bucket, and open body of water submersions.

Lead exposure is most often chronic, not acute, without obvious symptoms. Indeed, most lead exposure cases are not medically attended or even recognized as occurring. Therefore, we also drew on national surveillance data on lead poisoning to estimate low-level exposure.

Incidence

As prescribed in CPSC’s task order, this chapter largely focuses on counts of the medically treated injuries used to gain insight into medical treatment cost and outcome issues. Those interested in the subset of incidents associated with consumer products under CPSC regulation should consult the CPSC reports cited below. Among medically treated nonfatal cases, submersion incidence was highest, followed by CO poisoning, and then lead poisoning.

Lead Poisoning

Childhood lead poisoning is rarely fatal, with no fatalities under age nine in 2007–2010. Tables 1 and 2 present incidence of lead poisoning in children ages 0–4 both for hospital-treated fatal and nonfatal cases, as well as for nonfatal cases, by location of disposition. These events are rare. We recommend relying on the KID dataset for hospital-admitted lead poisoning (and child submersion) incidence counts because its sample size of child cases is much larger than in the 2007 HCUP–NIS, resulting in narrower confidence intervals. The NEDS estimate of lead poisoning admissions

Table 1. Incidence of Lead Poisoning at Ages 0-4 by Survival and Data Source

Data source	Year	Fatal or Nonfatal	Raw Case Count	Weighted Incidence	Linearized Standard Error	[95% Confidence Interval]	
NVSS	2007-2010	Fatal	0	0	0	0	0
NIS Inpatient Sample	2007	Nonfatal	51	247	69	111	384
		Fatal	0	0			
NEDS ED Visit Sample	2007	Treated & Released	10	45	*		
		Admitted	17	84	*		
		Fatal	0	0			
KID Child Inpatient Sample	2006	Nonfatal	120	211	34	144	279
		Fatal	0	0			

NB: NIS (nationwide inpatient sample) and KID (kid’s inpatient database) fatality counts are restricted to patients admitted to the hospital. NEDS counts only those fatalities where the victims died in the ED. KID counts only patients under age 21.* Empty strata prevented variance estimation.

is less reliable; it is based on such a small count that we could not estimate its standard error. The very low count of lead poisoning cases in EDs is matched by a virtual absence of cases in doctors' offices in National Ambulatory Medical Care Survey (NAMCS) data. NAMCS is an annual U.S. National Center for Health Statistics survey.

During 1999–2004, 1.4 percent of children ages 1–5 years had blood lead levels at least 10 micrograms per deciliter ($\mu\text{g}/\text{dL}$) (Jones et al., 2009). National data, collected in 2007–2008 (CDC, 2012), show that the median blood lead level (BLL) in children ages 1–5 years is 1.4 $\mu\text{g}/\text{dL}$; the 95th percentile is 4.1 $\mu\text{g}/\text{dL}$. Although children with BLLs in this lower range are unlikely to be medically attended, as documented in the wage loss section below, low lead exposure reduces IQ.

Table 2. Medically Attended Nonfatal Lead Poisonings by Case Disposition and Data Source

Data source	Year	Age-group	Disposition	Raw Case Count	Weighted Incidence	Linearized Standard Error	[95% Confidence Interval]	
Hospital-admitted								
NIS	2007	0–4	Discharged to Home	45	220			
NIS		0–4	Discharged to Nursing Home*	0	0			
NIS		0–4	Discharged to Another Facility**	0	0			
NIS		0–4	Discharged to Home Health Care	6	28			
Hospital-admitted								
KID	2006	0–4	Discharged to Home	111	195	30	136	254
KID		0–4	Discharged to Nursing Home*	0	0	0	.	.
KID		0–4	Discharged to Home Health Care	9	16	13	0	42

Note: Missing standard error due to stratum with single nonzero sampling unit.

* Nursing home includes both skilled nursing facilities and intermediate care facilities.

**The definition of another facility varies by state and may include residential care, hospice facility, rehabilitation center, psychiatric hospital, or other non-acute care.

CO Poisoning

Table 3 presents incidence data about CO poisoning. For 2007 to 2009, based on a hand-review of death certificates, CPSC estimates unintentional nonfire CO poisoning deaths associated with consumer products under CPSC’s jurisdiction averaged 169 per year (Hnatov 2012). NVSS indicates that an average of 477 people per year died of unintentional CO poisoning in 2007 to 2008 (excluding deaths in uncontrolled fires and motor vehicles). This estimate includes 14 deaths per year allocated from the undetermined intent category and 3.5 deaths per year involving carbon monoxide poisoning (ICD10 code T58) without codes that would indicate they were intentional or related to fires or motorized vehicles. That suggests roughly 35 percent of unintentional CO poisoning deaths not associated with fires or motor vehicles involve consumer products regulated by CPSC. That same percentage might apply to nonfatal cases. (Including intentional exposures and fire-related cases, CO deaths in 2007 to 2008 totaled 3,745.)

Table 3. Incidence of Carbon Monoxide Poisoning by Survival and Data Source

Data source	Year	Fatal or Nonfatal	Age-group	Raw Case Count	Weighted Annual Incidence	Linearized Standard Error	[95% Confidence Interval]	
NVSS + Death Certificates (Hnatov 2012)	2007-2009	Fatal Consumer Product	All	508	169	12	118	221
NVSS	2007-2008	Fatal	All	954	477	NA	NA	NA
HCUP NIS Inpatient Sample	2007	Nonfatal	All	243	1,210	186	846	1,574
		Fatal	All	6	30	12	6	53
HCUP NEDS ED Visit Sample	2007	Treated & Released	All	2,752	13,029	742	11,574	14,485
		Admitted	All	283	1,296	112	1,075	1,517
		Fatal	All	4	21	11	0	43
HCUP KID Child Inpatient Sample	2006	Nonfatal	0–20	93	153	25	105	201
		Fatal	0–20	2	3	2	0	8

NB: NA=Not Applicable (vital statistics counts are a census; HCUP = Healthcare Cost and Utilization Project, NIS = nationwide inpatient sample, NEDS = nationwide emergency department sample, KID = kid’s inpatient database. NIS and KID fatality counts are restricted to patients admitted to the hospital. NEDS fatalities died in the ED; NEDS does not track patients beyond hospital admission.

HCUP-NIS estimates 1,210 people were discharged alive after hospital inpatient stays for unintentional nonfire CO poisoning in 2007. According to HCUP KID, 153 of those survivors were children. NIS and KID show that almost none of the deaths occurred in hospital inpatient units. Finally HCUP-NEDS—which is an independent sample, not a subset of NIS—estimates that more than 14,000 CO poisoning patients were treated in the ED; an estimated 1,296 of these patients were admitted to a hospital and 21 died in the ED. The remainder were treated and released. NIS estimates about 30 died in the hospital after being admitted. It is not surprising that NIS and NEDS yield consistent estimates. Neither is a national probability sample. The NEDS emergency department sample (including cases admitted through the ED and cases that instead were treated and released) comes from a subset of the states that have NIS inpatient data, although the two systems sample cases from different data sets. In states included in both data sets, NEDS samples a much larger share of admissions through the ED, but it misses any admissions that bypass the ED (*e.g.*, if a hospital admitted the patient directly to a hyperbaric chamber). HCUP weights both samples using the same procedures.

Table 4 shows discharge destinations for hospital-admitted CO poisoning survivors. Patients discharged to places other than home typically have lasting disability. Effects of CO poisoning, however, are not always clear-cut during initial treatment. After initially appearing to have made a full recovery based on restoration of normal carboxyhemoglobin levels, even treated and released ED patients can experience delayed neurological sequelae resulting in lasting impairment. This is also true for patients treated with hyperbaric oxygen (HBO₂). Symptoms may not manifest until well after the patient returns home.

CO poisoning treatment generally involves administering oxygen, either normobaric (face mask at ambient pressure) or hyperbaric (high-pressure chamber). HCUP CO incidence (and related medical cost) estimates may miss some cases that receive hyperbaric oxygen treatment. Table 5 presents incidence of CO poisoning treated in hyperbaric treatment facilities between 1992 and 2002 (Hampson and Little, 2005). On average, approximately 1,500 cases, including fire-related and intentional CO poisonings, annually received one hyperbaric treatment, with a few receiving a second treatment. Of 1,907 survivors treated in a large convenience sample of 64 hyperbaric facilities between August 2008 and July 2011, 45 percent were treated and released (Hampson et al., 2012). Those patients probably would be missed in studies relying on hospital and ED discharge data. Wide variations in hyperbaric facility usage rates between

states (Hampson et al., 2012) suggest usage probably is determined by physician preference and facility presence more than by patient health status.

