

**Platform Session 1**

**Friday, September 15  
Abstracts #1–#5**

**10:45 am–12:00 pm**

**1 NEUTRALIZATION OF LATRODECTUS MACTANS AND L. HESPERUS VENOM BY REDBACK SPIDER (L. HASSELTII) ANTIVENOM.\***

Daly FFS, Hill RE, Bogdan GM, Dart RC. *Rocky Mountain Poison and Drug Center, Denver Health Authority, University of Colorado Health Sciences Center, Denver, CO*

**Objective:** To test the effectiveness of redback spider (*L. hasseltii*) antivenom in neutralizing the lethal effects of *L. hesperus* and *L. mactans* (North American black widows) venoms. **Methods:** LD<sub>50</sub> values for the *L. hesperus* and *L. mactans* venom preparations were determined in preliminary studies. A prospective, randomized, double blind experiment was then performed for each venom using a murine envenomation model. Power analysis determined that 7 animals per group conferred a 95% power to detect an 80% difference between the experimental groups with a two-tailed alpha of 0.05. The following treatments were pre-mixed and incubated at 25°C for 1 hour prior to intraperitoneal injection: 1) saline control + protein control (nonspecific equine IgG with a concentration equal to the antivenom) 2) saline control + *L. hasseltii* antivenom 3) venom (at 5× LD<sub>50</sub>) + protein control and 4) venom + *L. hasseltii* antivenom. The study endpoints were elapsed time until death and survival at 24 hours. Comparison of survival curves was performed with Log-rank and Wilcoxon tests. **Results:** The murine LD<sub>50</sub> values for *L. hesperus* and *L. mactans* venoms were 0.64 mg/kg and 0.26 mg/kg, respectively. All mice in the *L. hesperus* or *L. mactans* venom and nontoxic protein control groups died. All mice in group 4 in both experiments survived ( $p < 0.0001$ ). **Conclusion:** Redback spider antivenom is effective in neutralizing the lethal effects of *L. hesperus* and *L. mactans* venoms in a murine envenomation model. While our study is limited by the (optimized) premixing of antigen and antibody, it generates the hypothesis that redback antivenom would be effective in the treatment of latrodectism in humans caused by the North American widow spiders.

**\*Winner of the American Academy of Clinical Toxicology 1999 Research Award.**

**2 CONVERSION OF DIRECT COOMBS ANTIBODY SCREEN AFTER ADMINISTRATION OF CROTALIDAE ANTIVENIN (WYETH®).**

Ruha AM, Tanen D, Graeme K, Curry S, Beuhler M. *Department of Medical Toxicology, Good Samaritan Regional Medical Center, Phoenix, AZ*

**Background:** Crotalidae envenomation frequently produces coagulopathy requiring treatment with horse serum-derived Crotalidae antivenin (Wyeth). We hypothesized that antivenin administration would produce a positive direct Coombs antibody test (DAT). **Method:** 20 patients presenting with history, clinical and laboratory evidence of Crotalidae envenomation and requiring Crotalidae Antivenin (Wyeth) treatment had DAT measured before and 1–3 hours after antivenin administration. **Results:** Our patient population was typical of previously reported Crotalidae victims, with a mean age of  $24.8 \pm 2.5$  years and 85% being male. Upper extremities were involved in 75% of envenomations, and lower extremities in 25%. Patients received a mean of  $23 \pm 1.64$  vials of Crotalidae antivenin. Conversion from negative to positive DAT occurred in 35% of patients. All patients had negative DAT prior to antivenin administration. No patients required blood products or demonstrated clinical evidence of hemolysis. **Discussion:** A positive DAT commonly occurs

after administration of Crotalidae antivenin. Given the large load of immunoglobulin given with antivenin administration, the conversion of DATs may represent nonspecific binding of immune complexes and/or complement to human RBCs. **Conclusion:** Administration of Crotalidae Antivenin (Wyeth) may result in a positive DAT; and this, in and of itself, should not be taken as evidence of likely hemolysis in the absence of other convincing findings.

### 3 EFFICACY OF A NEW DIGOXIN-SPECIFIC FAB (DIGITAB) IN A RAT MODEL.

Hill RE, Bogdan GM, Dart RC. *Rocky Mountain Poison and Drug Center, Denver Health Authority, University of Colorado Health Sciences Center, Denver, CO*

**Background:** Digibind reverses the cardiovascular effects of digoxin, but efficacy of a new digoxin-specific Fab (DigiTAB) has not been demonstrated. **Objective:** To compare the efficacy of DigiTAB and Digibind in reversing digoxin cardiotoxicity in a rat model. **Methods:** Baseline (0 minutes) measurements were recorded. Digoxin toxicity was induced in 4 groups of 10 rats by IV administration of 1.0 mg/kg digoxin over 5 minutes. At 30 minutes postinfusion (time of toxicity), each rat was randomized to one of the following treatments: DigiTAB, Digibind, nondigoxin specific Fab control, and NaCl/Sorbitol control. Fab doses were equimolar to amount of digoxin infused. ECG recordings and serum potassium levels were determined at pre-defined intervals from 0–210 minutes and were the primary variables used to compare efficacy. **Results:** The following table summarizes mean values for variables ( $\pm$ SD) at pre-defined intervals:

Treatment	PTQ Index			Serum Potassium Levels (mEq/L)		
	Baseline	Toxicity	210 min	Baseline	Toxicity	210 min
Digibind	0.20 (0.02)	1.97 (0.90)	0.48 (0.20)*	3.5 (0.54)	7.4 (0.85)	4.6 (0.28)*
DigiTAB	0.19 (0.03)	1.83 (1.04)	0.66 (0.39)*	3.6 (0.60)	7.3 (0.92)	4.7 (0.67)*
Sorbitol	0.18 (0.02)	1.74 (1.06)	1.00 (0.52)	3.5 (0.44)	7.3 (0.90)	5.8 (1.10)
Fab control	0.19 (0.02)	1.71 (0.50)	0.91 (0.43)	3.8 (0.45)	7.6 (1.43)	6.4 (1.00)

PTQ index = [PR interval (sec)  $\times$  T score]/QT<sub>c</sub> (sec). An (\*) indicates groups that did not differ from each other ( $p > 0.05$ ), but differed from controls ( $p < 0.05$ ). **Conclusions:** There were no differences between DigiTAB and Digibind in reversing digoxin-induced increases in PTQ index and serum potassium levels. The primary limitation of the study was the use of an animal model.

### 4 PSILOCYBIN AND 5-SUBSTITUTED TRYPTAMINES CAUSE FALSE-POSITIVE REACTIONS WITH THE MEIXNER TEST.

Beuhler M, Lee DC. *North Shore University Hospital, Manhasset, NY*

**Background:** Meixner developed a simple colorimetric spot test for detection of amatoxin, an organic toxin found in mushrooms such as *Amanita Phalloides*. There are scant data studying specificity and interrater reliability of the Meixner test using amatoxin (A), psilocybin extract (P), 5-hydroxytryptamine (H), and 5-methoxy-*N,N*-dimethyltryptamine (M). **Methods:** Various quantities of test substances were deposited on lignin rich paper using standard volumes (A 2  $\mu$ g, 10  $\mu$ g; H 100  $\mu$ g, 200  $\mu$ g; M 100  $\mu$ g, 200  $\mu$ g; P 20  $\mu$ L, 70  $\mu$ L). P was prepared by extraction and filtration of a dried 1.5 gram sample of P containing mushroom using 15 mL of warm methanol. All test specimens were prepared as per the protocol described by Meixner, and one control (methanol only) was included in the presented set. Two noncolor-blind, blinded physicians were asked to determine if specimens had a sky blue color present (positive result) after 5 to 10 minutes. Amatoxin was purchased from Boehringer-Manheim Chemical Company (Indianapolis, IN), M and H were purchased from Sigma Chemical Company (St. Louis, MO). **Results:** Testers were able to correctly observe positives for all A specimens, and negatives for all H specimens. However, all testers declared the M and P containing samples positive ( $\kappa = 1$ ). **Conclusions:** The Meixner test is a sensitive test, allowing determination of A with as little as 2  $\mu$ g. It is not a specific test, resulting in 100% false positive rate with samples containing P and M.

## 5 VACCINATION AGAINST NICOTINE DURING CONTINUED NICOTINE ADMINISTRATION IN RATS: IMMUNOGENICITY OF THE VACCINE AND EFFECTS ON NICOTINE DISTRIBUTION TO BRAIN.

Hieda Y, Keyler DE, Ennifar S, Fattom A, Pentel PR. *Minneapolis Medical Research Foundation and Hennepin County Medical Center, Minneapolis, MN; Nabi, Rockville, MD*

**Background:** Vaccination against nicotine has been proposed as a potential treatment for nicotine dependence. Because vaccination may take months to elicit satisfactory antibody levels, the clinical usefulness of this approach will be enhanced if vaccination can be accomplished during continued nicotine intake (e.g. before a smoker quits). The current study examined the immunogenicity of a nicotine conjugate vaccine during continued nicotine dosing in rats, and its effects on nicotine distribution to the brain. **Methods:** Nicotine was administered over 11 weeks as either 20 IV bolus injections per day to simulate the usual pattern of nicotine intake from cigarette smoking, or as a continuous s.c. infusion of nicotine by osmotic pump for 11 weeks to provide 24 hour per day exposure. Rats were vaccinated during the 11 weeks of nicotine dosing. A single additional IV nicotine dose was administered at the end of each experiment. **Results:** Nicotine-specific antibody titers after the third booster dose were not compromised by either regimen of concurrent nicotine administration compared to those of rats receiving saline ( $p > 0.5$ ). The distribution of the final single nicotine dose to brain was reduced by 40–60% in vaccinated rats compared to controls ( $p < 0.05$ ). **Conclusions:** These data suggest that vaccination during concurrent nicotine administration is feasible, and that the ability of vaccination to reduce nicotine distribution to brain is preserved even after months of nicotine dosing at rates approximating cigarette smoking.

**Platform Session 2**

**Friday, September 15**  
**Abstracts #6–#11**

**4:00 pm–5:30 pm**

## 6 MEDICAL SURVEILLANCE OF CLANDESTINE DRUG LAB INVESTIGATORS.

Burgess JL, Kovalchick DF, Siegel EM, McCurdy SA. *University of Arizona, Tucson, AZ; University of Washington, Seattle, WA; University of California, Davis, CA*

**Objective:** To evaluate risk factors for longitudinal changes in medical surveillance data in law enforcement officers investigating clandestine drug laboratories. **Methods:** Lab investigators with initial training and at least one annual update and two medical surveillance examinations were eligible for the study. Participants completed a questionnaire evaluating occupational and personal health history. Medical surveillance examinations and administrative records were reviewed. Longitudinal analysis using a first-order random effects model (REM), which is similar to multiple regression, was implemented to identify risk factors for longitudinal changes in pulmonary function ( $FEV_1$ ), and laboratory tests (ALT, AST, hemoglobin, platelets, and WBC). **Results:** Forty-two (48%) of 88 eligible subjects participated in the study. Participants had an average of 3.8 medical evaluations for the period 1991 to 1998. Average annual decline in  $FEV_1$  was 0.11 mL/y (range 0.007–0.670). There were no associations between longitudinal changes in  $FEV_1$ , duration of exposure, and extent of respiratory protection used. For ALT, body mass index (coef. 1.38,  $p = 0.006$ ), minutes spent in level B protection during entry (coef. 0.16,  $p = 0.008$ ), and minutes spent in level D protection during entry (coef.  $-0.04$ ,  $p = 0.017$ ), but not alcohol use, were significant risk factors. For AST age (coef.  $-0.43$ ,  $p = 0.023$ ) and minutes spent in level D protection during entry (coef.  $-0.03$ ,  $p = 0.024$ ) were significant risk factors. No significant association was found between changes in hematologic parameters and measures of occupational exposure. **Conclusions:** No association was found between indices of exposure to methamphetamine labs and longitudinal changes in  $FEV_1$ . Contrary to expectations, ALT and AST decreased with longer periods of unprotected exposure during lab entry.

### 7 ADVERSE RESPIRATORY EFFECTS FOLLOWING OVERHAUL IN FIREFIGHTERS.

Burgess JL, Nanson CJ, Bolstad-Johnson DM, Gerkin R, Hysong T, Lantz RC, Sherrill DL, Quan SF, Witten ML. *University of Arizona, Tucson, AZ; City of Phoenix Fire Department, Phoenix, AZ*

**Objective:** To evaluate the respiratory toxicity associated with exposure to low concentrations of products of combustion during firefighter overhaul, which involves searching for and extinguishing possible sources of reignition. **Methods:** Firefighters were monitored at baseline and again after completing overhaul. Testing included spirometry and serum pneumoprotein [Clara cell protein (CC16) and surfactant-associated protein A (SP-A)] assessments. One group of firefighters wore no respiratory protection (NRP) while the other group wore air-purifying respirators (APR). **Results:** The results are summarized in the following table.

Group	n	CC16 ( $\mu\text{g/L}$ )	SP-A ( $\mu\text{g/L}$ )	n	FVC (L)	FEV <sub>1</sub> (L)
NRP-baseline	25	8.92 $\pm$ 3.52	287.0 $\pm$ 144.3	19	5.43 $\pm$ 0.70	4.06 $\pm$ 0.64
NRP-overhaul	25	12.34 $\pm$ 3.61	305.7 $\pm$ 156.7	19	5.36 $\pm$ 0.73	3.94 $\pm$ 0.65
		p = 0.003	p = 0.539		p = 0.228	p = 0.097
APR-baseline	26	9.55 $\pm$ 3.50	250.3 $\pm$ 117.4	24	5.38 $\pm$ 0.66	4.16 $\pm$ 0.48
APR-overhaul	26	14.57 $\pm$ 5.22	333.7 $\pm$ 140.7	24	5.23 $\pm$ 0.72	4.05 $\pm$ 0.64
		p < 0.001	p = 0.001		p = 0.001	p = 0.005

**Conclusions:** Overhaul increased CC16 in both groups. CC16 has been shown to increase with altered alveolar-capillary permeability, but the clinical significance of this change has not yet been determined. Contrary to expectations, SP-A increased and FVC and FEV<sub>1</sub> decreased in the firefighters wearing APR. Continuing analysis will focus on differences in levels of chemical exposure between the two groups. Firefighter respiratory exposures during overhaul have the potential to cause changes in spirometry and lung permeability.

### 8 THE PROTECTIVE EFFECTS OF POLY ADP-RIBOSE POLYMERASE (PARP) & CASPASE INHIBITION FOLLOWING CARBON MONOXIDE (CO) POISONING.

Gilmer B, Tomaszewski C, Watts JA. *Carolinas Medical Center, Charlotte, NC*

**Background:** *In vivo* models suggest the involvement of PARP in mediating necrosis while caspases initiate apoptosis following hypoxia. This study examines the efficacy of PARP and caspase 1,3 inhibition in preventing neurological sequelae after CO poisoning. **Methods:** Male Swiss-Webster mice (30–35 g) were exposed to CO: 1,000 ppm for 40 minutes and 50,000 ppm until loss of consciousness. 15 minutes later they received either 3-aminobenzamide (3-AB) 10 mg/kg ip or disulfiram (DSF) 200 mg/kg ip, while control poisoned animals received only normal saline (NS) ip. An additional control group was not poisoned. All animals underwent passive avoidance training at 24 hours before CO poisoning. Memory was assessed by measuring step-down latency (SDL) 7 days after poisoning. Histologic differences were compared in the CA1 region of the hippocampus. SDL was compared among groups with Kruskal-Wallis nonparametric ANOVA followed by Dunn's test for multiple comparisons; cell counts were compared with GLM ANOVA and Scheffe's. **Results:** Median (range) of SDL (sec) and number of pyknotic cells/40 $\times$  field ( $\pm$ SD) are reported (\*p < 0.05 vs nonpoisoned):

	Nonpoisoned + NS	CO + NS	CO + 3-AB	CO + DSF
SDL	300 (130–300; n = 15)	166 (2–300; n = 11)*	300 (154–300; n = 10)	300 (179–300; n = 3)
Cells	1 $\pm$ 1 (n = 3)	6 $\pm$ 1.5 (n = 6)*	2 $\pm$ 0.9 (n = 6)	—

**Conclusions:** Both 3-AB, an inhibitor of PARP, and DSF, an inhibitor of caspases 1 and 3, were found to be protective of memory in mice after acute CO poisoning. This suggests a role for both apoptosis and necrosis in explaining delayed neuro-deficits after CO poisoning.

## 9 PULMONARY ASPIRATION AND GASTROINTESTINAL OBSTRUCTION ASSOCIATED WITH THE USE OF MULTIPLE DOSE ACTIVATED CHARCOAL.

Dorrington C, Johnson DW, Brant R, and the MDAC Complication Study Group. *University of Calgary, Calgary, Alberta, Canada*

**Objective:** The objective of this study was to determine the incidence of and risk factors for the development of pulmonary aspiration and gastrointestinal obstruction with the use of multiple dose activated charcoal (MDAC). **Methods:** The study population was drawn from seven tertiary care hospitals (five general and two pediatric) in four North American cities. Medical records of all inpatients between March 1993 and March 1998 with a discharge diagnosis (primary or secondary) of *poisoning* (ICD9-CM codes 960-989.9) were reviewed to select those patients who had received MDAC (defined as receiving two or more doses of activated charcoal, within a twelve-hour period, during the course of their care). Charts of patients who received MDAC were reviewed for the following data: patient demographics, potential risk factors, and clinical information regarding complication occurrence. Accuracy of inclusion/exclusion of study patients and of data abstraction was determined by independent review of a randomly selected number of charts by the primary investigator and each site reviewer. A panel of three expert physicians experienced in emergency medicine and clinical toxicology, and not otherwise involved in chart abstraction, independently reviewed all cases of potential complications as to whether the cases met preset criteria. Differences in opinion were resolved by consensus. **Results:** Of the 5938 charts reviewed, 828 received MDAC. Five (0.6%, 95 percent C.I. 0.1-1.1%) cases (all adults) in this group were found to meet the criteria for pulmonary aspiration, and none (0%, 95 percent CI 0-0.3%) met the criteria for gastrointestinal obstruction. None of the patients with pulmonary aspiration died or were left with residual sequelae. Given the small number of complications, no further statistical analysis to determine independent risk factors was possible. **Conclusion:** Complications associated with the use of MDAC are uncommon.

## 10 LOW PRESSURE VENTILATION DOES NOT PREVENT MICROVASCULAR PERMEABILITY LUNG INJURY IN A RAT MODEL OF ACTIVATED CHARCOAL ASPIRATION.

Arnold TC, Cady FM, Zhang S, Carden DL, Conrad SA. *Department of Emergency Medicine, Louisiana State University Health Sciences Center, Shreveport, LA; Louisiana Poison Control Center, Monroe, LA*

**Background:** Previous animal data suggests that aspiration of activated charcoal (AC) is associated with pulmonary microvascular injury and has suggested a role for excessive airway pressures in the production of this injury. The purpose of this study was to test the hypothesis that the lung injury from AC aspiration is independent of injury caused by this volutrauma. **Methods:** Capillary filtration coefficient (Kf,c), a sensitive measure of lung microvascular permeability, was determined isogravimetrically prior to and after intratracheal instillation of 0.4 mL/kg (12% weight/vol. solution, pH 7.4) AC or an equal volume of sterile water in isolated, perfused rat lungs in which ventilation was either pressure-controlled at 10 cm H<sub>2</sub>O or volume-controlled at 5 mL/kg. In addition, arterial blood gas analysis was determined prior to and after tracheal instillation of AC or sterile water in a separate group of animals. Each group also had time-matched controls that did not receive interventions. **Results:** Lung injury was significantly increased in both AC groups regardless of ventilatory method compared to control lungs or lungs administered with sterile water (p < 0.05 ANOVA). Both groups receiving AC also had significantly greater hypoxemia than the other groups. **Conclusion:** The results of this investigation demonstrate that intratracheal instillation of AC is associated with a significant increase in lung microvascular permeability and arterial hypoxemia independent of injury related to excessive airway pressures.

## 11 PEDIATRIC LAMP OIL INGESTIONS FROM LAMPS.

Aitken SC, Barone S, Boja J, Morris CC. *US Consumer Product Safety Commission, Bethesda, MD*

**Background:** Between 1988 and 1998, an estimated 8,500 ± 2,100 incidents of lamp oil ingestion and/or aspiration by children less than 5 years old were reported in the CPSC's National Electronic Injury Surveillance System (NEISS). An estimated 31% of these poisoning victims were hospitalized, considerably higher than the annual hospitalization rate usually associated with pediatric poisonings reported in NEISS. CPSC in-depth investigations suggested that many (43/52) of these potentially serious lamp oil exposures occur directly from lamps rather than child-resistant (CR) bottles of lamp oil. **Methods:** During 1999, the CPSC contracted with the American Association of Poison Control Centers to conduct a prospective study of incident reports in which a child ingested and/or aspirated lamp oil directly from the lamp. Fifty-one of 63 poison control centers participated in administering a CPSC questionnaire investigating lamp access, type, design, attractiveness, and use. **Results:** Centers submitted 144 completed questionnaires. Exposures usually

occurred in a private home (78%); there was often no barrier to access to the lamp (89%); the lamp was usually of contemporary design (81%), made partially or entirely of glass (92%), and used for decorative purposes (90%); lamps were frequently obtained new (84%), not prefilled with oil (66%), and in a kit with a separate container of lamp oil (49%); the lamp oil was sometimes brightly colored (42%) but seldom scented (10%). **Conclusions:** Available injury data suggest that lamp oil aspiration directly from modern decorative lamps is a significant cause of serious injury to young children. **Disclaimer:** The views expressed are those of the authors and do not necessarily represent the views of the US Consumer Product Safety Commission. As this was written in the authors' official capacity, it is in the public domain and may be freely copied and reprinted.

## Poster Session 1

Saturday, September 16  
Abstracts #12–#58

10:00 am–4:00 pm

### 12 RETROSPECTIVE REVIEW OF 5 YEARS EXPERIENCE WITH CROTALIDAE ENVENOMATIONS IN ARIZONA.

Tanen D, Ruha AM, Graeme K. *Good Samaritan Regional Medical Center, Phoenix, AZ*

**Background:** Crotalidae envenomation (CE) in Arizona is most commonly due to *Crotalus atrox*. We present the largest study of CE in Arizona, examining the epidemiology, hematologic abnormalities and complications associated with CE. **Method:** A retrospective chart review of patients admitted to our referral center with CE over the past 5 years was performed. We identified 236 patients for whom 233 charts were obtained. Data were analyzed for epidemiologic parameters and hematologic abnormalities (coagulopathy, defibrination, thrombocytopenia). Time to antivenin, length of hospital stay and complications were recorded. **Results:** Overall, males comprised 81% of patients; 60% involved the upper extremity. Antivenin was used in 77% of patients and was initiated at  $5.3 \pm 0.3$  hour postenvenomation. Overall, 60% of patients developed a coagulopathy (PT > 14.0 sec), 49% defibrination (fibrinogen <170 mg/dL), and 33% thrombocytopenia (platelets <120 K/mm<sup>3</sup>). There were no differences in hematologic abnormalities between upper and lower extremity envenomations. Skin blebs were noted in 21% and dermatomy/fasciotomy was performed in 3.4%. Reactions during antivenin infusion was noted in 36%, mainly urticaria, with 7% exhibiting mild bronchospasm. No cases of severe anaphylaxis or death occurred. Hospital stay averaged  $2.5 \pm 0.1$  days. **Conclusion:** CE in Arizona is characterized by hematologic abnormalities, with coagulopathy being most common, followed by defibrination and thrombocytopenia. Dermatomy/fasciotomy was performed infrequently. Reaction to antivenin is common but tends to be mild and, in our practice, has not prevented further infusion of antivenom.

### 13 COMPARISON OF HEMATOLOGIC ABNORMALITIES IN CROTALIDAE ENVENOMATIONS OF THE UPPER AND LOWER EXTREMITIES IN PATIENTS NOT RECEIVING ANTIVENIN.

Tanen D, Ruha AM, Graeme K. *Good Samaritan Regional Medical Center, Phoenix, AZ*

**Background:** We noted upper extremity envenomations (UEE) tend to exhibit more rapid swelling compared to lower extremity envenomations (LEE). We sought to compare the temporal development of thrombocytopenia (TCP = platelets <120 K/mm<sup>3</sup>), coagulopathy (COAG = prothrombin time >14 sec), and defibrination (DEFIB = fibrinogen <170 mg/dL) between UEE and LEE. **Methods:** A retrospective chart review of patients admitted to our referral center over a 5-year period revealed 54 patients (28 UEE; 26 LEE) with suspected Crotalidae envenomation who did not receive antivenin or blood products. In those whom COAG, DEFIB, or TCP developed, we determined the time to highest documented PT, lowest documented fibrinogen, and lowest documented platelet count. **Results:**

Type of Bite	% with COAG	% with DEFIB	% with TCP	Mean Time to COAG (hour)	Mean Time to DEFIB (hour)	Mean Time to TCP (hour)
28 UEE	43%	39%	11%	$9.6 \pm 2.0$	$11.0 \pm 2.9$	$17.8 \pm 9.2$
26 LEE	35%	15%	12%	$16.77 \pm 4.02$	$15.9 \pm 6.25$	$27.2 \pm 6.2$

Differences between groups by 2-tailed *t*-test were not statistically significant. **Conclusion:** Our study found a trend toward delayed development of COAG, DEFIB, and TCP in LEE as compared to UEE. Although not statistically significant this trend may be explained by the increased distance required for spread of venom via the lymphatics in LEE vs UEE. Further study may be warranted.

#### 14 TOPICAL OCULAR EXPOSURE TO RATTLESNAKE VENOM.

Ness R, Akhtar J, Benitez J. *Toxicology Treatment Program, University of Pittsburgh Medical Center, Pittsburgh, PA*

**Background:** Few cases are reported in the literature about topical ocular exposure snake venom. We report the first such case from a rattlesnake, with a documented eye examination. **Case Report:** A 17-year-old healthy male was spat in the left eye from a distance of about 1 m by a western diamond-back rattlesnake (*Crotalus atrox*). The incident occurred at a herpetological society gathering while the patient was viewing the reptile through a wire cage. The patient's immediate symptom was of eye burning and irritation. The eye was immediately flushed for 30 minutes with tap water. His eye was again irrigated with two liters of normal saline in the emergency room. Examination of the eye showed mild conjunctival injection, with few areas of corneal de-epithelialization. No conjunctival edema was noted. Coagulation parameters were noted to be normal. The patient was discharged home on an antibiotic ointment. A follow up examination at 24 hours revealed complete resolution of symptoms without any eye damage. **Conclusion:** Spitting is a recognized method of defense of certain African snakes, and injuries to the eye are not uncommon. However, topical ophthalmic exposures to North American snake venom usually produce only transient symptoms, and follow a benign course without any systemic or hemotoxic manifestation.

#### 15 EASTERN CORAL SNAKE (*MICRURUS FULVIUS FULVIUS*) BITE OF THE TONGUE WITH FACIAL SWELLING.

Pancorbo D, Leon R, Weisman R, Bernstein J. *Florida Poison Information Center, Miami at University of Miami, Jackson Memorial Medical Center, Miami, FL*

**Background:** Coral Snake (*Micrurus fulvius fulvius*) envenomation occurs in an average of 63 times per year in the US. The systemic effects of elapid envenomation are diplopia, slurred speech, ptosis, dysphagia, stridor, paresthesiae, muscle weakness, fasciculation, and respiratory paralysis. Local effects are usually absent. We report a case of coral snake envenomation with both neurologic and local effects. **Case Report:** A 15-year-old male was playing with what he believed to be a "king snake", when he was bitten on the left side of his tongue. He arrived at the emergency department within 30 minutes with complaint of left-sided facial pain. Physical examination was remarkable for mild swelling of the left lateral aspect of the tongue, diaphoresis and tachycardia. The local poison information center was contacted. An infusion of 5 vials of *micrurus* antivenin was initiated 40 minutes after the bite. Gag reflex was absent; the patient was intubated and placed on a ventilator. Two hours later the tongue was grossly swollen and swelling progressed to the left side of the face and neck down to the upper thorax. Another infusion with 5 vials of antivenin was given. On day 7, the patient was discharged with persistent slurred speech and mild swelling of the tongue and face. At 2 weeks follow up, swelling of the tongue had almost resolved; however, mild slurred speech was still present. **Conclusion:** Signs of coral snake envenomation are primarily neurologic, but may present with soft tissue edema. Aspiration pneumonia is a common complication secondary to bulbar paralysis. The major cause of death is respiratory arrest; the paralysis is completely reversible with good airway protection, ventilatory support, and administration of antivenin.

#### 16 SEVERE PUFF ADDER (*BITIS ARIETANS*) ENVENOMATION.

Lavonas EJ, Tomaszewski CA, Ford MD, Rouse AM, Kerns WP. *Carolinas Medical Center, Charlotte, NC*

**Background:** Puff adder (*Bitis arietans*) envenomation, though common in Africa, is rare in North America. We report a severe puff adder envenomation with swelling, necrosis, thrombocytopenia, and the rare finding of coagulopathy. We detail the response to antivenom. **Case Report:** A puff adder bit a 37-year-old zookeeper on the finger. Within 30 minutes she developed severe pain, hypotension (88/57 mmHg), thrombocytopenia (28,000/mm<sup>3</sup>), and swelling to the wrist. Coagulation studies were initially normal. Four hours later, she was obtunded, more hypotensive (74/31), and

developed coagulopathy (PT 18 sec, INR 2.2, PTT 53 sec, fibrinogen 144 mg/dL, plts 25,000/mm<sup>3</sup>). Ecchymosis and swelling extended to the neck. Care included intubation and IV fluids. Due to known allergy to crotalid antivenom, she was pretreated with IV steroids, antihistamines, and epinephrine. Eight vials of antivenom (SAVP, Gauteng, SA) were infused beginning 4.5 hours after the bite. 30 minutes later, swelling and thrombocytopenia improved (plts 110,000), but coagulation worsened (PT 18.7, INR 2.4, PTT > 120, fibr 73). Clotting parameters stabilized after 15 total vials of antivenom (PT 14.1, INR 1.4, PTT 22, fibr 251, plts 255,000). FFP and platelet transfusions were not required. The patient bled into soft tissues, requiring 4 units of RBC transfusions. Vasopressor support was required for 6 days. The finger became necrotic and was amputated on day 10. Otherwise, the patient recovered completely. There was no adverse reaction to antivenom. Conclusion: Puff adder envenomation causes tissue necrosis, hypotension, coagulopathy, and thrombocytopenia. 15 vials of antivenom reversed the thrombocytopenia and coagulation defects, but hypotension persisted and local tissue necrosis still occurred.

### 17 SPIDER ON THE HEADBOARD, CHILD IN THE UNIT: SEVERE *LOXOSCELES ARIZONICA* ENVENOMATION CONFIRMED BY DELAYED SPIDER IDENTIFICATION AND TISSUE ANTIGEN DETECTION.

Boyer LV, Theodorou AA, Binford GJ, Gomez HF. *University of Arizona, Tucson, AZ; University of Minnesota, Ann Arbor, MI*

Background: Diagnosis of systemic arachnidism is challenging, especially in the absence of a spider. Envenomation by *Loxosceles arizonica* has rarely been described. An antigen-detection assay has been developed against venom of *L. deserta*, but has not previously been tested against other species. Case Report: A 10-year-old boy awoke with a sore spot on his arm. Within 12 hours he had fever, chills, and worsening forearm pain with local bruising. On presentation he had fever, tachycardia and generalized, blanching erythroderma. He developed Systemic Inflammatory Response Syndrome (SIRS) with hypotension, capillary leak syndrome (hypoalbuminemia, generalized edema), disseminated intravascular coagulation, elevated liver enzymes, and fever. This was followed by hemolytic anemia with a drop in hematocrit from 38 to 10. A mature male *L. arizonica* (endemic to the neighborhood) was discovered, after the fact, in the child's bed. Four days after presentation, routine dermatopathology showed diffuse necrosis. Immunoassay of the biopsy was strongly positive for *Loxosceles* venom antigen. Conclusions: Envenomation by *L. arizonica* may be clinically severe. The diagnosis of loxoscelism can be confirmed by detection of venom antigen in tissue. Antibodies raised against venom of *L. deserta* cross-react strongly against venom of *L. arizonica*.

### 18 *LOXOSCELES ARIZONICA* ENVENOMATION WITH RHABDOMYOLYSIS.

Seifert SA, Boyer LV, Murphy M, Madsen RJ, Martinez P. *Arizona Poison and Drug Information Center, Tucson, AZ; Carondelet Medical Group, Tucson, AZ*

Background: *Loxosceles* envenomations are reported to produce systemic reactions that include fever, chills, myalgias, hemolysis, renal insufficiency, thrombocytopenia, DIC, and/or shock. Rhabdomyolysis has not been previously reported. Case Report: A previously healthy 20-year-old male, bitten on the right side of the neck by an entomologist-identified *Loxosceles arizonica* spider developed rhabdomyolysis, thrombocytopenia, mild renal impairment, abnormal liver tests and possible hemolysis, as well as the typical dermal lesion. He initially developed pain and a small bleb at the site, tremulousness, muscle cramping, and fever to 103.4°F. He was treated with intravenous Valium®, Benadryl® and Toradol®, and oral Tylenol® with improvement and was discharged on oral Benadryl and Percocet®. Two days later he had a macular rash of the face, chest and arms, a hemorrhagic bleb, subcutaneous bruising of the neck and upper arm, and CPK 17,000 U/L; T.Bili 3.5 mg/dL; Platelets 120,000 /mm<sup>3</sup>; Creatinine 1.4 mg/dL; SGOT 175 U/L; and elevated urine myoglobin. He was admitted and treated with IV hydration, urinary alkalization and Kefzol®. Hemoglobin declined from 15.6 g/dL on day 2 to 12.8 g/dL on day 4. CPK peaked at 24,503 U/L, SGOT peaked at 182 U/L, and platelets declined to 101,000/mm<sup>3</sup>, but all labs were improving by 5 days postbite. At 1 week the patient only complained of minimal discomfort at the site, had generally resolving labs, and was given prednisone 60 mg/d × 3 days. At 2 weeks, the patient was systemically asymptomatic and the dermatonecrosis was resolving. Conclusion: Rhabdomyolysis may be seen following *Loxosceles arizonica* envenomation.



## 19 EVALUATION OF SCORPION STINGS IN ISRAEL.

Aloufy A, Taitelman U, Tamir A, Bentur Y. *Israel Poison Information Center, Rambam Medical Center and Department of Clinical Epidemiology, Carmel Hospital, Faculty of Medicine, Technion, Haifa, Israel*

**Background:** The most poisonous scorpions in Israel include *Leiurus quinquestriatus* ("yellow"), *Androctonus bicolor* and *Androctonus crassicauda* (both "black"). The venom is a neurotoxin that causes excessive adrenergic discharge resulting in cardiotoxicity. Publications on scorpion stings in Israel include mainly admission data from ICU. This has led clinicians to believe that most scorpion stings are severe and mandate an observation period of at least 12 hours regardless of symptomatology. **Objective:** To assess the characteristics of scorpion sting victims reported to the National Poison Information Center with emphasis on severity and time to presentation. **Methods:** Retrospective poison center chart review of 1996. **Results:** 252 calls were included. 84.5% of callers were physicians, 64% from emergency departments. 13%, 72% and 15% of patients were asymptomatic, mild and moderately to severely ill, respectively. The most frequent sign was pain (83%), followed by cardiovascular manifestations (23%) and ECG changes (4%). 94% of the patients presented within 6 hours, 86% within 3 hours. All moderate to severe patients presented within 6 hours, 92% within 3 hours. Among the mild patients, 93% and 85% presented within 6 and 3 hours, respectively. No correlation was found between degree of severity and time to presentation. Among the moderate to severe patients, there were more females than males. Antivenin was recommended in only 2% of patients. **Conclusions:** Most scorpion stings in Israel are mild. Most envenomated patients present for medical assistance within 3 hours, especially the moderate to severe cases. Because of potential complications, it is recommended that scorpion sting victims be observed for a period of 6 hours and be admitted if symptoms other than local pain develop. Reduction of unnecessary in-hospital observation time is expected to save public health money.

## 20 PROFILE OF MULTIPLE BEE ENVENOMATIONS.

Orletsky P, Thomas R. *Samaritan Regional Poison Center, Phoenix, AZ*

**Introduction:** The Africanized honey bee, *Apis mellifera scutellata*, through hybridization with the European and Italian races *Apis mellifera ligusta*, *Apis mellifera caucasia* and *Apis mellifera carnica*, is now colonized in Texas, Arizona and parts of California. Honeybees in these areas now contain varying degrees of the Africanized DNA. Given the Africanized honey bee's tendency to more aggressively defend their hives and to sting in larger numbers, increases in severe envenomations due to multiple stings is inevitable. **Methods:** The Southwest Multiple Bee Envenomation Registry was established to prospectively and retrospectively review reports of multiple bee envenomations from 1995 through 2000. Data analysis included patient demographics, how the stinging incident occurred, if and how sting numbers were counted, origin of the bees, clinical effects and patient outcome. **Results:** Twenty multiple bee exposure cases were reviewed. Two of these cases, or 10%, resulted in death. Six patients, or 30% had major outcomes to include rhabdomyolysis, thrombocytopenia, and renal failure. Eight patients, or 40% developed moderate effects, most commonly CPK elevations. Twenty percent developed only minor effects of local pain and edema. Most patients with adverse outcomes demonstrated delayed effects. Age, health and sting incident details proved predictive of potential for delayed effects. **Conclusions:** The majority of multiple bee envenomations, 80%, went on to develop at least a moderate effect from bee venom toxicity. Due to the potential for severe and delayed effects, treatment and admission guidelines for multiple bee envenomation patients should include a detailed history of patient demographics and details of the stinging incident rather than on presenting signs and symptoms alone.

## 21 RISK FACTORS FOR ADVERSE EFFECTS IN MULTIPLE BEE ENVENOMATIONS.

Orletsky P, Thomas R. *Samaritan Regional Poison Center, Phoenix, AZ*

**Introduction:** The Africanized honey bee is now considered well colonized in Texas, Arizona and parts of California. This study sought to identify risk factors for developing adverse effects from multiple bee stings. **Case Series:** The Southwest Multiple Bee Envenomation Registry was established in 1998 to prospectively and retrospectively review multiple bee envenomations. Data collection included patient demographics, details of the stinging incident, clinical effects and patient outcomes as defined by TESS criteria. Twenty cases were reviewed covering the years 1995-2000. Risk factors identified with the greatest potential for adverse effects were age 60 years or greater, prolonged attack time, high estimated number of stings (>100 stings or patient reportedly covered by bees), medical history including

cardiovascular problems, and early effects other than pain such as vomiting, diarrhea and hypo or hypertension. Of the two reported deaths, one patient had four and one had all five risk factors. Of those with major outcomes, including rhabdomyolysis, thrombocytopenia, and renal failure, 100% had three or more risk factors.

**Risk Factor Frequency by TESS Defined Outcomes**

Outcomes	Age >60 years	Prolonged Attack	High Sting Estimate	Medical History	Early Effects
Death-2 cases	2/2	2/2	2/2	2/2	1/2
Major-6 cases	2/6	6/6	3/6	3/6	5/6
Moderate-8 cases	3/8	1/8	4/8	1/8	7/8
Minor-4 cases	4/4	2/4	2/4	1/4	0/4

**Conclusion:** Victims of multiple bee envenomations who present with three or more risk factors are more likely to have a major outcome or death and should merit serious consideration for admission and observation.

## 22 THE EPIDEMIOLOGY OF AQUATIC ENVENOMATIONS IN THE US: MOST COMMON SYMPTOMS AND ANIMALS.

Hanley M, Tomaszewski C, Kerns W. *Carolinas Medical Center, Charlotte, NC*

**Background:** To date no comprehensive review exists of aquatic envenomations in the US. Our objective was to characterize reported exposures including most prevalent symptoms. **Methods:** AAPCC TESS data (1993–1998) was examined to identify all exposures relating to aquatic animals. A subsequent search focused on symptoms associated with the most common and most serious offenders. **Results:** There were 15,595 aquatic animal exposures reported during this 6-year period, with 7,208 (46%) occurring during the 3 summer months. Ages ranged from 30 days to 90 years, with men accounting for 61% of cases. The ten most common exposures accounted for 13,694 (88%) of all cases: jellyfish (28%), stingray (16%), lionfish (13%), catfish (11%), gastropods (6%), other fish stings (4%), *Physalia* (Portuguese man-o'-war) (3%), sea urchin (2%), coral (2%), and *Brachirus* (sole) (2%). Over 80% of each group had dermatological manifestations of toxicity, with less than 5% manifesting cardiovascular, neurological or gastrointestinal symptoms. Further analysis demonstrated 24 (0.2%) cases with major outcomes, which predominantly involved stingrays (5/24). In addition to dermatological symptoms (16/24), systemic symptoms in this group included: bradycardia (stingray and *Physalia*), conduction block (stingray), hypotension (stingray), peripheral neuropathy (*Physalia*) and ataxia (sea urchin, jellyfish, and fireworm). **Conclusions:** This is the first attempt to profile aquatic envenomations in the US. Although dermatological complaints are not unexpected, there was a disproportionate amount of systemic symptoms in the most severely exposed individuals. Knowledge of the most common species and symptoms can help target poison center education efforts.

## 23 ENVENOMATION FROM A CORAL CATFISH (*PLOTOSUS LINEATUS*).

Quail MT, Paul I, Stanger C, Woolf AD. *MA/RI Regional Poison Control Center, Children's Hospital, Boston Medical Center, Boston, MA; South Shore Hospital, South Weymouth, MA*

**Background:** Exotic fish stings or envenomation, exposure reports to poison control centers (PCC) rarely occur. 1998 TESS data report 1427 stings from the 2.24 million exposures. Unintentional injuries make up 99% of all exposures. Outcomes are: None (4%); Minor (30%); Moderate (9%); Major (0.1%). We report a case of a moderate outcome from envenomation of a Coral Catfish. **Case Report:** A 45-year-old male patient contacted the regional PCC ten minutes after being envenomated on his right index finger, from his pet fish, called a Coral Catfish. He immediately placed the finger in hot water, then in vinegar, and contacted the center after the pain became excruciating now radiating toward his elbow. He repeatedly "stated how poisonous it was." Poisindex and the American Zoo and Aquarium Association, Antivenom Index had no information about this species. He was told to continue with the hot water soak and seek medical attention immediately. He did not visualize any residual spines, but did have a puncture wound that was becoming reddened. The PCC made contact with the local Aquarium, who immediately disseminated information. The protein released from this species creates a local vasoconstrictor effect and inflammation at the site of the puncture

## 19 EVALUATION OF SCORPION STINGS IN ISRAEL.

Aloufy A, Taitelman U, Tamir A, Bentur Y. *Israel Poison Information Center, Rambam Medical Center and Department of Clinical Epidemiology, Carmel Hospital, Faculty of Medicine, Technion, Haifa, Israel*

**Background:** The most poisonous scorpions in Israel include *Leiurus quinquestriatus* (“yellow”), *Androctonus bicolor* and *Androctonus crassicauda* (both “black”). The venom is a neurotoxin that causes excessive adrenergic discharge resulting in cardiotoxicity. Publications on scorpion stings in Israel include mainly admission data from ICU. This has led clinicians to believe that most scorpion stings are severe and mandate an observation period of at least 12 hours regardless of symptomatology. **Objective:** To assess the characteristics of scorpion sting victims reported to the National Poison Information Center with emphasis on severity and time to presentation. **Methods:** Retrospective poison center chart review of 1996. **Results:** 252 calls were included. 84.5% of callers were physicians, 64% from emergency departments. 13%, 72% and 15% of patients were asymptomatic, mild and moderately to severely ill, respectively. The most frequent sign was pain (83%), followed by cardiovascular manifestations (23%) and ECG changes (4%). 94% of the patients presented within 6 hours, 86% within 3 hours. All moderate to severe patients presented within 6 hours, 92% within 3 hours. Among the mild patients, 93% and 85% presented within 6 and 3 hours, respectively. No correlation was found between degree of severity and time to presentation. Among the moderate to severe patients, there were more females than males. Antivenin was recommended in only 2% of patients. **Conclusions:** Most scorpion stings in Israel are mild. Most envenomated patients present for medical assistance within 3 hours, especially the moderate to severe cases. Because of potential complications, it is recommended that scorpion sting victims be observed for a period of 6 hours and be admitted if symptoms other than local pain develop. Reduction of unnecessary in-hospital observation time is expected to save public health money.

## 20 PROFILE OF MULTIPLE BEE ENVENOMATIONS.

Orlitsky P, Thomas R. *Samaritan Regional Poison Center, Phoenix, AZ*

**Introduction:** The Africanized honey bee, *Apis mellifera scutellata*, through hybridization with the European and Italian races *Apis mellifera ligustica*, *Apis mellifera caucasia* and *Apis mellifera carnica*, is now colonized in Texas, Arizona and parts of California. Honeybees in these areas now contain varying degrees of the Africanized DNA. Given the Africanized honey bee’s tendency to more aggressively defend their hives and to sting in larger numbers, increases in severe envenomations due to multiple stings is inevitable. **Methods:** The Southwest Multiple Bee Envenomation Registry was established to prospectively and retrospectively review reports of multiple bee envenomations from 1995 through 2000. Data analysis included patient demographics, how the stinging incident occurred, if and how sting numbers were counted, origin of the bees, clinical effects and patient outcome. **Results:** Twenty multiple bee exposure cases were reviewed. Two of these cases, or 10%, resulted in death. Six patients, or 30% had major outcomes to include rhabdomyolysis, thrombocytopenia, and renal failure. Eight patients, or 40% developed moderate effects, most commonly CPK elevations. Twenty percent developed only minor effects of local pain and edema. Most patients with adverse outcomes demonstrated delayed effects. Age, health and sting incident details proved predictive of potential for delayed effects. **Conclusions:** The majority of multiple bee envenomations, 80%, went on to develop at least a moderate effect from bee venom toxicity. Due to the potential for severe and delayed effects, treatment and admission guidelines for multiple bee envenomation patients should include a detailed history of patient demographics and details of the stinging incident rather than on presenting signs and symptoms alone.

## 21 RISK FACTORS FOR ADVERSE EFFECTS IN MULTIPLE BEE ENVENOMATIONS.

Orlitsky P, Thomas R. *Samaritan Regional Poison Center, Phoenix, AZ*

**Introduction:** The Africanized honey bee is now considered well colonized in Texas, Arizona and parts of California. This study sought to identify risk factors for developing adverse effects from multiple bee stings. **Case Series:** The Southwest Multiple Bee Envenomation Registry was established in 1998 to prospectively and retrospectively review multiple bee envenomations. Data collection included patient demographics, details of the stinging incident, clinical effects and patient outcomes as defined by TESS criteria. Twenty cases were reviewed covering the years 1995–2000. Risk factors identified with the greatest potential for adverse effects were age 60 years or greater, prolonged attack time, high estimated number of stings (>100 stings or patient reportedly covered by bees), medical history including

cardiovascular problems, and early effects other than pain such as vomiting, diarrhea and hypo or hypertension. Of the two reported deaths, one patient had four and one had all five risk factors. Of those with major outcomes, including rhabdomyolysis, thrombocytopenia, and renal failure, 100% had three or more risk factors.

**Risk Factor Frequency by TESS Defined Outcomes**

Outcomes	Age >60 years	Prolonged Attack	High Sting Estimate	Medical History	Early Effects
Death-2 cases	2/2	2/2	2/2	2/2	1/2
Major-6 cases	2/6	6/6	3/6	3/6	5/6
Moderate-8 cases	3/8	1/8	4/8	1/8	7/8
Minor-4 cases	4/4	2/4	2/4	1/4	0/4

**Conclusion:** Victims of multiple bee envenomations who present with three or more risk factors are more likely to have a major outcome or death and should merit serious consideration for admission and observation.

## 22 THE EPIDEMIOLOGY OF AQUATIC ENVENOMATIONS IN THE US: MOST COMMON SYMPTOMS AND ANIMALS.

Hanley M, Tomaszewski C, Kerns W. *Carolinas Medical Center, Charlotte, NC*

**Background:** To date no comprehensive review exists of aquatic envenomations in the US. Our objective was to characterize reported exposures including most prevalent symptoms. **Methods:** AAPCC TESS data (1993–1998) was examined to identify all exposures relating to aquatic animals. A subsequent search focused on symptoms associated with the most common and most serious offenders. **Results:** There were 15,595 aquatic animal exposures reported during this 6-year period, with 7,208 (46%) occurring during the 3 summer months. Ages ranged from 30 days to 90 years, with men accounting for 61% of cases. The ten most common exposures accounted for 13,694 (88%) of all cases: jellyfish (28%), stingray (16%), lionfish (13%), catfish (11%), gastropods (6%), other fish stings (4%), *Physalia* (Portuguese man-o'-war) (3%), sea urchin (2%), coral (2%), and *Brachirus* (sole) (2%). Over 80% of each group had dermatological manifestations of toxicity, with less than 5% manifesting cardiovascular, neurological or gastrointestinal symptoms. Further analysis demonstrated 24 (0.2%) cases with major outcomes, which predominantly involved stingrays (5/24). In addition to dermatological symptoms (16/24), systemic symptoms in this group included: bradycardia (stingray and *Physalia*), conduction block (stingray), hypotension (stingray), peripheral neuropathy (*Physalia*) and ataxia (sea urchin, jellyfish, and fireworm). **Conclusions:** This is the first attempt to profile aquatic envenomations in the US. Although dermatological complaints are not unexpected, there was a disproportionate amount of systemic symptoms in the most severely exposed individuals. Knowledge of the most common species and symptoms can help target poison center education efforts.

## 23 ENVENOMATION FROM A CORAL CATFISH (*PLOTOSUS LINEATUS*).

Quail MT, Paul I, Stanger C, Woolf AD. *MA/RI Regional Poison Control Center, Children's Hospital, Boston Medical Center, Boston, MA; South Shore Hospital, South Weymouth, MA*

**Background:** Exotic fish stings or envenomation, exposure reports to poison control centers (PCC) rarely occur. 1998 TESS data report 1427 stings from the 2.24 million exposures. Unintentional injuries make up 99% of all exposures. Outcomes are: None (4%); Minor (30%); Moderate (9%); Major (0.1%). We report a case of a moderate outcome from envenomation of a Coral Catfish. **Case Report:** A 45-year-old male patient contacted the regional PCC ten minutes after being envenomated on his right index finger, from his pet fish, called a Coral Catfish. He immediately placed the finger in hot water, then in vinegar, and contacted the center after the pain became excruciating now radiating toward his elbow. He repeatedly "stated how poisonous it was." Poisindex and the American Zoo and Aquarium Association, Antivenom Index had no information about this species. He was told to continue with the hot water soak and seek medical attention immediately. He did not visualize any residual spines, but did have a puncture wound that was becoming reddened. The PCC made contact with the local Aquarium, who immediately disseminated information. The protein released from this species creates a local vasoconstrictor effect and inflammation at the site of the puncture

wound. Excruciating pain radiating up the arm and lasting up to 48 hours occurs. Systemic effects could include: muscle fasciculations, syncope, hypotension, and death. In the emergency department the patient received meperidine 50 mg and hydroxyzine 25 mg for the pain radiating up his arm despite continuation of hot water soak. His ECG was normal, vital signs were stable. He was admitted to a telemetry bed and remained stable throughout his hospital stay. Conclusion: We report a rare envenomation of a Coral Catfish having moderate effects, with a good outcome.

#### **24 A PROSPECTIVE STUDY OF THE ACUTE THERAPY OF JELLYFISH ENVENOMATIONS.**

Lopez EA, Weisman RS, Bernstein J. *Florida Poison Information Center–Miami, and the University of Miami, Jackson Memorial Hospital, Miami, FL*

Objectives: Jellyfish envenomations are a frequent occurrence in the warm waters of South Florida. Traditional therapy involves the use of 5% acetic acid (vinegar), applying shaving cream and then scraping the area with the dull-side of a knife to remove adherent tentacles. The envenomated area is then immersed in hot water (110°F), or ice packs are applied for pain relief. There are no studies to determine whether the application of heat or cold is efficacious in relieving pain. We conducted a randomized study to evaluate hot versus cold therapy for the treatment of pain from jellyfish stings. Methods: After obtaining verbal informed consent, the patients or health care professionals were instructed to apply 5% acetic acid and to remove adherent tentacles. On odd days, instructions were provided for immersion of the envenomated area in hot water. On even days, instructions were provided for the application of cold packs. Follow-up calls at 1, 4, and 24 hours were made to collect data about the duration and intensity of pain, and the need for rescue analgesics or antihistamines. Results: There were a total of 27 patients with jellyfish stings enrolled in the study. Eighteen patients were treated with hot water. Sixteen of these patients reported pain relief within 60 minutes of the initiation of therapy, 2 patients were excluded because of protocol violations. Nine patients were treated with application of ice packs. Five reported pain relief within 60 minutes of the initiation of therapy, 3 patients failed to obtain pain relief with ice packs and experienced pain relief with immersion in hot water. One patient was excluded because of a protocol violation. Conclusion: Immersion of the jellyfish envenomated region in hot water (110°F) was statistically more effective ( $p < 0.05$ ) than the application of ice packs for analgesia.

#### **25 RHABDOMYOLYSIS FROM BUFFALO FISH CONSUMPTION.**

Burns D, Snyder L, Kirk M, Mowry J. *Indiana Poison Center, Clarian Health Partners, Indianapolis, IN*

Background: Buffalo fish toxicity is an infrequently encountered food borne disease believed to be caused by heat-stable fat-soluble toxins in blue green algae consumed by fish. Few previous cases of toxicity following consumption of Buffalo fish (*Ictiobus cyprinellus*) have been reported. We report two severe cases of poisoning and rhabdomyolysis following consumption of this fish. Case Report: A 50-year-old man with a previous history of coronary artery disease and hypercholesterolemia and his 48-year-old wife presented to a local emergency department with complaints of nausea, vomiting, myalgias of the lower extremities and debilitating neck pain. These symptoms began approximately 6 hours after ingestion of a meal of Buffalo fish. The fish was baked in a high temperature oven and then subsequently cooked on a stovetop. Treatment in the emergency department consisted of IV fluids, analgesics, and antiemetics. The husband's initial creatine phosphate (CK) was 244 U/L and rose to 42,260 U/L over the course of the next 11 hours. His CK peaked at 46,412 IU/L on the first hospital day. The wife's initial CK was 858 U/L and peaked at 10,643 U/L on the second hospital day. Additional symptoms noted in the husband included hypertension, bradycardia, hypothermia, myoglobinuria, and hepatic transaminase elevations with a peak AST of 1,330 U/L and ALT of 329 U/L on the first hospital day. The wife's only additional symptom was left arm pain and her AST peaked at 241 U/L on the second hospital day. Supportive treatment was provided along with urinary alkalinization of both patients. Over the course of 4 days their symptoms resolved with corresponding decreases in CK and hepatic transaminases. Conclusions: Ingestion of Buffalo fish can result in myalgia, severe rhabdomyolysis, and transient hepatic damage. Cooking does not seem to prevent toxicity. Treatment for this rare food borne toxicity is supportive.

#### **26 CAN PESTICIDE CONTAMINATED SOIL CAUSE HEALTH EFFECT?**

Baker BA, Topliff A. *Hennepin Regional Poison Center, Minneapolis, MN*

Objective: Description of health effects and experiences of four family members exposed to soil highly contaminated with multiple pesticides. Case Series: After moving to a farm in ND in 1989, a family noted yellow staining of the buildings when it rained. The father had forearm and ankle rashes when he cut the grass. The daughter had rashes and

frequent colds and children who swam in the pond also noted rashes. The wife developed headaches and eye irritation. In 1995, the family learned their land was a pesticide landfill. The EPA designated this the Manvel superfund site, and estimated 4,000 to 10,000 barrels of pesticides buried on the property. Sample soil pesticide levels were (in ppm): Dinoseb 22,900; Toxaphene 8,700,000; DDT 230; DDE 3.7; DDD 46; Dieldrin 74; Chlordane 52; Aldrin 28; Lead 22.8; and carbaryl .087. In 1996, the father had mildly elevated liver function tests, and the family moved. In 1998, the entire family had normal physical exams. Labs were within normal limits including: electrolytes, BUN, Cr, LFTs, TSH, estradiol, testosterone, rbc cholinesterase, plasma cholinesterase, serum DDT, serum DDE, serum DDT, and dieldrin level. Chromosomal analysis was normal in the wife and daughter but the father had inversion of part of chromosome 14 in one cell and additional material on chromosome 14 in another. The son had elevated levels of chromatid breaks (10% versus 4.6% in controls). The father developed thyroid cancer within 2 years. **Conclusion:** This family had mild irritant symptoms and rashes while exposed and the father developed thyroid cancer. Toxaphene exposure has been associated with thyroid cancer in animal studies. The family continues to be plagued with concerns about long-term health effects and financial concerns about the land cleanup.

### 27 VALONE RODENTICIDE INGESTION: SHORT ANTICOAGULATION PERIOD.

Burkhart KK, Treon B, Donovan JW. *Central Pennsylvania Poison Center, The Pennsylvania State University, Hershey, PA*

**Background:** Indandiones are considered long-acting anticoagulants often used as rodenticides. The 4-hydroxycoumarin products such as brodifacoum when ingested have produced anticoagulation that has lasted months. Few reports exist that describe the time course following indandione ingestions. The following case is the first reported overdose of valone with documented levels. **Case Report:** A 30-year-old male ingested an unknown amount of PMP Tracking Powder and Drano®. He also injected Drano into both antecubital fossa. He was found unresponsive and was intubated. The alkaline corrosive injuries included moderate pharyngeal edema, second degree gastric burns, and minimal skin injury in the antecubital fossae. Surgical procedures were not required. His initial PT was 18.1 sec. That same day the peak PT was 21.6 prior to any treatment. The valone level determined by high performance thin-layer chromatography on admission was 0.96 mmol/L. Aggressive treatment was given to prevent hemorrhage from any of his alkaline corrosive injuries. Treatment included four units of fresh frozen plasma and 10 mg vitamin K intravenously and ordered every 12 hours, three additional doses would be given. A few hours after the initial treatment his PT was 13.3 sec, valone level 0.70 mmol/L. Twenty-four hours after admission the PT was 13.5 sec, while the valone level was 0.40 mmol/L. Approximately 12 hours later his PT was 11.9 sec and remained normal thereafter. On the third day of admission his valone level remained at 0.40 mmol/L. Activated charcoal was not given because of the alkaline corrosive ingestion. **Conclusions:** Valone-induced anticoagulation is short-lived, when compared to 4-hydroxycoumarin ingestions.

### 28 ASSESSMENT: GLYPHOSATE DATA FROM THE CAL-EPA PESTICIDE DATABASE.

Goldstein DA. *The Monsanto Company, St. Louis, MO*

**Background:** Activist groups allege that glyphosate (GLY) is a leading cause of pesticide "poisoning" in California based upon the volume of calls to the California-EPA Pesticide Poisoning Information System (PPIS). The PPIS logs calls which include general inquiries and asymptomatic exposures. Thus, call volume may be a poor indicator of clinical poisoning (CP). This study evaluated the incidence of CP due to GLY in the PPIS data to determine whether call volume accurately reflects CP. **Methods:** Data for GLY calls for 1982-1997 were obtained from Cal-EPA. Data included: type of exposure (agricultural/other); system(s) affected (skin/eye/respiratory/systemic); exposure(s), causal relationship (possible, probable, or definite); and limited medical text. Data were tabulated by target organ and degree of relatedness. Cases were reviewed to assess the nature of the reported symptoms. **Results:** Of 776 total calls, most involved only eye (330), skin (232), topical upper airway irritation (6), or combinations of these (31); all without systemic symptoms, serious injury, or persistent effect. The remaining 178 cases were classed as systemic alone (112) or combined with respiratory (31) or topical (35) involvement. Of the 178 systemic cases, 130 were reported as "possible", leaving 47 "probable" or "definite" cases of which 16 had multiple exposures and could not be attributed specifically to GLY; 7 had no reported illness; and 3 had only topical eye/skin complaints. Only 20 of 776 cases (2.6%) had systemic symptoms probably or definitely related to unconfounded GLY exposures. **Conclusion:** For GLY, call

volume appears to grossly over-estimate CP. Allegations regarding the high rate of GLY “poisoning” in California are not supported by the Cal-EPA data. While agents other than GLY have not been evaluated, care is clearly necessary when utilizing Cal-EPA or similar call data to address the epidemiology of pesticide poisoning.

### 29 ZINC PHOSPHIDE POISONING: A RETROSPECTIVE STUDY OF 21 CASES.

Lohani SP, Casavant MJ, Ekins BR, Walson PD, Wolowich WR. *Nepal Poison Center, Katmandu, Nepal; United Hands to Nepal, Central Ohio Poison Center, Columbus, OH*

**Introduction:** Zinc phosphide is a commonly used household rodenticide. It releases phosphine gas on contact with water and acid in the stomach. Due to its low cost and easy availability, it is emerging as a common self-poisoning agent in adults. **Case Series:** A retrospective study of all zinc phosphide exposure calls to a poison center during the period January 1998 to December 1999. A total of 21 exposure cases were reported to the poison information center. The results are summarized in the following table:

Age Group (in years)	<6	6-12	13-19	>19
No. of exposure	3	1	9	8
Unintentional	3	1	0	0
Intentional	0	0	9	8
Asymptomatic	2	1	7	7
Symptomatic	1	0	2	1
Death	0	0	0	1

In the majority of cases, the amount ingested was reported to be  $\leq 1$  gm except in the fatality where  $>20$  gm was ingested. Only 4 (19%) were symptomatic at the time of call. Most common presentation was vomiting and abdominal pain. **Conclusion:** All zinc phosphide exposures in children under 12 years of age involved unintentional ingestion. Intentional self-poisoning was found to be the reason for all patients above 12 years of age. Regardless of the reason for exposure, zinc phosphide ingestion of  $\leq 1$  gm had a favorable outcome.

### 30 VISUAL CHANGES AFTER ACUTE IATROGENIC IVERMECTIN POISONING.

Graeme K, Giancola J, Curry S. *Good Samaritan Regional Medical Center, Phoenix, AZ*

**Background:** Acute ivermectin toxicity is generally characterized by CNS toxicity, but not ophthalmological toxicity. We report a case of acute iatrogenic ivermectin poisoning resulting in both mental status and visual changes in a man without previous ophthalmologic disease. **Case Report:** A 46-year-old man with Demadex folliculitis, a mite infestation, of the scalp was to receive 25 mg oral ivermectin. Pharmaceutical error resulted in a single 200 mg oral dose, which he ingested the evening prior to admission. Within hours of ingestion, he noted marked drowsiness and fell asleep. Upon awakening, he was weak, ataxic, and unable to ambulate. He felt dizzy, nauseated, and “drugged.” He noted blurred vision with black spots and flashing lights. Examination revealed normal fundi, pupil size and reactivity, but mild photophobia. His visual acuity was 20/50 OD, 25/50 OS. With the exception of ataxia, slightly diminished DTRs, and difficulty rising from a lying to sitting position, the neurologic exam was normal. His ataxia and drowsiness resolved by the next morning. Visual blurring had improved, but persisted. Visual acuity was 20/30 OD, 20/25 OS. An ophthalmologist was consulted; he found blurring of the central vision (OD) on Amsler grid testing and felt the presentation was most consistent with a resolving optic neuritis. On follow-up, the patient’s vision had returned to baseline. **Conclusion:** While visual changes, including transient blindness, have been reported previously in other mammals exposed to ivermectin, such toxicity has not been reported in humans without underlying ophthalmologic disease. Visual changes are reported in the treatment of onchocerciasis (river blindness), but have been attributed to inflammation from death of the parasite. Our case, and previous animal reports, suggest that ivermectin may produce transient visual deficits after single large oral doses.

### 31 ACUTE BORIC ACID TOXICITY IN AN ADULT.

Topliff A, Hornfeldt C. *Hennepin Regional Poison Center, Minneapolis, MN*

**Background:** Two large retrospective studies have suggested that most individuals remain totally asymptomatic after the acute ingestion of boric acid. This case illustrates however that significant toxicity from acute ingestions does occur.

**Case Report:** A 64-year-old female ingested approximately three ounces of boric acid (approximately 24 hours prior to arrival), rapidly developing emesis, "dark" diarrhea, and an erythematous boiled lobster-like rash. In the emergency room she appeared moderately lethargic, clinically dehydrated and mildly hypotensive (systolic blood pressure of 80). Her exam revealed a rash over most of her upper body and large but reactive pupils. Initial labs revealed a leukocytosis of  $11.6 (\times 10^3/uL)$ , a glucose of 124 (mg/dL), and a BUN/creatinine ratio of 24/1.6 (baseline creatinine was 0.9 mg/dL). Liver function tests, urinalysis and urine toxicology were nonremarkable. The patient's CK became mildly elevated and serum boron level returned at 1770 ug/dL. Because of the massive overdose and signs of toxicity including possible early acute renal failure the patient received hemodialysis after which the boron level was 1250 ug/dL. During hemodialysis the patient's pruritus decreased and her rash partially resolved although she continued to have dyspepsia. She did well postdialysis and was transferred two days later to psychiatry. **Conclusions:** Although the majority of single acute boric acid ingestions are benign and most individuals remain asymptomatic, large doses can cause typical dermatologic findings, gastrointestinal symptoms with fluid loss and hypotension, renal compromise, mental status changes and rarely death.

### 32 DIGITAL IMAGING: A PROMISING TOOL FOR MUSHROOM IDENTIFICATION.

Fischbein C, Mueller G, Wahl M, Aks S. *Illinois Poison Center, Field Museum of Natural History, and Mercy Medical Center, Chicago, IL*

**Background:** In general, the identification (ID) of mushrooms is a slow and inconvenient process for the clinician. Decisions regarding treatment following mushroom ingestion are usually made without a firm ID of the fungal species and tend to be more aggressive than necessary. The use of digital images (DIs) sent over the internet may provide an important tool for more rapid ID and therefore facilitate optimum patient care. **Case Reports** Cases 1 & 2: Two 40-year-old males developed vomiting, diarrhea and severe abdominal cramping 4 hours after ingesting white "puffballs." On admission the following day both patients were dehydrated and one had mildly elevated liver enzymes. The "puffballs" had opened to reveal mushrooms which, described over the phone, could not be ruled out as an *Amanita* species. A DI E-mailed to the expert mycologist (EM) indicated these were the GI irritant species, *Chlorophyllum molybdites*, and the patients were discharged home after 2 days of observation. A positive ID for *C. molybdites* was later obtained after examination of the actual mushroom specimen. **Case #3:** A 17-month-old female was found eating white mushrooms bearing features consistent with the amatoxin-containing species, *Amanita virosa*. While the patient received activated charcoal at a local ED, DIs were E-mailed to the EM who was able to presumptively ID the mushroom as a nonamatoxin containing lepiota or agaricus species. The patient was discharged home and the specimen was positively ID'd as *Lepiota naucina*, an edible species. **Conclusion:** While standard digital images alone will not permit positive ID, they often contain sufficient information to help the EM rule out the possibility of a severely toxic species. Data accumulated to date indicate that digital imaging will be a powerful tool for the identification of mushrooms and other biologicals.

### 33 A RARE CAUSE OF AMATOXIN POISONING RESULTING IN LIVER TRANSPLANTATION AND DEATH.

Donnelly M, Brouxhon S, Schneider S, Wax P. *Department of Emergency Medicine, University of Rochester Medical Center, Rochester, NY*

**Background:** Over 90% of fatal mushroom poisonings in the United States are caused by the ingestion of *Amanita phalloides*. We report the ingestion of a much lesser known amatoxin-containing mushroom for which there is one previous report of ingestion and death in this country. **Case Report:** A 62-year-old previously healthy woman from upstate New York made a meal of mushrooms she picked from her lawn. She developed nausea and diarrhea the next day but waited until 40 hours after the ingestion before seeking medical care. She was found to have elevated liver transaminases and was transferred to a regional poison treatment center where she rapidly developed fulminant hepatic failure and underwent liver transplantation. The postoperative course was complicated by ARDS, sepsis, and renal failure. The patient died 18 days after transplant. Samples collected from where the patient had picked the mushrooms included 3 known edible species and several small *Lepiota* which were later identified as *Lepiota josserandii*. The



patient admitted having no knowledge of what species she had picked. Conclusion: *Amanita phalloides* and other amanitas are well known as causes of amatoxin poisoning. Some species of *Galerina* and *Lepiota* are also known to contain amatoxin but are less frequently encountered as toxic ingestions. This case represents only the second reported ingestion and death caused by *Lepiota josserandii* in this country.

### 34 ACCIDENTAL INGESTION OF DEATH CAMAS (*ZIGADENUS* SP.) IN A 4-YEAR-OLD BOY.

Higgins T, Selden B, Beuhler M, Brooks D. *Department of Medical Toxicology, Good Samaritan Regional Medical Center, Phoenix, AZ*

Background: *Zigadenus* sp. are commonly mistaken for wild onions and contain veratrum alkaloids that open voltage-gated sodium channels to produce bradycardia and hypotension, mimicking toxicity from a variety of medications. Case Report: A previously healthy 4-year-old Native American boy developed nausea and vomiting 30 minutes after visiting his grandmother's dwelling. Upon presentation he was noted to have persistent emesis, depressed mental status and a systolic blood pressure (SBP) of 60 mm Hg with a heart rate (HR) of 58–60 bpm. A fluid bolus did not result in resolution of his bradycardia or hypotension and a dopamine infusion increased his SBP to 100 mm Hg. Upon transfer to our institution a 12-lead ECG was significant for a NSR of 64 bpm with a QT<sub>c</sub> of 455 msec while on dopamine. The QRS duration was 78 msec. Electrolytes were normal. A digoxin level was <0.2 ng/mL. GC-MS screening of urine was positive for metoclopramide (iatrogenically administered) and caffeine. At this time the family verified that the patient had consumed a "wild onion" shortly prior to onset of symptoms and that no vasoactive medications were available at the grandmother's home. Over the course of the next 12 hours the patients symptoms resolved. His SBP was 94–121 mm Hg without pressors. A repeat 12-lead ECG showed SR of 119 BPM, a QRS of 62 msec and a QT<sub>c</sub> of 402 msec. Conclusion: Veratrum alkaloid toxicity should be included in the differential diagnosis of hypotension with bradycardia.