In 2008, the Centers for Disease Control and Prevention (CDC) teamed with the Undersea and Hyperbaric Medical Society (UHMS) to monitor cases of carbon monoxide poisoning treated in U.S. hyperbaric facilities (Hampson et al., 2012). This system went live nationally and was open voluntarily to all U.S. hyperbaric facilities on August 1, 2008. Given the estimates from Table 5, it probably captures around 42 percent of cases annually (636/1500). Exhibit 1 presents surveillance system data from Hampson et al. (2012), showing incidence, patient demographics, and details of exposure and treatment. These data probably were representative of hyperbaric use in their catchment areas. Importantly, 68 percent of cases were unintentional nonfire, non-motor-vehicle (auto or boat) cases. Most nonoccupational cases in this category resulted from defective furnaces and improper use of generators. Intubation often means the patient was unconscious when hyperbaric treatment began, but it may not necessarily imply a bad outcome.

Table 4. Medically Attended Nonfatal Carbon Monoxide Poisoning by Case Disposition and Data Source

Data source	Year	Age-group	Disposition	Raw Case Count	Weighted Incidence	Linearized Standard Error	[95% Confidence Interval]	
Hospital-admitted only								
NIS	2007	All	Discharged to Home	208	1029	158	719	1339
NIS		All	Discharged to Nursing Home	0	0	.	.	.
NIS		All	Discharged to Another Facility*	22	113	31	51	174
NIS		All	Discharged to Home Health Care	13	68	19	32	105
Hospital-admitted children only								
KID	2006	0–20	Discharged to Home	89	147	24	99	195
KID		0–20	Discharged to Nursing Home	3	4	3	0	11
KID		0–20	Discharged to Home Health Care	1	2	2	0	4

Note: Missing standard error due to stratum with single nonzero sampling unit

*The definition of another facility varies by state and may include residential care, hospice facility, rehabilitation center, psychiatric hospital, or other non-acute care.

Table 5. Incidence of Carbon Monoxide Poisoning Treated with Hyperbaric Oxygen (HBO₂), 1992–2002

Year	Patients with CO Poisoning Annually Treated with HBO₂
1992	1291
1993	1450
1994	1502
1995	1714
1996	1684
1997	1448
1998	1492
1999	1452
2000	1441
2001	1388
2002	1505

Source: Hampson and Little (2005).

Exhibit 1. Demographic Characteristics of Carbon Monoxide Poisoning Patients in the (Voluntary) National Hyperbaric Oxygen Surveillance System, 8/1/2008–7/31/2011

- One thousand nine hundred seven cases were reported between August 1, 2008 and July 31, 2011.
- The six states that reported the most cases are: Utah (197), Pennsylvania (189), Washington (169), Michigan (146), Illinois (144), and Maryland (139).
- The incidence by gender is 1,128 males (59%), 787 females (41%), including 46 pregnant women (total of 1915 conflicts with other counts in Hampson et al, 2012).
- A total of 59.6 percent of cases were non-Hispanic whites.
- Among 1,513 cases responding to this question, 3.7 percent had experienced prior CO poisoning.
- Furnace (38%), generator (20%), and fire (13%) were the three most common sources of nonoccupational, non-transport-related CO poisoning cases. Together they accounted for 71 percent of those cases.
- Of known cases, 1,505 (85%) were unintentional (including fire and motor vehicle), and 273 (15%) were intentional.
- Occupational incidents (292) accounted for 15 percent of cases.
- Virtually all cases already had been treated with normobaric oxygen.
- A total of 15.7 percent of cases were intubated.

Age Distribution (1,905 cases with known ages)

Age Group	Cases	Percent
0–9	167	8.8%
10–19	203	10.7%
20–29	279	14.6%
30–39	295	15.5%
40–49	362	19.0%
50–59	275	14.4%
60–69	170	8.9%
70–79	92	4.8%
80–89	57	3.0%
90+	5	0.3%

Source: Hampson et al. (2012).

Submersion

Table 6 presents submersion incidence in children ages 0–14, for fatal and non-fatal discharges from the ED, and Table 7 breaks these cases down by place of disposition. In Table 7, ED data systems record whether children were admitted to the hospital but do not track status post-admission. NIS and KID show discharge destinations and survival for these cases. NEDS’s estimate of child submersion cases treated and released, which comes from a much larger sample (1617 cases versus 101 for NEISS, Consumer Product Sample (CPS) and 32 for NEISS-AIP) and far more EDs than the NEISS estimates, is two to three times the NEISS CPS and NEISS AIP estimates and exceeds their 95 percent confidence intervals. In contrast, admission estimates (weighted incidence) from the five data systems range from 1,951 to 2,586, with the preferred KID estimate at 2,107, including 1,914 non-fatal discharges and 203 deaths in the hospital.

Table 6. Incidence of Submersion by Survival and Data Source

Data source	Year	Fatal or Nonfatal	Age-group	Raw Case Count	Weighted Incidence	Linearized Standard Error	[95% Confidence Interval]	
NIS Inpatient Sample	2007	Nonfatal Inpatient	0–14	375	1805	297	1223	2387
	2007	Fatal	0–14	38	178	.	.	.
NEISS-CPS ED Consumer Product Sample	2008	Nonfatal ED	0–14	185	4981	1616	1771	8192
NEISS-AIP ED All Injury Sample	2008	Unintentional Nonfatal ED	0–14	103	3536	817	1936	5136
NEDS ED Sample	2007	Nonfatal ED	0–14	2180	10083	.	.	.
	2007	Fatal	0–14	98	452	.	.	.
KID Child Inpatient Sample	2006	Nonfatal Inpatient	0–14	1160	1914	135	1650	2179
	2006	Fatal	0–14	121	203	26	153	253

Table 7. Medically Attended Nonfatal Submersions by Case Disposition and Data Source

Data source	Year	Age-group	Disposition	Raw Case Count	Weighted Incidence	Linearized Standard Error	[95% Confidence Interval]	
NIS	2007	0-14	Admission Discharged to Home	365	1755	289	1189	2322
NIS	2007	0-14	Discharged to Nursing Home or Another Facility*	0	0	.	.	.
NIS	2007	0-14	Discharged to Another Institution	3	14	.	.	.
NIS	2007	0-14	Discharged to Home Health Care	7	35	.	.	.
NEISS-CPS	2008	0-14	ED Treated & Released-All	101	2783	895	1005	4561
NEISS-CPS	2008	0-14	ED Treated & Released-Injured at Home	53	1521	521	486	2555
NEISS-CPS	2008	0-14	Admitted thru ED-All	84	2198	795	619	3777
NEISS-CPS	2008	0-14	Admitted thru ED-Injured at Home	49	1490	653	192	2788
NEISS-AIP	2005	0-14	ED Treated & Released-All	32	1169	354	460	1878
NEISS-AIP	2005	0-14	ED Treated & Released-Injured at Home	5	88	41	5	171
NEISS-AIP	2005	0-14	Admitted thru ED-All	49	1951	551	848	3054
NEISS-AIP	2005	0-14	Admitted thru ED-Injured at Home	24	877	269	339	1415
NEDS	2007	0-14	ED Treated & Released	1617	7496	.	.	.
NEDS	2007	0-14	Admitted thru ED	563	2586	.	.	.
KID	2006	0-14	Admission Discharged to Home	1112	1835	130	1580	2091
KID	2006	0-14	Discharged to Nursing Home or Another Facility*	30	49	10	29	69
KID	2006	0-14	Discharged to Home Health Care	18	30	7	16	43

Note: Missing standard error due to stratum with single nonzero sampling unit

* The definition of another facility varies by state and may include residential care, hospice facility, rehabilitation center, psychiatric hospital, or other non-acute care.

Admitted submersion cases with sequelae almost always emerge dependent on tracheotomy tubes and possibly feeding tubes. This means these children have expected short lifespans, cannot breathe independently, typically require round-the-clock nursing care, and are medically fragile and prone to infection.

Based on narrative record review, Laosee et al. (2012) estimate that at ages 0–4, 50.1 percent of deaths and 64.6 percent of ED visits for submersion result from pool and spa incidents. Among nonfatal events, they estimate 45.7 percent are in private pools; 31.7 percent are in public pools; and 22.6 percent are in unspecified pools. At ages 5–14, they estimate 29.9 percent of deaths and 76.0 percent of ED visits are pool-related submersions. The pattern among pool types differs with only 20.8 percent in private pools; 62.4 percent in public pools; and 16.8 percent in pools of unspecified type. Overall, at ages 0–14, 43.4 percent of drowning deaths and 67.8 percent of nonfatal submersions occur in pools and spas. Gipson (2011a) estimates pool and spa fatalities under age 15 averaged 383 in 2006–2008, higher than the Laosee et al. estimate of 332 per year (with place of occurrence unknown for many cases). (Another 351 pool submersion deaths occurred annually among adults.) Gipson (2011b) further estimates 660 non-pool and non-spa submersion incidents occurred in 2005–2009 for children younger than 5 years old. These include 431 fatalities, 212 nonfatal injuries, and 17 incidents with no apparent injury or unknown injury.

Short-Term Medical Costs

Tables 8–11 present the mean costs of acute medical care for hospital admissions for the target conditions. We computed costs by multiplying hospital-and-service-specific cost-to-charge ratios developed by AHRQ times charge data on NIS. We generally did not add separately charged professional fees and post-discharge costs. CPSC’s Injury Cost Model (ICM) estimates lifetime medical costs of a hospital-admitted nonfatal injury by multiplying acute inpatient costs times the ratio of total costs, including inpatient costs, professional fees, and post-discharge costs, to acute costs. The last column in Table 8 shows the ratios the ICM uses for CO poisoning, lead poisoning, and submersion. the ratios of total costs – including inpatient costs,

professional fees, and post-discharge costs – to inpatient costs that our existing injury cost algorithm would multiply, times hospital inpatient costs, to estimate lifetime costs. Reliability of those ratios is uncertain for the conditions under study. It was originally planned to analyze diagnosis-specific utilization/medical payments in a claims file to provide better estimates, but lack of funding precluded this analysis.

Among admitted cases, acute medical costs per case were highest for submersion, followed by CO poisoning, with lead poisoning much lower (Table 8). For nonfatal submersion, we could not establish case severity because HCUP datasets lack the five-digit procedure codes needed to identify cases requiring critical care or ventilator management. Data sparseness also prevented us from calculating standard errors.

In 1998, average Medicare payments for a covered hyperbaric treatment of CO poisoning were approximately \$405, including \$140 for physician supervision and \$265 for outpatient services (Office of Inspector General, 2000). Hampson and Little (2005) estimate most patients receive only one treatment.

Quest Laboratories charges the military and other volume purchasers \$6–\$7 for a blood lead test, with retesting indicated if blood lead level (BLL) exceeds 35–40 µg/dL. Although CDC only recommends chelation therapy if child BLL exceeds 45 µg/dL, concerns about effects of lower BLLs has increased use on lesser exposures, despite potential side effects. A single chelation treatment usually lasts 2–4 hours. It cost \$77 on average in 2007 (Nahin et al. 2009). In the first month, patients typically receive 5–30 treatments, with 30 being most common (Keogh & Boyer 2011). Thus, the typical cost is around \$2,000.

By design, this task order did not probe the costs of care in the post-acute period. Estimating those costs would require a longitudinal analysis of a very large claims file like a multistate Medicaid file. That analysis also would greatly improve our knowledge of the severity distribution and of survival time following catastrophic injury.

A parent gave us permission to disclose long-term medical costs of her child's submersion injury that occurred in California in July 1979 when he was 14 months old. He is tracheotomy-dependent and tube-fed. He spent his first year post-injury in hospital. He then came home to 24-hour registered nursing (RN) care at \$8 per hour. The mother sued and got him admitted to school; the schools paid his nurse as an attendant. He was home for 25 years with 24-

hour nursing care. Because RNs now cost \$40/hour in California, the mother has been forced to shift her son to a subacute hospital facility with costs of \$18,000 per month. Consistent with this anecdote, life care planners tell us that \$8 million in lifetime medical costs is typical as a plaintiff's claim for cases related to catastrophic birth outcomes that involve similar care needs.

Table 8. Mean Hospital Costs per Admission for Carbon Monoxide Poisoning, All Ages; Lead Poisoning, Ages 0–4, and Submersions, Ages 0–14, HCUP-NIS, 2007

Age-group	Type of Case	Mean Hospital Costs (2007\$)	Lifetime Medical Cost Multiplier*
All	CO Poisoning	15,769	1.86
0–4	Lead Poisoning	7,857	1.73
0–14	Submersions	25,896	3.36

* This multiplier is the ratio of lifetime medical costs, including professional fees and post-discharge care to acute hospital care from PIRE's existing injury cost models.

Note: Missing standard error due to stratum with single sampling unit.

Table 9. Mean Hospital Costs per Carbon Monoxide Poisoning Admission by Disposition, HCUP-NIS, 2007

Disposition	Mean Hospital Costs (2007\$)
Discharged to Home	9,657
Discharged to Another Institution	70,588
Discharged to Home Health Care	17,542

Table 10. Mean Hospital Costs per Lead Poisoning Admission by Disposition, HCUP-NIS, 2007

Disposition	Mean Hospital Costs (2007\$)
Discharged to Home	7,131
Discharged to Home Health Care	13,654

Table 11. Mean Hospital Costs per Submersion Admission by Disposition, HCUP-NIS, 2007

Disposition	Mean Hospital Costs (2007\$)
Discharged to Home	22,438
Discharged to Another Institution	130,346
Discharged to Home Health Care	156,453

Work Loss Costs

This section provides the present value of future productivity losses (wage work and household work) at 3 percent and 7 percent discount rates.

Lead Poisoning

The relationship between childhood lead poisoning and subsequent IQ loss is well established in the literature. Attention originally focused on productivity losses associated with child blood lead levels (BLL) ≥ 10 $\mu\text{g}/\text{dl}$ (Grosse, 2002; Gould, 2009, Needleman, 2004; Pocock et al., 1994; Schwartz, 1994a; Schwartz, 1994b). More recent research documents impacts of lower range BLLs of 2–10 $\mu\text{g}/\text{dl}$ (Canfield et al., 2003; Lanphear et al., 2005).

Lanphear et al. (2005) is widely accepted as the definitive study (CPSC, 2012; EPA, 2006; Gould, 2009; Muennig, 2009). It estimates a nonlinear relationship between increasing lead exposure and changes in IQ. “Estimated IQ point decrements associated with an increase in blood lead from 2.4 to 10 $\mu\text{g}/\text{dL}$, 10 to 20 $\mu\text{g}/\text{dL}$, and 20 to 30 $\mu\text{g}/\text{dL}$ were 3.9 (95% CI, 2.4–5.3), 1.9 (95% CI, 1.2–2.6), and 1.1 (95% CI, 0.7–1.5), respectively. For a given increase in blood lead, the lead-associated intellectual decrement for children with a maximal blood lead level < 7.5 $\mu\text{g}/\text{dL}$ was significantly greater than . . . for those with a maximal blood lead level ≥ 7.5 $\mu\text{g}/\text{dL}$ ($p = 0.015$).” With adjustment for covariates, the best-fitting model resulted in a relationship between BLL and change in IQ given by:

$$\text{Change in IQ} = \ln(\text{BLL}) * (-2.7).$$

Consistent with these findings, Chandramouli et al. (2009) established that lead poisoning adversely affects academic performance and echoed findings by Chen et al. (2007) that it causes behavioral problems. Importantly, Dietrich et al. (2004) found that chelation therapy did not reduce the long-term neuropsychological and behavioral effects of child lead poisoning.

Muennig (2009), Grosse (2002), and Schwartz (1994b) (all relying heavily on classic work by Needleman) directly analyzed the impact of lower IQ levels on future earning potential. Like IQ impact, this relationship is relatively invariant over time. Thus, future work loss cost can

be estimated by applying their estimates of percentage impact on average potential earnings and household production per IQ point lost to CPSC’s lifetime productivity estimates by age and sex.

Muennig (2009) concludes that Schwartz (1994b) uses the soundest design for assessing the impact of lower IQ levels on future earning potential. Schwartz (1994b) estimated that a one-point difference in IQ results in a 1.76 percent difference in earnings, an estimate that also accounts for a differential rate of high school graduation. Grosse et al. (2002) calculate that analyses in Salvever (1995) indicate the difference averages 2.37 percent, while Neal and Johnson (1996) estimate the difference averages 1.66 percent. So earnings loss for a given BLL change would be $(\ln(\text{BLL}_{\text{post}})-\ln(\text{BLL}_{\text{pre}}))*(-2.7) * .0176 * \text{lifetime earnings}$.