### 35 THE ACUTE TOXICITY OF TOBACCO EXTRACTS IN RATS.

Fukumoto M, Shirai M, Akahori F, Ogamo A. *Division of Toxicology, School of Pharmaceutical Sciences, Kitasato University, Tokyo, Japan; Department of Veterinary Pharmacology, School of Veterinary Medicine, Azabu University, Kanagawa, Japan*

Introductions: Accidental cigarette ingestion by children is a frequent occurrence in Japan. However, experimental information on the mammalian toxicity of tobacco is limited. We therefore investigated its acute effects and lethal concentrations in rats. Methods: Male Sprague-Dawley rats 6 weeks old were randomly divided into 4 groups of 4 animals/group. Cigarette extracts were given orally at 90, 135, and 180 mg/kg for each of the 4 rats. Lethal serum concentrations were determined using HPLC. Results: The following table lists the values obtained:

Dosage <sup>1</sup>	Mortality <sup>2</sup>	Time to Death (min)	Lethal Concentrations (µg/mL)	LD <sub>50</sub> (mg/kg)
Tobacco extracts				
90	2/4	142.0 ± 25.45	—	
135	4/4	88.5 ± 123.74	18.48 ± 13.62	62.1
180	4/4	47.75 ± 36.59*	16.37 ± 7.62	(36.0 ~ 93.6)†
Nicotine				
90	4/4	54.75 ± 61.83	13.15 ± 17.82	
135	4/4	3.5 ± 3.1	18.81 ± 15.13	57.6
180	4/4	3.25 ± 2.06*	26.93 ± 27.57	(34.2 ~ 90.0)†

<sup>1</sup> Calculated as nicotine. <sup>2</sup> Expired treatment rats. † Confidence limits. \* Significantly different (p < 0.05).

**Conclusion:** The LD<sub>50</sub> values were not significantly different between the 2 groups. Time to death values were significantly longer for the tobacco extracts than for nicotine groups, suggesting that onset of toxic effects are slower in cigarette ingestion cases.

### 36 COMPARISON OF *IN VIVO* ACUTE LETHAL POTENCY AND *IN VITRO* CYTOTOXICITY OF 9 CHEMICALS.

Fukumoto M, Mitsuoka E, Sugiyama T, Kondo R, Ogamo A. *School of Pharmaceutical Sciences, Kitasato University, Tokyo, Japan; Kitasato University Medical Center, Kanagawa, Japan*

**Introductions:** To compare the toxicities of tobacco extracts with those of other substances implicated in the acute poisoning exposure, the cytotoxicity of 9 chemicals was evaluated by colony formation and other assays. **Methods:** [Cells] Chinese hamster V79 cells and HL 60 cells. [Chemicals] Tobacco extracts, nicotine, cotinine, phenol, paraquat, thioridazine hydrochloride, amitriptyline hydrochloride, acephate, acetaminophen. [Colony formation assay] IC50 values were calculated from the colony formation frequency (relative percentage of the control). [Other assays] MTT and WST assays. [Rodent toxicity data] The oral rodent LD<sub>50</sub> values were all taken from the NOISH Registry of Toxic Effects. **Results:** The cytotoxicities of tobacco extracts were 10 to 52 times those of nicotine, perhaps because of multiple toxic components contained in tobacco extracts. Significant correlations ( $r = 0.92-0.98$ ) were obtained with IC50 values of 9 chemicals in V79 vs HL 60 cells, and colony formation vs MTT assay, colony formation vs WST assay and MTT vs WST assay, respectively. When IC50 values from colony formation and WST assays of these chemicals were compared with rat oral LD<sub>50</sub> values, close correlations ( $r = 0.75-0.87$ ) were seen between data from *in vitro* and *in vivo* tests. **Conclusions:** The number of substances used in this study is too small for general conclusions concerning the relative predictive values of cytotoxicity tests. However, these results suggest that further validation of more chemicals with the *in vitro* methods is essential and may be worthwhile to evaluate toxicities of substances implicated in acute poisoning exposures.

### 37 SIGNIFICANT TOXICITY AFTER THE INGESTION OF ARNICA.

Topliff A, Grande G. *Hennepin Regional Poison Center, Minneapolis, MN*

**Objective:** Description of the clinical course of the mistaken ingestion of Arnica (*Arnica montana*) which resulted in severe cardiotoxicity, hyperthermia, renal and hepatic toxicity and weight loss. **Case Report:** A 19-year-old male with a history of scoliosis and chronic back pain ingested an unknown amount of tea made from Arnica leaves and flowers in an attempt to control his pain. Apparently the bottle may not have had sufficient precautions concerning internal use and he did not realize that the product was intended to be applied as a poultice. Approximately 2 hours after consumption he began to have myalgias, a headache, and shaking chills. He presented to an ER where he was found to be hyperthermic (eventually developing a temperature of 105.1° F despite cooling attempts), tachycardic (heart rate of 150) and hypotensive (systolic blood pressure of 80). Fluids and dopamine were begun. While the initial laboratory results were within normal limits he eventually developed a creatinine of 1.8 (mg/dL), an AST of 2200 (IU/L) and an ALT of 1000 (IU/L). A urine toxicology screen and initial serum acetaminophen level were negative. Over the next six days the patient's symptoms slowly improved, dopamine was no longer required and the patient was eventually discharged. Since the event the patient has regained the 25-30 pounds that he lost and his liver function tests have normalized. **Conclusion:** Although herbal products are rarely associated with significant toxicity some products may have inadequate directions on the label. Misinformed use may lead to significant morbidity and occasionally mortality.

### 38 PARTIAL PARALYSIS AND ALTERED BEHAVIOR IN DOGS TREATED WITH MELALEUCA OIL.

Kaluziński M. *Georgia Poison Center, Atlanta, GA*

**Background:** Melaleuca Oil is widely marketed as an antiseptic and fungicide for humans and animals. Also known as Tea Tree Oil, it is derived by distillation from the leaves of *Melaleuca alternifolia*, an Australian tree. The active ingredients of commercially available oil are predominantly cyclic terpenes. **Case Report:** Approximately 7-8 drops of 100% Melaleuca Oil were applied topically as a flea repellent along the spinal columns of two healthy dogs. By the following morning, the smaller dog (14 lbs.) had developed partial paralysis of the hind legs, loss of coordination, and depressed behavior. The larger dog (19lbs.) displayed depressed behavior only. Upon recognition of symptoms, the owner washed both dogs with noninsecticidal shampoo, but noted no immediate improvement. The dogs were taken

to a veterinarian where each received a second course of dermal decontamination, IV fluids, and a 2 mg intramuscular injection of butorphanol tartrate. Symptoms resolved quickly and both were admitted for overnight observation. They fully recovered and were discharged the following afternoon. Conclusions: Melaleuca Oil is commonly used on dogs to treat various skin conditions. Typically, it is incorporated in low concentrations in various topical preparations, including shampoos. External application of pure Melaleuca Oil to the spinal area of small dogs can produce transient paralysis, loss of coordination, and depression. Dermal decontamination, treatment of clinical symptoms, and supportive care has been sufficient for an uncomplicated recovery within 2-3 days. Warnings regarding the potential health effect of highly concentrated Melaleuca Oil products, particularly when applied to small animals, should be considered.

### 39 USE OF DIGOXIN IMMUNE FAB IN STROPHANTHUS POISONING.

Ogunlana V, Caraccio T, Mofenson H. *Long Island Regional Poison Center, Winthrop University Hospital, Mineola, NY*

Background: Strophanthus is a cardiac glycoside derived from *strophanthus gratus* seed. It is poorly absorbed orally so it is usually administered IV/IM. It is contained in various homeopathic remedies and plants. Intoxication is infrequent but produces manifestations similar to cardiac glycoside toxicity. This is the first reported case where cardiac manifestations developed in a patient on a herbal product containing Strophanthus who was successfully treated with Digibind®. Case Report: A 76-year-old woman with a PMH of increased BP and CHF presented with vomiting and HR 40/m. She had been on strophanthus tincture (15 drops daily) for many years prescribed in Hungary. It was refilled as a homeopathic preparation by a pharmacy. ECG showed 3° heart block, atrial fibrillation with many PVCs. Digoxin level was 0.4 ng/mL. Labs: ↓ Na<sup>+</sup> 126, CO<sub>2</sub> 19.7, K<sup>+</sup> 4.7, ↑ glucose 210 mg/L. The patient was placed on an external pacemaker. 17 hours postadmission she developed SOB and HR 30/min. She was given 5 vials (200 mg) IV Digibind and within 30 minutes the ECG showed NSR. She developed no further ECG abnormalities. Discussion: A patient on a herbal product containing Strophanthus developed cardiac toxicity with a low digoxin level. Strophanthus levels are not commercially available. Patients with cardiac glycoside plant poisonings may have low digoxin blood levels. Conclusion: Digibind may be useful in the treatment of Strophanthus intoxication despite low digoxin levels.

### 40 A PROSPECTIVE CASE SERIES OF PEDIATRIC SENNA INGESTIONS.

Smyth D, Gallo A, McGuigan M. *Ontario Regional Poison Information Centre, The Hospital For Sick Children, Toronto, Ontario, Canada*

Background: Senna (*cassia acutifolia* leaves) is classified as one of the anthraquinones laxatives. Pediatric ingestions of senna-containing laxatives have become a more common occurrence with the withdrawal of phenolphthalein laxative products from the market. A literature review found a paucity of documentation regarding senna toxicity and more specifically no information on a pediatric range of toxicity. A retrospective review of senna ingestions in children from June 1997 to October 1998 at our Poison Information Centre (PIC) revealed treatment inconsistencies. Method: A prospective case series of all calls related to the accidental ingestion of senna containing laxatives in children ≤12 years, received by the PIC between November 17, 1998 and April 12, 2000. Children received no initial decontamination. CSPIs classified symptoms as either none, minor, moderate or severe according to a modified World Health Organization Outcome Severity Scale. Parents treated children at home with fluids for minor symptoms, or rehydration using an oral electrolyte solution (OES) for moderate symptoms. PIC staff instructed parents to call back if symptoms worsened. A planned PIC follow up was to occur within 24 to 48 hours postingestion. Results: 45 cases were enrolled. Ten cases were lost to follow up, 3 cases lacked data on weights. Reported dose ingested ranged from 0.5 to 15.4 mg/kg (mean = 5.2, median = 4.8), in children ranging from 1.6 to 9 years (mean = 3.8, median = 4.5). Of the 35 cases followed, four (11.4%) remained asymptomatic, fifteen (42.9%) experienced minor, and sixteen (45.7%) developed moderate symptoms. No children developed severe symptoms. Conclusion: Unintentional ingestion of senna containing laxatives by young children are generally benign. Senna laxative ingestions in children may be safely managed at home.

### 41 EVALUATION OF SELECTED MEDICAL HERBAL REFERENCES COMPARED TO PUBLISHED REPORTS OF ADVERSE EVENTS RELATED TO COMMON HERBS.

Haller C, Anderson IB, Kim S, Blanc PD. *University of California at San Francisco/California Poison Control System (CPCS), San Francisco, CA*

Background: There has been a recent proliferation of medical texts to guide practitioners whose patients use herbal medicines. Herbal toxicity information contained in such texts has not been systematically assessed. Methods: We

reviewed herbal-related calls to the CPCS-SF in 1998, and identified the 12 most common herbs (defined as botanical dietary supplements). We searched MEDLINE to identify published reports of adverse effects potentially related to these herbs. We then evaluated five major herbal references for the adequacy of their toxicologic information in light of published adverse events. **Results:** The herbs, listed in order of CPCS frequency (no. of published case reports showing toxicity) are: St. John's Wort (14), Ma Huang (11), Echinacea (0), Guarana (0), Ginkgo (7), Ginseng (12), Valerian (5), Tea Tree Oil (10), Goldenseal (0), Arnica (1), Yohimbine (12), Kava kava (6). The herbal reference comparison is shown below:

	% Listed	Precautions	Overdose Info	Side Effects	Interactions
PDR for Herbal Medicines	100%	++	+	++	+
Comm. E Monographs	75%	+	0	+	+
Review of Natural Products	100%	+ / ++	+	++	+
Botanical Safety Handbook	92%	++	0 / +	++	+
Herbal Medicines-A Guide	58%	++	+	++	++

Rating: (-) wrong info; (0) no info.; (+) insufficient; (++) mostly adequate; (+++) detailed, useful.

**Conclusion:** All of the references lacked sufficient information on overdose management and drug-herb interactions. Practitioners who rely solely on these references may not be able to adequately assess toxic manifestations of herbal therapy.

#### 42 CLINICAL PRESENTATION OF "MUTI" POISONING.

Tagwireyi D, Ball DE. *Drug and Toxicology Information Service (DaTIS), University of Zimbabwe, Medical School, Harare, Zimbabwe*

**Introduction:** Traditional medicines ("muti") account for a significant number of poisoning cases in Africa. A knowledge of common clinical presentations of this poisoning linked with reasons for taking the "muti" may help identify culprit toxins. **Methods:** Available records for all poisoning cases diagnosed as due to "muti" at the largest referral hospital in the country for the period January 1995–December 1999 were collected and assessed. **Results:** There was a total of 33 cases of poisoning due to "muti". The following table compares the clinical presentation of common adverse effects with common reasons for taking the "muti".

Clinical Presentation	Reasons for taking "Muti"			
	Abdominal Pains (n = 8)	Unknown (n = 7)	Musculoskeletal Pain (n = 3)	Aphrodisiac (n = 3)
GIT pain	2	4	1	3
Vomiting	4	1	1	2
Diarrhea	2	1	1	1
Dysuria	5	1	1	2
Hematuria	6	1	2	1
Frequency	1	—	1	1

**Conclusion:** All patients taking "muti" for abdominal pains suffered from either dysuria and/or hematuria. "Muti" taken for this complaint may therefore contain a nephrotoxic agent. Further studies are required to confirm this.

#### 43 SEROTONIN SYNDROME ASSOCIATED WITH THE USE OF ST. JOHN'S WORT (*HYPERICUM PERFORATUM*) AND PAROXETINE.

Waksman JC, Heard K, Jolliff H, Daly FFS, Bogdan GM, Dart RC. *Rocky Mountain Poison and Drug Center-Denver Health; University of Colorado Health Sciences Center, Denver, CO*

**Objective:** Serotonin syndrome has not been reported following the use of St. John's Wort. We report a patient who developed serotonin syndrome after using St. John's Wort and paroxetine, a selective serotonin reuptake inhibitor (SSRI). **Case Report:** A previously healthy 61-year-old female presented to an emergency department complaining of restlessness and uncontrollable movements of all four extremities. She discontinued St. John's Wort (600 mg per day) 3 days prior to presentation and commenced paroxetine on the day of presentation. The patient noted agitation and marked akathisia 8 hours after ingesting a single dose of 20 mg of paroxetine. On admission blood pressure (BP), heart rate (HR) and temperature were normal. The patient was alert and diaphoretic. Neurologic examination revealed involuntary movements of all extremities. Hyperreflexia and rigidity was noted in all extremities (legs greater than arms). Fasciculation was not observed. The initial creatine kinase was 212 U/L and later peaked at 1024 U/L. Thyroid function tests and other laboratory values were normal. Following admission, her BP increased to 200/116 mmHg and her HR increased to 145 BPM. The patient was managed with supportive care and lorazepam. She was discharged 2 days after presentation. **Conclusion:** Concomitant administration of a SSRI and St. John's Wort may cause serotonin syndrome. Given the prolonged elimination half-life of St. John's Wort, physicians may be advised to wait at least one week following discontinuation of St. John's Wort before initiating SSRI therapy.

#### 44 MA HUANG-INDUCED MYOCARDIAL INFARCTION WITH NORMAL CORONARY ARTERIES.

Sharma AN, Cinci PM, Hoffman RS. *New York City Poison Control Center, New York, NY*

**Background:** Ma Huang (*Ephedra sinica*) is a natural form of ephedrine commonly sold as a nutritional supplement. Ephedrine is rarely reported to cause chest pain syndrome. However, we were unable to find any other cases of Ma Huang-induced myocardial infarction with angiographic evaluation. **Case Report:** A 30-year-old male body builder took four pills of a weight loss product containing Ma Huang (334 mg each) as instructed by the product label. The patient also reported taking testosterone, but his last cycle was one month prior. He presented to the emergency department complaining of right-sided chest pain. Initial vital signs were: HR 112 bpm; BP, 132/82; RR 16 rpm; 97.8°F and physical examination only revealed mild right chest wall tenderness. Initial therapy with ketorolac was ineffective. Subsequently, and ECG revealed T wave inversions in leads III and AVF with poor R wave progression. Aspirin, sublingual nitroglycerin and morphine were given to control pain and a repeat ECG now showed normal R wave progression. Urine toxicology screen was negative for cocaine and amphetamines. The patient's first cardiac enzymes were as follows: CPK 447 IU/L; Troponin I 14.4 ng/mL; MB 6.2%. An emergent cardiac catheterization demonstrated normal coronary arteries, mild global LV hypokinesis and mild LVH. He was admitted to the CCU with subsequent normalization of cardiac enzymes and was discharged on day 3 with warnings regarding stimulant use. **Conclusion:** Ma Huang (ephedrine) is a vasoactive stimulant capable of causing cardiac injury in "recommended doses" even in the absence of underlying heart disease. As such, stricter regulation of these agents should be considered.

#### 45 A TOXICITY PROFILE OF SIBUTRAMINE.

Mrvos R, Cubberley V, Krenzelo EP. *Pittsburgh Poison Center, Children's Hospital of Pittsburgh, Schools of Pharmacy and Medicine, University of Pittsburgh, Pittsburgh, PA; Knoll Pharmaceutical Company, Mount Olive, NJ*

**Introduction:** Sibutramine (Meridia®) is a serotonin and norepinephrine reuptake inhibitor marketed for weight control. No case series or case reports have described sibutramine toxicity. The purpose of this toxicosurveillance project was to profile the acute toxicity and adverse reactions associated with sibutramine. **Methods:** Poison centers were recruited voluntarily to participate in a toxicosurveillance program directed at monitoring exposures to sibutramine. Centers submitted all exposures and adverse reactions involving sibutramine on a blinded poison center record and a completed FDA MedWatch form. These data were analyzed using an MS Excel® database and descriptive statistics. **Results:** Thirty-six centers representing 58.2% of the population participated. Over a two-year period, 41 sibutramine reports were submitted. Eight involved children age ≤2 years (mean = 20.4 months ± SD 3.02) and the amount ingested ranged from 5–210 mg (median = 10 mg). Four children received no treatment and 4 had GI decontamination. All 8 remained asymptomatic. Sixteen unintentional or intentional exposures occurred in adolescents and adults (mean = 28 years ±

SD 16.97). Eight ingested sibutramine alone and 8 involved co-ingestants. The median amount ingested was 30 mg. Tachycardia, dizziness and hypertension were reported in the sibutramine alone group while the multi-drug ingestant group experienced symptoms consistent with the co-ingestants. Seventeen adverse drug reactions/interactions were reported. In those where follow-up was available, symptoms subsided with discontinuation of the drug. No sibutramine abuse cases were reported. **Conclusion:** In this toxicosurveillance project, acute sole exposures to sibutramine were not associated with significant morbidity and no mortality occurred. Adverse reactions were not life-threatening.

#### **46 DEMONSTRATION OF CLOBENZOREX EXPOSURE AS AN EXPLANATION FOR GC/MS-CONFIRMED POSITIVE URINE TESTS SPECIFIC FOR AMPHETAMINE.**

Wallace KL, Gerkin R, Gatewood P. *Good Samaritan Regional Medical Center, Phoenix, AZ*

**Background:** Exposure to prescription diet drugs other than amphetamine and methamphetamine may cause GC/MS-confirmed, and therefore chemically specific, positive urine drug tests for amphetamine. This is explained by the metabolism of these drugs, e.g., clobenzorex, to amphetamine, which is then excreted in amounts that exceed federal drug test cut-off levels. Drug reference information and laboratory standards needed to verify exposure to such drugs are largely unavailable. A case is presented that demonstrates the value of analytical laboratory investigation beyond the initial GC/MS confirmation of the urinary presence of amphetamine. **Case Report:** Investigation of a GC/MS-confirmed laboratory positive workplace urine test for amphetamine suggested use of an unidentified Mexican diet drug by the specimen donor. Pharmaceutical reference source consultation failed to reveal the identity of donor-provided drug samples. Discussion with a US representative of the suspected pharmaceutical manufacturer and GC/MS analysis of the drug samples strongly suggested, but did not confirm, that the samples contained clobenzorex. Self-administration of drug sample by one of the authors, followed by chemical derivatization, solid phase extraction, and GC/MS analysis of urine collected at 4 hours postexposure revealed acetylated derivatives of clobenzorex (S-(+)-N-(2-chlorobenzyl)-amphetamine) and associated metabolites, including amphetamine, 4-OH-amphetamine, and norephedrine. **Conclusion:** Demonstration of the urinary presence of clobenzorex metabolites other than amphetamine by GC/MS analysis corroborates exposure to clobenzorex as an explanation for a GC/MS-confirmed positive urine drug test for amphetamine.

#### **47 PEDIATRIC EXPOSURES TO PHENTERMINE.**

Mrvos R, Krenzelok EP. *Pittsburgh Poison Center, Children's Hospital of Pittsburgh, Schools of Pharmacy and Medicine, University of Pittsburgh, Pittsburgh, PA*

**Introduction:** Phentermine is a sympathomimetic amine used as an anorectic drug for the treatment of obesity. Stimulant effects, both CNS and cardiovascular, can be seen in overdose and leads to serious concerns regarding morbidity in pediatric ingestions. A toxicosurveillance project profiled phentermine exposures over a two-year period. **Methods:** Poison centers were recruited voluntarily to participate in a toxicosurveillance program directed at monitoring exposures to phentermine. Centers submitted all phentermine exposures on a blinded poison center record and a completed FDA MedWatch form. These data were compiled in a MS Excel® database and analyzed using descriptive statistics. **Results:** Over a two-year period, 195 phentermine exposure patients were enrolled: 113 were adult exposures and 82 were pediatric. Fifty two of the pediatric exposures were  $\leq 24$  months of age and had known amounts and outcomes. Ages ranged from 10–24 months (mean 20 months). Total amounts ranged from 4–90 mg (mean 31 mg,  $\pm$ SD 19.5). Mg/kg amounts ranged from 0.3 to 9 mg/kg (mean of 2.64 mg/kg,  $\pm$ SD 1.85). Six of the 52 (11.5%) developed mild symptoms and did not require intervention. Seventeen patients received activated charcoal (32.7%), 2 were given ipecac (3.8%), and 33 no therapy (63.5%). Thirty five (67.3%) were treated in an emergency department and 17 (32.7%) remained at home. In this cohort of patients, the amount ingested did not appear to be a determinant in the development of symptomatology with one child becoming hyperactive after 15 mg (1.3 mg/kg) while others remained asymptomatic at 90 mg (9 mg/kg). **Conclusions:** In this case series, children aged 24 months or less ingesting phentermine exhibited minor, if any, symptomatology and required minimal therapy.

#### **48 ACUTE PSYCHOSIS FROM A DIETARY WEIGHT CONTROL SUPPLEMENT.**

Wahl M, Grant C, Jespersion R. *Illinois Poison Center, Illinois Masonic Medical Center, Chicago, IL*

**Background:** Dietary supplements have become increasingly popular with the general public. Some patients believe that dietary supplements are essentially harmless and that higher than listed doses are without toxic effects. We present a case of chronic overdose of an ephedra containing product which led to an acute paranoid psychosis. **Case Report:**

A 54-year-old female was brought to an urban ED for paranoid delusions regarding her husband persecuting and attempting to leave her. She had elements of tactile, olfactory, visual and auditory hallucinations. She also had aggressive paranoid behavior with interpretation of everyday details alluding to a conspiracy of strangers involved with her husband to cause her emotional and financial harm. The patient was afebrile, BP 137/78, HR 80, RR 18. She had a normal physical and neurological exam. She was alert and oriented with a clear sensorium other than her paranoid delusions. Upon questioning she admitted to taking multiple dietary supplements in large amounts, among them Amp II Pro Drops for weight control. She started out at recommended doses years ago, but had gradually increased to a 30 mL bottle a day for the past few weeks. PoisIndex (PI) lists the ephedra component as 241 mg/7 drops with 120 doses in a 30 mL bottle. She was consuming over 28,000 mg per day. The patient agreed to stop taking the supplement and was admitted to a psychiatric floor for observation. She was discharged the next day on zyprexa and at her one and two week follow up visits she had no evidence of paranoid thinking after cessation of Amp II. The zyprexa was discontinued by the second follow up visit. Conclusion: Ephedra containing supplements have been associated with cardiac effects, HTN, intracerebral hemorrhage, nephrolithiasis, mania, and death. We present a case of acute paranoid delusional psychosis from chronic overdose of an ephedra containing product.

#### 49 HYPERKALEMIA FOLLOWING ACCIDENTAL L-ARGININE INGESTION.

Christianson G, Mowry J, Kirk M. *Indiana Poison Center, Clarian Health Partners, Indianapolis, IN*

Introduction: L-arginine is a semi-essential amino acid used as a treatment for a variety of medical conditions. While treatment for L-arginine ingestions is listed in Poisindex as nontoxic or symptomatic drug ingestion, it has been reported to cause hyperkalemia when given intravenously in persons with pre-existing hepatic and/or renal dysfunction. No reports of hyperkalemia have been found in the literature after oral ingestion. We report a case of hyperkalemia following an accidental ingestion of L-arginine in a child with end-stage renal disease. Case Report: A 15-year-old male with chronic renal failure due to dysplastic kidneys, status postbilateral nephrectomy and on hemodialysis, accidentally ingested 1.5 gm of his father's L-arginine (3-500 mg tablets). His initial serum potassium at 1 hour postingestion was 5.4 mmol/L (nl 3.5-5.5 mmol/L) rising to 6.1 mmol/L at two hours postingestion. He received sodium polystyrene sulfonate (SPSS) at approximately 4 hours postingestion. His serum potassium at 5.5 hours postingestion (1.5 hours postSPSS) was 5.4 mmol/L and 5.3 mmol/L at 8.5 hours postingestion. At no time during the observation period did he develop any cardiac abnormalities or GI symptoms. Discussion: L-arginine is well absorbed from the gastrointestinal tract with a peak plasma concentration at approximately 2 hours. L-arginine contains significant amounts of nitrogen and chloride and can induce extracellular shifts of potassium from cells, resulting in hyperkalemia. Life-threatening hyperkalemia has been associated with its intravenous use in patients with renal failure who have an inability to excrete excessive potassium. Conclusion: It is reasonable to argue that a person with no renal function who ingests L-arginine could be at risk for hyperkalemia. This case report supports this conclusion at a dose well below the dose used clinically in children or adults. Care should be used when interpreting information from Poisindex in the context of pre-existing disease states.

#### 50 A CREAM OF TARTAR MISADVENTURE.

Sanftleben J, Smith J, Rusyniak D, Mowry J. *Indiana Poison Center, Clarian Health Partners, Indianapolis, IN*

Background: There are no reports of overdose on Cream of Tartar in the medical literature. We report an intentional ingestion of this product and review its toxic characteristics. Case Report: A 16-year-old body builder ingested an 82-gram bottle of cream of tartar mixed in Mountain Dew to "clean himself out". Four hours later his sister called the poison center stating that he had been vomiting for about an hour and was "feeling bad". The poison center recommended treatment in his local emergency department. In the emergency department, an ECG showed sinus rhythm at a rate of 60 to 80/min with flattened P-waves and markedly peaked T-waves. Initial laboratory values 5 to 6 hours after ingestion showed a potassium of 8.5 mmol/L, chloride 121 mmol/L, CO<sub>2</sub> 20 mmol/L, BUN 12 mg/dL, and creatinine 1.2 mg/dL. One dose of sodium polystyrene sulfonate was administered in the emergency department and the patient was admitted to the ICU. In the ICU, his ECG showed junctional alternating with sinus rhythm. Treatment consisted of calcium chloride, D<sub>50</sub>W, insulin, sodium bicarbonate and aerosolized albuterol. Serum potassium 2 hours after presentation and treatment was 7.2 mmol/L dropping to 5.2 mmol/L at 4 hours with a normal ECG. The patient was released to home the next day with a potassium of 4.2 mmol/L and a normal ECG. Discussion: Cream of Tartar is potassium hydrogen tartrate, KC<sub>4</sub>H<sub>5</sub>O<sub>6</sub>, the acidic potassium salt of tartaric acid. It is used as a leavening agent in

baking powders. Poisindex lists laxatives and then potassium as managements, and notes that saline cathartics are poorly absorbed with systemic toxicity not expected unless massive amounts are ingested. The tartrate salts of potassium or sodium are listed as examples of saline cathartics. This patient ingested an entire 82-gram bottle, equivalent to 420.5 mEq of potassium and developed serious toxicity. Conclusion: This case demonstrates that health care providers must be alert to the fact that everyday food products may pose a toxicologic hazard if taken in excess.

### 51 HYPOKALEMIA RELATED TO STRYCHNINE INGESTION.

Fernandez X, Fernandez MC, Schumaker A, Watson W. *South Texas Poison Center, University of Texas Health Science Center San Antonio, San Antonio, TX*

Background: Strychnine exposures are often associated with a high incidence of mortality. We report a case of strychnine ingestion in which significant hypokalemia developed. The patient survived with prompt medical care. Case Report: A 16-year-old healthy female, on no chronic medications, intentionally ingested an undetermined amount of coyote bait containing strychnine. The patient presented to the ED shortly after exposure with tremors, nystagmus, abdominal pain, nausea, and vomiting. Intubation and paralysis were utilized. The patient's initial potassium reading was 2.1 mEq/L. Supplemental potassium salts were administered. The patient remained paralyzed with Norcuron® for 3 days. Potassium readings varied from 2.8 to 3.7 mEq/L while on potassium salts. Four days postexposure the patient developed elevated potassium readings of 6.0 mEq/L that resolved spontaneously. Conclusion: This case illustrates hypokalemia to be an under reported effect of strychnine exposures. Only one other episode of hypokalemia related to strychnine ingestion has been reported in the literature. Strychnine exposures may produce significant hypokalemia that may be under reported due to the severe symptomatology and high mortality rate of strychnine.

### 52 SEIZURE AND HYPONATREMIA FROM INGESTION OF CREMORA® NONDAIRY CREAMER IN AN INFANT.

Hendrickson RG. *Department of Emergency Medicine, Toxicology Division, Medical College of Pennsylvania/Hahnemann University, Philadelphia, PA*

Objective: This is the report of a case of seizure, severe dehydration and hyponatremia in an infant from 3 week ingestion of nondairy creamer as his sole source of nutrition. Case Report: A 6-month-old boy presented to the emergency department after having a seizure at home. His mother reported that he had had decreased activity and feeding over 3 weeks. He had developed severe diarrhea one month ago after receiving formula. The mother, assuming that the child was allergic to milk, used a "milk-substitute" instead; a nondairy creamer mixed 2 teaspoons per 8 oz bottle. The boy arrived to the emergency department with sunken fontanelle, dry mucosa, lethargy, hypotonia. HR was 180/min, RR was 28/min, rectal temperature 99.9°F and pulse oximeter of 97% RA. Blood glucose was 90 and he was fluid resuscitated with two 20 cc/kg boluses of normal saline and vitals normalized, but he remained lethargic. Laboratory results were: Na 107, K 4.5, Cl 80, HCO<sub>3</sub> 10, glucose 80, BUN/Cr 40/1.0, Ca 7.1, Mg 1.2, PO<sub>4</sub> 3.0. White blood cell count was 8.0, hemoglobin was 10.2 and platelet count was 250. CT scan of his head was normal. His sodium was corrected to 122 with 3% saline, then to normal range over two days with D5½NS. His mental status improved after sodium correction. Nondairy creamer is a powder intended for use in coffee and tea that contains saturated oils with few electrolytes, carbohydrates or minerals. Nondairy creamer has not been previously reported as a cause of severe hyponatremia. Conclusion: We report a unique cause of seizures secondary to severe hyponatremia and dehydration from ingestion of dilute nondairy creamer in an infant as substitute for formula.

### 53 ELECTROMAGNETIC INTERFERENCE FROM A CELLULAR PHONE AS A CAUSE OF ACUTE EPINEPHRINE POISONING.

Hahn I, Schnadower D, Dakin RJ, Hoffman RS, Nelson LS. *New York City Poison Control Center, New York University, New York, NY*

Background: Although electromagnetic interference (EMI) with medical equipment is described in the engineering community, little documentation exists in the medical literature to address this largely theoretical concern. The FDA collects data on EMI, but often faces resistance from health care facilities during their investigations. Case Report: An 18-year-old male with metastatic chondrosarcoma, who was receiving an epinephrine infusion for septic shock, developed acute onset of agitation, hematemesis, and chest pain nine hours after the infusion began. Physicians diagnosed acute epinephrine poisoning and noted that the infusion bag contained less than the expected volume. Calculations



determined that the patient received 10.5 mg of unintended epinephrine. The infusion pump was turned off and, despite myocardial ischemia and pulmonary edema, the patient ultimately recovered with supportive care. Examination of the implicated infusion pump found it was in normal working condition. Further scene investigation noted that a cellular phone in “standby mode” as present in the immediate area during the event. When exposed to a similar “standby” cellular phone in a laboratory setting, the infusion pump reproduced its bolus delivery. During the event the pump displayed “999” mL/h and corrected to the previous setting upon cessation of the signal. Interrogation of the pump’s memory revealed that it did not record this event. The investigators thereby concluded the EMI was responsible for the infusion pump failure. Conclusions: This is the first documented case in which EMI with sensitive medical equipment caused substantial human morbidity. The implications of this interference are potentially grave and the health care industry should be cognizant of such possibilities. Reporting of all potential EMI events to the FDA and assistance in their inquiry is suggested.

#### **54 A COMPARISON OF SELECTION CRITERIA IN MECONIUM DRUG TESTING.**

Kraner J, Brown H, Evans L, Sweeney B, McCoy D, Waller C, Bloom E, Long W, Winkler C, Simmonds S, Evans M. *Laboratories, St Vincent’s Hospital and the Indiana State Board of Health, Indianapolis, IN*

Background: Drug abuse during pregnancy has been associated with a high incidence of premature birth, low birth weight, and physical/developmental deficiencies. While history of drug abuse is often used in identifying at-risk infants, analysis of meconium for drugs of abuse has also been used as criteria for establishing drug use during pregnancy. We have compiled a database of meconium drug testing results from three criteria. These criteria are (1) specimens from infants of low birth weight (<2500 grams) and head circumference (<third percentile for gestational age) (Growth Deficient Infants), (2) specimens per request of the physician (Physician Requested), and (3) specimens submitted from all consecutive births at 6 separate birthing institutions (No Selection Criteria). The results from the meconium analysis were used to evaluate selection criteria for predicting drug use during pregnancy. Methods: The drugs/drug classes included in this study were amphetamines (amphetamine, methamphetamine), cannabinoids, cocaine, and opiates (codeine, morphine and hydrocodone). All meconium specimens were screened by Radioimmunoassay (RIA) and presumptive positives were confirmed by gas chromatography/mass spectrometry. Results: Specimens (total 842) submitted from the No Selection Criteria group demonstrated a 4.2% positive rate. This group was also tested for cotinine and 6.9% of the specimens were found to be positive. A positive rate of 12.6% was found in the specimens from the GDI group (total 222). The Physician Requested group (total 758) demonstrated a 28.1% positive rate. This finding suggests that while growth deficiency may be useful in predicting drug abuse during pregnancy, the physician’s evaluation is more often predictive.