We recommend that the CPSC estimate productivity losses from its lifetime earnings estimates, assuming an initial BLL of one. Table 12 shows how the losses of IQ points associated with various blood lead levels determine earnings losses. The age-earnings model built into the CPSC’s ICM estimates lifetime productivity at age 2 averages \$939,691, including \$653,559 in wages and \$286,132 in household production (present value at a 3 percent discount rate, in 2007 dollars). These estimates exclude productivity loss associated with other chronic health effects of lead exposure.

Table 12. IQ Points and Resulting Percentage of Earnings Lost by Blood Lead Level

Blood Lead Level (ug /dL)	IQ Points Lost	% of Earnings Lost
2	1.9	3.3%
5	4.3	7.6%
7	5.3	9.2%
10	6.2	10.9%
20	8.1	14.2%
30	9.2	16.2%
40	10	17.5%
50	10.6	18.6%
60	11.1	19.5%
90	12.1	21.4%

Earnings Loss Unrelated to IQ Loss. Although the task order focuses on childhood lead poisoning, some cases will continue undiagnosed into adulthood for two reasons. First, chelation therapy reduces but does not eliminate blood lead. Second, some parents decline chelation. Moreover, although a month of intensive treatment often is indicated, side effects or other burdens on parents and the exposed child may lead to drop out mid-course. For analyses that

need to account for enduring effects of lead exposure, Table 13 summarizes signs and symptoms by BLL. Of greater relevance, the table estimates net present value of disability payments that the Veteran’s Administration would make to a claimant by BLL. Those payments proxy lost earnings due to the listed health effects.

Table 13. Adverse Health Effects and Estimated Veteran’s Administration Disability Costs of Adult Lead Exposure by Blood Lead Level, in 2007 Dollars

BLL (µg/dL)	Signs & Symptoms Making Onset	Disability Cost/VA Claim
≤10	ALAD* inhibition, linear homocysteine level rise with BLL, nephrotoxicity, increased risk for ischemic heart disease, hypertension, and end-stage renal disease; decreased glomerular filtration rate; adverse pregnancy outcomes including spontaneous abortion, decreased birth weight and head circumference	
15–20	Erythrocyte protoporphyrin increases, EPO inhibition; subtle, non-specific neurologic effects at 20-60 µg/dL	\$15,075
30	Increased simple reaction time, cognitive CNS deficits, anemia, [†] peripheral nerve dysfunction, proteinuria, enzymuria, hypertension; changes in FSH, LH and testosterone levels; changes in immune parameters	\$58,685
40	Increases in urinary ALA and coproporphyrin, sperm changes, gout, decreased fertility. Changes in T ₄ and TSH levels. Neurobehavioral changes	\$148,237
50	Decreased hemoglobin production, disturbed sleep, anorexia	\$262,915
60	Abdominal pain, arthralgia, clumsiness, vomiting, irritability, inability to concentrate, fatigue, diarrhea, headache, behavioral changes, inhibition of Vitamin D activity, severe renal disease with chronic exposure	\$374,631
80	Frank anemia	\$416,626
100–120	Ataxia, vomiting, convulsions, stupor, constipation, coma; mental retardation, epilepsy, blindness, sterility, severe renal damage	\$664,017

*Abbreviations: ADD (attention deficit disorder); ALA (5-aminolevulinic acid); ALAD (δ-aminolevulinic acid dehydratase); EPO (erythropoietin); FSH (follicle-stimulating hormone); Ig (immunoglobulin); LH (luteinizing hormone); T₄ (thyroxine); TSH (thyroid stimulating hormone)

[†]Anemia can appear at BLLs as low as 20 µg/dL, but may be more commonly associated with higher levels (50-80 µg/dL).

Source: Compiled from various VA disability scales and coding systems and analyzed by the author.

CO Poisoning

Surprisingly, although many studies (reviewed in the section on quality of life loss) have analyzed longer term impairments associated with CO poisoning, a literature search did not reveal data about impact on work-related disability. Data on 92 jury verdicts in carbon monoxide cases included only three, where past wage losses were stated, with losses of \$3,600, \$49,947, and \$95,500. We gained additional insight by calling a leading forensic specialist in vocational

rehabilitation (VR), Robert Taylor. Mr. Taylor reports that brain damage severity is strongly predicted by carboxyhemoglobin level at intake and that brain damage often is diffuse and quite severe in its impact. We suggest a reasonable assumption might be a 15 percent loss in earning capacity (proportional to the quality of life loss estimate below) for those with enduring cognitive sequelae. Hampson et al. (2009) report that regardless of severity, nonfatal CO poisoning does not shorten lifespan, although lifestyle factors cause those experiencing CO poisoning (including intentional self-harm) to have elevated risks of subsequent death from suicide, violence, substance abuse, or unintentional injury.

For people with the age and sex distribution of CO poisoning victims from Exhibit 1, the age-earnings model built into CPSC's ICM predicts lifetime productivity would average \$937,815. This includes \$640,282 in wages and \$297,533 in household production (present value at a 3 percent discount rate, in 2007 dollars). A 15 percent lifetime earnings loss equates to \$140,672, including \$96,042 in wages and \$44,630 in household production.

Submersion

For submersion, children with severe sequelae generally do not survive to working age, meaning their lifetime productivity is lost (Zamula, 1987). Unless the child is placed in residential care, one parent also typically will lose the majority of their productivity until the child dies.

Our current injury cost model includes imperfect estimates of probability of work-related disability for hospital-admitted submersion survivors with moderate sequelae. The best data available are the broadly based but not-fully-representative submersion estimates in Zamula (1987). We supplemented that information with estimates by VR specialist Robert Taylor. Cases with severe sequelae require 24-hour skilled nursing care and greatly reduce lifespan. Mr. Taylor tells us that submersion cases with moderate (as opposed to severe) sequelae are rare, maybe 2 percent of those with sequelae—and those involve substantial deficits, even though the child is ambulatory. National Drowning Prevention Alliance Advisory Council Chair Nadina Riggsbee had similar impressions and reported that pediatricians generally tell parents that typical survival is 6 years. The longest surviving patient with severe sequelae currently is in his 34th year.

The sequelae of submersion are brain injuries. Therefore, one alternatively could calculate the cost of submersion injuries with the work loss estimates for ages 0–4 developed for CDC for traumatic brain injury of different severities (Miller et al., 2009). In 2000 dollars, the short-term earnings losses (which should approximate parental work loss) and percentage of lifetime earnings lost appear in Table 14. Table 15 shows equivalent information for household production. Unfortunately our only insight into the traumatic brain injury (TBI) severity distribution for submersion injuries is information from key informants Taylor and Riggsbee. That information suggests that 98 percent of the cases might face 100 percent disability (meaning it is associated with 100 percent loss and is not covered in Tables 14-15; and 2 percent might be Abbreviated Injury Scale 3 (AIS 3, <http://www.trauma.org/archive/scores/ais.html>) (e.g., a case that resulted in deafness).

Table 14. Short-Term Wage Loss and Percentage of Lifetime Earnings Lost to Traumatic Brain Injury by Abbreviated Injury Scale (AIS) Threat to Life Severity

AIS	Admitted	ED & Released	Outpatient Dept	MD Office	All
Short-Term Earnings Loss (in 2000 dollars)					
1-Minor Injury	\$2,289	\$1,229	\$82	\$112	\$842
2-Moderate Injury	\$2,179	\$468	\$86	\$285	\$686
3-Serious Injury	\$3,561	\$1,926		\$242	\$2,600
4-Severe Injury	\$3,000	\$550		\$412	\$2,016
5-Critical Injury	\$3,613				\$3,613
Percentage of Lifetime Earnings Lost					
1-Minor Injury	6.811%	0.102%	0.114%	0.041%	1.642%
2-Moderate Injury	4.683%	0.398%	0.337%	0.399%	1.009%
3-Serious Injury	5.729%	0.290%		0.102%	3.111%
4-Severe Injury	5.884%	0.316%		0%	3.647%
5- Critical Injury	20.646%				20.646%

Table 15. Short-Term Household Production Loss and Percentage of Lifetime Household Production Lost to Traumatic Brain Injury by Abbreviated Injury Scale (AIS) Threat to Life Severity

AIS	Admitted	ED & Released	Outpatient Dept	MD Office	All
Short-Term Household Production Lost (in 2000 dollars)					
1	\$732	\$249	\$94	\$96	\$275
2	\$678	\$162	\$101	\$137	\$232
3	\$1,186	\$400		\$113	\$780
4	\$1,033	\$158		\$49	\$681
5	\$1,009				\$1,009
Percentage of Lifetime Household Production Lost					
1	7.081%	0.096%	0.093%	0.083%	1.725%
2	4.775%	0.445%	0.500%	0.483%	1.068%
3	5.689%	0.373%		0.061%	3.117%
4	6.716%	0.281%		0.024%	4.132%
5	19.238%				19.238%

Quality of Life Loss

This section reviews existing evidence on quality of life loss, willingness to award compensation for, and willingness to pay to reduce the risk of CO and lead poisoning and submersion. It relies primarily on existing quality-adjusted life year (QALY) rating scales to quantify quality of life loss. A QALY is a health outcome measure defined such that a year in perfect health is 1, death is 0, and fates worse than death are allowed. Most Americans and Canadians rate a persistent unconscious state as worse than death (see, *e.g.*, the systematic review in Miller et al., 1995).

One family of QALY rating scales proved especially applicable to the health conditions under study. Versions 1 and 2 of the Health Utility Index (HUI) were designed to assess QALY loss associated with birth-related problems and chronic childhood conditions (Torrance et al. 1982, 1996). Torrance et al. (1982, 1996) surveyed 112 Ontario parents to calibrate HUI-1 and 194 to calibrate HUI-2. HUI-1 estimates 20 percent QALY loss is associated with needing special education due to trouble learning or remembering. QALY loss rises to 48 percent if the condition will make it impossible for the child to work in a normal setting and 81 percent if the child also will need long-term help with self-care (eating, dressing, bathing, toileting).

Torrance et al. (1996) consider HUI-2 a refinement of HUI-1. It assesses more cognitive and self-care states but does not address work-readiness. Table 16 shows HUI-2 scores for the range of health states commonly associated with CO poisoning, lead poisoning, and submersion. The mildest retardation causes a 5 percent lifetime QALY loss, with retardation serious enough to require special education associated with a 13 percent loss and severe retardation a 51 percent loss. Because lead poisoning harms reproductive health, it adds a further 2 percent to 3 percent loss. Blood lead levels above 60 $\mu\text{g}/\text{dL}$ create habitual irritability, which raises losses by 15 percent to 30 percent. Both CO poisoning and submersion can impair hearing. Depending on degree of hearing loss and severity of retardation, a further 15 percent to 30 percent loss of lifetime QALYs results. The limitation of these estimates is that frequency data on these health status changes do not exist.

Several studies focus more narrowly on retardation losses. Miller (2004) reviewed QALY loss estimates related to reduced IQ loss and retardation. Table 17 summarizes its estimates. They tend to be higher than the estimates in Table 16, with maximum loss exceeding 80 percent.

Table 16. Health Utility Index 2 Estimates of Utility Loss for Brain Injury by Functional Impact

	Normal self-care	Independent self-care with difficulty	Needs help to eat, bathe, dress, or toilet
With No Impact on Mobility, Emotional Health, Pain, or Reproductive Health			
Learns and remembers more slowly than classmates	5.3%	8.3%	25.4%
Requires special education because learns and remembers very slowly	12.7%	15.5%	31.4%
Unable to learn and remember	37.1%	39.2%	50.9%
With Mild Impact on Reproductive Health (any positive lead level)			
Learns and remembers more slowly than classmates	8.3%	11.3%	27.9%
Requires special education because learns and remembers very slowly	15.5%	18.2%	33.6%
Unable to learn and remember	39.2%	41.2%	52.5%
With Habitual Irritability (BLL\geq60 μg/dL) + Reproductive Impact			
Learns and remembers more slowly than classmates	37.6%	39.7%	51.3%
Requires special education because learns & remembers very slowly	42.7%	44.6%	55.3%
Unable to learn and remember	59.2%	60.6%	68.6%
With Hearing Limitations (possible sequelae, submersion or CO poisoning)			
Learns and remembers normally	14.8%	17.6%	33.1%
Learns and remembers more slowly than classmates	19.4%	22.0%	36.7%
Requires special education because learns and remembers very slowly	25.8%	28.2%	41.8%
Unable to learn and remember	46.7%	48.5%	58.6%
With Deafness (possible sequelae, submersion or CO poisoning)			
Learns and remembers normally	41.3%	43.3%	54.3%
Learns and remembers more slowly than classmates	44.6%	46.4%	56.9%
Requires special education because learns and remembers very slowly	49.1%	50.8%	60.5%
Unable to learn and remember	64.0%	65.2%	72.4%

Computed with the Torrance et al. (1996) scoring algorithm.

Table 17. Utility Loss Associated with Retardation

Condition	% Utility Loss	Source
Very severely retarded	83	Torrance (1996)
	75+	Am Med Assoc. (1984)
	91	MacKenzie (1994)
Retarded, needing help with care	57	Kaplan (1976)
	54–57	Torrance (1996)
	55–75	Am Med Assoc. (1984)
	77	MacKenzie (1994)
Moderately retarded with self-care	42–51	Kaplan (1976)
	48–51	Torrance (1996)
	25–50	Am Med Assoc. (1984)
	49	MacKenzie (1994)
Mildly retarded	33	Kaplan (1976)
	20–27	Torrance (1996)
	23	Sintonen (1994)
	23	Glotzer (1995)
	10–20	Am Med Assoc. (1984)
	26	MacKenzie (1994)

Source: Miller et al. (2004)

In Global Burden of Disease studies (World Health Organization (WHO) 2008), Dutch EQ-5d survey data indicated mild mental retardation attributable to lead exposure results in a 36.1 percent QALY loss. This estimate is lower than WHO’s earlier 45.9 percent loss estimate for mental retardation, which was based on expert judgment (World Health Organization, 2008). It is consistent with moderate retardation estimates in Table 17 but higher than the HUI-2 estimates in Table 16.

EPA took a jury verdict approach to value the quality of life loss associated with developmental toxicity (Lopez et al. 1995). From 132 jury verdicts for severe birth defects (cerebral palsy with severe mental retardation, quadriplegia, other symptoms that require lifetime custodial care, deafness, and blindness), they estimated willingness to award for a severe birth defect was \$8.0 million (inflated to 2010 dollars) with a standard deviation of \$7.6 million. That far exceeds other estimates. We also experimented with a jury verdict approach, using jury verdict data that CPSC purchased on near-drowning (9 cases) and nonfire-related carbon monoxide poisoning (92 cases). Unfortunately, virtually none of the cases stated past or future medical or earnings losses. Without data on these monetary losses, which are included in the compensation paid, it was impossible to develop a regression equation to estimate non-monetary

losses from monetary losses. The data also included minimal information on victim health status, with the large majority of cases merely coded as CO poisoning. Among the cases with diagnostic information, seven were coded as headaches, eight as mild brain damage, one as moderate brain damage, one as seizure disorders, one as paraplegia, and one as death. Circumstance data also were quite spotty, with most cases merely coded as accidents at different locations (*e.g.*, rental homes, motels).

Torrance et al. (1982) also provides a basis for computing QALY loss from work loss for impairments like IQ loss that primarily impact earning capacity. Torrance estimates lost ability to work causes a 32.66 percent QALY loss. Applying that estimate in conjunction with the QALY valuation that CPSC's ICM uses in its sensitivity analyses, monetized QALY loss from lost work is 1.43 times associated wage, fringe benefit, and household production losses, implying an incremental quality of life loss of 0.43 times productivity loss.

Lead Poisoning

Several studies estimated lifetime losses from lead poisoning directly. Agee and Crocker (1996) examined household decisions about obtaining chelation therapy for 256 lead-exposed children (with much higher exposures than currently are typical). They estimated willingness to pay (WTP) was \$24 for a 1 percent reduction in a child's lead burden, with a range from \$16 for households that decided against chelation therapy to \$155 for ones that chose it. WTP was 3.1 times medical plus work losses a weighted average of ratios of 20.0 for those choosing chelation and 2.1 for those choosing against it. Perhaps because they are for high lead exposures, all three ratios (3.1, 20.0, and 2.1) exceed the all-cause ratio of 1.43 from Torrance et al. (1982). Lutter (2000) claimed to convert Agee and Crocker's estimates into WTP of \$1,100 to \$1,900 per IQ point gained; his estimate that WTP is about 1/6 of the wage loss is inconsistent with Agee and Crocker's underlying estimate and with Torrance's estimate.