#### **55 PATIENTS “INTOXICATED” BY MULTIELEMENT URINE, HAIR OR STOOL RESULTS: A TOXICOLOGY CLINIC EXPERIENCE.**

Senecal PE, Chalut D. *McGill University Health Centre, Montreal, PQ, Canada*

Background: United States Food and Drug Administration and Federal Trade Commission studies have reported low reproducibility of some consumer-aimed biological analyses. An increasing number of patients are referred to our medical toxicology clinics for evaluation of “toxic” chemical element poisoning, following inductively coupled plasma-mass spectrometry (ICP-MS) assays of urine, hair and even stool. Case Series: The aforementioned cases, referred to a tertiary care university health centre (adult and pediatric), were reviewed. Most pediatric cases referred were autistic. Adult cases resulted mainly from self-testing (with medical referral) and alternative medicine practitioner prescriptions. “Toxic” levels of lead, aluminum and mercury were noted. Rarely encountered analyses (i.e., boron or zirconium) were common, as was information appended for “detoxication” treatment. Reported analytical fees spread from \$50 to \$300, usually covered by insurance. The reasons reported for testing generally did not meet accepted medical toxicology criteria (i.e., compatible symptoms or signs). Taking care of these cases was very time-consuming as patients and their relatives were often confused by the detailed computerized interpretations of the urine, hair or stool results. Standard medical toxicology investigation did not confirm any poisoning in our series. However, despite these findings, research on the marketing of saliva, sweat and cerumen multielement analyses is proceeding. Conclusion: Pending more regulations and research, multielement analysis is still, at best, a screening tool. Abnormal results should be investigated and confirmed in a different setting before concluding to toxicity.

## 56 AN ANALYSIS OF 25,394 COIN EXPOSURES REPORTED TO POISON CENTERS.

White NC. *Holton-Arms School and National Capital Poison Center, Washington, DC*

**Background:** The medical literature is replete with conflicting recommendations regarding the management of ingested coins and the role of x-ray evaluation to identify esophageal coins. **Methods:** Coin exposures reported to the Toxic Exposure Surveillance System (TESS) from 1993 through 1999 were analyzed. **Results:** 25,394 coin exposures were reported. The 22,921 cases with known coin type included pennies (77.3%), dimes (8.9%), quarters (7.6%), and nickels (6.3%). Outcome analysis focused on 10,463 coin exposures with sufficient follow-up to determine a definitive outcome. In this group, the percentage of patients with an outcome of "no effect" or "minor effect" varied inversely with coin diameter: dime (98.5%), penny (97.8%), nickel (94.7%), and quarter (92.6%). Similarly, the frequency of moderate or major effects increased with increasing coin diameter: dime (1.5%), penny (2.2%), nickel (5.3%), and quarter (7.4%). Only 12 patients had a major effect (0.11%), and there were no fatalities. The most frequently observed related clinical effects (n = 25,394) were: cough/choke (6.8%), throat irritation (3.0%), vomiting (2.5%), abdominal pain (1.3%), chest pain (1.0%), dysphagia (0.4%), and nausea (0.4%). Of the 10,463 patients with adequate follow-up, 58.9% were not evaluated in a health care facility, thus no initial x-ray could have been obtained. **Conclusion:** Most coin ingestions were benign. Rare, serious outcomes occurred more frequently with large diameter coins. More than half of coin ingestions were managed without radiographic determination of coin position. The generally benign clinical course and large number of patients successfully managed at home suggest that health-care facility utilization for coin ingestions was excessive.

## 57 ARE ACCIDENTAL PEDIATRIC INGESTIONS OF AMOXICILLIN OR AMPICILLIN TOO AGGRESSIVELY TREATED WITH GI DECONTAMINATION?

Alsop J, Walsh M. *California Poison Control System-Sacramento Division, Sacramento, CA*

**Background:** Pediatric ingestions of amoxicillin or ampicillin (A/A) are common, usually resulting in mild GI symptoms. In 3 case reports of A/A ingestions (amounts of 574 mg/kg, 580 mg/kg, and an estimate of between 333-733 mg/kg), nonallergic nephrotoxicity resulted. **Methods:** Poison Centers (PC) were surveyed to determine current recommendations for A/A ingestions. A hypothetical situation of a healthy 32 pound child ingesting 150 mg/kg, 250 mg/kg, or 450 mg/kg A/A was presented. The child had received A/A before with no allergic reactions and this was a sibling's medication ingested 15 minutes before the call to the PC. Medical Directors (MDs), Managing Directors (Ds), and Poison Specialists (SPIs) were asked to provide treatment recommendations for the 3 scenarios. **Results:** 23 MDs, 29 Ds, and 60 SPIs responded. MDs, Ds and SPIs at the same site often had different recommendations. GI decontamination was recommended for 0% in the 150 mg/kg group, 24.1% in the 250 mg/kg group, and 62.5% in the 450 mg/kg group. SPIs recommended treatment most often (75%) for the 450 mg/kg group compared to 48.2% of the Ds and 47.8% of the MDs. At ingestions of 450 mg/kg, ipecac was recommended by 36.7% SPIs, 31.0% Ds, and 30.4% MDs. At ingestions of 450 mg/kg, activated charcoal was recommended by 35% SPIs, 17.2% Ds, and 17.4% MDs. Lavage and charcoal were recommended by 3.3% of SPIs. **Conclusion:** With only three case reports of nephrotoxicity, it appears that PCs are over-treating these ingestions. Observation without GI decontamination is a reasonable treatment for oral A/A exposures in healthy nonallergic children. Replacing fluids from any severe vomiting and diarrhea with electrolyte solutions is suggested. Instructing the caregiver to observe for changes in urine color or volume is prudent.

## 58 ACUTE CANINE C-4 EXPLOSIVE INGESTION RESULTING IN PROLONGED STATUS EPILEPTICUS.

Stork CM, Kos S, Langden B, Cantor R. *Central New York Regional Poison Control Center, Department of Emergency Medicine, Upstate Medical University, Cornell University, School of Veterinary Medicine, Syracuse, NY*

**Background:** C-4 (cyclonite) explosive ingestions are rarely reported in the literature. We report the case of a canine ingestion of cyclonite that resulted in seizures unresponsive to conventional treatment. **Case Report:** A 3-year-old, other wise healthy, male Rottweiler ingested approximately 1 cubic cm of C-4 explosive during an exhibition exercise. The owner reported vomiting after 30 minutes and generalized seizure-like activity 4 hours after ingestion. The dog was brought to a health care center where 8 further generalized seizures occurred over 10 hours. Treatment consisted of several doses of benzodiazepines and barbiturates in addition to supportive care. After the eighth seizure, the dog was placed on a continuous intravenous infusion of propofol, which was continued until 60 hours after ingestion. A granular odorous material was retrieved rectally after several high warm saline enemas. The dog made a complete recovery and

was released after 72 hours. **Conclusion:** C-4 (cyclonite) ingestion is rarely reported in the literature. Seizures that occur after exposure may be prolonged and recalcitrant to conventional therapy warranting aggressive treatment.

### Platform Session 3

**Saturday, September 16**  
**Abstracts #59–#62, #64–#66, #191**

**3:00 pm–5:00 pm**

#### 59 PRETREATMENT OF CD-1 MICE WITH 4-METHYLPYRAZOLE (4-MP) BLOCKS 1,4-BUTANEDIOL (BD) TOXICITY.\*

Quang L, Maher T, Shannon M, Woolf A. *Children's Hospital, Massachusetts/Rhode Island Poison Control System, Harvard Medical School, Massachusetts College of Pharmacy and Allied Health Sciences, Boston, MA*

**Objective:** BD is a prodrug that undergoes biotransformation by hepatic alcohol dehydrogenase to gamma-hydroxybutyrate (GHB). This study investigated if pretreatment of CD-1 mice with 4-MP blocks BD toxicity. **Methods:** Male CD-1 mice were pretreated with intraperitoneal (ip) injections of 4-MP (25mg/kg) or deionized, distilled water (controls). Thirty minutes later, mice in both groups were given BD 600–6000 mg/kg ip. Toxicity was assessed by the righting reflex and the roto-rod test at 10-minute intervals up to 60 minutes. **Results:**

	1,4-BD Dose (mg/kg)							
	600	700	800	900	1000	3000	5000	6000
<b>Righting Reflex</b>								
4-MP Group	10/10	10/10	10/10	10/10	10/10	9/9	8/10	0/10
Control Group	2/10	4/10	1/10	2/10	3/10	0/9	0/10	0/10
p-value	<.001	.01	<.001	<.001	<.001	<.001	<.001	NS
<b>Roto-rod Test</b>								
4-MP Group	9/10	10/10	10/10	10/10	8/9	8/9	0/10	0/10
Control Group	0/10	1/10	1/10	1/10	0/10	0/9	0/10	0/10
p-value	<.001	<.001	<.001	<.001	<.001	<.001	NS	NS

**Conclusion:** Pretreatment of CD-1 mice with 25 mg/kg 4-MP increases the TD<sub>50</sub> for BD at doses of 600–3000 mg/kg, presumably by inhibiting alcohol dehydrogenase biotransformation of BD to GHB.

\*Winner of the American Academy of Clinical Toxicology 1999 Research Award.

#### 60 IMIPRAMINE CARDIOTOXICITY AND SARCOPLASMIC RETICULUM FUNCTION.

Wang R. *Division of Emergency Medicine, Brown University School of Medicine, Providence, RI*

**Objective:** To evaluate sarcoplasmic reticulum (SR) function during imipramine-induced cardiotoxicity. **Methods:** Post-extrasystolic potentiation (PESP) was used to study SR function in an isolated perfused rat heart model. The hearts were perfused with the physiologic buffer (KHB) at a coronary flow of 10 mL/min for 30 minutes. Experiments included 15 minute intervals of basal, and treatment. Left ventricular (LV) pressures were measured with a balloon-tipped catheter placed in the LV via the mitral valve. LV generated pressure was calculated by subtracting LV end diastolic pressure from LV peak systolic pressure. LV contractility (dP/dt) was used as an index of cardiac function. PESP was obtained by introducing an extrastimulus (50, 100, 150 ms) between two regular stimuli separated by a 200 ms interval, corresponding to PESP intervals of 150, 100, and 50 ms, respectively. This was initiated after 10 minutes of treatment exposure. Treatments included KHB (control) and imipramine (3000 ng/mL). N = 6 in each group. Values represent means. Data were analyzed by ANOVA and p < 0.05 determined significance. **Results:** 1) Imipramine decreased LV dP/dt to 60% of control at the end of the treatment period. 2) LV dP/dt increased from basal at increasing PESP intervals of 50, 100, and 150 ms (113%, 142%, and 178%, respectively) with KHB treatment. 3) Imipramine treatment increased

LV dP/dt from basal at PESP intervals of 100ms (144%) and 150 ms (146%), which was less than control. **Conclusions:** Imipramine decreases LV contractility, which is associated with an abnormal PESP response. This suggests sarcoplasmic reticulum dysfunction. Further studies are necessary to clarify this relationship.

### 61 THE EFFECTS OF SODIUM BICARBONATE IN IMIPRAMINE-INDUCED CARDIOTOXICITY.\*

Wang R, Raymond R, Lawler R, Jackson D. *Departments. Biology, Medicine, and Chemistry, Brown University, Providence, RI*

**Objective:** To evaluate the effects of sodium bicarbonate in imipramine-induced cardiac dysfunction. **Methods:** Isolated rat hearts were perfused on a Langendorff apparatus. Coronary flow was maintained at 10 mL/min. A silastic balloon was placed in the left ventricle to measure left ventricular peak systolic pressure (LVSP) and left ventricular end diastolic pressure (LVEDP). Left ventricular generated pressure (LVGP) = LVSP-LVEDP. All hearts were electrically paced at 300 bpm. Hearts were allowed to equilibrate with KHB and then randomized to individual treatments and perfused for 15 minutes. Control groups included KHB, imipramine (IMIP 1500 ng/mL), NaHCO<sub>3</sub> (20 mM, pH 7.65–7.70), NaGluconate (NaG, 20 mM), and HipH (pCO<sub>2</sub> 16 mmHg, pH 7.65–7.70). Treatment groups included IMIP + NaHCO<sub>3</sub>, IMIP + NaG, IMIP + HipH. N = 4 in each group. Measurements included cardiac function (LVGP, LVSP, LVEDP, HR), myocardial oxygen consumption (MVO<sub>2</sub>), myocardial work (LVSP × HR), and heart afferent and efferent buffer content (pO<sub>2</sub>, lactate, pCO<sub>2</sub>). Means were compared by ANOVA and alpha < 0.05. **Results:** 1. IMIP treatment decreased LVGP, myocardial work, and MVO<sub>2</sub> to 51%, 63%, and 78% of basal, respectively. 2. IMIP + HipH increased LVGP, MVO<sub>2</sub>, and myocardial work to 150%, 121%, and 132% of IMIP control. 3. IMIP + NaHCO<sub>3</sub> increased LVGP, MVO<sub>2</sub> and myocardial work to 123%, 107%, and 130% of IMIP control. Lactate production increased as well. 4. IMIP + NaG decreased LVGP, MVO<sub>2</sub> and myocardial work vs IMIP control. **Conclusion:** 1. IMIP cardiotoxicity decreases myocardial contraction, work, and oxygen consumption. 2. HipH is more effective than NaHCO<sub>3</sub> in reversing these effects. 3) The role of ion exchangers in these effects will require further investigations.

\*Winner of the American College of Medical Toxicology 1999 Research Award.

### 62 INFLUENCE OF ACTIVATED CHARCOAL ON THE ABSORPTION OF FERROUS SULFATE IN RATS.

Gades NM, Chyka PA, Virgous CK, Butler AY, Mandrell TD. *University of Tennessee and Southern Poison Center, Memphis, TN*

**Objective:** To determine whether activated charcoal alters the gastrointestinal absorption of iron as ferrous sulfate. **Methods:** Male Sprague-Dawley rats (236.0 ± 8.6 g; N = 75) were randomly assigned to one of five groups: 1) control given only distilled water, 2) 400 mg/kg elemental iron (approximately one-half of LD<sub>50</sub>) and water, 3) 1:1 ratio of charcoal to iron, 4) 2:1 ratio of charcoal to iron, and 5) 4:1 ratio of charcoal to iron. All treatments were administered consecutively by gavage within 5 minutes. Physiological measurements and blood samples were taken at 0, 1, 4, and 8 hours after treatment. Necropsies were performed at the end of the study period. Data were analyzed with ANOVA with an alpha of 0.05. **Results:** A total of 53 rats completed the study with the others excluded due to technical complications or premature death. There were no significant differences in physiological parameters among the 5 groups. There were no significant differences in mean (±SEM) serum iron concentrations (µg/dL) among groups 2, 3, 4, and 5 except at 1 hour between groups 4 and 5 (\*p < 0.004).

Group	N	Baseline	1 hour	4 hours	8 hours
1	13	243 ± 84	292 ± 84	364 ± 84	169 ± 84
2	14	224 ± 81	1010 ± 91	855 ± 87	536 ± 87
3	11	215 ± 91	908 ± 91	847 ± 107	440 ± 91
4	7	265 ± 114	737 ± 135*	717 ± 135	443 ± 114
5	8	211 ± 107	1251 ± 114*	963 ± 107	450 ± 114

**Conclusion:** Activated charcoal did not alter the extent of iron absorption in the experimental model.

### 63 IATROGENIC ADMINISTRATION OF PROPYLENE GLYCOL WITH ACTIVATED CHARCOAL.

Higgins T, Curry S, Brooks D, Beuhler M. *Department of Medical Toxicology, Good Samaritan Regional Medical Center, Phoenix, AZ*

**Background:** During routine GC-MS toxicology screening of admitted patients, we have occasionally noted the unexplained presence of propylene glycol (1,2-propanediol) in urine. We hypothesized that propylene glycol (PG) may be used as an excipient in activated charcoal preparations. **Methods:** A GC assay to identify and quantify PG was prepared, and 7 commonly available brands of activated charcoal were tested. **Results:** Four of the 7 products tested contained significant concentrations of PG.

Charcoal Brand	[PG] mg/L (grams PG/container)	Container Size
Actidose-Aqua (long tube)	100,480 mg/L (12.05 g/container)	25 gm AC/120 mL
Actidose-Aqua (short tube)	109,930 mg/L (13.2 g/container)	25 gm AC/120 mL
JP-Jones: Sorbitol Base	108,720 mg/L (13 g/container)	25 gm AC/120 mL
JP-Jones Aqueous Base	17,680 mg/L (212 g/container)	25 gm AC/120 mL
Charflo	None-Detected	25 gm AC/120 mL
Kerr Insta Char	None-Detected	25 gm AC/120 mL
Requa: Charcoaid	None-Detected	50 gm AC/240 mL

**Discussion:** Since activated charcoal minimally adsorbs PG, the administration of 100 g of some activated charcoal products to a 70 kg adult would theoretically raise serum osmolality by about 7 mOsm/kg water. Repeat activated charcoal dosing in children might result in even greater elevations in serum osmolality. **Conclusion:** Propylene glycol is administered iatrogenically with some brands of activated charcoal.

### 64 SYSTEMIC PHOSPHOROTHIOATE OLIGODEOXYNUCLEOTIDE AS AN IRON CHELATOR.

Angle CR, Iversen PL, Mata JE. *University of Nebraska Medical Center, Omaha, NE*

**Background:** Phosphorothioate oligodeoxynucleotides (PS-ODNs), synthetic antisense nucleotides for chemotherapy, may have potential as heavy metal chelators. PS-ODNs are modified DNA molecules with sulfur replacing nonbinding oxygen on the phosphate backbone, creating a stable poly-anionic molecule that interacts with drugs as well as with other cellular components. *In vitro*, PS-ODNs bind iron as a function of the number of sulfurs present in the backbone. We estimate that the log of the stability constant of an unsaturated iron-PS-ODN complex is greater than the 14.4 for iron with EDTA. **Methods:** Iron excretion was measured in 16 patients with relapsed or refractory acute myelogenous leukemia or myelodysplastic syndrome (MDS) participating in studies to test the safety of OL(1)p53, a 20-mer PS-ODN complementary to p53 mRNA. Patients were given OL(1)p53, 0.05 to 0.25 mg/kg/h  $\times$  10 days, by continuous intravenous infusion. 24 hour urines before and daily were analyzed for Fe, Cu, Zn, and Cd. **Results:** *In vitro*, PS-ODNs have a high affinity for Fe as well as Hg, Sn, Pb, Cd. Following administration of OL(1)p53 at low doses (0.05 mg/kg/h), urine iron increases 7.5-fold. In a patient with MDS who was regularly transfused with packed red blood cells during the study, urine iron increased 3.7-fold to  $1387 \pm 177$   $\mu$ g/d. **Conclusions:** PS-ODNs, with  $t_{1/2}$  of about 24 hours, may have therapeutic potential as heavy metal chelators. Renal clearance of iron-PS-ODN complexes most likely involves secretion into proximal tubules.

## 65 EFFECTS OF COMBINED IRON DEFICIENCY AND LEAD POISONING ON MEMORY AND DOPAMINE CONCENTRATION.

Wright RO, Bhole G, Pilipovic L, Amarasiriwardena C, Hu H, Maher T. *Department of Pediatrics, Brown Medical School, Providence, RI; Massachusetts College of Pharmacy, Boston, MA; Channing Laboratory, Harvard Medical School, Boston, MA*

**Background:** Among children, iron (Fe)-deficiency anemia and lead (Pb)-poisoning are known to cause cognitive impairment, possibly via effects on dopamine metabolism. The objective of this study was to determine the effects of combined developmental iron deficiency and lead poisoning on spatial learning and to correlate these findings with brain extracellular dopamine (DA) concentrations in rats. **Methods:** Male rats were exposed prenatally and postnatally with: 1) Fe-deficient diet and control water; 2) Fe-deficient diet and 300 ppm Pb in water; 3) Iron replete diet and control water; and 4) Iron replete diet and 300 ppm Pb in water. Morris Water Maze (MWM) testing was performed on the 6th to 7th week of life, followed by DA microdialysis in the nucleus accumbens. Iron deficiency was assessed by hematocrit and blood lead level by Inductively Coupled Plasma-Mass Spectrometry. Multiple linear regression models using MWM probe test results as the dependent variable and hematocrit, blood lead and extracellular DA concentrations as both independent variables and effect modifiers were generated to assess their association with spatial learning. **Results:** With respect to MWM probe tests, Fe-deficient, and iron deficient/lead poisoned groups performed worse than control diet groups ( $p < 0.05$  for both). Extracellular DA levels did not differ among the four treatments groups; however, DA concentrations were directly correlated with probe test performance ( $R^2 = 0.24$ ,  $p < 0.05$ ). In the multiple regression model, hematocrit, blood lead level and dopamine concentration were all significantly associated with probe test results. No significant interaction terms were generated among the 3 independent variables, suggesting a lack of synergism. **Conclusions:** Iron deficiency, lead poisoning and extracellular dopamine are all independent predictors of spatial learning. Our results suggest that extracellular dopamine levels do not change with chronic developmental iron deficiency and lead exposure. Chronic lead poisoning and iron deficiency may still influence dopaminergic systems via receptor function or number.

## 66 USE OF REVERSIBLE CHOLINESTERASE INHIBITOR IN MANAGEMENT OF THE COMPLICATIONS AFTER ALCOHOL INTOXICATION.

Tonkopi DV, Aksenov IV. *Department of Military Field Therapy, Military Medical Academy, St. Petersburg, Russia*

**Introduction:** Presently due to the fast growth of alcoholism there is an increase in the cases of alcohol delirium (AD). Generally, we consider AD complication of alcohol withdrawal syndrome (AWS), which is a severe, life-threatening situation, requiring intensive treatment. The aim of this work was to investigate the possibility of using aminostigmine, which is central reversible cholinesterase inhibitor, in complex therapy of AWS. **Methods:** 18 men between ages of 31–53 years old, who have developed AD on day 3–5 of the AWS, were studied. The patients were divided into 3 groups: 2 experimental groups and 1 control group. Patients from the first experimental group were given intravenous injections of aminostigmine in the researched dosage in the beginning of the AD development (first hours). Patients from the second experimental group were given aminostigmine late (day 1–2) in AD development. All the patients were given standard infusion and medication therapy. The dynamic of psychic and somatic status was evaluated. **Results:** It was found that in the first experimental group intravenous injection of aminostigmine together with standard medication and infusion therapy at early hours of AD development interrupted AD completely and immediately. At the same time, in the second experimental group, in cases of developed AD, aminostigmine administration had no influence on the clinical course of delirium in comparison with control group. **Conclusion:** On the basis of the observed data it is possible to assume that a cholinergic mechanism plays an important role in the onset of AD. Later, in the case of developed delirium, the course of AD is defined probably by other mechanisms.

**Poster Session 2****Sunday, September 17  
Abstracts #63, #67-#124****10:00 am-4:00 pm****67 JUST A CLICK AWAY: STUDENT INTERNET SURFING FOR RECREATIONAL DRUG INFORMATION.**

Wax P, Reynolds N. *Department of Emergency Medicine & University Health Service, University of Rochester, Rochester, NY*

**Objective:** An increasing number of recreational drug related ED visits appear to be precipitated by drug use suggestions garnered off the Internet. The study objective is to determine the frequency of Internet drug site visitation by students and its association with drug use. **Methods:** A convenience sample of undergraduate college students (school size = 3800), and a 1st year medical school class (class size = 100) were asked to fill out an anonymous written questionnaire about their own recreational drug use. Specific questions were also asked about whether they had surfed the Internet for drug information and its impact on subsequent drug use. Chi-squared statistics were used as appropriate. **Results:** 168 students (20% of whom had been to a rave) were surveyed (94 college and 74 1st year medical students). 19% reported that they surfed the Internet for information on recreational drug use (24% college and 14% medical students). Of those who surfed the Internet, 9% said this increased their likelihood of using recreational drug (27% college and 0% medical students), 19% said it decreased likelihood of using recreational drug (27% college and 10% medical students) and 72% stated this had no effect (45% college and 90% medical student). Those who surfed for recreation drug information were much more likely to have used MDMA 53%, "magic mushrooms" 50%, and LSD 22% than those who had not surfed the internet 7%, 11%, and 6% respectively ( $p < 0.05$ ). There was no statistical difference between web surfers and nonsurfers regarding their likelihood of having attended a rave. **Conclusions:** About 1 in 5 students surveyed have visited recreational drug web sites. Comparable numbers of college students reported that web site visitation increased or decreased their drug taking likelihood. The impact on medical students was considerably less. There appears to be an association between hallucinogenic drug use and drug site visitation but causation can not be ascertained.

**68 EVALUATION OF AGE OF INHALANT ABUSE PATIENTS REPORTED TO POISON CENTERS.**

Spiller HA. *Kentucky Regional Poison Center, Louisville, KY*

**Background:** The TESS database provides a broad picture of exposures in the US involving data from all age groups from 45 of the 50 states and including cases reported by the lay public and health professionals. **Method:** All patients reported to the TESS database where the reason for exposure was abuse and the route of exposure was inhalation were evaluated for the years 1995 through 1998. **Results:** 8.6 million patients were reported in TESS, of which 118,377 (1.4%) were abuse patients. 22,015 of these patients (19%) were inhalation abuse. Inhalant abuse patients overall were young: children (<18 years) represented 47% of inhalant abuse patients and 78% were <30 years. The peak years of inhalant abuse were in adolescence: 14 to 15 years of age (17% of total) and 16 to 17 years of age (16% of total). 73 patients were less than 6 years and 489 were less than 10 years. Children were more likely to abuse volatiles (72% of nonpharmaceutical/volatile abuse population), while adults were more likely to abuse pharmaceuticals (64% of pharmaceutical inhalant abuse population). HCF usage increased progressively with age, from 45% in <6 year age group to 82% in the adult group. When comparing the distribution of fatalities by age, adolescents (13 to 19 years) represented 6% of all TESS fatalities but were 31% of the inhalant-abuse group fatalities. Inhalant-abuse patients represented 1% of the total TESS adolescent patient group but represented 19% of all adolescent TESS fatalities. **Conclusion:** Incidence of inhalant abuse begins very early in childhood and peaks in adolescence. Inhalant abuse represents a major cause of death in the adolescent age group of the TESS database.

## 69 EVALUATION OF FATAL INHALANT ABUSE CASES REPORTED TO POISON CENTERS.

Spiller HA. *Kentucky Regional Poison Center, Louisville, KY*

**Background:** The TESS database provides a broad picture of exposures in the US involving data from all age groups from 45 of the 50 states and including cases reported by the lay public and health professionals. **Method:** All patients reported to the TESS database where the reason for exposure was abuse and the route of exposure was inhalation were evaluated for the years 1995 through 1998. **Results:** 8.6 million patients were reported in TESS, of which 118,377 (1.4%) were abuse patients. 22,015 of these patients (19%) were inhalation abuse. There were 178 fatalities of which 109 (61%) and 69 (39%) were related to pharmaceuticals and nonpharmaceuticals (volatiles), respectively. Air fresheners and hydrocarbons represented a disproportionate share of the fatalities in the volatile group (24% and 58%, respectively). When comparing inhalant-abuse fatalities vs all poison center case fatalities, children (<19 years) represented 33% vs 9% of fatalities, respectively. The fatality rate (deaths/100,000 patients) for all patients reported to poison centers was 34, while all abuse patients and inhalant-abuse patients were 393 and 809, respectively. When comparing inhalant abuse patients and all poison center patients there was a relative risk of 24.5 for a fatal outcome (95% confidence interval,  $p < 0.01$ ). In comparing all abuse patients with inhalant-abuse patients there was a relative risk of 2.7 for a fatal outcome (95% confidence interval,  $p < 0.01$ ). **Conclusion:** Inhalant abuse patients represent a dangerous sub-group of poison center patients, with a significantly increased risk for a fatal outcome.

## 70 OTC COUGH AND COLD MEDICATION ABUSE IS INFREQUENT AND OCCURS IN CLUSTERS: POISON CENTER DATA AS A SURVEILLANCE METHOD.

Watson WA, Hellsten JJ, Fant PM, Shepherd JG, George DJ. *South Texas Poison Center, San Antonio, TX; Texas Department of Health, University of Texas-Austin School of Pharmacy, Austin, TX; North Texas Poison Center, Dallas, TX; National Toxicology Network, Bedminster, NJ*

**Background and Objective:** Surveillance of infrequently occurring events such as OTC cough and cold medication (OTCCC) abuse (dextromethorphan, anticholinergics, and sympathomimetics) is difficult and is best determined with multiple passive and active surveillance methods. The objective of this retrospective study is to determine whether TESS data can identify OTCCC abuse clusters. **Methods:** Human exposure cases (without patient identifiers) of OTCCC from a state TESS database serving a population of 19,840,595 in 1998 and 1999 were identified using the 6 groupings of generic codes in the TESS format. Intentional exposures cases were reviewed using predefined criteria for abuse. Duplicate cases were deleted and miscoded records were corrected or deleted. The data is presented using descriptive statistics. **Results:** During the study period, 16,534 OTCCC cases were identified from 314,372 total cases; 331 cases were defined as OTCCC abuse. Cases appear to be clustered, with 38% of cases reported within a 5-day period from the same county. Cases were reported from 68 of the 254 state counties. Median patient age was 16 years (IQR 15 to 20 years); 37% of calls were from health care facilities and 41% from residences. Multiple cases (2–4) were reported during 20 calls and 23 calls came from schools. The most common brands involved were Coricidin ( $n = 180$ ), Robitussin ( $n = 21$ ), and Nyquil ( $n = 12$ ); 28 of the 38 different products were national brand names. **Conclusion:** These findings demonstrate that OTCCC abuse clusters can be identified using TESS data. Active monitoring of TESS cases would be a useful addition to other surveillance methods of abuse for these drugs.

## 71 CORICIDIN® ABUSE IN OHIO TEENS AND YOUNG ADULTS.

Simone KE, Bottei EM, Siegel ES, Tsipis GB. *Cincinnati Drug & Poison Information Center (DPIC), Cincinnati Children's Hospital Medical Center, University of Cincinnati, Cincinnati, OH*

**Background:** Teens and young adults are taking overdoses of Coricidin HBP™ Cough & Cold tablets, instead of drinking ounces of DM-containing syrup, for a phencyclidine-type "high" that may be due to the dextropropranolol metabolite of DM. The chlorpheniramine present in the Coricidin product increases the toxicity of overdose by adding an anticholinergic component. **Methods:** Calls about DM abuse made to the Cincinnati DPIC between 1/1/00–4/28/00 are examined. Exposures are described. **Results:** The DPIC received 75 calls about DM abuse from 16 different counties. 44 calls were exposures; these exposures involved Coricidin HBP Cough/Cold (67%), a different Coricidin product (21%), and either Robitussin DM or Maximum Strength Cough (12%). Those taking a Coricidin product as one-time, acute ingestions ( $n = 36$ ) took an average of 13 tablets (range 6–23). Those abusing Coricidin on a daily basis ( $n = 3$ ) averaged 51 tablets per day (range 24–56). The patient ages ranged from 13–21 years. 40 callers reported symptoms: CNS (46%), CV (22%), other (14%), ocular (13%), and GI (5%). The most common symptoms, in order of frequency, were drowsiness, tachycardia, other (e.g. numbness, tremor, pallor, etc.), mydriasis, hypertension, and ataxia. At least 72% were



seen at a health care facility. Outcomes of the 44 exposures were: no effect (5%), minor effect (50%), no more than minor (14%), moderate effect (20%), potentially toxic (11%). The duration of effects (n = 30) were: <8 hours (60%), <24 hours (33%), and <72 hours (7%). Conclusion: We have noted a disturbing increase of Coricidin abuse among teens and young adults. It is possible that the added anticholinergic effects are bringing more DM abusers to medical attention.

## 72 CORICIDIN® USE AND ABUSE IN TEXAS DURING 1998 AND 1999.

Baker SD, Borys DJ. *Central Texas Poison Center, Texas Poison Center Network, Scott & White Hospital, Temple, TX*

Objectives: In recent years Coricidin products seemed to be one of the most common over-the-counter (OTC) medications being abused by adolescents. The objective of this retrospective chart review is to investigate the occurrence of abuse, develop a patient profile, and define the clinical effects resulting from the abuse of Coricidin products in Texas during 1998 & 1999. Methods: Data was collected from the Texas Poison Center Network TESS® database. Inclusion criteria included human exposures between 1998–1999, patients >10 years old, reason coded as intentional use or abuse, single substance ingestion of one of the tablet formulations of Coricidin products. Results: All Coricidin cases for 1998 & 1999 were extracted from the Texas Poison Center Network TESS database. Thirty-three cases from 1998 (36%) and 59 cases from 1999 (64%) for a total of 92 cases were reviewed. Of these cases, 78 (85%) met the inclusion criteria. Of the seven medications searched by AAPCC code, only four substances were coded for, Coricidin D, Coricidin D (Long Acting), Coricidin D (Cold, Flu & Sinus) and Coricidin HBP (Cold & Flu). Out of the 78 cases, 49 (63%) were male and 29 (38%) were female. The top five most common ages were 14 (23.1%), 16 (21.8%), 15 (15.4%), 13 (10.3%), and 17 (6.4%). Eighteen different symptoms were reported, including tachycardia (50%), somnolence (24.4%), mydriasis and hypertension (16.7%), agitation (12.8%), disorientation (10.3%), slurred speech (9%), ataxia (6.4%), vomiting (5.1%), dry mouth and hallucinations (3.9%), tremor (2.6%), headache, dizziness, syncope, seizure, chest pain, and nystagmus (1.3%). Another observation of significance is that 12.8% of the calls originated from a school nurse. Conclusions: The incidence of abuse of Coricidin products increased 60% from 1998 to 1999 in Texas. Two thirds of the patients were male. Of the cases reported 77% were between the ages of 13 and 17 and a significant amount of calls originated from a school nurse.

## 73 THE EFFECT OF CODEINE IN ANTI-COUGH SYRUP ON MORPHINE SCREEN.

Hsu C, Hung D, Yang D. *Division of Toxicology, Emergency Department, Taichung, Taichung Veterans General Hospital, Taiwan*

Background: Codeine is a lawfully permitted ingredient of anti-mid and anti-cough medicine that is readily available to the public at drug stores in Taiwan. Drug abusers often use it as a substitute for morphine while out of heroin and try to make an excuse to evade the law and say that they have only taken anti-cough syrup when morphine is detected in their urine. The purpose of this study is to try to propose criteria for codeine intake using a multiple lower dose model in normal therapeutic situations. Methods: Ten healthy volunteers each received, in two days, a total of 8 doses of cough syrup containing 4.5 mg of codeine per dose. Urine specimens were collected regularly during and in 3 days after syrup use. The urine specimens were analyzed by EIA screening test and GC-MS confirmation test. Results: Positive screen was noted during syrup use and 18–30 hour after the last dose of medication. The ratios of morphine to codeine were all less than one during syrup use and 12 hour after the last dose of syrup. Only 3.1% of the urine specimens, the ratio of the concentrations of morphine to codeine were between 1 and 3 when the concentrations of morphine and codeine were both very low i.e. below the cut-off values. Conclusion: The results may be used as a reference for the interpretation of urine analysis after the intake of multiple doses of anti-cough syrup with codeine. The criteria for codeine intake may be proposed as codeine concentration larger than 300 ng/mL and the ratio of morphine to codeine to be less than 3.