At current exposure levels, Muennig (2009), using 2000–2002 MEPS EQ5d data, estimated lifetime QALY loss per lead-exposed child averages 20 percent (computed at a 3 percent discount rate, with standard deviation of 8 percent). That estimate is slightly above Table 16 estimates, close to moderate retardation estimates in Table 17, and below the Dutch EQ5d

estimate of 36 percent. We consider it the best estimate for lead poisoning, with sensitivity analysis recommended at 15 percent and 35 percent loss.

CO Poisoning

Without a CO alarm in a home, CO poisoning often continues for some time before it is detected. Table 18 shows the signs and symptoms that may accumulate prior to detection. Treatment with oxygen, either normobaric (face mask) or hyperbaric (high-pressure chamber) clears many of these symptoms, at least initially. Neurologic symptoms, however, often persist or develop days to weeks after apparent recovery. UHMS data indicate that 17.7 percent of those treated with hyperbaric oxygen who were tested (15.1 percent of all hyperbaric patients) had cardiac abnormalities consistent with cardiac injury.

A randomized trial of hyperbaric oxygen treatment gives probabilities of longer term sequelae among 224 patients treated in emergency departments or admitted (Weaver et al. 2007, Weaver 2009). Initial carboxyhemoglobin levels in this population averaged 22 percent with a standard deviation of 11 percent. Those levels are greatly influenced by how long the victim was removed from the CO exposure and how long they received normobaric oxygen en route/at the treating facility. Hampson et al. (2012) reported initial levels for 1,394 hyperbaric patients averaged 23.4 percent with a standard deviation of 10.4 percent and a range from 0.1-77.0 percent. Their samples were obtained an average of 2.2 hours from the end of the CO exposure (standard deviation 6.9 hours, range 0 to 198 hours).

Table 18. Acute Symptoms in 1144 Patients with Carbon Monoxide Poisoning

Symptom	% of Patients
Headache	85%
Dizziness	69%
Fatigue or generalized weakness	67%
Nausea or vomiting	52%
Trouble thinking or confusion	37%
Loss of consciousness	35%
Dyspnea	7%
Chest pain	2%

Source: Lavonas, 2007, pooled data from 7 studies.

Table 19 shows the probability of neurocognitive sequelae over time, with and without hyperbaric oxygen. Without hyperbaric oxygen, 34 percent of patients reported headaches at four

weeks (Weaver 2009). In a follow-up study, six years after poisoning, 37 percent (17/42) had abnormal neurologic evaluations, and 19 percent (10/52) had cognitive problems, with some unstated overlap (Weaver 2009). Of 17 patients with neurologic abnormalities, 88 percent had cerebellar, 29 percent cranial, 29 percent motor, 18 percent deep tendon reflex, and 12 sensory abnormalities (Weaver et al. 2008). Of 10 patients with cognitive deficits, all had deficits in visual memory, 80 percent in attention, 70 percent in verbal memory, and 70 percent in executive function in the Hopkins and Weaver abstracts for which no paper has been published (Hopkins & Weaver 2008). None had slow mental processing speed.

Table 19. Probability of Neurocognitive Sequelae of Carbon Monoxide Poisoning by Treatment Mode and Time Post-Exposure

	No Oxygen	Normobaric Oxygen	Hyperbaric Oxygen
Sequelae at 6 weeks	53% (9/17)	41% (60/146)	24% (18/75)
At 6 Months	30% (44/146)		17% (10/58)
At 12 Months	18% (27/149)		14% (9/62)

Source: Weaver et al., 2007 The source did not differentiate sequelae at 6-12 months between no oxygen and normobaric oxygen.

Delayed sequelae were common. Among 75 patients with neurocognitive sequelae who participated in early repeat testing, 68 percent had persistent losses over the first six weeks; 12 percent experienced declines from function immediately post-treatment during weeks 1–2; and another 20 percent experienced declines during weeks 3–6. Only 6 of 17 patients with abnormalities after six years had cognitive sequelae at six weeks and only seven did at 12 months (Hopkins & Weaver 2008). The Hopkins and Weaver abstracts seemed to suggest some of the symptom increase was unrelated to the CO poisoning, but it was unclear how much.

Procedure codes in HCUP were too coarse to provide rates of hyperbaric oxygen use versus oxygen administration by face mask. The 1,500 hyperbaric chamber cases in 2002, and the 14,236 medically treated cases in 2006, suggest that perhaps 10 percent to 12 percent receive hyperbaric treatment. In the nonrandomized portion of Weaver’s three-state study, 37 percent of those without hyperbaric oxygen received no oxygen (Weaver, 2009) (All hyperbaric oxygen was randomly assigned, so the study provided no insight into its usage frequency.) Applying that ratio suggests 26 percent to 27 percent of CO patients get no oxygen therapy. Sequelae rates would average 43 percent at six weeks, 29 percent at six months, 17 percent at 12 months, and 19 percent to 37 percent at six years. Severity of cognitive sequelae was quite large, an average

score three standard deviations below the population norm on a battery of seven neuropsychological tests: general orientation, digit span, Trail Making (Parts A and B), digit-symbol, block design, and story recall (Weaver et al. 2007). Unfortunately, we could not translate that severity into HUI-2 categories. Based on the estimates in Table 16, we conservatively suggest assuming a 15 percent QALY loss for those with sequelae, with sensitivity analysis at 20 percent and 35 percent. With the \$5 million value of statistical life that CPSC uses, lifetime QALYs are valued at \$4,062,185 (\$5,000,000 to \$937,815 in productivity losses from above), so a 15 percent loss would be valued at \$609,328.

Submersion

No data on submersion severity was available because HCUP procedure codes lacked sufficient detail to identify intubation or ICU usage. We suggest assuming that 90 percent of admitted cases will be tracheotomy-tube dependent with utility losses in the 90 percent range, with the rest experiencing losses of perhaps 20 percent to 25 percent. We assume non-admitted cases would suffer no sequelae.

Future Research

For submersions and CO poisoning, if Medicaid claims data were available, we would use discharge to inpatient rehabilitation or long-term care, intensive care unit use, and intubation procedure codes to infer the frequency of severe sequelae. Receipt of services like physical and speech therapy could be used to infer the frequency of more moderate sequelae.

Summary

In summary, this report provides incidence data and a wide range of data on costs per case for CO and lead poisoning and submersion, by exposure severity. Those data will improve ICM estimates for these severe health challenges.

Unfortunately, severity distribution data are unavailable, so we can only provide average figures by assuming a distribution. The easiest and least burdensome way to get accurate severity distributions for CO poisoning and submersion is to analyze Medicaid or possibly private health insurance claims files. In the interim, our best estimates are:

- For CO poisoning, quality-adjusted life-year (QALY) loss and lifetime earnings loss are perhaps 15 percent per year with sequelae, with at least 20 percent of patients experiencing lifetime sequelae. That implies a 3 percent lifetime loss.
- For lead poisoning, a 20 percent QALY loss is the average for child exposures, with losses rising with blood lead level. Lead poisoning also results in ADHD and crime that are costly and further reduce quality of life.
- For submersion, perhaps 90 percent of hospital inpatients experience a 90 percent QALY loss, with the remainder experiencing losses of 20 percent to 25 percent, yielding a combined average of 83 percent. Treated and released cases probably experience minimal QALY loss.

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Appendix A: Other Costs of Lead Poisoning

Special Education. EPA analyzed the costs of special education for children with lead-induced IQ deficits (Agarwal, 1998). We used their estimates of the frequency of special education usage and updated associated costs with current data from the U.S. Department of Education. “The number of children with IQ less than 70 and the number of children with blood lead levels above 20 µg/dL can be used to measure cognitive effects seen mostly in children with high levels of lead exposure. An IQ score of 70 is two standard deviations below the population mean; it is used as an indicator of mental retardation and as the cut-off for special education requirements. Blood lead levels above 20 µg/dL are the level at which CDC traditionally recommended complete medical evaluation, environmental assessment, and necessary environmental remediation for the child and his/her environment. EPA (Agarwal, 1998) used NHANES III data to indicate the fraction of one to two-year olds falling within these categories. An estimated 0.115% of children with lead poisoning have IQs under 70 as a result, and 0.588% have blood lead levels above 20 µg/dL.” (Agarwal, 1998). These data suggest that 19.6% (.115/.588) of children with lead levels exceeding 20 µg/dL require special education.