## 74 DETERMINATION OF 1,4-BUTANEDIOL TOXIC DOSE<sub>50</sub> IN CD-1 MICE.\*

Quang L, Maher T, Shannon M, Woolf A. *Children's Hospital, Massachusetts/Rhode Island Poison Control System, Harvard Medical School, Massachusetts College of Pharmacy and Allied Health Sciences, Boston, MA*

Objective: To determine the Toxic Dose 50 (TD<sub>50</sub>) of 1,4-butanediol (BD) in CD-1 mice. Methods: Male CD-1 mice received intraperitoneal (ip) injections of BD or deionized, distilled water (BD excipient; controls). Toxicity was observed at 10-minute intervals up to 60 minutes by assessing righting reflex (ability to regain an upright posture within

10 seconds after being placed on its back) and the roto-rod test (ability of the mouse to log roll on a 1-inch diameter rod revolving at 6 RPM). Results:

	BD Dose (mg/kg)								
	100	200	300	400	500	600	700	800	900
<b>Righting Reflex</b>									
BD Group	8/8	10/10	10/10	10/10	5/10	4/10	4/10	2/10	0/8
Control Group	8/8	10/10	10/10	10/10	10/10	10/10	10/10	10/10	8/8
<i>p</i> -value	NS	NS	NS	NS	.01	<.001	<.001	<.001	<.001
<b>Roto-rod Test</b>									
BD Group	8/8	0/10	0/10	0/10	0/10	0/10	0/10	0/10	0/8
Control Group	8/8	10/10	10/10	10/10	10/10	10/10	10/10	10/10	8/8
<i>p</i> -value	NS	<.001	<.001	<.001	<.001	<.001	<.001	<.001	<.001

The TD<sub>50</sub> of BD for loss of righting reflex is 554 mg/kg (95% CI, 442–693 mg/kg). The TD<sub>50</sub> of BD for failing the roto-rod test ranges from 100–200mg/kg. The TD<sub>50</sub> of BD for both loss of the righting reflex and failure of the roto-rod test appears to be 600 mg/kg. The roto-rod test is more sensitive than the righting reflex for detecting BD toxicity. Conclusions: These results are important for animal studies investigating BD toxicity.

**\*Winner of the American Academy of Clinical Toxicology 1999 Research Award.**

#### 75 FATAL OVERDOSE FROM INGESTION OF 1,4-BUTANEDIOL, A GHB PRECURSOR.

Kraner J, Plassard J, McCoy D, Rorabeck J, Witeck M, Evans M. *AIT Laboratories, Indianapolis, IN; The Lake County Coroner's Office, Waukegan, IL*

Background: Gamma hydroxybutyrate (GHB) is a central nervous system depressant that has become popular as a drug of abuse. Its effects include sedation, dizziness, nausea, seizures, and respiratory depression and coma. While sale of GHB has been banned, several products are available that contain 1,4-butanediol (BD) which is converted *in vivo* to GHB. Case Report: A healthy 40-year-old female was reported to have purchased several bottles of a commercially available product containing BD and began consuming the product on a regular basis. She was found unresponsive on the couch with foam present around the nose and mouth and was declared dead at the scene by medical personnel. At autopsy, the pulmonary parenchyma was soft, spongy, dark purple to black, severely congested and edematous. Routine toxicology screening revealed only the presence of caffeine and nicotine metabolite. No alcohol was found in either the blood, urine, or vitreous fluid. GHB and BD were assayed by GC/MS:

	Blood	Vitreous	Urine	Bile
1,4-Butanediol	7.6 µg/mL	12.3 µg/mL	146 µg/mL	ND
GHB	280 µg/mL	324 µg/mL	6171 µg/mL	218 µg/mL

An opened container of the commercial product at the scene was analyzed by GC/MS and found to contain BD with no GHB detected. Conclusion: The blood GHB level is consistent with other GHB-related fatalities. Here we report for the first time a GHB fatality occurring from the apparent ingestion of the GHB precursor, 1,4-butanediol.

#### 76 CHILD NEGLECT LEADING TO GAMMA-HYDROXYBUTYRATE INGESTION.

Boyer EW, Fearon D, Anderson AC, Woolf A, Shannon M. *The Children's Hospital, Boston, MA; Hasbro Children's Hospital, Providence RI*

Background: Gamma-hydroxybutyrate (GHB) is a drug of abuse that is increasing in popularity. We report the respiratory arrest of a child who ingested GHB. Case Report: A 4-year-old boy ingested an unknown amount of GHB stored

in a beverage container that he found while riding with his parents in the family car. Gamma-butyrolactone products were later found in the passenger compartment of the same vehicle. After ingestion he vomited and rapidly became unresponsive. He was intubated for respiratory arrest; upon arrival in the ED he received flumazenil and naloxone without effect. Urine organic acid screens identified GHB. Analysis of the contents of the beverage container also identified GHB. Other toxicologic screens were negative. The patient's mental status normalized approximately 6 hours following ingestion, at which point he was successfully extubated. He had a normal neurologic exam but required treatment for left lower lobe aspiration pneumonia. Conclusion: GHB ingestion is a cause of coma and respiratory arrest in children. Clinicians should recognize that children presenting in toxic coma or respiratory arrest may be victims of chemical child neglect or abuse. In this case flumazenil was an ineffective treatment for GHB toxicity. Clinicians may be able to identify GHB intoxications using urine amino acid screens.

### 77 GAMMA BUTYROLACTONE EXPOSURE FROM NAIL POLISH REMOVER.

Leblanc F, Blais R. *Quebec Poison Control Centre, Quebec City, Quebec, Canada*

Background: In recent years Gamma hydroxybutyrate (GHB) has become an increasingly popular street drug. It is a central nervous system (CNS) depressant wrongly used for bodybuilding and sometimes as a rape drug. Gamma butyrolactone (GBL) is the main ingredient of commercially available products such as nail polish remover; it is rapidly transformed into GHB when ingested. We report two cases illustrating this type of intoxication. Case Report #1: A 12-month-old girl swallowed more than 5 cc of nail polish remover; 65 minutes later, she became apneic and required intubation. Heart rate was 80 to 120 beats/minute; her epiglottis was slightly edematous. Her Glasgow coma score was 6. Blood gas analysis showed a pH of 7.36, PO<sub>2</sub> at 115 mm Hg, PCO<sub>2</sub> at 19.5 mm Hg and HCO<sub>3</sub> at 11.6 mmol/L; salicylate blood level was negative. Six hours after ingestion, the child was extubated; she recovered uneventfully. Case Report #2: 45 minutes after ingestion of the same brand of nail polish remover, a 2-year-old boy vomited several times and became ataxic; upon arrival at the emergency room 30 minutes later, the child was asymptomatic; vital signs and blood gas were normal. GHB was detected in the urine. The product involved in both cases was Finelle® nail polish remover, alcohol and ketone free, containing 75% GBL. Discussion: GBL toxicity is the same as GHBs: rapid CNS depression sometimes followed by sudden arousal. Although Finelle Cosmetics no longer manufactures nail polish remover containing GBL, the product could still be commercially available. Also it must be remembered that it has a long shelf-life and can be a cause of intoxication (accidental or intentional) for many years to come. Conclusion: Nail polish remover without acetone does not mean without toxicity. Many people still unknowingly have in their homes a product that can cause serious poisoning.

### 78 SEVERE GAMMA BUTYROLACTONE WITHDRAWAL.

Sharma AN, Nelson L, Hoffman RS. *New York City Poison Control Center, New York, NY*

Background: Although gamma hydroxybutyrate (GHB) withdrawal has been previously reported, only one prior abstract reports gamma butyrolactone (GBL) withdrawal. We report two nearly identical cases of severe GBL withdrawal. Case: Two 29-year-old males presented separately to the ED agitated. Both patients suffered GBL overdoses complicated by unresponsiveness (2) and intubation (1) at different times during the preceding 48 hours. They were discharged home within 12 hours and had subsequently discontinued their GBL use abruptly. On readmission, both reported being unable to sleep since discontinuation of GBL, were confused, agitated, hallucinating and had autonomic instability. Initial vital signs were as follows: patient #1: HR 130 bpm; BP 140/106 mmHg; patient #2: HR 133 bpm; BP 142/100 mmHg. Both had normal physical examinations except for tremor and slight diaphoresis. They received diazepam (>50 mg) over 4 hours with reduction in HR to <100 bpm and BP to <130/90 mmHg. However, they still alternated between periods of lucidity and confusion. Subsequently large doses of diazepam (>100 mg) were required over the next 12 hours and the patients became less confused, but somnolent. Patient #2 had an elevated CPK level (5770 IU/L) that resolved quickly. Both required high dose benzodiazepine therapy followed by a taper for the remainder of their admissions. Conclusion: GBL withdrawal is clinically similar to GHB withdrawal, but can be cause severe life threatening agitation consistent with alcohol withdrawal induced delirium tremens and may require high dose benzodiazepine treatment.

### 79 APNEA FROM FENTANYL PATCH SMOKING.

Souders C, Branton T, Wax P. *Department of Emergency Medicine, University of Rochester Medical Center, Rochester, NY*

**Background:** The abuse of fentanyl is a well described phenomenon. Fentanyl patch smoking has rarely been reported. A case of apnea immediately following one inhalation of fentanyl in this manner is described. **Case Report:** A 37-year-old female with a history of drug abuse was found unresponsive (GCS = 3), apneic, and with miosis by her friends after smoking a "pipe with fentanyl." Upon arrival of EMS, the patient was intubated and given 2 mg naloxone to which she rapidly responded. In the ED, the patient was very agitated but calmed after extubation. Her urine drug screen was positive for benzoylcegonine and negative for opiates. After one additional dose of 2 mg naloxone, the patient remained alert and required no further treatment. Upon further questioning, the patient admitted to past history of nasal insufflation of heroin and fentanyl smoking. She had refrained from opioid abuse during the previous 6 months. She described her fentanyl smoking technique as follows: "Take a 75 or 100 µg fentanyl patch, cut the edges and peel it apart. Place the gel portion in the oven on 500° and bake for approximately 10 minutes until the gel becomes brown and bubbles. Remove from the oven and let it cool and scrape the residue into a glass pipe (eg. crack pipe). Light it and inhale. The high is similar to heroin and peaks within 20 minutes and lasts 60 minutes." She reports each patch provides enough fentanyl for only one inhalation. **Conclusion:** We report an unusual case of fentanyl abuse by smoking the fentanyl obtained from a patch. The smoking of fentanyl from a patch may produce apnea.

### 80 NERVOUS REGULATION OF BREATHING IN OPIATE DEPENDENT PATIENTS.

Pach J, Kolarzyk E, Targosz D. *Department of Clinical Toxicology, Department of Hygiene and Ecology, Collegium Medicum Jagiellonian University, Kraków, Poland*

**Objective:** Neurotoxic properties of opioids which are central nervous system depressants may have also depressive action on the brain stem complex responsible for the breathing control. Disorders of breathing regulation are reflected in respiratory efficiency. The aim of this study was to evaluate the regulation of breathing by measuring respiratory pattern parameters, occlusion pressure and also by evaluation of respiratory efficiency in opiate abusers. **Material and Methods:** There were 76 persons under examination: group I—36 opiates abusers treated at the Department of Clinical Toxicology; group II (control group)—40 healthy persons not dependent of opiates and never treated at the Department. During hospitalization the functional state of the respiratory system was monitored. Ventilation efficiency was determined on the basis of the results from a "flow-volume" loop, spirometry and the measurements of the respiratory tract resistance in a computerized system (Lungtest-MES company, Poland). Respiratory regulation was evaluated by means of synchronic "on line" measurements of the respiratory pattern and occlusion pressure during unrestrained breathing with atmospheric air. **Results:** Insignificantly higher frequency (from 33.3 to 38.5%) of obturative disorders, reflected by respiratory resistance elevation, was noted in the group of opiate dependent patients after 9 days hospitalisation. Also an increase in occlusion pressure, neuro-muscular respiratory drive, minute ventilation and shorter duration of expiration and of total breathing cycle in opiate dependent patients compared to the control group were noted. **Conclusion:** An individual variability and significant neural effect on ventilation efficiency at the first stage (at the beginning) of abstinence was shown.

### 81 DEATH AS A COMPLICATION OF ULTRARAPID OPIOID DETOXIFICATION (UROD).

Olmedo RE, Hoffman RS, Howland M, Nelson L. *New York City Poison Control Center, St. John's University, New York, NY*

**Background:** Since its inception in 1988, proponents of UROD claim high degrees of safety and efficacy. The procedure reportedly minimizes early intolerable opioid withdrawal symptoms. Treatment consists of administration of opioid antagonists under general anesthesia, followed by maintenance therapy with a long lasting subcutaneous naltrexone implant. This method of naltrexone administration is nonFDA approved. Subsequent withdrawal symptoms are managed as an outpatient. **Case Report:** A 30-year-old opioid-dependant man (300 mg methadone daily for 2 years) underwent treatment with UROD under general anesthesia with propofol, midazolam, and ketamine. Withdrawal symptoms were treated with clonidine and octreotide. The patient was discharged and was advised to administer octreotide, trazodone, baclofen, acetaminophen, and diphenhydramine for withdrawal symptoms. Three days following the procedure, the patient's family brought him to the hospital comatose and with hematemesis. Vital signs were BP 170/100 mmHg, P

140 BPM, and T 104° C. After immediate sedation and intubation, he was cooled, fluid resuscitated, cultured and given antibiotics. Four naltrexone pellets were removed from his abdominal subcutaneous tissue. Laboratories were significant for marked prerenal azotemia. (BUN 72 mg/mL, creatinine 2.6 mg/mL) Endoscopy revealed nonbleeding esophageal varices. His ICU course was complicated by seizures and cardiovascular instability characterized by persistent tachycardia, hypertension and episodic ventricular dysrhythmias requiring cardiopulmonary resuscitation. Despite treatment, he died within 18 hours of arrival. Autopsy revealed an esophageal tear with mediastinitis, and alcoholic liver disease. Pre-mortem blood analysis revealed a naltrexone level of 39 ng/mL (therapeutic 10–60 ng/mL). Drugs of abuse testing was negative. Conclusion: UROD has many potential complications including intractable vomiting with resultant dehydration, esophageal tears, hemorrhage, mediastinitis, and ultimately death. This case may represent unrecognized sedative-hypnotic or alcohol withdrawal. Public awareness of the potential dangers of UROD is necessary.

## 82 REFRACTORY SEDATIVE-HYPNOTIC WITHDRAWAL TREATED WITH PROPOFOL.

Sharma AN, Nelson L, Hoffman RS. *New York City Poison Control Center, New York, NY*

Background: Sedative-hypnotic withdrawal is generally treated with benzodiazepines or barbiturates. Propofol, a sedative-hypnotic with unique pharmacokinetics, may theoretically be useful for the management of patients not responding to conventional therapy. We report the first case of refractory sedative-hypnotic withdrawal successfully treated with propofol. Case Report: A 35-year-old female with a history of migraine headaches, who had been taking 750–1500 mg of butalbital per day, started outpatient chlordiazepoxide-based detoxification the week prior to admission. The morning prior to presentation the patient took over 200 mg of chlordiazepoxide and presented asking for further detoxification. Initial evaluation revealed a normal physical examination and normal vital signs. A routine psychiatric evaluation revealed a subtle cognitive disorder that required admission to the medical service for evaluation. The following day the patient developed severe psychomotor agitation, auditory hallucinations and autonomic instability (HR of 166 bpm and BP of 158/106 mmHg). Initial therapy including 3 mg of IV lorazepam and 600 mg of IV phenobarbital failed to improve the patient's clinical status. Subsequently, the patient received thiopental, succinylcholine, was intubated and started on a propofol infusion at 50 mcg/kg/min. Within an hour, the patient's heart rate was 95 bpm and her blood pressure was 124/84 mmHg. Twenty-four hours later the propofol drip was discontinued and the patient remained calm, had a normal mental status and was extubated. No further therapy was required. Conclusion: Propofol should be considered as an alternate therapy for refractory sedative-hypnotic withdrawal.

## 83 PROPOFOL SAFELY CONTROLS DELIRIUM TREMENS.

Olmedo RE, Nelson L, Howland M, Hoffman RS. *New York City Poison Control Center, St. John's University, New York, NY*

Background: Severe alcohol withdrawal resistant to high doses of benzodiazepines (over 1 gram of diazepam) is reported. Such patients usually require the administration of barbiturates, which frequently causes hypotension, the need for ventilatory support, and unpredictable awakening. Propofol, a GABA<sub>A</sub> agonist, is used in sedation of critically ill patients because of its predictably rapid pharmacodynamics. Although a single previous report suggested that propofol was efficacious in delirium tremens, that patient's diagnosis was debated. We describe the use of propofol for a clear case of delirium tremens. Case Report: A 34-year-old alcohol-dependent man presented to the hospital with one-day history of alcohol abstinence. Within a few hours in the Emergency Department, he developed tremulousness, diaphoresis, and had a self-limited seizure with a short postictal period. He was confused, delirious, agitated, and was hallucinating. His vital signs were: HR 110–120 BPM, BP 170/120 mmHg, and T 99.2°C. Administration of diazepam (1130 mg) and phenobarbital (650 mg) over 36 hours failed to control his agitated delirium. 40 mg of Propofol was administered and the patient was endotracheally intubated. A propofol infusion of 50 µg/kg/min was continued for 80 hours during which time the patient remained sedated with normal vital signs. The propofol infusion was stopped and he was extubated on hospital day 5. Although mild alcohol withdrawal symptoms continued following discontinuation of propofol, they were easily controlled with conventional therapy and he was transferred to psychiatry asymptomatic. Conclusion: Propofol may be safely used for delirium tremens after conventional therapy fails. Propofol's rapid and short-lived pharmacodynamics makes it ideal for following signs of alcohol withdrawal syndrome.

**84 ACIDOSIS WITH "NORMAL" ANION GAP DURING PROPOFOL INFUSION.**

Senecal PE, Chalut D, Simard Y, Lefebvre M. *McGill University Health Centre, Montreal, PQ, Canada; Centre de Toxicologie du Quebec, Ste-Foy, PQ, Canada*

**Background:** Propofol (2,6-diisopropylphenol), an iv anesthetic formulated as a white oil, is used in children, despite its innocuity for sedation not being well established. In pediatrics, propofol-associated acidosis has been reported. **Case Report:** Day 1, a 6.1 kg 5-month-old male received propofol during and after cleft lip surgery. Day 3, when the infusion rate was up to 15 mg/kg/h, acidosis (pH 7.21, pCO<sub>2</sub> 46, BIC 19; Na 132, Cl 108), green urine and gross lipemia were noted. Vitals: BP 75/35, HR 127 with normal temperature. Osmolality, creatinine, glucose, L-lactate: normal. Propofol was stopped (after a total of 720 mg/kg in 62.5 hours). Serum propofol then was 171 mg/L (adult therapeutic range: 1.5–6 mg/L). Day 4, one hour after an ionized calcium of 1.0 mmol/L: bradycardia (90/min) and ventricular tachycardia developed requiring CPR. Seven hours after: ABG pH 7.23, pCO<sub>2</sub> 36, BIC 15; Na 137, Cl 113, L-lactate 6.5 mmol/L, and ketones negative. N-acetylcysteine (50 mg/kg), and L-carnitine 50 mg/kg were given to limit the peaks of AST (6459), CK (7695), INR (12.3), creatinine (1.7 mg%) and lactate (9.6). Hemodialysis (HD) and hemoperfusion had little effect on serum propofol: 1.2 mg/L (day 4 before CPR and HD) and 1.3 mg/L (day 8) although HD corrected the base deficit transiently. ICU stay was 23 days. Propofol was measured, after chloroform extraction at pH 11.7, by splitless injection in an HP 5973 GC with MS single ion monitoring at m/z of 163 and 178 (or 107 and 122 for the 2,4-dimethylphenol internal standard). **Conclusion:** We report a near-fatal pediatric adverse event preceded by an elevated propofol concentration and a metabolic acidosis, with some features suggestive of renal tubular acidosis. In pediatrics, caution is advised if propofol is to be used for sedation at rates >10 mg/kg or for more than 8 hours duration.

**85 METABOLIC ACIDOSIS, HYPERTHERMIA, RHABDOMYOLYSIS AND SUBSEQUENT DEATH AFTER PROLONGED PROPOFOL INFUSION.**

Olmedo RE, Hoffman RS, Howland M, Nelson L. *New York City Poison Control Center, St. John's University, New York, NY*

**Background:** Propofol (2,6-diisopropylphenol) is a rapidly acting intravenous sedative-hypnotic agent unrelated to other available agents. Propofol is formulated in an emulsion of 10% soybean oil, 2.25% glycerol, and 1.2% purified egg phosphatide. It is widely used for sedation of mechanically ventilated adult patients. In children, however, prolonged high-dose propofol infusion (>6 mg/kg/h) is associated with hyperthermia, metabolic acidosis, rhabdomyolysis, hepatic steatosis, and lipemia. Nine of twelve cases previously reported resulted in death. Critics declare that the evidence is circumstantial and without clear etiology. We report the death of a child who developed these characteristics after a 42-hour propofol infusion. **Case Report:** A 28-month-old boy with history of Apert Syndrome underwent elective surgical correction of craniosynostoses. Sedation was maintained with a propofol infusion up to 18 mg/kg/h (total 372 mg/kg). On postop day 2, he developed hyperthermia (T<sub>max</sub> = 103°C), hypertension (BP = 150/85 mmHg), and tachycardia (Pulse = 140 BPM). Laboratory findings revealed metabolic acidosis (pH 7.32, pCO<sub>2</sub> 35 mm Hg, HCO<sub>3</sub> 17 mm Hg), rhabdomyolysis (peaked CPK 246,380 U/L on postop day 3), hypocalcemia (6.0 mg/dL), and hyperphosphatemia (8.5 mg/dL). Additionally, his blood was noted to be lipemic. Despite discontinuation of the propofol infusion, he was unresponsive. Electrolyte abnormalities were corrected. On postop day 3 he developed ventricular tachycardia unresponsive to standard therapy and he died. Autopsy revealed CNS changes consistent with Apert Syndrome (status postcraniotomy), hepatic microvesicular steatosis and degenerative changes in skeletal muscle fibers. All cultures were negative. A postmortem propofol level was 2.2 µg/mL (therapeutic 1.0 µg/mL). Autopsy findings in other propofol-associated deaths were similar. **Conclusion:** Prolonged propofol infusion may have caused this patient's rapidly deteriorating clinical syndrome. The finding of microvesicular steatosis points to a toxic-metabolic etiology of this syndrome. Propofol's safety in critically ill children remains undetermined.

**86 SIGNIFICANT OSMOLAR GAP AND ANION GAP METABOLIC ACIDOSIS FROM HIGH-DOSE LORAZEPAM INFUSION IN A CRITICALLY ILL ADOLESCENT.**

Pali M, Kallas H. *California Poison Control System—Sacramento, and University of California, Davis Medical Center, Department of Pediatrics, Sacramento, CA*

**Background:** Intravenous (IV) lorazepam preparations commonly include propylene glycol (PG), and benzyl alcohol (BA) in the vehicle. We present a case of an adolescent on high dose IV lorazepam who developed an osmolar gap from the PG vehicle, with an associated acute anion gap metabolic acidosis likely due to BA. **Case Report:** A critically

ill 13-year-old, 75 kg female developed acute respiratory distress syndrome requiring high-frequency oscillatory ventilation and neuromuscular blockade with a resulting permissive hypercapnia ( $\text{PaCO}_2$  up to 88 mmHg). She had no evidence of extrapulmonary organ system dysfunction. She developed hypertension and tachycardia as a result of her hypercapnic state, which was misinterpreted as agitation. She was then treated with increasing doses of IV lorazepam infusion (40% v/v propylene glycol and 3% BA in the vehicle) up to 500 mcg/kg/h (37.6 mg/h) for 4 hours. Her osmolar gap 3 hours after this change was 18.4 mOsm/L. She developed an acute anion gap nonketotic metabolic acidosis with a serum lactate of 1.5 mEq/L. A calculated PG level (using a conversion factor of 7.2) was estimated at 132.5 mg/dL; the actual serum PG level returned at 115 mg/dL. Her infusion also delivered 188 mg/h of BA, likely leading to the associated acute metabolic acidosis. Her lorazepam infusion was subsequently lowered to 250 mcg/kg/h with a corresponding resolution of her osmolar gap and anion gap metabolic acidosis. **Conclusion:** The PG vehicle in IV lorazepam preparations may cause significant osmolar gap widening when large doses are administered, even in patients with normal hepatorenal function. BA, which is converted to benzoate and hippurate, likely caused the acute anion gap metabolic acidosis seen in this patient.

### 87 GHB: EFFECT ON SERUM OSMOLALITY.

Ingels M, Kelner M, Ly B, Clark RF. *University of California at San Diego, Medical Center, San Diego, CA*

**Background:** Physicians are often called upon to treat patients with coma of unclear etiology. Gamma hydroxybutyrate (GHB) is one possible cause. Although GHB levels can be obtained, the lab turnaround time is usually several days. GHB has a relatively small molecular weight (MW = 104). The purpose of this study was to determine 1) whether GHB's measured contribution to serum osmolality correlates with the expected calculated value and 2) whether this is significant at clinically relevant serum concentrations. **Methods:** The sodium salt of GHB was dissolved in pooled human serum to make GHB concentrations of 3000 mcg/mL, 2000 mcg/mL, and 1000 mcg/mL. These samples and a control were analyzed on an osmometer to determine serum osmolality by freezing point depression, and were compared to the calculated expected osmolality. To determine clinical relevance, reference levels for GHB-associated coma were obtained from a national laboratory and local poison center records. **Results:** The expected changes in osmolality due to GHB at these concentrations are 28.8, 19.2, and 9.6 mOsm/L, respectively. The measured change in osmolality from GHB in this study were 28.5, 18, and 10.5 mOsm/L. Reference laboratory materials state that levels greater than 260 mcg/mL produce deep sleep/coma. The highest blood GHB level seen by this poison center in a comatose patient was 645 mcg/mL. The calculated contributions to an osmol gap of these levels are 2.5 and 6.2 mOsm/L, respectively. **Conclusion:** The measured contribution of GHB to serum osmolality can be predicted by calculations based on GHB's molecular weight. Patients may be comatose due to GHB and have only minimal elevation in serum osmolality. While the contribution of GHB must be considered when evaluating an osmol gap, GHB alone is unlikely to cause significant elevations at clinically encountered serum concentrations.

### 88 CAN THE DIAGNOSIS OF ETHYLENE GLYCOL (EG) TOXICITY BE MADE WITHOUT SERUM EG AND OSMOLALITY VALUES?

Jolliff HA, Dart RC, Bogdan GM, Daly FFS. *Rocky Mountain Poison & Drug Center—Denver Health, University of Colorado Health Sciences Center, Denver, CO*

**Objective:** Serum EG and osmolality values are not available in many regions. In such cases, the development of acidosis may indicate significant EG ingestion. This concept has never been validated using an evidence based approach. We test the hypothesis that all patients who ingest EG will develop an acidosis (serum  $\text{HCO}_3^- \leq 20$  mEq/L or base deficit  $\geq 4$ ) unless the resulting serum EG  $< 20$  mg/dL. **Methods:** A systematic analysis was performed using a predefined search procedure of MEDLINE and the Cochrane Databases (Ovid) for English language articles (1960–1999). Bibliographies, personal files, poison center consults (1997–1999), and extensive communications with authors identified additional cases. Structured data collection extracted time of ingestion, EG levels, serum  $\text{HCO}_3^-$ , and ethanol levels. **Results:** 162 articles (367 patients) and 11 consults were reviewed; 27 articles and 2 consults were excluded due to lack of patient data on acidosis and EG levels. Overall, 135 articles and 9 consults provided data on a total of 220 patients. Of these, 199 (90%) were acidotic and 21 (10%) were not acidotic at presentation. However, of these 21 patients, 12 had ingested ethanol, 8 presented within 4 hours (before acidosis would develop) and one patient was treated with a  $\text{HCO}_3^-$  infusion prior to assessment. Kappa scores for intra-rater and inter-rater reliability were 0.95 and 0.93 respectively. **Conclusion:** Although this study is limited by its retrospective nature, all patients with significant EG ingestion manifested acidosis

unless alcohol dehydrogenase was blocked or they presented prior to 4 hours of ingestion. Decreasing HCO<sub>3</sub> levels at least 4 hours after ingestion may be a sensitive indicator of significant EG ingestion when serum EG levels are not available.

### 89 USE OF FOMEPIZOLE TO AVOID HEMODIALYSIS.

Najafi CC, Korbet SM, Hertko L, Burda AM, Leikin JB. *Rush-Presbyterian-St. Luke's Medical Center, Toxikon, Chicago, IL*

**Introduction:** Fomepizole is approved as an adjunct to hemodialysis for ethylene glycol (EG) poisoning. We present a case of an elderly patient who ingested EG; the combination of Fomepizole and close metabolic monitoring avoided hemodialysis. **Case Report:** A 61-year-old woman with a history of depression was admitted after attempting suicide by ingesting EG. Upon arrival to the emergency department she was stuporous. Her pulse rate was 110/min; respiratory rate 30. Physical exam was remarkable for a lethargic patient able to move all extremities and follow simple commands. Laboratory values revealed: sodium 144 mmol/L; chloride 106 mmol/L; bicarbonate 13 mmol/L; blood urea nitrogen 15 mg/dL; creatinine 0.9 mg/dL; glucose 147 mg/dL. The lactic acid level was 3.2 mEq/L and acetone was negative. The arterial pH was 7.17; pCO<sub>2</sub>, 28 mmHg. The anion gap was 25 mmol/L. The measured plasma osmolality was 365 mOsm/kg with an osmolar gap of 61 mOsm/kg. Urinalysis revealed calcium oxalate crystals. She was hydrated with normal saline and given sodium bicarbonate. Fomepizole (15 mg/kg) was given followed by 5 additional doses (10 mg/kg × 4; 15 mg/kg × 1) q12 hours. The initial EG level was 202 mg/dL. Within 24 hours the anion gap metabolic acidosis had resolved. At 36 hours the EG level was 19 mg/dL with a normal osmolar gap. Her renal function remained normal with excellent urine output. The patient never required hemodialysis. **Conclusion:** In the setting of normal renal function, Fomepizole and supportive care can effectively treat EG intoxication even at levels regarded as an absolute indication for hemodialysis.

### 90 METHEMOGLOBINEMIA FROM ARTIFICIAL NAIL BUILDER AND FILLER.

McArthur NJ, Moore K, Sangalli B, Chiang WK. *Hudson Valley Poison Control Center, Sleepy Hollow, NY*

**Background:** There are numerous fingernail products available in the market. Most of these products contains acetone and other solvents. They usually pose minimal toxicity except for aspiration. We report here an unusual toxicity from one type of artificial nail builder and filler product. **Case Report:** A 57-year-old male inadvertently ingested a mouthful of a commercial artificial nail builder and filler instead of a cough syrup. Approximately 2 hours later, the patient experienced some shortness of breath and noted some bluish color skin changes. The patient presented to an emergency department with cyanosis. The rest of the examination was unremarkable and the patient was not in any distress. The initial pulse oximetry reading on room air was 80%. The pulse oximetry oxygen saturation was unchanged with oxygen supplement. An arterial blood gas was obtained: pH 7.38, pCO<sub>2</sub> 42 mm, Hg pO<sub>2</sub> 78 mm Hg, Hb 15.0 g/dL on room air. Co-oximeter reading demonstrated a methemoglobin level of 31%. Methylene blue (1 mg/kg) was administered intravenously with complete resolution of the cyanosis within 1–2 hours. Repeated methemoglobin levels over the 24 hour period were low and no further methylene blue treatment was required. This artificial nail builder and filler product contains 1–5% *N,N*-dimethyl-*p*-toluidine, 80–90% ethyl methacrylate monomer, and 10–20% ethylene glycol dimethacrylate. Although *N,N*-dimethyl-*p*-toluidine does not directly cause methemoglobinemia, but its metabolite *p*-methylphenylhydroxylamine is a potent methemoglobin inducer. **Conclusion:** *N,N*-dimethyl-*p*-toluidine containing products are capable of causing methemoglobinemia. It is important for poison centers to clearly identify the specific nail product and alert to the possibility of *N,N*-dimethyl-*p*-toluidine containing products.

### 91 POSTCOLLECTION RISE IN METHEMOGLOBIN LEVEL IN FROZEN BLOOD.

Wallace KL, Curry SC, Brooks D. *Department of Medical Toxicology, Good Samaritan Regional Medical Center, Phoenix, AZ*

**Background:** A 72-year-old woman presented with nonspecific complaints (headache, malaise, fatigue), history of residential sewer gas exposure, and 4 prior elevated venous methemoglobin (metHb) levels (15.6–20.1%), reported by an outside lab. Clinic evaluation was negative for previous history of methemoglobinemia, exposure to a likely exogenous cause of methemoglobinemia, or objective clinical or other lab test evidence to suggest true methemoglobinemia. Venous cooximetry performed immediately after collection in our lab revealed a metHb level of 0.8% (0.10 g/dL), while that



reported by the original reference lab on a simultaneously collected specimen was 14.9% (1.95 g/dL). The reference lab disclosed that cooximetry was performed 6 days postcollection on a frozen specimen. Literature search yielded no report of rise in metHb as an effect of frozen storage of blood specimens. We sought to determine the effects of specimen storage time and temperature on metHb level. Methods: Aliquots from a single collection of venous blood from a healthy volunteer were tested by cooximetry after various storage times (10 minutes to 6 days) at room temperature (22–24°C), refrigerated (1–4°C), and frozen (–14 to –12°C). Cooximetry was performed within 30 minutes of thawing frozen specimens in cool tap water and/or removal from refrigeration. Results: MetHb levels (% total Hb; g/dL) in frozen-thawed specimens (y) rose over time (x) above baseline levels (0.5%; 0.08 g/dL) in a manner that best fit a quadratic function [ $y = 0.156 + (0.620)x - (0.061)x^2$ ;  $r = 0.982$ ], beginning at 6 hours (1.8%; 0.29 g/dL) to a maximum at 6 days (10.9%; 1.81 g/dL). MetHb levels in nonfrozen specimens never exceeded 2.6% (0.40 g/dL). Conclusions: Progressive increase in metHb levels occurs in frozen blood specimens.

## 92 METHYLENE BLUE SAFELY AND EFFECTIVELY TREATS METHEMOGLOBINEMIA IN A KNOWN G6PD DEFICIENT PATIENT WITH HEMOLYTIC ANEMIA.

Sharma AN, Chawla S, Nelson L, Hoffman RS. *New York City Poison Control Center, New York, NY*

Background: Treatment of methemoglobinemia in G6PD deficient patients may induce hemolysis and be ineffective. We report a case of successful treatment in such a patient with profound hemolytic anemia and methemoglobinemia without any further complications. Case Report: A 40-year-old male with end stage AIDS was admitted for respiratory depression. The patient was empirically treated for pneumonia with primaquine and clindamycin. An initial CBC was positive for bands but had no evidence of hemolysis. The patient's condition deteriorated despite therapy and he was intubated. A subsequent CBC revealed a decrease in hemoglobin from 8.0 to 5.6 gm/dL and the presence of schistocytes and reticulocytes on a peripheral blood smear. Further evaluation revealed a methemoglobin level of 14.7% that rose to 23.1% over 2 days. As the patient's religious convictions prevented transfusion the risk of further hemolysis from methylene blue therapy was of great concern. Intravenous methylene blue was administered at 0.5 mg/kg with a subsequent drop in the methemoglobin level to 15.5% within a few hours of therapy. The patient's tachycardia resolved, mental status improved and his hemoglobin stabilized at 3.6 gm/dL with minimal evidence of hemolysis on subsequent peripheral blood smears. His methemoglobin level returned to normal within 48 hours without further therapy. Conclusion: This case demonstrates that methylene blue can be used effectively, in appropriate doses, for the treatment of methemoglobinemia in G6PD deficient patients.