Exclusive of capital spending and debt service, average spending per pupil in U.S. public elementary or secondary school was \$10,441 in FY 2007–2008 (National Center for Education Statistics, 2011, Table 190, in Feb 2009 dollars).² Based on a massive national study, Chambers et al. (2003) reports that spending per special education student is 1.6 times spending on other students. Swanson (2008) reports that 9 percent of U.S. students have special educational needs. Thus the additional spending per US special needs student is \$5,944 ($0.6 * \$10,441 / (.91 + .09 * 1.6)$). The special education multipliers average (a) 2.3 for mental retardation (or for orthopedic impairment) and (b) 2.5 for traumatic brain injury (Chambers et al. 2003, Exhibit 1), implying annual special education costs of (a) \$22,784 and (b) \$24,765 for these conditions. Thus special education spending per lead poisoning case at ages 0-4 averages \$4,660 per school year or \$46,713 in present value from grades K–12 (assuming exposure at age 2).

² This estimate excludes \$1,676 per pupil in annual capital spending and debt service. Those costs would not be higher for special education students.

ADHD. Braun et al. (2006) analyzed data from the National Health and Nutrition Examination Survey (NHANES-III) and found that children with blood lead concentrations greater than 2 µg/dl were at a 4.1-fold increased risk of ADHD. Medical costs of ADHD average \$1,586 per year (or \$14,984 from ages 6 through 18, brought to present value using a 3 percent discount rate; inflated to 2009 dollars) (Swensen et al., 2003). This estimate is based on cost differentials relative to a matched control group in a privately insured population. The present value of QALY losses through age 18 for ADHD diagnosed at age 6 averages 1.9. Secnik et al. (2005) estimated the mean QALY level for a child with an acute case of ADHD at 0.74. They rated the utility of someone with chronic ADHD treated with no side effects at 0.96 compared to unmedicated utility of 0.88, implying an annual utility loss of at least 0.08 and possibly as much as 0.12 from chronic ADHD. These estimates were based on children in the United Kingdom assessed using the EQ5d QALY instrument. Data were not readily available on the QALY norm for the age group studied. No comparable United States data were available. Coghill et al. (2004) estimated utility of an ADHD responder to treatment at 0.837 (standard error 0.039) and of a non-responder to treatment at 0.773 (standard error 0.039), independent of treatment received, suggesting a loss of at least 0.064 (0.837–0.773).

Crime. Wright et al. (2008) finds that child lead poisoning increases criminal behavior. “Adjusted total arrest rates” for violent and non-violent crime “at ages 18–24 were greater for each 5 µg/dl increase in blood lead concentration.” Relative risk (RR) “= 1.07 (95% CI 0.88–1.29) for average childhood blood lead through age 6, and 1.27 (95% CI 1.03–1.57) for blood lead at age 6. Adjusted arrest rates for violent crimes at ages 18–24 were greater for each 5 µg/dl increase in blood lead: RR = 1.30 (95% CI 1.03–1.64) for average childhood blood lead, and 1.48 (95% CI 1.15–1.89) for blood lead at age 6.” We assume these odds ratios apply to both youth and subsequent adult crime, and that the differential arrest rates match the differential crime rates. If lead poisoning is relatively rare, with RR=1.27, per 5 µg/dl, crime associated with lead poisoning will equal roughly 0.27 times the average crime rate. With crime incidence from Miller and Hendrie (2013), RR for non-violent crime would be 1.23 (0.27 * 98,827,502 crimes - 0.48 * 20,380,407 violent crimes)/ 78,447,095 non-violent crimes) for each 5 µg/dl increase in blood lead.

Using these RRs requires separate costs for violent and non-violent personal crimes covered in arrest data. We relied on estimates from Miller and Hendrie (2013), but substituted the \$5 million value of a statistical life that CPSC uses in its regulatory analyses.

Table 20 summarizes unit costs by crime used to estimate costs per crime. Table 21 presents incidence, percentage of crimes reported to police, and percentage leading to arrest. Costs per police-reported crime averaged \$49,727 for violent crime and \$3,328 for property crime. Costs per crime were lower at ages 8–18 than at ages 18 and older.

Table 22 shows the age distribution of arrestees and associated crimes per person under the assumption that virtually all arrests at ages 0–14 occur at ages 8–14. Applying the rise in relative risk from above to the crime rate per capita, then multiplying times cost per crime yields the annual crime costs per 5 µg/dl rise in blood lead level by age shown in the right-hand columns. We computed present value of these annual costs over a lifespan starting at age 2, accounting for life table probabilities of survival from year to year. Conservatively, except in sensitivity analysis, we restricted Table 22 to police-reported crimes and excluded impaired driving crashes, intimate partner violence, and child maltreatment. Present value of estimated costs per 5 µg/dl rise in blood lead level are \$77,125, including \$74,898 in violent crime costs and \$2,228 in non-violent crime costs. Had we assumed crimes not reported to the police are reduced proportionately to police-reported crimes and included all personal crimes, estimated crime costs per 5 µg/dl rise in blood lead level would be \$181,824, including \$176,516 in violent crime costs and \$5,308 in non-violent crime costs.

Blood lead level elevation from consumer products depends on method and duration of exposure. Licking a toy regularly, swallowing something, or licking a product on a single access are quite different exposures. To estimate resulting blood lead levels, a regulatory analysis presumably would draw on medical or engineering expertise or on historical data on elevation with similar exposures.

Table 20. Costs per Police-Reported Crime by Cost Category in 2010 Dollars

	Medical	Mental Health	Work	Property Damage	Public Services*	Quality of Life	Adjudication & Sanctioning	Perpetrator Work Loss	Total
Murder	\$16,183	\$10,735	\$1,577,758	\$175	\$2,852	\$3,422,242	\$368,336	\$143,023	\$5,541,304
Rape	\$2,887	\$5,830	\$5,765	\$157	\$790	\$128,196	\$24,679	\$9,517	\$177,821
Robbery	\$1,313	\$176	\$2,716	\$1,143	\$405	\$14,754	\$11,866	\$4,413	\$36,786
Assault	\$1,141	\$361	\$1,879	\$70	\$335	\$12,708	\$5,655	\$1,878	\$24,027
Burglary	\$0	\$0	\$34	\$2,564	\$450	\$0	\$2,041	\$693	\$5,782
Larceny/Theft	\$0	\$0	\$27	\$936	\$442	\$0	\$778	\$168	\$2,351
Motor Vehicle Theft	\$0	\$0	\$102	\$6,423	\$269	\$0	\$1,778	\$571	\$9,143
Arson	\$2,373	\$40	\$2,924	\$17,411	\$3,561	\$5,548	\$2,310	\$603	\$34,770
Forgery/Counterfeiting	\$0	\$0	\$0	\$414	\$266	\$0	\$2,525	\$682	\$3,887
Person-to-Person Fraud	\$0	\$0	\$0	\$1,687	\$65	\$0	\$2,289	\$682	\$4,723
Embezzlement	\$0	\$0	\$0	\$414	\$266	\$0	\$2,743	\$682	\$4,105
Buying Stolen Property,	\$0	\$0	\$0	\$0	\$266	\$0	\$4,791	\$1,169	\$6,226
Vandalism	\$0	\$0	\$88	\$261	\$70	\$0	\$2,075	\$724	\$3,218
Weapons Carrying,	\$0	\$0	\$0	\$0	\$70	\$0	\$2,289	\$799	\$3,158
Prostitution/Pandering	\$0	\$0	\$0	\$0	\$70	\$0	\$229	\$80	\$379
Other Sex Offenses	\$1,278	\$3,682	\$3,947	\$145	\$316	\$86,102	\$24,679	\$9,517	\$129,666
Drug Possession/Sales	\$0	\$0	\$0	\$0	\$4,490	\$0	\$3,202	\$1,118	\$8,810
Gambling	\$0	\$0	\$0	\$0	\$70	\$0	\$229	\$80	\$379
DUI, No Crash	\$0	\$0	\$0	\$0	\$19	\$0	\$415	\$145	\$579
Liquor Law Violations	\$0	\$0	\$0	\$0	\$70	\$0	\$1,093	\$381	\$1,544
Drunkenness	\$0	\$0	\$0	\$0	\$70	\$0	\$1,093	\$381	\$1,544
Disorderly Conduct	\$0	\$0	\$0	\$0	\$70	\$0	\$1,093	\$381	\$1,544
Vagrancy	\$0	\$0	\$0	\$0	\$70	\$0	\$1,093	\$381	\$1,544
Curfew/Loitering Violations	\$0	\$0	\$0	\$0	\$70	\$0	\$1,093	\$381	\$1,544
All Other Nontraffic Offenses	\$0	\$0	\$0	\$0	\$70	\$0	\$229	\$92	\$391
All Violent Crime	\$1,254	\$619	\$7,016	\$377	\$396	\$28,681	\$8,410	\$2,974	\$49,727
All Other Personal Crime	\$0	\$0	\$27	\$856	\$622	\$0	\$1,386	\$436	\$3,328
Perpetrator Ages 8–17 Years									
All Violent Crime	\$1,202	\$475	\$3,358	\$452	\$403	\$18,215	\$6,855	\$2,359	\$33,320
All Other Personal Crime	\$0	\$0	\$39	\$969	\$489	\$0	\$1,425	\$449	\$3,371
Perpetrator Ages 18 & Over									
All Violent Crime	\$1,280	\$691	\$8,835	\$340	\$392	\$33,885	\$9,183	\$3,280	\$57,885
All Other Personal Crime	\$0	\$0	\$24	\$831	\$652	\$0	\$1,405	\$444	\$3,356