## 93 FATAL PNEUMONITIS IN A CHILD FOLLOWING ASPIRATION OF A HIGH VISCOSITY HYDROCARBON.

White S, Baltarowich L, Warfield S, Smolinske S, Scott D. *Wayne State University, Detroit, MI*

Background: Hydrocarbon (HC) aspiration pneumonitis is a common, serious pediatric injury. While high viscosity HCs have rarely been associated with lipoid pneumonia following chronic oral use, pediatric fatality following a single accidental ingestion has not been previously described. Case Report: An 11-month-old male was witnessed to "taste" a hair and body oil (safflower oil 50–80%; mineral oil 30–50%; castor, avocado, sesame, jojoba <2% each) from a bottle on the bathroom counter. Immediate coughing was noted, milk was administered and one episode of pre-hospital emesis occurred. 15 minutes later in the ED, fever, tachycardia, tachypnea, persistent coughing, hypoxia, and wheezing were noted. The initial chest X ray was negative. Treatment included oxygen and bronchodilators. Over the next 12 hours, respiratory distress and oxygen requirements increased, and bilateral perihilar infiltrates developed. At 24 hours, bilateral pneumothoraces and pneumomediastinum corresponded to further desaturation, necessitating intubation and tube thoracostomies. Despite high frequency oscillatory ventilation, clinical and radiographic deterioration ensued, and ECMO was initiated on hospital day 5. Complications included further barotrauma, major hemorrhage from chest tube sites requiring massive transfusions, and inability to wean from ECMO. Nitric oxide was of no benefit. ECMO was discontinued on day 30, and the patient expired. Conclusion: We report the first pediatric fatality following the acute accidental ingestion of a high viscosity hydrocarbon. The lack of child-resistant packaging and the product similarity to a baby bottle are of concern. Furthermore, given the widespread use of this product, enhanced preventative educational efforts should be targeted at certain high-use populations.

#### 94 POISONINGS AND OTHER HEALTH-RELATED EFFECTS DUE TO THE GUAGUA PICHINCHA VOLCANIC ERUPTION OF 1999 (50 CASES) IN QUITO, ECUADOR.

Maldonado A, Sánchez A. *Quito-Ecuador Hospital VozAndes, Quito, Ecuador.*

**Objective:** To provide a general overview of the direct or indirect health effects of a volcanic eruption in the Andean city of Quito, Ecuador during the Guagua Pichincha volcanic eruption of 1999. **Methods:** This is a prospective study of all patients presenting to our hospital in Quito, Ecuador, with complaints related during and after the volcanic over a period of twelve days while the orange alert was active and when the volcanic ashes were in the environment. **Results:** Although we expected mainly respiratory problems generated by the direct effects of airborne volcanic ash, there were several unexpected findings of interest. Of patients presenting during the orange alert period of volcanic activity, 34% experienced carbon monoxide (CO) poisoning and 32% complained of falls from a height of 4–5 meters while cleaning rooftops. Just 10% of patients complained of bronchospasm, 8% experienced panic attacks, 8% allergic rhinitis, and 4% had other complaints. Most cases of CO poisoning were related to windows and doors having been sealed while the ash was falling and while gas heaters were being used. Two severe incidents involved coma and seizures, and involved more than one family member. Volcanic gases did not cause direct toxicity during the eruption. **Conclusion:** During the orange alert period of volcanic activity, the use of improperly ventilated space heaters caused the largest percentage of health-related effects detected at our community hospital by causing CO poisoning. Patients complaining of other respiratory-related effects accounted for less than half as many cases. Injury cases related to falls were almost as numerous as CO poisoning cases.

#### 95 HAZARDOUS MATERIALS EVENTS: EVALUATION OF TRANSPORT TO HEALTH CARE FACILITY AND EVACUATION DECISIONS.

Burgess JL, Kovalchick DF, Lymp JF, Harter L, Kyes KB, Brodtkin CA. *University of Arizona, Tucson, AZ; University of Washington, Seattle, WA; Washington State Department of Health, Olympia, WA*

**Objective:** To analyze hazardous materials event and victim factors associated with transportation of victims to a health care facility, and evacuation or shelter-in-place of nearby populations. **Methods:** A retrospective review was conducted on hazardous materials events in Washington State from 1993 to 1997. Multivariate logistic regression was used to identify risk factors for transportation, evacuation, and shelter-in-place. **Results:** Over five years, 2,654 victims from 457 events were reported, with 1,859 (70%) transported to a health care facility. Evacuation occurred in 279 (61%) events and shelter-in-place in 14 (3%) events. After excluding 14 deaths, regression analysis indicated that victims with trauma (OR 5.87, 95% CI 1.41–24.5), thermal burns (6.90, 1.15–41.3), dizziness/other CNS symptoms (1.59, 1.00–2.54), and headache (1.54, 1.01–2.35) were most likely to be transported. Chemical releases inside buildings (2.09, 1.06–4.10, compared with transportation events), and involving 3–5 victims (2.86, 1.54–5.31, compared to 1 victim) or  $\geq 6$  victims (8.74, 4.01–19.0), were most likely to involve evacuation or shelter-in-place. Events involving sulfuric acid (0.15, 0.05–0.49) and sodium hydroxide (0.19, 0.04–0.94) were least likely to involve evacuation or shelter-in-place. **Conclusions:** Prehospital decisions to transport victims to a health care facility and evacuate or shelter-in-place nearby populations are associated with event and victim factors. Further research is needed to determine if these factors also predict need for medical care or removal from exposure, and to develop evidence-based prehospital care protocols for hazardous materials exposure victims.

#### 96 HEALTH EFFECTS OF HAZARDOUS MATERIALS EXPOSURES.

Burgess JL, Kovalchick DF, Lymp JF, Kyes KB, Robertson WO. *University of Arizona, Tucson, AZ; University of Washington; Washington Poison Center, Seattle, WA*

**Objective:** To study adverse outcomes in individuals exposed to hazardous materials. **Methods:** Individuals exposed during hazardous materials incidents were contacted to complete a questionnaire within 8–40 days, and medical records were reviewed when available. Logistic regression analysis adjusting for correlation using GEE was used to identify predictors of adverse outcome. **Results:** From 12/97 to 10/99, 87 incidents were reported. 202 (59%) of 339 subjects with contact information were surveyed. 51 (25%) subjects had persistent medical symptoms. 18 (9%) left work or school for  $>2$  days. Medical records were available for 79 (66%) of 119 subjects evaluated in a health care facility. Medical treatment was reported in 46 (58%) and objective abnormalities in 57 (72%). For persistent symptoms, dermal exposures (OR 3.75, 95% CI 1.47–9.59),  $\geq 3$  alcoholic drinks/week (3.16, 1.13–8.80), and present or past use of psychiatric medications (2.68, 1.15–6.28) were significant predictors. For time loss, being divorced, widowed, or sepa-

rated (8.36, 1.40–50.1), asthmatic (4.92, 1.29–18.7), or having initial dermal symptoms (6.77, 1.34–34.2) were significant predictors. Of patients with medical records, those with preexisting hypertension (10.6, 1.82–61.3) were more likely to receive medical treatment or have objective medical findings, while those with inhalation exposures (0.17, 0.04–0.78) and those decontaminated at the scene (0.14, 0.03–0.69) were at reduced risk. **Conclusions:** Both incident and individual patient factors were associated with persistent symptoms, time loss, and medical treatment or objective medical findings.

### 97 INADVERTENT ADMINISTRATION OF NEBULIZED ACETIC ACID.

Sigg T, Pallasch E, Leikin JB. *Illinois Poison Center and Rush-Presbyterian-St. Luke's Medical Center, Chicago, IL*  
**Background:** Acetic acid inhalation has caused reactive airway disease, pulmonary edema, pneumonitis, reactive airway disease, and other respiratory difficulties. We describe a case where inhaled acetic acid produced relatively significant respiratory effects: acidosis, hypercapnea, and tachypnea in a 10-month-old female. **Case Report:** This child was initially hospitalized for RSV pneumonia. Inadvertently, her albuterol treatment was substituted with 5% acetic acid in 0.9% sodium chloride, diluted 1:1, then administered via nebulizer. She developed an immediate cough, tachypnea, and a mild respiratory acidosis; initial ABG: pH = 7.35, pCO<sub>2</sub> = 60, pO<sub>2</sub> = 67, O<sub>2</sub> saturation = 92% on FiO<sub>2</sub> = 100%. In an ABG twelve hours prior to this error: pCO<sub>2</sub> = 42. Over the next twelve hours, she received continuous albuterol nebulizer treatments while her respiratory rate increased from 40 to 65 and became more labored, repeat ABG: pH = 7.30, pCO<sub>2</sub> = 67, pO<sub>2</sub> = 72, O<sub>2</sub> sat = 93% on FiO<sub>2</sub> = 100%. The next morning, she became more playful and alert, the albuterol administration was changed to every two hours, and her respiratory rate decreased back to 40 while O<sub>2</sub> saturation on room air was 95%. Her other vital signs remained stable throughout this event, except for a low-grade fever. All chest X-rays showed no new infiltrates. **Discussion:** This child developed transient hypercapnea, cough, and respiratory difficulty after the inadvertent administration of nebulized acetic acid. She was treated for approximately 24 hours with 100% oxygen and continuous nebulized albuterol. Her acute symptoms resolved within 36 hours of the accident. No metabolic consequences were seen, possibly demonstrating a lack of systemic absorption. Treatment consisted of oxygen and bronchodilators; resolution was complete within 36 hours with no permanent sequelae. She was subsequently discharged home.

### 98 CRITICAL INJURIES FROM A CHLOROSILANE EXPLOSION.

Martin TG, Burgess JL, Tadaki K, Robertson WO. *University of Washington Medical Center, Washington Poison Center, Seattle, WA*

**Introduction:** Trichlorosilane and tetrachlorosilane are involved in the production of semiconductor grade silicon. This is the first report of critical injuries following exposure. **Case Reports:** Four male workers were injured by exposures from a ruptured pipe carrying 330°F pressurized trichlorosilane and tetrachlorosilane with subsequent explosion and fire. All 4 were decontaminated at the scene and sustained a toxic inhalation, corneal burns and patchy partial and full thickness dermal burns. Case 1, a 26-year-old, and Case 2, a 55-year-old, experienced severe corneal burns, necrotic laryngotracheobronchitis, and pneumonia, which progressed to severe ARDS despite steroids. Case 1 also had posttraumatic glaucoma (intraocular pressure  $\cong$  80 mmHg) resulting in retinal and corneal ischemia and expired on day 38. Case 2 also had hypotension requiring pressor support; acute renal failure, sepsis and atrial fibrillation and expired on day 16. He had a previous history of myocardial infarction. Case 3 was a 24-year-old with mild corneal burns, moderate respiratory insufficiency and laryngotracheobronchitis, which progressed to bronchiolitis obliterans; and was discharged moderately disabled on day 8. Pulmonary function tests 11 months later showed severe airflow obstruction, air trapping and mildly reduced diffusing capacity. Case 4 was a 29-year-old with mild corneal burns, mild respiratory insufficiency and oropharyngeal and laryngeal burns who was discharged on day 15. Pulmonary function tests 1 year later showed mild restrictive lung disease. **Conclusions:** Chlorosilanes react vigorously with water, releasing hydrogen chloride, which may also produce injury. These patients had persistent respiratory effects more severe than previously reported, and ocular findings not previously reported in the peer-reviewed medical literature.

### 99 ARSINE POISONING FROM RECYCLING OF COMPUTER CHIPS.

Caravati EM, Grover J. *Utah Poison Control Center, College of Pharmacy, University of Utah, Salt Lake City, UT*  
**Background:** Gallium arsenide (GaAs) is commonly used in the semiconductor industry and its recycling is placing an increasing number of workers at risk for arsine poisoning. This is the first report of arsine poisoning from GaAs chip

recycling. **Case Report:** A metal recycling plant reclaims gallium by heating GaAs chips in a furnace then rinsing in an acid wash to remove the arsenic and other impurities. A 25-year-old male was "in a hurry" and neglected to use the ventilation hood during the acid wash procedure. He reported getting a "few whiffs" of the fumes. He became progressively ill over two days and developed dark urine. The patient was diagnosed with jaundice and "gross hematuria" and was admitted for fluids and observation. His initial hematocrit (Hct) was 45.3%, platelets 223K, total bilirubin 7.9 mg/dL, indirect bilirubin 7.5 mg/dL, LDH 3570 U/L, and creatinine 1.0 mg/dL. Urinalysis was dark red with only 3–5 rbc's. The Hct decreased to 37.7% over the next 48 hours. Blood arsenic levels were 1162 mcg/L on day 6, 364 mcg/L on day 14, and 103 mcg/L on day 47 after exposure. The patient denied seafood/shellfish ingestion. The patient's subjective symptoms and myoglobinuria cleared by day 5 and he was discharged. The patient never received chelation therapy or blood transfusions. Elevated blood arsenic levels persisted for greater than 45 days postexposure. There were no long-term effects noted. **Conclusion:** This patient developed hemolytic anemia and increased blood arsenic from arsine poisoning. Recycling of GaAs chips for the semiconductor industry is a risk for arsine exposure.

### **100 EPIDEMIOLOGY OF US WORKPLACE HYDROGEN SULFIDE FATALITIES—AN IMMEDIATELY FATAL TOXIN WITH MULTIPLE CASUALTIES**

Hendrickson R, Hamilton RJ, Greenberg MI. *MCP-Hahnemann School of Medicine, Philadelphia PA*

**Objective:** To study the characteristics of US workplace fatalities caused by hydrogen sulfide (H<sub>2</sub>S). **Methods:** Bureau of Labor Statistics fatal occupational data for the years 1992 to 1998 (inclusive) was analyzed with EPI INFO software (6.04b, 97). The demographics of the worker and the workplace, survival after injury, and activity at the time of death was some of the characteristics analyzed. **Results:** Of the 33,520 fatal occupational injuries in this 7-year period, 45 deaths were caused by H<sub>2</sub>S. The exposure was immediately fatal in 38 (84%) of the cases, 3 (7%) more died within 24 hours, and the remaining 4 died over the next two months. There were 8 episodes with two or more deaths from the same incident. Of these, 5 episodes were simultaneously exposed workers and 3 were workers who attempted to rescue exposed co-workers. Total numbers of fatalities were highest among those who had been employed less than one year (12) and in companies with greater than 100 employees (16). Workers were often cleaning, inspecting, or repairing vats, tanks, and trucks. A large number of exposures (25) occurred after entering an enclosed space. Accidents were predominantly in the oil and natural gas industries. **Conclusion:** H<sub>2</sub>S is a highly lethal workplace toxin. Fatalities, when they occur, often involve multiple casualties and co-worker rescuers. Larger companies in the oil and natural gas industries should be particularly concerned about new workers.

### **101 PETROCHEMICAL EXPOSURE RESULTING IN TOXIC ENCEPHALOPATHY AND POSSIBLY PEYRONIE'S DISEASE—A CASE SERIES.**

Leikin JB, Carlson A, Rubin R, Webb MC, Secest CL. *RushPresbyterian-St. Luke's Medical Center, Toxikon, Chicago, IL*

**Introduction:** While toxic encephalopathy has been associated with solvent exposure, it has not been well defined. Furthermore, urologic consequences have not been associated. **Case Series:** Seven previously healthy male boilermakers (ages: 41 to 58 years old) were exposed to varying amounts of benzene and toluene along with tetra ethylene glycol while cleaning heat exchangers during a petroleum plant turnaround in October 1994. Spot urinary phenol was measured in five of these workers (range <5 to 16 mg/L). Following an initial flu-like illness during the first month postexposure, over the intervening six years all individuals complained of decreased appetite, headaches, fatigue, decreased libido, and sleep disturbances with all of the symptoms originating since the exposure. Serial neuropsychiatric evaluations documented attention, concentration and short-term memory impairment along with increased depression indices. Six of these individuals have been diagnosed with depression; they also exhibited erectile dysfunction presenting 6 months postexposure. Peyronie's disease with penile curvature ranging from 30° to 70° was noted in 3 workers (general population prevalence: 2%) at about 18 months postexposure eventually requiring surgery. **Discussion:** The persistent cognitive dysfunction in these 7 workers along with depression in six of them are attributable to a relatively brief exposure to petroleum solvents. Sexual dysfunction involving erectile abnormalities, decreased libido and Peyronie's disease may not be manifested until several months postexposure.

### 102 RESPIRATORY FAILURE FROM INHALATIONAL NICKEL CARBONYL EXPOSURE TREATED WITH CONTINUOUS HIGH VOLUME HEMOFILTRATION AND DISULFIRAM.

Scott LK, Grier LR, Arnold TC, Conrad SA. *Louisiana State University-Health Science Center Shreveport, Division Critical Care, Department of Emergency Medicine and the Louisiana Poison Control Center, Shreveport, LA*

**Background:** Nickel carbonyl is a colorless gas that can cause severe pulmonary and cerebral toxicity when inhaled. Acute poisonings are rare but often fatal. Treatment of acute poisoning is based on chelation of the nickel with sodium diethyldithiocarbamate (Dithiocarb). Disulfiram has been proposed as a substitute. Toxicokinetic studies have shown nickel may be transported by albumin, however, nickel may also circulate in a form that may respond to ultra-filtration. This case report describes the use of disulfiram combined with high volume hemofiltration in the treatment of severe toxicity from nickel carbonyl. **Case Report:** The patient is a 39-year-old chemist who suffered inhalational nickel carbonyl exposure in an industrial accident. He rapidly developed respiratory failure requiring mechanical ventilation. The initial 24-hour urine nickel level was 1021 mcg/L. Because diethyldithiocarbamate was unable to be obtained, the patient was started on disulfiram and continuous high volume hemofiltration. The rationale for this therapy was to use the disulfiram as a chelator and to remove the ultra-filtratable forms using continuous high volume hemofiltration. After 24 hours of this therapeutic regimen, a repeat 24-hour urine nickel level was reduced to 102 mcg/L. The patient's acute lung injury resolved over several weeks and the patient survived. Six month follow up reveals the patient is doing well with minimal pulmonary dysfunction. **Conclusions:** We present a case of severe respiratory failure from acute nickel carbonyl inhalation that was successfully treated with disulfiram and high volume hemofiltration combination therapy.

### 103 INHALATIONAL EXPOSURE TO HYDROGEN FLUORIDE TREATED WITH NEBULIZED CALCIUM GLUCONATE.

Boyer EW, Walker N, Woolf A, Shannon M. *The Children's Hospital, Boston, MA; Memorial Hospital, Providence, RI*

**Background:** Hydrogen fluoride (HF) may produce pulmonary injury and hemorrhage when inhaled. Nebulized calcium salts are an infrequently-used therapy for HF exposure. **Case Report:** A 26-year-old male glass worker suffered a 1 minute exposure to fumes from an anhydrous HF spill in an enclosed room. He experienced immediate upper airway irritation and shortness of breath. He was treated at the work site with 3 nebulized Ca<sup>++</sup> gluconate treatments with resolution of respiratory complaints. In the ED his physical exam was normal except for mild oropharyngeal erythema. ECG and CXR were normal. Initial laboratories showed a serum Ca<sup>++</sup> concentration of 8.2 mg/dL. He was treated with 35 grams of intravenous Ca<sup>++</sup> gluconate over a 5-day period but remained mildly hypocalcemic (range: 7.5-9.0 mg/dL) with normal PTH levels. He also had hypomagnesemia (range: 1.2-2.4 mg/dL) that was treated with 23g Mg<sup>++</sup> sulfate. Serum Ca<sup>++</sup> and Mg<sup>++</sup> levels returned to normal after 5 days. Pulmonary function testing at that time was also normal. He had no evidence of renal wasting of Ca<sup>++</sup> or Mg<sup>++</sup>. **Conclusion:** The use of nebulized Ca<sup>++</sup> salts led to symptomatic improvement and may have protected this patient from pulmonary injury. Although HF manufacturers educate on-site occupational clinicians about nebulized Ca<sup>++</sup> salts as therapy for inhaled HF, material safety data sheets (MSDS) often do not recommend it. MSDS should suggest this treatment. Clinicians should administer nebulized Ca<sup>++</sup> salts following inhalational HF exposure and after selected dermal exposures where inhalation of fumes may occur.

### 104 FATAL PULMONARY EDEMA FOLLOWING EXPOSURE TO AN ACROLEIN HERBICIDE.

Daly FFS, Kosnett MJ. *The Rocky Mountain Poison and Drug Center-Denver Health; University of Colorado Health Sciences Center, Denver, CO*

**Background:** Acrolein is a highly reactive and toxic aldehyde, recognized as a pulmonary irritant following smoke inhalation. It is also found in herbicides used in irrigation systems. **Objective:** To report delayed pulmonary edema and death following acute exposure to an acrolein-containing herbicide in an occupational setting, the first report of such a case. **Case Report:** A previously well 38-year-old male nonsmoker was applying Magnacide-H (95% acrolein), using a high pressure application system. At approximately 0800, a hose disconnected, reportedly sending undiluted herbicide 60 feet into the air at 0.15 gallons/sec. Without protective apparatus, the patient was "soaked" as he tried to stem the

flow. Immediate symptoms were a choking sensation, cough and burning of the eyes, nose and mouth. He jumped into nearby water to wash. He presented to a local ED later that morning complaining of chest discomfort and dyspnea. Physical examination showed a respiratory rate of 24 breaths/minute and conjunctival suffusion. Auscultation of the lungs and chest radiograph were interpreted as normal. Arterial blood gases revealed a room air PaO<sub>2</sub> of 59 mmHg. He was discharged in apparently stable condition 2.5 hours later with reportedly "good O<sub>2</sub> saturations on room air." Later that afternoon, he complained of respiratory symptoms and restlessness. He was found dead in a chair at home 20 hours after the exposure. Postmortem demonstrated acute bronchitis, laryngitis and "massive pulmonary congestion and edema". **Conclusion:** Delayed pulmonary edema may complicate inhalational exposure to acrolein-containing herbicides. Prolonged observation may be warranted in the management of exposed patients with initial pulmonary symptoms.

### 105 PHOSPHINE EXPOSURE FROM A METHAMPHETAMINE LAB INVESTIGATION.

Burgess JL. *University of Arizona, Tucson, AZ; Washington Poison Center, Seattle, WA*

**Background:** Phosphine gas may be produced during manufacture of methamphetamine in clandestine laboratories. Exposure to this gas is a potential threat to law enforcement officers. **Case Report:** A 28-year-old forensic specialist with a clandestine drug laboratory investigation team was investigating a red phosphorus-hydriodic acid methamphetamine lab in an automobile repair shop. Four warm 22 liter reaction flasks half full of red liquid were found, vented into separate five gallon buckets of ice. Phosphine gas was initially detected during entry, and the building was ventilated until the levels decreased to below detectable limits. During subsequent lab processing without respiratory protection, the patient noted a fishy garlic odor, and a phosphine concentration in her work area was measured at 2.7 ppm. Estimated duration of exposure was 20–30 minutes. Shortly following exposure she developed dizziness, cough, headache, and diarrhea. Initial physical examination, CXR and laboratory tests within 3–4 hours of the exposure were normal, as was spirometry four days later. Pulmonary exam at 7 days postexposure revealed bilateral rhonchi. The cough persisted despite B-agonist inhaler treatment, and was worse with exertion. Repeat spirometry tests were unremarkable. High resolution CT scan 4 months following exposure was essentially normal. Methacholine challenge test at that time demonstrated mild reactive airways disease without evidence of asthma. Symptoms improved on inhaled steroids. **Conclusions:** Phosphine gas has been measured previously in methamphetamine labs, and deaths in lab "cooks" from presumed phosphine exposure have been reported. This is the first case of documented occupational phosphine gas exposure in a law enforcement officer with resulting persistent adverse effects.

### 106 CLINICAL ASSESSMENT OF PATIENTS WITH CHRONIC ENVIRONMENTAL EXPOSURE TO LEAD.

Lugo AM, Haynes JF, Artalejo L. *West Texas Regional Poison Center, El Paso, TX*

**Background:** We evaluated a community chronically exposed to lead and other heavy metals from a nearby smelter plant, "Met Mex Peñoles", located in Torreon, Coahuila, Mexico. From March to September 1999, blood lead levels (BLL) were obtained from 7,512 children ages 0–18 years old in the vicinity of the plant: 13.4% (1,007) of the samples had a BLL <10 µg/dL; 49.7% (3,732) had 10–24 µg/dL; 27.6% (2,073) had 25 to 44 µg/dL; 8.9% (671) had 45 to 69 µg/dL; and 0.4% (29) had >69 µg/dL. Approximately 9.3% (700) of children had a BLL ≥45 µg/dL. An extensive environmental cleanup is ongoing. **Methods:** A physical exam of 107 patients were conducted to determine any clinical signs and symptoms of lead toxicity. The study included 25 hospitalized patients with BLL from 45 to 91 µg/dL, with a median of 55.71 µg/dL, who were hospitalized to receive chelation therapy with DMSA, and 82 ambulatory patients with a BLL of 5 to 51 µg/dL, with a median of 28.35 µg/dL. All patients were residents from neighborhoods where the indices of contamination in the air and soil were the highest. **Results:** We did not detect a single case of encephalopathy, nor any other clinical evidence of serious health problems related to a high BLL. **Conclusion:** There was a lack of symptoms in a large part of the patients and mild symptomatology in some of the cases with a BLL >70 µg/dL. These findings are comparable with other large series of chronic environmental exposures to lead. The high BLL require continued monitoring and a careful search for neurotoxicity. There is a need to perform further epidemiological studies in the region.

**107 LEAD EXPOSURES FROM INDOOR FIRING RANGES.**

Stromness J, Crouch BI, Caravati EM. *Utah Poison Control Center, College of Pharmacy, University of Utah, Salt Lake City, UT*

**Background:** Lead poisoning is an occupational hazard of instructors at indoor firing ranges. We report seven individuals exposed to lead at firing ranges, six of whom has elevated blood lead concentrations (BLC). **Case Series:** Cases 1–5:

	Age/Gender	Initial BLC	Repeat BLC	Exposure type	Symptoms
Case 1	27 yr M	62 mcg/dL	19.6 mcg/dL	Owner operator	Yes
Case 2	14 yr M	39 mcg/dL	35 mcg/dL	Reclaims lead/sweeps	No
Case 3	3 yr M	20.8 mcg/dL	11 mcg/dL	Plays/eats on floor	No
Case 4	Adult F	7 mcg/dL	3 mcg/dL	Reception desk	No
Case 5	3 mo M	12 mcg/dL	10.5 mcg/dL	Breastfed by #6	No
Case 6	38 yr M	45 mcg/dL	27.8 mcg/dL	Inspects q 2 weeks	No
Case 7	34 yr M	35 mcg/dL	31.5 mcg/dL	Reclaims bullets	No

The PCC was called about a 27-year-old male with an elevated BLC who worked full time at an indoor firing range without using personal protective equipment (PPE). He had diarrhea and nausea, and was treated with succimer. He was a sentinel case and all family members were potentially exposed to lead. The ventilation system at the firing range was subsequently upgraded. Two additional occupational cases are reported. Case 7 did not wear PPE Family members of #7 were monitored and did not have elevated BLC. **Conclusion:** Lead exposure is still a risk at indoor firing ranges and may involve more than just those with direct lead contact. Family members may also need to be screened and poison control centers can play a proactive roll in this process.

**108 PLUMBISM IN PREGNANCY TREATED WITH DMSA.**

Mirkin D, Sudakin D, Parker S, Horowitz Z, Bizovi K, Warden C. *Oregon Poison Center, Oregon Health Sciences University, Portland, OR*

**Background:** Data on the treatment of lead toxicity in pregnancy is limited. We were unable to find published experience in the use of 2,3-dimercaptosuccinic acid (DMSA) in human pregnancy. **Case Report:** We present a case of chronic lead toxicity in pregnancy. One year earlier and prior to pregnancy, the patient had been evaluated for abdominal pain and was found to have anemia with basophilic stippling and a blood lead level of 67 mcg/dL. She was lost to follow up until presentation in the 25th week of pregnancy with recurrence of her abdominal pain. Ultrasound showed a normal intrauterine fetus. Blood count showed a Hgb of 7.4 gm/dL, and Hct 22.9%. The blood lead level was 62 mcg/dL. The patient was treated with oral DMSA 10 mg/kg every 8 hours for 5 days followed by 10 mg/kg every 12 hours for 13 days. Blood lead just prior to the initiation of chelation was 44.0 mcg/dL. The recommended 19 day treatment period was shortened by 1 day due to worsening of her recurrent nausea and vomiting. Blood lead at the conclusion of DMSA therapy was 43.9 mcg/dL. No environmental cause has been clearly identified despite inquiry into folk remedies, pottery, hobbies, occupation and a home visit by the public health department. At parturition, maternal blood lead was 57.6 mcg/dL and Hct was 16. The patient is undergoing further chelation postpartum. **Conclusion:** A single course of oral chelation therapy with DMSA did not change the maternal blood lead level. Its efficacy may have been affected by ongoing mobilization of maternal blood lead or continued unrecognized environmental lead exposure. The treatment appeared safe despite some nausea and vomiting.

**109 AN UNUSUAL CASE OF IN-UTERO LEAD EXPOSURE.**

Walsh MJ, Pali M, Ferguson T. *California Poison Control System–Sacramento Division, Sacramento, CA*

**Background:** This report describes an unusual case of lead poisoning in a newborn due to in-utero lead exposure. **Case Report:** A 30-year-old Hispanic female, one day postpartum, told her physician she had eaten glazed pottery from Mexico as part of a cultural ritual one week prior to her child's birth. The newborn's lead level was 84 mcg/dL. The baby was treated for lead poisoning with succimer 10 mg/kg mixed with formula and given every 8 hours for 5 days,

along with IV hydration. After 5 doses of succimer the lead level dropped to 46 mcg/dL. The following depicts the baby's blood lead levels over time:

Date	Dose	Lead Level
Day 6	dose changed to succimer 10 mg/kg bid	
Day 10	Succimer 10 mg/kg bid	34 mcg/dL
Day 23	Succimer 10 mg/kg bid	30 mcg/dL
Day 35	medication stopped	34 mcg/dL
Day 59	Succimer restarted 10 mg/kg bid	
Day 89	Succimer 10 mg/kg bid	31 mcg/dL
Day 276	Succimer 10 mg/kg bid	18 mcg/dL
Day 296	Succimer 10 mg/kg bid	9 mcg/dL, med stopped

**Conclusion:** Cultural rituals during pregnancy can result in toxicity to the infant. Although this infant seemed to be developing normally while undergoing treatment for lead poisoning, the long-term effects of lead poisoning from in-utero exposure are unknown. The long-term effects of succimer treatment in children under the age of one year are unknown as well.

#### 110 LEAD CHELATION IN THE NEONATE.

Mirkin D, Sudakin D, Parker S, Horowitz Z, Bizovi K, Warden C. *Oregon Poison Center, Oregon Health Sciences University, Portland, OR*

**Background:** Neonatal cord bloods have been used to screen for lead exposure. We present a case of a healthy-appearing neonate with a cord blood lead of 126 mcg/dL. **Case Report:** A woman with chronic lead toxicity had a maternal blood lead level of 57.6 mcg/dL the day of delivery and a Hct of 15. The neonate's Hct was 48, and she was morphologically normal, appropriate size for gestational age, alert and responsive. Based upon the elevated cord blood level, chelation was begun four days postpartum with BAL 50 mg/m<sup>2</sup> IM every 4 hours and CaNa<sub>2</sub>EDTA 1000 mg/m<sup>2</sup>/d via continuous IV infusion. A blood lead level collected just prior to chelation was 74.7 mcg/dL. Seven hours after the commencement of chelation the blood lead level was 46.7 mcg/dL. The urine was alkalized with sodium bicarbonate. 24-hour urine collected during the second day of parenteral therapy had a total lead content of 682 mcg. There were no adverse effects. Therapy was discontinued after 3 days when another 24-hour urine lead was collected showing a lead content of 206.2 mcg. After 24 hours off therapy, the patient was begun on DMSA 350 mg/m<sup>2</sup> po every 8 hours for 5 days followed by 350 mg/m<sup>2</sup> po every 12 hours for 14 days. A 12-hour urine during the first day of DMSA therapy showed a total of 45.9 mcg lead. **Conclusion:** Differences among maternal, cord and fetal blood lead levels of a healthy neonate remain unexplained. Possible reasons include a threefold difference in hematocrit between mother and child, presence of fetal hemoglobin, and mobilization of bone lead during parturition.

#### 111 CALCIUM EDTA AND DMSA CHELATION OF A NEONATE WITH CONGENITAL LEAD INTOXICATION.

Guzman DD, Velez LI, Shepherd JG, Goto CS. *The University of Texas Southwestern Medical Center and the North Texas Poison Center, Dallas, TX*

**Background:** The placenta is permeable to lead, and the adverse effects of lead on fetal and early childhood development have been well documented. We report a case of congenital lead intoxication in a 3-week-old neonate who was chelated with IV calcium EDTA followed by oral DMSA. **Case Report:** A 3-week-old full-term Hispanic female was referred for an elevated blood lead level (BLL). The maternal BLL was 50 µg/dL during the third trimester and 60 µg/dL after delivery. The infant had a normal physical examination and normal vital signs. Admission laboratory tests showed: lead 37 µg/dL, hemoglobin 12.2 g/dL, hematocrit 35.1%, reticulocytes 0.8%, free erythrocyte protoporphyrin 229 µg/dL, creatinine 0.4 mg/dL, and normal urinalysis. The infant was chelated for 5 days with a continuous IV infusion of calcium EDTA (50 mg/kg/d) and the BLL decreased to 17 µg/dL. There were no adverse effects except for a mild anemia attributed to frequent phlebotomy. Four weeks later, the BLL had increased to 26 µg/dL and she was chelated



with 3 courses of oral DMSA (10 mg/kg tid for 5 days, then bid for 14 days) over 3 months until her BLL stabilized under 20 µg/dL. Conclusion: We report the use of IV calcium EDTA and oral DMSA in treating a neonate with congenital lead intoxication. It is not known whether early chelation will improve the long-term neurologic outcome of such infants. This case supports the need for prenatal screening in mothers who are at high risk for elevated BLL, such as recent immigrants from areas with a high prevalence of lead exposure or those with other children who have elevated BLL.

### **112 THE SUCCESSFUL MANAGEMENT OF SERIOUS LEAD INTOXICATION IN A SIX-MONTH-OLD CHILD WITH ORAL CHELATION THERAPY.**

Speranza V, Gaar G, Chambers-Emerson J, McNutt T. *Florida Poison Information Center-Tampa, Tampa, FL*

Objective: To describe the presentation and treatment of a serious lead exposure in a 6-month-old child with oral Succimer (Chemet) therapy. Case Report: A six-month-old boy was referred to our hospital from the local health department for evaluation, after it was discovered that the older sibling had an elevated serum lead level of 68 mcg/dL. When a screening intervention was performed on the six month old, an initial serum lead level of 83.5 mcg/dL was revealed. Upon further inquiry it was found that the child's parent had been giving the infant Azarcon, a Mexican home remedy used to treat stomach cramps. This product contains as much as 94% lead by weight. The mother had been administering this product daily to both children for as long as one week prior to admission. The Poison Center was consulted on the use of chelation therapy. Since the child was neurologically intact, it was decided to initiate chelation therapy with oral Succimer instead of parenteral chelation treatment. Within two days after initiation of Succimer therapy, the blood lead level dropped to 56 mcg/dL. A repeat serum lead level obtained 72 hours after Succimer therapy noted a reduction in the level to 40 mcg/dL. The child remained asymptomatic and was discharged home on the remaining doses to complete full course oral chelation therapy. The Succimer therapy was well tolerated in this patient. Conclusion: The use of oral chelation agents like Succimer can be successfully utilized to treat small children with significantly elevated blood lead levels (>70 mcg/dL) who present with no clinical evidence of neurological toxicity from lead exposure.

### **113 BITE THE BULLET: LEAD POISONING AFTER INGESTION OF 206 LEAD BULLETS.**

McNutt TK, Dethlefsen M, Shah R, Chambers-Emerson J. *Florida Poison Information Center, Tampa General Hospital, Tampa, FL; James A. Haley V.A. Hospital, Tampa, FL*

Objective: To describe presentation and management of an ingestion of 206 22-caliber lead bullets. Case Report: A 45-year-old male with a history of schizophrenia was admitted to a local VA psychiatric unit. Five days later, an endoscopy was performed due to abdominal pain, gastrointestinal bleeding, and a Hg of 5.6 g/dL. Results revealed the presence of bullets in the gut. On a subsequent KUB, greater than 50 bullets were visualized in the stomach and intestines. The Poison Center was consulted. Recommendations included whole bowel irrigation and a blood lead level. After poor results of gastrointestinal decontamination and a repeat KUB showing >100 cartridges, surgical intervention was considered. However, surgery was not performed due to risk of detonation of the bullet mass by electrocautery. Five days after the lead level was drawn, the results were reported as 391 mcg/dL. Calcium EDTA was initiated, followed by aggressive gastrointestinal decontamination. Four days of whole bowel irrigation with GoLyteLy facilitated passage of a total of 206 cartridges over the next 10 days. The patient was discharged on a 14-day course of Succimer 600 mg tid to treat radiographic evidence of lead deposits in the bone and a lead level of 49 mcg/dL. An outpatient visit six weeks later showed the lead level had dropped to 24 mcg/dL. Conclusion: Aggressive gastrointestinal decontamination, Calcium EDTA and a course of Succimer successfully treated an ingestion of a large quantity of lead bullets and subsequent lead poisoning.

### **114 ADVERSE EFFECTS IN 5 PATIENTS RECEIVING EDTA IN AN OUTPATIENT CHELATION CLINIC.**

Singleton K, Morgan B, Thomas J, Pettigrew D. *Georgia Poison Center, Atlanta, GA; Athens Regional Medical Center, Athens, GA*

Background: Calcium EDTA is a chelating agent primarily used in the treatment of lead poisoning. Despite limited scientific evidence, parenteral EDTA chelation therapy has been advocated for a variety of conditions including atherosclerosis. Case Series: Five patients presented to the ED with symptoms that developed 30 minutes to 2 hours into IV

chelation therapy at an outpatient clinic. All patients received an infusion of sterile water with 3 g EDTA, 2 g MgCl, 100 mg B-12, 100 mg B-6, 1 cc Vit B Complex and 15 g Vit C. One patient received 10 cc of 50% DMSO soln IV. All patients initially experienced GI and musculoskeletal symptoms. Additional signs and symptoms include headache 4/5, excessive thirst 4/5, and diaphoresis 4/5. On presentation patients were hypotensive 5/5, tachycardic 4/5 and febrile 5/5. Therapy included IV fluids 5/5, dopamine 1/5, and IV antibiotics 4/5 pending cultures. Initial laboratory data showed leukopenia 5/5, thrombocytopenia 3/5, bandemia 4/5. All patients had ECG abnormalities of unknown acuity. Four of 5 patients were admitted with length of stay from 3–5 days including one ICU admission. Of those admitted, 3 of 4 had transient, mild rise in serum creatinine. Blood cultures in all 4 showed no growth. All patients were discharged without permanent sequelae. **Conclusion:** We present a case series of 5 patients who developed adverse effects after receiving outpatient IV chelation therapy. It is unclear if effects were related to dose, rate of administration or contaminant. Investigation by state authorities is ongoing.

### 115 INGESTION OF MERCURIC OXIDE POWDER WITHOUT CLINICAL TOXICITY.

Ly BT, Rangan C, Ingels M, Williams SR, Clark RF. *California Poison Control System, University of California at San Diego, San Diego, CA*

**Background:** Most inorganic mercury poisonings reported have been due to mercuric chloride. Experience with mercuric oxide (HgO) exposure is mainly in children ingesting button batteries containing HgO. We report a case of a HgO powder suicidal ingestion. **Case Report:** A 31-year-old male presented to an emergency department (ED) 45 minutes following a witnessed ingestion of approximately 40 g of HgO powder mixed in water. Prior to arrival he had vomited 3 times. In the ED, he only complained of mild abdominal cramping. Except for HR 106 his exam was unremarkable. KUB revealed dense radiopaque material primarily in the stomach. Activated charcoal was administered prior to transfer to our institution. Electrolytes, BUN, Cr, and CBC obtained at the initial facility and 4 hours postingestion was entirely normal, except for minimal transaminase elevation. Whole bowel irrigation with polyethylene glycol was initiated 5 hours postingestion at 1.5 L/h and was continued for 24 hours until KUB was negative. BAL was initiated at 4 mg/kg with subsequent doses at 3 mg/kg q 6 hours. Repeat labs showed transient decrease in HCO<sub>3</sub> to 19 with normal BUN/Cr. The blood and first 24 hour urine Hg levels were 130 µg/dL and 2220 µg/L, respectively. Blood Hg level peaked on day 2 (185 µg/dL). Urine concentrations declined on all subsequent collections. On the 5th day, chelation therapy was changed to oral DMSA (10 mg/kg q8 hours for 10 days). At discharge on hospital day 7, he was asymptomatic with normal renal function. Repeat urine and blood Hg measurements 18 days after ingestion showed levels of 308 µg/L and 15 µg/dL, respectively. The patient remained asymptomatic at 6 weeks. **Conclusion:** Despite high levels of Hg in urine and blood, our patient did not develop the classic findings of inorganic Hg toxicity. It is unclear whether distinctions exist in the toxicity of different salts of Hg.

### 116 CHRONIC PEDIATRIC ARSENIC POISONING FROM PRESSURE-TREATED WOOD BURNED IN A FIREPLACE.

Hahn I, Kline SA, Howland MA, Hoffman RS, Nelson LS. *New York City Poison Control Center, St. John's University, New York, NY; Eden Prairie Clinic, Eden Prairie, MN*

**Background:** Pressure-treated wood contains copper, chromium, and arsenate (CCA) to prevent decay. Arsenic poisoning from burning pressure-treated wood usually presents with immediate symptomatology. **Case:** An 11-year-old-male presented with a 6–8 week history of leg cramping, paresthesias, and disabling pain in his feet prior to evaluation. His past medical history was significant for lactose intolerance and chronic sinusitis secondary to a mild IgG subclass deficiency. His medications were Augmentin® and guaifenesin/pseudoephedrine. There was no patient or family history of pica, vitamin deficiency, metabolic disorders, neuropathy, or illicit drug use. While building an indoor fire alone, the patient unknowingly used pressure-treated wood and was singularly exposed for 10–15 minutes several months prior to his initial medical evaluation. Apparently the damp wood was smoldering and caused smoke to enter the room because the flue was initially closed before the parents realized the problem. Irritability, a gastrointestinal illness, and lower extremity symptoms began 2 weeks after the experience. On his day of presentation, the patient only complained of bilateral pain and tingling of his feet as well as difficulty sleeping and walking secondary to the pain. The patient had normal vital signs. His neurologic examination, which was reportedly normal 2 months earlier when he was evaluated for

a minor head contusion after snowboarding, was now significant for bilateral symmetric hyporeflexia, decreased sensation to vibration and position sense of feet, and hypesthesia. Soft tissue swelling was also noted in the dorsum of both feet. No skin changes or nail abnormalities were noted. The patient had normal EMGs of the lower extremities. Laboratory evaluation was normal except for a 24 hour urine total arsenic test of 128  $\mu\text{g}/950\text{ mL}$  urine, inorganic fraction was 32  $\mu\text{g}/\text{g}$  creatinine ( $>25\text{ }\mu\text{g}/\text{g}$  creatinine toxic). Four days after the initiation of succimer therapy, his vibration and position sense normalized but his hypesthesias remained. The patient is still being followed. **Conclusions:** This case suggests a single exposure to inhalational arsenic can cause significant and persistent toxicity that may be reversible with chelation.

### 117 FATAL IRON POISONING ASSOCIATED WITH MASSIVE IRON LEVEL (18,570 $\mu\text{g}/\text{dL}$ ).

Perrone J, Lawless ST, Quintana E, De Roos F. *Delaware Valley Poison Control Center, Philadelphia, PA*

**Background:** Despite FDA mandated warning labels on iron preparations since 1997 serious iron exposures continue to occur. Despite early diagnosis, gastrointestinal decontamination with whole bowel irrigation (WBI), endoscopy, and gastrotomy, and aggressive supportive care, we report a fatality in a 17-month toddler with the highest serum iron level ever reported of 18,570  $\mu\text{g}/\text{dL}$ . **Case Report:** A 17-month-old toddler presented within 1 hour postingestion of ferrous gluconate tablets, actively vomiting blood and pill fragments. He was lethargic with vital signs: T 96.9, RR 35/min, HR 130 beats/min, BP 95/55 mmHg. An abdominal radiograph (AXR) revealed numerous radioopaque fragments. Orotracheal intubation, orogastric lavage, WBI and IV deferoxamine at 15 mg/kg/h were initiated. Laboratory abnormalities included a serum iron level 18,570  $\mu\text{g}/\text{dL}$ , pH 7.15, and an INR 4.5. After transfer to a tertiary pediatric ICU, the patient remained in shock (HR 188 beat/min, BP 70–80 mmHg) despite fluid boluses and vasopressors. An AXR revealed persistent iron tablets. Endoscopy was unsuccessful in removing pills which were tightly adherent to the gastric mucosa. Emergent laparotomy and gastrotomy was ultimately successfully in removing the remaining tablets. Within 8 hours after exposure and with a deferoxamine infusion of 15 mg/kg/h, the patient developed acute lung injury, diagnosed by increased arterial-alveolar oxygen gradient and typical radiographic findings. His oxygenation and hemodynamics progressively deteriorated and he died despite aggressive ventilatory support, vasopressors, and blood product transfusions. **Conclusion:** Despite early diagnosis, standard dose IV deferoxamine, and early surgical gastrointestinal decontamination, this massive iron exposure resulted in acute lung injury and a fatal outcome. Acute lung injury appears to be a complication of severe iron poisoning possibly independent of deferoxamine therapy. Further interventions should explore the perceived pulmonary toxicity of high dose deferoxamine as well as preventive measures to decrease fatal iron exposures.

### 118 FERRIC CHLORIDE COPPER ETCHANT INGESTION: DECODING DEGREES BAUMÉ.

Dougan C, Tominack R, Thompson MW, Scalzo AJ. *Missouri Regional Poison Center, Cardinal Glennon Children's Hospital, St. Louis, MO*

**Background:** Ferric chloride ( $\text{FeCl}_3$ ) solution, used by the hobbyist and artist to etch printed circuitry boards and intaglio prints, is a less familiar source of iron exposure than vitamin supplements. The literature contains only two reports of  $\text{FeCl}_3$  ingestion, neither in the US. Assessing the exposure requires an understanding of the typical products and the hydrometer terminology that indicates the dilution. **Case Report:** A 17-month-old child drank from a cup containing a 1:3 dilution of stock  $\text{FeCl}_3$  (43 degree Baumé [Bé]) used by his father to etch copper circuitry boards. Multiple episodes of vomiting ensued. In the ED, no oral burns were noted. A stat serum iron level was 403 mcg/dL. An IV infusion of deferoxamine was begun. Repeat level 2 hours later was 378 mcg/dL and deferoxamine was discontinued. The child was lethargic overnight but normal the next day.  $\text{FeCl}_3$  is available through electronics hobby shops, specialty chemical and arts sources as an anhydrous powder and as premixed stock solution of 41–43 degrees Bé. This is an arbitrary scale of specific gravities measured by floating a heavy-liquid hydrometer in the solution. The typical  $\text{FeCl}_3$  43 Bé stock solution has a Sp. Gr. of 1.42 and is 40% w/w. Thus, 1 mL weighs 1.42 grams and contains 568 mg of  $\text{FeCl}_3$  (193 mg Fe). Addition of 3 equal parts of water produce a 14 degree Bé solution. This patient's ingestion contained 48.25 mg Fe/mL (241 mg/5 mL), 22.6 mg/kg. **Conclusion:**  $\text{FeCl}_3$  etching solution represents a source of toxic iron ingestion in the home. Because of its unusual units of measure, ascertainment of iron dose is not straightforward. Triage decisions can be made by calculating dose from the known initial concentration of the typical stock solution.

### 119 RETROSPECTIVE REVIEW OF CARBONYL IRON INGESTION.

Wahlen HS, Stephens TL, Spiller HA, Krenzelok EP, Benson B, Peterson J, Dellinger JA. *Kentucky Regional Poison Center, Louisville, KY*

**Background:** There are limited published data concerning carbonyl iron ingestion and no data on overdose. Iron carbonyl is a nonionized insoluble form of elemental iron, which requires conversion by stomach HCL for absorption of the iron to occur. This rate-limiting step may result in reduced toxicity of large ingestions. **Method:** Retrospective chart review of all patients with carbonyl iron ingestion reported to 5 regional poison centers from the years January 1998 to April of 2000. **Results:** 33 patients with carbonyl iron ingestion were reported. 18 patients (55%) were male. 27 patients (82%) were managed outside a HCF, of which 11 (41%) had continued telephone follow-up. Mean and median age of these patients was 3 years and 20 months, respectively. Mean dose ingested was 11.2 mg/kg with a range of 2.2 to 34.5 mg/kg. 1 patient received syrup of ipecac. The remaining 10 were observed with no intervention. No effects were noted in any of these patients. 6 patients were evaluated in an ED. Mean dosage ingested was 34 mg/kg with a range of 12 to 72 mg/kg. Serum iron levels were drawn on 4 of the 6 patients. Mean peak serum iron concentrations were 82 mcg/dL, with a range of 36 to 177 mcg/dL. One child with a recent history of flu-like symptoms reported diarrhea, fever and lethargy and had a serum iron concentration of 36 mcg/dL. The symptoms were believed unrelated to the exposure. No other symptoms were reported in any patient. Two of these patients were suicide attempts with reported histories of large ingestions. **Conclusion:** This is the first report of iron carbonyl overdose. In this limited case series of iron carbonyl ingestion evidence of serious toxicity did not occur. Further study may be warranted.

### 120 HEPATITIS AND HYPERAMYLASEMIA CAUSED BY GOLD POTASSIUM CYANIDE.

Wu ML, Tsai WJ, Ger J, Deng JF. *Division of Clinical Toxicology, Department of Medicine, Veterans General Hospital-Taipei, Taiwan*

**Introduction:** Therapeutic gold salts are known to have variable adverse effects which include gastrointestinal (GI), dermatological, hematological, renal, neurological and cardiovascular involvement; whether the other gold salts have the same effect is still unknown. The toxicity of gold potassium cyanide is thought to be cyanide intoxication, however we present a unique case of gold potassium cyanide poisoning with presentations of GI upset, hyperamylasemia and hepatitis. **Case Report:** A 27-year-old man attempted suicide by ingesting 3-5 mL gold potassium cyanide solution. He developed vomiting and abdominal pain 3 hours later and was sent to a hospital nearby. After emergent decontamination, he was referred to our service for fear of cyanide intoxication and the unavailability of antidote. On arrival, the vital signs and respiration remained stable. The blood cyanide test was negative. The arterial blood gas was normal. Laboratory tests (13 hours postingestion) were unremarkable except hyperamylasemia (amylase 1566 U/L), hypokalemia (3.3 mEq/L), mild leukocytosis (WBC 10,600/mm<sup>3</sup>). Jaundice was noted on the second admission day. The laboratory tests (37 hours postingestion) revealed elevated aspartate aminotransaminase (273 U/L), aspartate aminotransaminase (149 U/L), alkaline phosphatase (117 U/L),  $\gamma$ -glutamyl transferase (224 U/L), lactate dehydrogenase (233 U/L), bilirubin (total 5.6 mg/dL, direct 3.5 mg/dL). The abdomen sonography was normal. The liver biopsy showed centrilobular cholestasis with eosinophilic infiltration. Marked elevation of gold level was documented later. The whole blood and serum gold were 4361, 6011  $\mu$ g/L respectively, and the 24 hour urine gold was 429  $\mu$ g/d. **Conclusion:** This patient demonstrated that gold potassium cyanide could result in significant systemic toxicity of gold. The mechanism is still not known, however it may be attributed to immune complex hypersensitivity reaction as in gold drug.

### 121 ARGYRIA SECONDARY TO CHRONIC INGESTION OF COLLOIDAL SILVER.

Chomchai S, Kim SY. *California Poison Control System, San Francisco. University of California at San Francisco, San Francisco, CA*

**Background:** Colloidal silver protein (CSP) has been used as a popular nutritional supplement for many years. CSP has been promoted to boost immune response and is claimed to be effective against many forms of infections. Although silver-containing drugs are no longer available for OTC sale, CSP is available in health food stores as well as over the Internet. We report a case of argyria due to chronic use of CSP. **Case Report:** A 35-year-old woman presented with bluish-gray discoloration of the skin and nail beds. The discoloration was more pronounced in sun exposed areas. The patient is a housewife with no hobbies involving chemicals or excessive outdoor activities. She has no underlying diseases and is on no medications except for a nutritional supplement called "mild colloidal silver protein" containing 25 mcg/tsp of elemental silver. The patient had been taking this product orally, in accordance with the labeled instruc-

tions, daily for one year. Laboratory studies, including liver and renal functions, are all normal. Heavy metal screens of urine were all negative, including silver, lead, mercury, and arsenic. Skin biopsy was not done. Based on the patient's history of CPS use, the total cumulative dose of silver before the onset of skin discoloration was 66 mg. **Discussion:** Chronic exposure to silver-containing products has been known to cause silver deposition in various organ systems including skin, liver, kidney, gastrointestinal tract, brain and cornea. Such deposition results in chronic skin discoloration, which is effectively irreversible and untreatable. Although argyria is thought to be a relatively benign condition, silver deposition has been shown to be associated with central nervous system dysfunction in animals. In humans, there is one case report of seizures in a patient who had been taking a silver-containing product for 40 years and had a very high serum silver level. Although argyria has been reported with chronic ingestion of colloidal silver products, the estimated accumulated amount of elemental silver consumed in our patient is low compared to previously reported cases. **Conclusion:** We report this case to illustrate the potential for permanent, disfiguring complications of a commonly used nutritional supplement product.

### 122 ARGYRIA IN THE EMERGENCY DEPARTMENT.

Newman M, Kolecki P. *Department of Emergency Medicine, Thomas Jefferson University Hospital, Philadelphia, PA*  
**Background:** The following is a report of argyria associated with the chronic ingestion of colloidal silver in an attempt to treat Lyme disease. **Case Report:** A 38-year-old previously healthy female presented to the emergency department because of a grayish discoloration to her face. Two years ago, the patient had a positive Lyme titer. After consulting homeopathic Internet web sites, the patient purchased colloidal silver touted as a natural alternative to antibiotics. She ingested approximately 1/4 of a cup of the solution 3 times a day for 8 months. The patient had no other complaints, denied any other medical problems, and denied taking any other medications. Physical examination revealed: T 98.9°F (PO), R 18, BP 146/105, P 100, pulse ox 100% (room air). Examination of the head and neck was normal except for a grayish discoloration of her face. Cardiac exam revealed a regular rate without murmurs, gallops, or rubs. Pulmonary examination revealed clear, equal breath sounds. The abdomen was soft without tenderness, masses, distention, rebound, rigidity, or guarding. The neurologic examination revealed no focal deficits. Laboratory values revealed a normal CBC, SMA<sub>7</sub>, and liver enzymes. An x-ray of the abdomen revealed no radio-opaque foreign bodies. The patient was diagnosed with argyria, and she was instructed to stop ingesting the colloidal silver. **Conclusion:** Colloidal silver is available for purchase via the Internet, health food stores, and pharmacies for the treatment of many diseases, all of which are without substantiation. Patients using silver products need to be aware of the potential dermatologic complication (argyria) that may result following chronic absorption.

### 123 THE COFFEE POT POISONINGS: A TALE OF THALLIUM AND ARSENIC POISONING IN A GROUP OF AUTOMOTIVE WORKERS.

Rusyniak D, Furbee B, Kirk M. *Indiana Poison Center, Indiana University School of Medicine, Clarian Health Partners, Indianapolis, IN*

**Objective:** We present a case series of ten automotive workers poisoned with thallium and arsenic, describing clinical presentation, lab results, EMG results, treatments, and outcomes. **Case Series:** Several workers at a local automotive plant began simultaneously complaining of myalgias, paresthesias, and dysesthesias. After hospitalization and testing of one worker revealed an elevated urine arsenic level, he along with eleven co-workers were referred to our service for further testing and treatment. Two patients were hospitalized secondary to the severity of their symptoms. Based on the similarity and severity of symptoms, 6 patients were empirically started on DMSA. Urine and hair analysis revealed 2 patients with arsenic and thallium poisoning, 8 with thallium poisoning alone, and 2 with no evidence of poisoning. After the confirmation of thallium, patients were taken off DMSA and as Prussian blue was unavailable, treated with multiple dose activated charcoal (50 grams bid) until 24 hour urine collections of thallium were less than 500 mcg/specimen. The source of exposure was traced to break room coffee pots, one showing elevated levels of arsenic and one based on epidemiologic data linked to thallium (pot was unavailable for testing). Clinical symptoms varied with the most common being myalgias 90%, arthralgias 90%, joint stiffness 70%, abdominal pain 70%, insomnia 70%, feet dysesthesias/paresthesias 60%, myoclonus 60%, fatigue 50%, alopecia 50%. EMG testing revealed sensorimotor axonopathies without conduction slowing in 7 of the 10 patients: three severe, two moderate and two mild. All of the patients improved with treatment. At this time, 8 have returned to work and 2 remain in physical therapy but with

improvement of symptoms. Conclusion: Thallium and arsenic poisoning should be considered in patients presenting with myalgias, arthralgias, paresthesias, insomnia, alopecia and EMG evidence of axonopathy.

#### 124 A DARKER SHADE OF PRUSSIAN BLUE: THE DIFFICULT QUEST FOR THE THALLIUM (TI) ANTIDOTE.

Senecal PE, Chalut D. *McGill University Health Centre, Montreal, PQ, Canada*

Background: Prussian blue (Merck Index: ferric ferrocyanide, ferric hexacyanoferrate) is often quoted in medical literature as *the* antidote for thallium poisoning. Unfortunately, uncertainty surrounds its name and exact composition. This can create confusion when it is urgent to locate Prussian blue for thallium, or even radiocesium, poisoning or contamination. Case Report: While attempting to get some Prussian blue, a literature review was conducted so as to verify its exact chemical nomenclature and composition. The search revealed several other names used for Prussian blue: 'Radio-Gardase'<sup>™</sup> (European trademark); 'ferrocin' (Russian literature); 'potassium ferri(c) hexacyanoferrate (II)'; and 'colloidal ferrihexacyanoferrate (II)'. There were dangerous Prussian blue name sound-alikes: 'ferrocins' (bacterial antibiotics of a new class), potassium ferrocyanide (i.e., potassium hexacyanoferrate II, yellow) and potassium ferricyanide (i.e., potassium hexacyanoferrate III, red), none of which is reputed to bind thallium. Moreover, the inorganic cyano-compound literature distinguishes a whole spectrum of Prussian blues based on their mode of synthesis, ferric/ferro ratio, water and alkali (sodium, potassium and ammonium) content, solubility and mesh size. Exact chemical differentiation of Prussian blue compounds may in fact need X-ray diffraction analysis of the crystal lattice. Conclusion: Identifying and obtaining a stable Prussian blue, certified for clinical use, from the whole family of Prussian blues, is challenging—at least at our location. If need be, the substitution of activated charcoal which is widely available and has known thallium-binding capacity, should be considered.

**Platform Session 4**

**Sunday, September 17  
Abstracts #125–#128**

**10:15 am–11:15 am**

#### 125 COMPARATIVE EFFICACY OF PRALIDOXIME VS SODIUM BICARBONATE IN RATS AND HUMANS SEVERELY POISONED WITH O-P PESTICIDE.

Wong A, Sandron CA, Magalhães AS, Rocha LCS. *CEATOX (São Paulo Regional Poison Center), Instituto da Criança, School of Medicine, University of São Paulo, São Paulo, Brazil*

Introduction: Poisoning, often lethal, with O-P insecticides is very common, especially in developing countries. Oximes, the classic antidotes, are very expensive, often unavailable, may be ineffective in serious O-P poisoning and have certain side effects. NaHCO<sub>3</sub>, when given to fully correct serum bicarbonate, has been shown to be effective in certain noncontrolled experimental studies. We tested the hypothesis in a comparative, controlled study in rats and present data of 22 human subjects treated with IV bicarbonate, 19 of whom survived. Methods: Five groups of ten male Wistar rats were given lethal doses (ip) of DDVP (18 mg/kg). The animals were previously anesthetized with ether and the right jugular vein was cannulated, then separated in treatment groups: **I**. No treatment; **II**. Atropine (17 mg/kg) only; **III**. Atropine + Pralidoxime (1 G/kg); **IV**. Atropine + NaHCO<sub>3</sub> (3 mEq/kg) and **V**. Atropine + NaCl 0.9% (1.9 mL/kg). Blood samples were obtained for blood gases and AChE activity at T<sub>1</sub> (before poisoning), T<sub>2</sub> (at time of maximal seizure or critical point-CP) and T<sub>3</sub> (time of death or 10 minutes after CP). All animals were beheaded for determination of brain AChE. Results: There were NO survivors in group I, 3 in group II, 4 in group III, 9 in group IV, and 5 in group V. There were no significant differences at T<sub>1</sub> among the groups; at T<sub>2</sub>, AChE was highest in group III and serum bicarbonate was highest in group IV. Serum AChE was not significantly greater in the survivors in any group. Brain

AChE was lowest in group III. **Conclusion:** Full correction of serum bicarbonate was the best treatment for severe O-P poisoning, serum AChE levels did not reflect survival. Volume expansion increased survival, but significantly less than bicarbonate. Survival using oximes was not significantly better than atropine alone, besides being more toxic.

### 126 NEW INDOL DERIVATIVE EFFECTIVELY RESTORES PHYSICAL WORK CAPACITY AFTER ACUTE ORGANOPHOSPHATE INSECTICIDE POISONING.

Spivakova RP, Tomchin AB, Tonkopi DV, Aksenov IV. *Department of Pharmacology, Military Medical Academy, St. Petersburg, Russia.*

**Introduction:** The aim of the investigation was to explore the influence of hydrochloride 3(3-morpholinopropyltio)-1,2,4,-triazino-5,6-b-indol (Substance 1) on restoration of the physical work capacity (PWC) after poisoning with organophosphate insecticides (OPI). **Methods:** Experiments were performed on rats. PWC of the rats was measured by the running test on a treadmill. Rats performed the running tests; after 3 days, when the initial PWC was measured, they were given malathion in doses that cause convulsions in 30 minutes. Investigated drugs: Atropine, Bemetil and Substance 1 were administered after 15 minutes from the start of convulsions. Control animals were given 0.9% solution of NaCl. PWC was tested 24, 48, 72 hours and 7 days from the time of poisoning. Investigated drugs were administered once a day. **Results:** The following table summarizes the results.

Time of Testing After Intoxication	Increase in Running Time (%) Compared to the First Test Before Intoxication, Which is Considered as 100%			
	Control	Atropine	Bemetil	Substance 1
24 hours	16	40	35	40*
48 hours	50	60	40	80*
72 hours	55	60	45	95*
7 days	75	66*	70*	110*

\* Difference from control ( $p \leq 0.05$ )

**Conclusions:** Substance 1 accelerates restoration of PWC after poisoning with OPI and more active than Atropine and Bemetil.

### 127 ACTOPROTECTOR ETOMERSOL IS EFFECTIVE FOR ACCELERATION OF RECOVERY AFTER MALATHION POISONING.

Aksenov IV, Tonkopi DV. *Department of Clinical Medicine, Saba University School of Medicine, Saba, Netherlands-Antilles*

**Objective:** Purpose of this investigation was to explore clinical efficiency of etomersol from a new class known as actoprotectors as a remedy for acceleration recovery after acute malathion poisoning. **Methods:** 34 cases of acute poisoning with malathion (20 men and 14 women) were explored. Patients were divided into control and experimental groups. All patients received regular treatment. Patients of experimental group received etomersol intramuscularly 2 mL of 4% solution 1-2 times in a day in addition to a "traditional" scheme of treatment. Control patients received the same amount of 0.9% NaCl. The investigation included clinical examination data, indices of physical work capacity, conditions of cardiovascular and respiratory systems, main biochemical indices, and emotional status. **Results:** The investigation showed that poisoning with malathion led to a significant decrease in physical work capacity, disturbance of the central nervous system, disorders of systemic blood circulation, hormonal imbalance, inhibition of the nonspecific defense and immune status, infringement of the biochemical homeostasis and activation of lipid peroxidation. Administration of etomersol promoted normalization of liver and kidney function, indices of lipid peroxidation, decreased the level of endotoxiosis, stimulated nonspecific resistance of the body and led to the early restoration of physical work capacity

(to 7th day in experimental group and to 10th day in control group). The obtained clinical data showed that administration of etomorsol allowed a reduction of the period of treatment and accelerated recovery. Conclusion: Etomorsol is an effective medication to accelerate recovery after acute poisoning with malathion.

### 128 A PROSPECTIVE STUDY OF ACUTE UNINTENTIONAL PEDIATRIC SUPERWARFARIN INGESTIONS WITHOUT DECONTAMINATION.

Ingels M, Lai C, Tai W, Rangan C, Williams SR, Manoguerra AS, Clark RF. *California Poison Control System, San Diego, Sacramento, and San Francisco, CA*

Objective: Published case series of superwarfarin rodenticide ingestions in children show a minimal incidence of clinically significant bleeding, but these studies are retrospective and do not account for gastric decontamination. We performed a prospective study of acute unintentional pediatric superwarfarin ingestions without decontamination. Methods: We prospectively studied patients reported to our poison center who were under age 6 years with acute unintentional superwarfarin ingestions. Patients who underwent GI decontamination or received prophylactic vitamin K were excluded. 48- to 96-hour PT-INR blood tests were recommended and phone contact was attempted at least 3 days postingestion. Results: 604 consecutive patients were enrolled in the study in a 16-month period. 45 were excluded (activated charcoal in 27, induced emesis in 16, and prophylactic vitamin K in 2) and 94 were lost to follow up. Follow-up was obtained on 465 patients: 222 by telephone contact, 39 by 48–96 hour INR, and 204 by both methods. None of the patients had clinically significant coagulopathy. 2 patients had an INR  $\geq 1.5$  (1.5 and 1.8) without symptoms. Single nosebleeds were reported in 2 patients who had normal 48-hour INRs. Another child had a small amount of blood crusted in the nose, with no other symptoms and no labs available. One child had blood-streaked stools, thought to be due to an anal fissure. His 48-hour INR was normal. Conclusion: Neither gastric decontamination nor laboratory evaluation is necessary in the management of acute unintentional superwarfarin ingestions in children.

## Poster Session 3

Monday, September 17  
Abstracts #129–#184

8:00 am–1:30 pm

### 129 REGIONAL VARIATION IN THE INCIDENCE OF INTENTIONAL EXPOSURES.

Sudakin DL. *Veterans Administration Medical Center, Portland, OR*

Background: Geographic information systems (GIS) are an epidemiological tool that can be utilized when spatial identifiers are linked to human health data. The purpose of this investigation was to utilize GIS and poison center data to evaluate regional variation in the incidence of intentional exposures within a single state. Methods: All intentional human exposure calls to the poison center were analyzed by county of origin utilizing ArcView GIS V3.1. Incidence rates by county were defined as the number of intentional human exposures per 10,000 population. Maps demonstrating variation in the incidence rates were constructed for analysis. Results: The overall number of intentional exposure calls by county was strongly associated with the population within the county ( $r = .86$ ,  $p < .01$ ). There was no significant association between incidence rates and population by county. When examined as rates (calls/10,000 population), there was considerable variation in the overall incidence of intentional exposure calls by county (mean 10, range 0–22). Counties with the highest overall incidence rates were mainly located in rural central and southern regions of the state. The incidence of suicidal exposure calls also varied by region (mean 7, range 0–13). Most counties with higher incidence rates were located in rural southern regions of the state. Less variation was observed in the regional incidence of intentional abuse/misuse exposures, although a consistent pattern of higher incidence was observed in counties without urbanized areas. Conclusions: The incidence of intentional exposure calls varied considerably by county, with higher incidence rates in nonurbanized, less-populated areas. GIS can be effectively utilized to evaluate regional variation in incidence.



### 130 POISONING WITH CHEMICAL COMPOUNDS AMONG ADULT INHABITANTS OF KRAKÓW IN 1998.

Targosz D, Pach K, Pach J. *Department of Clinical Toxicology, Department of Pediatric Nephrology, Collegium Medicum Jagiellonian University, Kraków, Poland*

**Objective:** In Kraków all sick above 14 years of age with a stated or suspected poisoning with chemical compounds are treated at the Department of Clinical Toxicology. This enables a precise recording of all the treated cases of acute poisonings. The permanent co-operation over many years with the Department of Forensic Medicine of Jagiellonian University enables recording of all persons who died at the place of accident prior to any treatment, so a full epidemiologic analysis of acute poisonings among adult Kraków residents can be done. The aim of this study was to evaluate the frequency, kind, and structure of acute poisonings which occurred among Kraków adult inhabitants in year 1998. **Methods:** Under analysis there were 3236 people treated at the Department of Clinical Toxicology and 108 poisoned who died at the scene before any treatment was conducted. The group of hospitalized persons consisted of 2093 (64.7%) men and 1143 (35.31%) women, and the group of people who died at the place of accident consisted of 92 (85.2%) men and of 16 (14.8%) women. **Results:** The general incidence rate of poisonings per 10 000 inhabitants in year 1998 was 43.7; 60.2 for men and 29.1 for women. Ethanol (38.8%) followed by drugs (21.3%), drugs + ethanol (6.2%), carbon monoxide (4.7%), and narcotics (3.8%) were the most common causes of acute poisonings. 37% of all poisonings treated at the Department in 1998 involved abusers intoxicated with ethanol and narcotics (mostly opiate derivatives). A significant number of poisonings were caused by narcotics and drugs simultaneously. The overall mortality rate of patients treated at the Department of Clinical Toxicology was low (0.46%), but after including those who died at the scene, the mortality rate due to poisoning rose from 0.46 to 3.55%. That increase in the mortality rate was caused mainly by fatal cases due to ethanol, carbon monoxide and drug poisoning.