* Public services include police, fire, EMS, victim services. Violent crimes are rape, robbery, assault, arson, other sexual offenses, intimate partner violence, and child maltreatment.

Table 21. Percentage of Crimes Reported, Percentage of Reported Crimes that Lead to an Arrest, and Number of Arrests in 2010

	Reported Crimes	% Reported	% Arrested of Reported	% Under Age 18	Arrests
Murder	16,259	100.0%	73.9%	9.0%	11,201
Rape	92,322	9.8%	21.8%	14.1%	20,088
Robbery	409,584	65.6%	27.4%	24.1%	112,300
Assault	4,414,537	43.8%	38.1%	15.4%	1,700,937
Burglary	2,385,971	50.7%	12.1%	22.7%	289,769
Larceny/Theft	8,460,936	31.0%	15.0%	22.3%	1,271,410
Motor Vehicle Theft	761,669	85.4%	9.4%	22.1%	71,487
Arson	65,763	100.0%	20.4%	48.5%	13,416
Forgery/Counterfeiting	293,828	31.0%	26.6%	2.2%	78,101
Fraud	1,166,474	31.0%	16.1%	3.2%	187,887
Embezzlement	46,681	31.0%	35.6%	2.7%	16,616
Buying Stolen Property,	157,806	100.0%	60.1%	15.6%	94,802
Vandalism	3,594,637	8.3%	7.0%	30.6%	252,753
Weapons Carrying,	322,300	100.0%	49.3%	19.8%	159,020
Prostitution	66,926	100.0%	93.6%	1.7%	62,668
Other Sex Offenses	232,953	9.8%	31.2%	18.0%	72,628
Drug Possession/Sales	2,242,480	100.0%	73.1%	10.4%	1,638,846
Gambling	11,458	100.0%	86.8%	13.8%	9,941
Intimate Partner Violence	628,331	3.7%	100.0%	3.5%	111,062
Child Maltreatment	512,790	22.9%	100.0%	6.0%	689,435
Driving Under the Influence	560,718	31.2%	100.0%	0.9%	1,412,223
Liquor laws	615,172	100.0%	100.0%	18.9%	512,790
Drunkenness	32,033	100.0%	100.0%	2.3%	560,718
Disorderly conduct	94,797	100.0%	100.0%	25.1%	615,172
Vagrancy	3,720,402	100.0%	100.0%	6.8%	32,033
Curfew/Loitering Violations	5,231,417	100.0%	100.0%	100.0%	94,797
All Other Non-Traffic Offenses	25,675,409	100.0%	100.0%	8.0%	3,720,402
All Violent Crimes	16,259	27.9%	48.5%	14.8%	2,731,067
All Non-Violent Crimes	92,322	33.8%	25.0%	12.2%	512,790

Table 22. Annual Arrest Rate and Estimated Crimes Committed per Person by Age Range, 2010, and Estimated Annual Violent and Property Crime Costs Over Different Age Ranges per 5 µg/dl Rise in Blood Lead Level at Age 0–4 (in 2010 dollars)

Age Group	% Arrested	Police-Reported Crimes per Person	Police-Reported Only; No DWI, Domestic Violence		All Crime	
			Violent Crime Cost	Property Crime Cost	Violent Crime Cost	Property Crime Cost
8 to 14 years	1.14%	0.045	\$1,041	\$31	\$1,919	\$41
15 to 19 years	8.68%	0.342	\$7,918	\$235	\$14,600	\$313
20 to 24 years	9.45%	0.218	\$5,640	\$168	\$15,276	\$521
25 to 29 years	7.18%	0.166	\$4,285	\$128	\$11,605	\$396
30 to 34 years	5.57%	0.129	\$3,324	\$99	\$9,004	\$307
35 to 39 years	4.33%	0.100	\$2,582	\$77	\$6,995	\$239
40 to 44 years	3.79%	0.087	\$2,261	\$67	\$6,124	\$209
45 to 49 years	3.16%	0.073	\$1,887	\$56	\$5,112	\$174
50 to 54 years	2.15%	0.050	\$1,285	\$38	\$3,482	\$119
55 to 59 years	1.23%	0.028	\$732	\$22	\$1,983	\$68
60 to 64 years	0.64%	0.015	\$382	\$11	\$1,034	\$35
65 years & over	0.19%	0.004	\$111	\$3	\$302	\$10

Arrest rates from Table 4.4, Sourcebook of Criminal Justice Statistics 2010. Washington DC: Bureau of Justice Statistics, 2012.

Appendix B: Narrative Descriptors for International Classification of Disease Codes and NEISS Codes Included

Codes from the 9th Edition of ICD, Clinical Modification,
<http://www.cdc.gov/nchs/icd/icd9cm.htm>

984	Toxic effects of lead and its compounds (including fumes)
986	Toxic effect of carbon monoxide
994.1	Drowning and nonfatal submersion
E830	Accident to watercraft causing submersion
E832	Other accidental submersion or drowning in water transport accident
E861.5	Accidental poisoning by lead paints
E866.0	Accidental poisoning by lead and its compounds and fumes
E868	Accidental poisoning by other utility gas and other carbon monoxide
E868.0	Liquefied petroleum gas distributed in mobile containers Butane or carbon monoxide from incomplete combustion of these gases Liquefied hydrocarbon gas NOS or carbon monoxide from incomplete combustion of these gases Propane or carbon monoxide from incomplete combustion of these gases
E868.1	Other and unspecified utility gas Acetylene or carbon monoxide from incomplete combustion of these gases Gas NOS used for lighting, heating, or cooking or carbon monoxide from incomplete combustion of these gases Water gas or carbon monoxide from incomplete combustion of these gases
E868.2	Accidental poisoning by motor vehicle exhaust gas
E868.3	Accidental poisoning by carbon monoxide from incomplete combustion of other domestic fuels
E868.8	Accidental poisoning by carbon monoxide from other sources
E868.9	Accidental poisoning by unspecified carbon monoxide
E910	Accidental drowning and submersion

Codes from the 10th Edition of ICD,
<http://apps.who.int/classifications/icd10/browse/2010/en> (regardless of medical treatment, multiple cause of death data typically include both a diagnosis code and an external cause code)

Diagnosis Codes

T56.0 Toxic effect of lead and its compounds
T58 Toxic effect of carbon monoxide

External Cause Codes

W 16	Diving or jumping into water causing injury other than drowning or submersion
W 29	Contact with other powered hand tools and household machinery (washing machines)
W65	Drowning and submersion while in bath-tub
W66	Drowning and submersion following fall into bath-tub
W67	Drowning and submersion while in swimming-pool
W68	Drowning and submersion following fall into swimming-pool
W73	Other specified drowning and submersion
W74	Unspecified drowning and submersion
X47	Accidental poisoning by and exposure to other gases and vapors
Y17	Poisoning by and exposure to other gases and vapors, undetermined intent
Y21	Drowning and submersion, undetermined intent

NEISS Codes

64	Submersion (including drowning)
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