### 131 INCIDENCE OF HOMICIDE BY POISONING AS IDENTIFIED BY MEDICAL EXAMINERS' OFFICES IN THE UNITED STATES.

Snowman P, Greene T, Greenberg MI, Dougherty T. *MCP-HUH, Philadelphia, PA*

**Background:** Over 25,000 homicides are reported annually in the United States (US). It is unknown what percentage of these homicides are due to poisonous substances although it is assumed that homicide by poisoning (HBP) is an uncommon event. This study provides a current update regarding the incidence of HBP in the US. **Methods:** Data was gathered via telephone survey of official Offices of Medical Examiners (OMEs) in 10 regions of the US. Each OME contacted was questioned regarding the incidence of documented HBP in their region during the previous 10 years. Survey questions identified specific substances involved in homicide cases as well as OME laboratory testing for specific substances of homicidal intent. **Results:** Average OME cases/site was 3500 (n = 1000-7000) with an average of 1600 (n = 500-2500) autopsies (with toxicology tests)/OME site annually. The average number of cases of HBP within the past decade was 0.7/region. This results in an incidence rate of 2 HBP/100,000 OME deaths. Specific substances identified in HBP included: arsenic, cocaine, cyanide, pancuronium, paraquat, and thallium. Toxicology testing for "homicidal substances" is not undertaken on a routine basis by all OMEs. None of the OMEs surveyed routinely tested for heavy metals, strychnine, digoxin, nondepolarizing muscle relaxants, or paraquat. Additionally, while many OMEs utilize computers resources for testing and data compilation, some OMEs use manual resources exclusively. **Conclusions:** This study validates earlier assumptions that HBP is a relatively uncommon event. Significant variability in OME procedures makes regional comparisons difficult. The complete lack of standardization of procedures nationwide may result in under-detection and under-reporting of HBP.

### 132 INFLUENCE OF CERTIFIED POISON CONTROL CENTERS ON STATE POISONING MORTALITY RATES IN THE UNITED STATES.

Chyka PA, Somes GW. *Southern Poison Center, The University of Tennessee, Memphis, TN*

**Objective:** To compare poisoning mortality rates of states that are served by a poison control center certified by the American Association of Poison Control Centers (AAPCC) to those that are not served by a certified center. **Methods:** Poisoning mortality rates for 1993 to 1997 were obtained from a public use database of US death certificates (WISQARS) maintained by the Centers for Disease Control and Prevention. Data were stratified by state and circumstance. Each state and the District of Columbia were classified as being fully served (FC), partially served (PC) or not served (NC) by an AAPCC-certified center. Partial service was defined as at least one certified and one noncertified center serving

the same state. States that were consistently in one category of service were selected for analysis. **Results:** During this 5-year period 39 states had consistent FC (N = 17), PC (N = 7), or NC (N = 15) poison control center services. The crude mortality rates per 100,000 population for each of these 5 years were 5.9, 6.1, 6.0, 6.2, and 6.6, respectively ( $p < 0.05$ ). The 5-year mortality rate for FC states (7.1) was higher ( $p < 0.05$ ) than those for PC (6.2) or NC (5.2) states. Changes in 5-year mortality rates (slope of rate versus year) indicated increasing mortality ( $p < 0.05$ ) in FC states (0.21) compared to nonsignificant changes in NC (0.18) or PC (0.08) states. There was no change in intentional poisoning rates for FC (-0.04), PC (-0.03) or NC (-0.06) states. Unintentional poisoning rates increased ( $p < 0.05$ ) for FC states (0.18); whereas those for PC (0.10) or NC (0.13) states did not significantly change. **Conclusion:** Increased poisoning mortality rates were associated with AAPCC certification status and year. Poisoning mortality rates may not be appropriate to serve as a valid outcome measure of the impact of AAPCC-certified poison control centers.

### 133 THE USE OF FOCUS GROUPS TO PLAN POISON PREVENTION PROGRAMS.

Schwartz L, Howland MA, Mercurio-Zappala M, Hoffman R. *New York City Poison Control Center, New York City Department of Health, St. John's University, New York, NY*

**Background:** Children under the age of five are at highest risk for potential poisoning exposures in the home. General poison prevention programs may not adequately address the concerns of patients in targeted socioeconomic populations. A review of poison prevention literature produced few programs developed using a focus group approach to define pertinent issues. **Methods:** Two focus groups were held with members from a convenience sample selected at the Women, Infants, and Children (WIC) program in a large urban public hospital. **Results:** The focus groups' participants were low-income parents of young children enrolled in the WIC program (N = 20). The age of participants ranged from 17 to 44 years old with a mean of 29 years old. The number of children of participants ranged from 1 to 4 (mean of 2) and the children's ages ranged from 13 months to 26 years old. None of the group members had ever called the poison center, although 35% (n = 7) had the poison center number posted on the telephone. Barriers for calling poison centers included statements that participants would be more likely to call 911 after a potential poisoning exposure. In addition, participants felt that they would be blamed by city child protective services agencies if something happened to the child. Respondents also stated that they felt more comfortable with assistance in person rather than over the telephone. **Conclusion:** Qualitative methods are useful for planning future poison prevention programs to overcome barriers to calling the poison center. Programs for parents of young children should focus on education about calling the poison center prior to 911 when appropriate, issues related to documentation and confidentiality of poison center calls, and self-confidence for home management of potential poison exposures.

### 134 THE RIGHT CALL FOR POISON HELP.

Stone E, Pope L. *San Francisco Division, Fresno/Madera Division, California Poison Control System, San Francisco, CA*

**Background:** The mission of our education program is to reduce the frequency of childhood (<6 years) poisoning exposures in low-income families and to increase their appropriate utilization of poison control services. Childcare providers are enthusiastic about an educational program targeting parents. **Methods:** An English/Spanish training program for use by childcare providers and educators with parents/caretakers was piloted at 17 Head Start, State Preschool, Women, Infant Children (WIC) programs. The self-contained kit included videos, teacher guides, parent handouts, telephone stickers, and evaluation forms. The program reached 1449 people in 55 classes, 52% of whom were Spanish speaking. Evaluation components included: written forms for instructors and participants, telephone call backs to 138 participants with more detailed questions, and PCC education staff observation. **Results:** 80% of participants ranked the total program and the video as very good and 11%, above average. 77% of participants ranked the handout (low literacy and mainly pictorial) as very good. Only 44% of total participants (less of the Spanish speaking) knew the "hotline" number before the class. Afterwards 69% of those not previously knowing the phone number now had it on their phones. In addition, many parents reported making significant changes regarding poison prevention in their home environments. **Conclusion:** In this intervention group, less than half of the parents had been aware of the "hotline" number. The pilot program showed that a half-hour class can make a significant difference in poison awareness and prevention action. Based on the evaluation analysis, appropriate changes were made in the materials. Revised kits are being duplicated and distributed free of charge to 1300 sites of these three agencies in summer 2000.

**135 FOLLOW UP STUDY WITH PARENTS GIVEN CABINET SAFETY LOCKS.**

Schwartz L, Howland MA, Mercurio-Zappala M, Hoffman RS. *New York City Poison Control Center, New York City Department of Health, St. John's University, New York, NY*

**Background:** Poison prevention often includes using cabinet safety locks to deter young children from accessing household toxins. Few poison prevention programs report parents' use of cabinet safety locks. **Methods:** Poison prevention kits containing telephone stickers, brochures, and a slide cabinet safety lock were distributed to parents enrolled in a Women, Infants, and Children (WIC) program. Poison education was also provided to participants. Telephone follow up calls were made approximately four weeks later to determine if the cabinet safety lock was used, in which room, reasons for not using the lock, and if the poison control center's telephone number was posted. **Results:** Preliminary data of an ongoing study (n = 21) showed that 62% (13) of participants reported that the safety lock was installed in the home. The locks were usually used in the kitchen (85%, n = 11). The respondents (n = 8) who did not use the lock cited reasons that the lock did not fit the cabinets in the home (n = 4), have gates blocking entry to the kitchen (n = 1), didn't know how to install (n = 1), products are kept out of reach (n = 1), and one didn't remember receiving the lock. Twenty respondents reported at least one child in the home under the age of five years old. The majority of respondents (76%) stated that they knew the poison center telephone number and 71% (n = 15) of respondents had the poison center telephone number posted on or near the telephone. **Conclusion:** Parents will usually use cabinet safety locks when distributed if the lock fits the cabinets in the home. Distributing a variety of types of locks may accommodate a wider range of cabinets and improve compliance. Poison prevention programs should continue to focus on the importance of using cabinet safety locks and posting the poison center's telephone number in the home.

**136 ROLE OF A POISON CENTER IN PREVENTING SEVERE POISONING: INDUSTRIAL AUTOMATIC DISHWASHING DETERGENTS AS A CASE.**

Ballesteros S, Martinez-Arrieta R, Ramón MF, Torrecilla JM. *Spanish Poison Center. Servicio de Información Toxicológica, Instituto Nacional de Toxicología. Madrid, Spain*

**Background:** Industrial automatic dishwashing machine detergents are strongly alkaline usually transparent liquids intended for professional use. An unusual number of calls due to these products were detected in our Poison Control Center. The aim of this study was to describe the situation of exposure and the preventive measures undertaken as well as the first results notified. **Methods:** All human exposures to these compounds reported to our PCC from January 1995 to December 1999 were collected. Data including age, route, clinical features, outcome, etc. were analyzed. **Results:** A total of 377 cases were reported in the studied period. The most common route of entry was ingestion (81.7%). Oral accidental exposures occurred in 81% of cases, and suicide attempts only in 0.5%. The site of exposure was a residence in 194 cases, 114 occurred in small restaurants, bars or hotels. Peak call volumes were noted at mealtime in restaurants and food services with a weekend predominance. Seventy-eight percent of reported exposures resulted in a moderate or major effect. As a result of these data in 1997 some enterprises advised by our PCC voluntarily introduced a dye into their detergents. A law was introduced in May, 1999 in which the addition of a dye was compulsory, in order to avoid confusion with water, among other requests. Even when the law required 12 months to be implemented a decrease in the incident of exposures was observed afterwards: From 1997 to 1999 a 50% decrease was observed for oral exposures specially of the severe cases, and a 83.3% decrease in children less than 2 years. **Conclusion:** Some of the tasks of a poison center include identifying hazards early, focus prevention measures, and supporting regulatory actions. Detection of a severe and frequent poisoning, analysis of the circumstances and implementation of the appropriate measures, were the main steps undertaken in order to avoid accidents with professional dishwashing detergents.

**137 CHILDREN'S ENVIRONMENTAL HEALTH: ONE YEAR IN A PEDIATRIC ENVIRONMENTAL HEALTH CLINIC (PEHC).**

Shannon M, Woolf A, Goldman R. *Children's Hospital, Harvard Medical School, Cambridge Hospital; Massachusetts/Rhode Island Poison Control System, Boston, MA*

**Background/Objective:** An increasing desire for clinical centers which can evaluate children exposed to environmental toxins has led to the creation of a network of PEHCs. This descriptive study profiles the children seen in such a program. **Setting:** A New England, university-affiliated PEHC. **Methods:** Review of all children seen in the PEHC in 1999. Outcome measures of interest were demographic factors, chief complaint and management. **Results:** Over the study interval there were 857 visits to the PEHC. Children fell into 4 groups: new visit for lead poisoning (Group I, n =

148), return visit for lead poisoning (Group II, n = 663), new visit, exposure to a nonlead toxin (Group III, n = 31) and return visit for a nonlead toxin (Group IV, n = 15). Among Group III the most common chief complaints were: exposure to solvent-contaminated water (n = 7, mean age 5 years), pesticide exposure (n = 6, mean age 6 years.), illness associated with proximity to a hazardous waste site (n = 5, mean age 3 years), autism, r/o mercury poisoning (n = 4, mean age 5 years.), and sick school syndrome (n = 4, mean age 11 years.). Families traveled as far as 450 km for assessment. Every child was evaluated by a pediatrician/medical toxicologist. Therapeutic interventions consisted of chelation therapy, relocation, removal from school and termination of chelation therapy (n = 2, both with autism). All visits were reimbursed fully by third party payors. **Conclusions:** Based on this program's annual census, there appears to be a substantial need for PEHCs. Chief complaints are diverse. Parents and physicians desiring such an evaluation are willing to travel extensive distances, suggesting the need for a broader network of such centers. HMOs and third party payors have provided appropriate compensation for these toxicologic services. (Supported by a grant from ATSDR and the AOEC)

### 138 A MEDIA AND LEGISLATIVE CAMPAIGN TO FUND POISON CONTROL CENTERS.

Heard S, Pope L. *California Poison Control System, University of California at San Francisco, San Francisco, CA*  
**Background:** Unstable funding is a well-known problem among poison control centers across the country. Until recently, the California Poison Control System (CPCS) contract with the State of California provided only 20% of our budget. The Governor expected the private health care industry to voluntarily fund the balance. By this time, we had exhausted the initial seed funding from a federal Medicaid matching program and failed to achieve voluntary funding from the health insurance industry as requested by the previous Governor. Our survival instincts ultimately launched a dual strategy-marketing plan. **Methods:** A member of the state Legislature agreed to carry a bill imposing a fee on health plans to fund poison control services. The Budget Committees of both houses of the legislature agreed to hear a concurrent appeal for increased General Fund (taxpayer) subsidy. The services of a pro-bono professional lobbyist and a paid media consulting firm were also enlisted. The legislative member advanced the bill in the Health Committee while PCC staff testified in the Budget Committee hearings and he conducted a press conference to alert California residents (34 million) that the CPCS was in financial jeopardy. The media firm was involved in developing information materials, contacting spokespersons, organizing a Coalition of grass root supporters of CPCS, and newspaper editorial boards. They published Editorials and "Letters to the Editor" in major newspapers throughout the state. **Results:** The legislative bill met with strong opposition by the Health Committees. However they were influential in convincing the Budget Committees to vote for augmentation to the poison service budget. The media presence was influential on the newly elected Governor, who ultimately approved the Budget augmentation. **Conclusion:** Our multi-strategy effort utilizing a professionally managed media campaign combined with high profile legislative activities proved effective.

### 139 AN EXPANDING RELATIONSHIP: EMERGENCY MEDICAL SERVICES AND POISON CENTER COOPERATION.

Haynes JF, Jr, Loflin JR, Artalejo L, Lugo AM. *West Texas Regional Poison Center, Texas Tech Health Science Center—El Paso, Department of Emergency Medicine, Division of Emergency Medical Service, El Paso, TX*  
**Background:** There are few formal models for interaction between Poison Centers (PCC) and Emergency Medical Service (EMS). When established, such cooperative agreements may result in substantial medical cost savings. We established a formal relationship between our poison center and El Paso City EMS (serving a metro area of >700,000). Our agreement provides for direct communication between the EMS dispatcher, the PCC-SPI, and the caller resulting in a three way conversation. We also established direct radio communication between EMS field personnel and our SPI. This system makes transport and treatment decisions on exposure calls. Cases determined safe for home management were not transported and their care was transferred to the PCC. **Methods:** We reviewed 4 years of data. We determined cost savings, case severity, program satisfaction, and future directions. **Results:** There were 1420 exposure calls: (1996) 371, (1997) 440, (1998) 302, (1999) 307. There were 610 (43%) "no transport" cases: (1996) 182, (1997) 136, (1998) 138, (1999) 154. Costs were determined as \$350 for basic EMS transport, and \$305 for basic Emergency Department charges (ED + MD). "No transport" cases saved an estimated \$399,550 over the 4 years. We found no adverse outcomes from these cases. On site radio communication resulted in both upgrading and downgrading of call severity. **Conclusion:** We confirmed previous experience of cost savings. Radio communication provided an extra margin

of safety for our patient population. EMS experienced a decrease in unnecessary use and the PCC had increased performance of service. Expansion of this mutually beneficial program is cost effective.

#### 140 UNNECESSARY EMS DISPATCH—WHAT'S THE COST?

Herrington L. *Georgia Poison Center, Atlanta, GA*

**Background:** One function of poison centers is to maximize the use of health care resources while decreasing unnecessary health care costs. This could include utilization of EMS resources. The purpose of this study is to determine the incidence and financial impact of unnecessary EMS dispatch for subtoxic poisoning exposures. **Methods:** All exposure calls to the PC from EMS where ambulance dispatch was made prior to the PC call were identified at the time of the initial call and coded accordingly. **Results:** This regional PC handled 76,470 human exposure calls from 1/1/1999–12/10/1999, of which 1692 (2.2%) were initiated by EMS. Of the EMS calls, 418 (24.7%) were placed to the PC after an ambulance had been dispatched. Where ambulance dispatch occurred prior to the PC call, 150 of these cases (36%) were left at home. The substances involved included: 53 (35.3%) OTC or prescription medicinals; 42 (27.3%) household, cleaning or laundry products; 17 (11.3%) personal use or cosmetic products. 13 patients had no known or expected symptoms; 11 had known minor effects that resolved within 2–8 hours. The remainder had only minimal expected symptoms, though outcome was not confirmed. Assuming 80% of cases follow PC recommendations, had the PC been consulted prior to ambulance dispatch, at an average cost of \$390 per ambulance dispatched an estimated annual savings of \$46,800 could have been realized in addition to helping to maximize utilization of EMS resources. **Conclusion:** Where transport times are not adversely effected, PC consultation by EMS prior to ambulance dispatch can help contain health care costs and maximize utilization of EMS resources.

#### 141 FACTORS INFLUENCING POISON CENTER SEND-IN RATES.

Alsop J, Marquardt K, Lamb J. *California Poison Control System—Sacramento Division, Sacramento, CA*

**Background:** In California (CA), the clinical expertise of the poison center staff is one of the biggest factors in poison center send-in rates: the more experienced the staff, the fewer cases sent in for evaluation. Other factors thought to influence send-in rates include AAPCC certification, written send-in guidelines, call volume, number of FTEs, and the type of staffing mix. **Methods:** In CA, a mix of pharmacists, nurses and providers and use of an extensive send-in guideline resulted in a 4.6% send-in rate. All poison centers were surveyed to compare send-in rates and to determine what factors influence the send-in rate. Call volume, number of cases referred to a HCF, and type and number of staffing were obtained, along with availability of written send-in guidelines. **Results:** 29 poison centers responded to the survey, of which 22 were certified centers. The average send-in rate was 13.2% (range 4.7% to 37.5%). Noncertified centers had an average send-in rate almost double that of certified centers (24.2% vs 12.9%). Sites with both the highest and lowest call volume had higher than average send-in rates (23.5% and 37.5% respectively). Sites that were not adequately staffed or that did not staff with licensed professionals had the highest send-in rates (37.5% and 33.9% respectively). Sites with written send-in guidelines had an average send-in rate of 13.8% while the average send-in rate for sites without guidelines was 15.3%. Sites staffed with any number of pharmacists had an average send-in rate of 11.3% while sites without pharmacists had an average send-in rate of 18.1%. Sites with providers had an average send-in rate of 12.7% while sites without providers had an average send-in rate of 14.2%. **Conclusion:** AAPCC certification, adequate staffing, and utilization of licensed professionals, including pharmacists, in the staffing mix appear to be the main factors in decreasing poison center send-in rates.

#### 142 CONSUMER ACCESS TO ACTIVATED CHARCOAL PRODUCTS.

Rose R, Waring E. *Virginia Poison Center, Medical College of Virginia Hospitals at Virginia Commonwealth University, Richmond, VA*

**Background:** Activated charcoal (AC) is now marketed for consumer use. Some poison centers recommend that AC be included in home first aid kits, and/or administered by prehospital providers. We surveyed retail pharmacies in diverse geographical areas within our center's service region to assess whether AC was available for purchase. **Methods:** Staff poison specialists (all RN) administered a six-item questionnaire by telephone to 53 retail pharmacies throughout the center's region; divided by locality as either rural, urban or suburban. Respondents were either a pharmacist (87%) or the store manager (13%). Respondents were specifically told to distinguish AC products intended for use in poisonings. Stores considered to be primary pharmacies or supermarkets with a pharmacy were included. **Results:** Eleven

(21%) of the 53 stores surveyed had AC available for public purchase. Pharmacies in urban areas were least likely (13%) to stock AC products. The AC was located on a store shelf in 9 of the 11 stores that carried it, and 2 stores kept the AC behind the pharmacy counter only. One store stocked the AC both on the shelf and in the pharmacy. None of the pharmacies surveyed provided any printed materials describing the use of AC. Only two pharmacists recalled selling AC in the three months prior to the survey. **Conclusions:** AC is not widely available to consumers in our poison center's region. Patient education materials that discuss the use of AC are nonexistent in the retail outlets surveyed. Pharmacists have limited experience in dispensing AC, and may not be familiar with information on correct use. Poison centers must work with retail pharmacies to increase availability and knowledge of AC prior to advocating its use by consumers in cases of accidental poisoning.

#### 143 PEDIATRIC HEALTH CARE PROVIDERS' ADVICE ABOUT IPECAC SYRUP—WHAT ARE THEY DOING AND HAS IT CHANGED?

Liebelt E, Woolf A. *Department of Pediatrics, Johns Hopkins School of Medicine, Baltimore, MD; Children's Hospital, Boston, MA*

**Background:** Emerging research and practice guidelines have generated discussion about the utility of ipecac syrup as a beneficial GI decontaminant. **Objective:** To describe pediatric health care providers' (PHCP) current practice about advising the home storage and use of ipecac syrup in children with potentially toxic ingestions. **Methods:** Cross-sectional survey of PHCPs which asks specific questions about their practices on advising caregivers to store and use ipecac syrup. Three scenarios were presented involving an unintentional ingestion by a young child, asking the PHCP whether they would advise the caregiver to give ipecac at home. **Results:** 271 surveys were distributed at postgraduate courses for PHCPs. 144 surveys were completed (53% return rate). Only 41% (n = 59) *always* advise to store ipecac at home while 8% (n = 11) *never* advise to store ipecac. 25% (n = 36) *never* or *seldom* explain when/how to use ipecac. If a child ingested a toxic dose of iron, 70% (n = 101) would advise to give ipecac at home before going to the hospital or calling the poison center; 66% (n = 95) would advise for a toxic dose of acetaminophen; and 51% (n = 72) would advise for one unknown mushroom. 21% reported that they advise home storage of ipecac *less* than they did 5 years ago and 21% reported that they advise it *more*. 63% (n = 89) were physicians. Significantly more physicians *never* or *seldom* advise to store ipecac at home (20%) compared to nonphysicians (8%),  $p = 0.01$ . Significantly more physicians have changed their practice in the last 5 years on advising home availability of ipecac *less* (31%) compared to nonphysicians (6%),  $p < 0.01$ . There was no significant differences in responses based on years in practice or age. **Conclusion:** PHCPs are not routinely advising storage and use of ipecac syrup. Physicians' practices have significantly changed in the last 5 years compared to nonphysicians.

#### 144 AVAILABILITY OF ACTIVATED CHARCOAL IN THE METROPOLITAN AREA OF OKLAHOMA CITY.

McGoodwin L, Schaeffer S. *Oklahoma Poison Control Center, University of Oklahoma College of Pharmacy, Oklahoma City, OK*

**Background:** Prompt use of activated charcoal for mild poisoning exposures can prevent the need for costly Emergency Department referrals and offers several advantages over home use of ipecac in selected exposures. Callers to our center have reported difficulty in locating activated charcoal in slurry or powdered formulations, and in some cases, they have inappropriately purchased activated charcoal in tablets or capsule formulations. A telephone survey of area pharmacies was utilized to determine the availability of activated charcoal in the metropolitan area of Oklahoma City. **Methods:** All pharmacies in the metropolitan area were surveyed by telephone. Pharmacists were asked to report if their current pharmacy stock included any form of activated charcoal as well as to indicate the type of formulation. **Results:** All 200 pharmacies responded to the survey. Only 23.5% of all pharmacies in the metropolitan area of Oklahoma City reportedly stocked an appropriate formulation of activated charcoal for use in poisoned patients. Activated charcoal tablets or capsules alone were reportedly stocked by 39% of pharmacies in this survey, while 37.5% of the pharmacies stocked no type of activated charcoal formulation. **Conclusions:** In most instances, the correct formulation of activated charcoal for the home treatment of the acutely poisoned patient is unavailable in the metropolitan pharmacies in Okla-

homa City. More extensive professional programs are needed to educate Oklahoma pharmacists of the importance of stocking and using the correct formulation of activated charcoal appropriately for poisoned patients.

#### **145 AVAILABILITY OF ANTIDOTES AT ACUTE CARE HOSPITALS IN ONTARIO.**

Juurlink DN, McGuigan MA, Paton TW, Redelmeier DA. *Departments of Clinical Pharmacology and Epidemiology, University of Toronto, Toronto, Canada*

**Background:** Several authors have documented poor antidote availability at hospitals in the United States. We sought to determine the availability of ten antidotes at hospitals throughout Ontario and explore hospital characteristics related to stocking. **Design:** A mailed survey was sent to the pharmacy directors of all acute care hospitals in Ontario (n = 184). Survey results were linked to hospital characteristics using multiple administrative databases. We defined an adequate supply as a sufficient amount of antidote to initiate treatment for a single adult poisoning. **Results:** After repeated reminders, we obtained replies from 179 hospitals (97%). Only one site reported an adequate supply of all ten antidotes. Adequacy rates were greatest for flumazenil (92%) and poorest for digoxin immune Fab (9%). In the univariate analysis, teaching hospital status, annual emergency department volume and designation as a regional trauma center were associated with better stocking, while small hospital status and greater distance from the nearest neighboring hospital were associated with poorer stocking. No association was found between antidote stocking and 30-day myocardial infarction survival rates. In the multivariate analysis, annual emergency department volume (p < 0.001), small hospital status (p = 0.002), and designation as a regional trauma center (p = 0.046) were independently predictive of antidote supply. **Conclusion:** Many Ontario hospitals stock insufficient amounts of several antidotes to initiate treatment for even a single poisoned patient.

#### **146 HAVE THE ANTIDOTE STOCKING PRACTICES OF HOSPITALS IMPROVED?**

Manney F, Geller RJ, Tarantino ML, Parramore CS. *Georgia Poison Center and Emory University Department of Pediatrics, Atlanta, GA*

**Background:** Several studies have demonstrated suboptimal antidote stocking by hospitals (HCF). Since 1996, our state has hosted the Centennial Olympic games and had several well-publicized bioterrorism hoaxes, resulting in attempts to improve its ability to handle such events. We sought to learn whether the antidote stocking practices by HCF in our state have changed. **Methods:** We had previously surveyed all HCF in our state about their patient care capabilities and antidote stocking practices (for activated charcoal, antivenins, heavy metal chelators, ethanol, pralidoxime, *n*-acetylcysteine, naloxone, and rabies biologicals). After categorizing the HCF by capability into 4 groups, we surveyed every third HCF in the list by telephone. Nonresponders were replaced with the next HCF on the list that responded. Complete data was gathered on 53 HCF, with a similar proportion of HCF from each category participating. Each HCF's response was compared to its own early 1996 response. Statistical significance was determined using the paired *t*-test. **Results:** Of 17 tabulated agents, the average HCF now had  $12.5 \pm 2.5$  agents, compared with  $11.6 \pm 2.9$  antidotes in early 1996 (p = 0.043, paired *t*-test, two tailed). There was no statistically significant trend in any individual group, but the power to detect small differences was poor due to the limited size of each group. **Conclusions:** Georgia HCF have improved their stocking practices of antidotes by a small but significant amount. Nonetheless, as we did not study the quantity each HCF stocks, it remains likely that no individual HCF could handle large numbers of simultaneous poisoned victims.

#### **147 THE IMPACT OF AN EDUCATIONAL INTERVENTION ON DECREASING DRUG INTERACTIONS IN THE EMERGENCY DEPARTMENT.**

Benson BE, Cenicerros O, Ritch LJ, Vo C, McKinney PE. *New Mexico Poison and Drug Information Center, University of New Mexico, Albuquerque, NM*

**Introduction:** It is estimated that 20% to 40% of patients presenting to the emergency department are discharged with medications that either interact with each other or with a prior medical condition. There are no data published detailing the effect of an educational intervention on reducing drug-drug and drug-disease interactions in the emergency department. **Methods:** We prospectively assessed patients for potential drug-drug and drug-disease interactions for four weeks using a computerized drug interaction program (DrugReax<sup>®</sup>, Micromedex), before and after they were examined by a physician. Patients were excluded if they presented between 1AM and 8AM, were admitted, took no medications, or

had incomplete medication histories. Halfway through the study period, emergency department physicians attended a mandatory inservice on the detection and prevention of drug interactions that included a hands-on demonstration of the computerized department drug interaction software. **Results:** Of the 1319 patients enrolled in the study, 391 were excluded, leaving a final population of 928 patients. The final population had a mean age of 35 years ( $\pm 16$  years), and took an average of 0.5 medications. The overall incidence of drug interactions (drug-drug and drug-disease) was 16%. The number of drug interactions did not increase after the patient was seen by a physician. The median number of interactions in the pre-education phase (2.4 interactions, 1.8–3.0 95% CI) did not differ significantly with the posteducation phase (2.8 interactions, 2.2–3.4 95% CI) ( $P = 0.4$ , Mann Whitney) **Conclusions:** A physician-directed educational intervention did not reduce drug-drug and drug-disease interactions in an our emergency department population.

#### 148 “DRUG IMPRINTING” AND IDENTIFICATION WOES.

Marder S, Winkler T, Tadaki K, Bobbink S, Robertson WO. *Washington Poison Center, Seattle, WA*

**Background:** “Drug Imprinting” was first implemented by Eli Lilly and Company in 1962 for all its products. The state of Washington mandated the use of imprints on all prescription drugs in 1980 and for “OTCs” in 1991. The FDA implemented federal requirements in 1995. Unfortunately, the FDA permitted the continued use of symbols, logotypes, and trademarks as code components, limiting the use of automated recognition systems, which we demonstrated in both 1992 and 1995. In December 1999, we sought a measure of our success in responding to phone inquiries seeking decoding of imprints. **Method:** Analyses of several week-long samples of phone inquiries were conducted, documenting imprinting calls, the staff’s ability to respond with an identification, the information sources used, and the apparent reasons for any failure. **Results:** Surprisingly, in our first week’s sample, we received a total of 666 decoding requests, which—projected for the year—amounted to more than 25,000 annual calls. A review of 1999 total data confirmed we had exceeded that number. Staff was able to reach an identification in 93.8%. Both uninterpretable symbols and absence of code listings contributed to the 36 failures. **Conclusion:** While we were pleased to achieve a greater than 90% success rate, we were overwhelmed by the number of inquiries and repeated our study on several subsequent week samples. Projecting our findings and our penetrance to the rest of the United States would suggest as many as 1.25 million possible calls costing poison centers as much as \$25 million per year. Using either a touch-tone telephone or website response system could permit a totally automated response with enormous reduction in human costs. Neither solution is feasible absent elimination of symbols or logotypes, so as to permit an exclusively alpha-numeric code. Efforts are underway to achieve same.

#### 149 THE INTERNET: SOMETIMES HELPFUL, SOMETIMES NOT.

Finke D, Roll D, Sunshin M, Simone K. *Cincinnati Drug & Poison Information Center (DPIC), Cincinnati Children’s Hospital Medical Center, University of Cincinnati, Cincinnati, OH*

**Background:** Even the most novice computer user can disseminate opinion as well as fact over the Internet. Poison control center staff can use the Internet to keep abreast of current trends and statistics, and act as a gatekeeper for the dangerous misinformation obtained by callers. **Methods:** Some of the websites significantly contributing to the poisoning of callers and used by center staff for ingredients, identification or other information are listed. **Results:** A poor website contributed to cyanide toxicity in a 71-year-old female who purchased B-17 (laetrile) and apricot pits, which she ground and ingested ([www.christianbrothers.com](http://www.christianbrothers.com)). In another case, severe CNS depression occurred in a 20-year-old male who purchased and deliberately abused 1,4-butanediol ([www.jlfcatalog.com](http://www.jlfcatalog.com)). Gamma-hydroxybutyrate analogue identification (<http://www.witchy-woman.com/CA/dream.html>, <http://www6.ios.com/~knpo5719/revive.html>) and dextromethorphan abuse information (<http://third-plateau.lycaum.org/beginner/index.html>) are used for case management and abuse trend insight. More generally helpful websites provide quick information about: new drug approvals, drugs in the news and drug manufacturer websites ([www.pharmacology.tqn.com](http://www.pharmacology.tqn.com)); recalls of drugs, foods and household products ([www.safetyalerts.com](http://www.safetyalerts.com)); and reverse look-up of addresses from phone numbers ([www.anywho.com](http://www.anywho.com)). The [www.anywho.com](http://www.anywho.com) site was used to send a life squad to the home of a confused, elderly male whose granddaughter overdosed on clonidine. **Conclusion:** The proficiency with which the Cincinnati DPIC staff accesses Internet information has proven invaluable in dozens of instances. The Center has provided needed drug information, substance identification, education as to the dangers of drugs of abuse and herbal products, and recalls.



**150 INTRANET SYSTEM—A VALUABLE TOOL.**

Kell S, Oneida B, Thompson J, Holstege C. *Blue Ridge Poison Center, University of Virginia Health System, Charlottesville, VA*

**Introduction:** Efficient information retrieval of poisonous substances and resources to manage poison exposures and queries is critical to poison center competency. Presently, poison center computer technology primarily serves to access the Poisindex® database and Toxicall® systems. Information concerning poison center consultants, internal and external phone directories, triage protocols, nomograms, staffing schedules, memos, policies, etc., typically find residence on bulletin boards, in 3 ring binders, or as dog-eared pages and post-it notes. These bits of information are often critical to effective phone management and can be difficult to locate for busy poison information specialists. **Methods:** Our poison center developed an intranet system called "InfoWeb," containing more than 20 linked files, which incorporate text, databases, calculators, internet links, and messaging capabilities. This system is accessible via a shortcut icon on the desktop of each poison center workstation. **Results:** "InfoWeb" continues to grow as new applications have become evident. Our poison specialists access "InfoWeb" more frequently as the value of this useful tool is realized. **Conclusion:** Intranet sites can provide a valuable tool to poison centers nationwide. Perhaps along with the implementation of the national 800 number and awareness campaign, a standard intranet skeleton might be developed and provided to all poison centers.

**151 IMPLEMENTATION OF TOXICALL®: IMPACT ON DOCUMENTATION.**

Gopalan D, Gopalan Y, Robertson WO. *Washington Poison Center, Seattle, WA*

**Background:** On January 1, 1999, the Washington Poison Center implemented Toxicall; 6 months later we sought to measure its impact on time consumed by staff members for each of 3 missions—A) telephone interchange, B) information retrieval from other than Poisindex® and C) documentation, and to compare it with prior studies of our conventional paper-pencil system. We conducted previous "time and motion" studies over the past decade, using random alarm devices (Devilbiss Electronics) and had data for comparative purposes. **Method:** Two investigators (DG and YG) developed a computer-based alarm system for both timing and station selection and for recording the observed events. **Results:** Over a 6-week period, 211 calls were analyzed; the percentage of time involved in each call was recorded (shown in Table) and compared to results from our 1993 study. An increase, rather than anticipated decrease, in documentation effort was observed (<0.05), but staff participation in other activities than those itemized fell even more dramatically, suggesting increased staff efficiency.

Percentage of Time Allocation			
Behavior Observed	1993	1999	$\chi^2$
Phone Interchange	31.4	35.4	NS
Info Retrieval	6.2	7.9	NS
Documentation	24.4	33.4	<0.05
All Other	38.0	23.3	<.001

**Conclusion:** We were disappointed to discover that documentation efforts consume so much time. We are working on solutions to that problem. Toxicall was successful in providing one nondebtable benefit; it finally eliminated illegibility of staff handwriting!

**152 EXPERIENCE WITH INPATIENT CHARTING OF POISON CENTER ADVICE USING AN EXISTING CLINICAL PHARMACOLOGY SERVICE.**

Stork CM, Lehmann DF, Medicis JJ, Cantor R. *Central New York Regional Poison Center, Emergency Medicine & Clinical Pharmacology, Upstate Medical University, Syracuse, NY*

**Background:** Poison Centers (PC) often interact with health care professionals in order to provide information and advice. A messenger often relates PC advice to the primary caregiver, which at times leads to misinterpretation of PC information. To attenuate this, a clinical toxicology service within an existing clinical pharmacology service at a teaching hospital was initiated to ensure primary caregivers knowledge of PC advice and involvement. **Description:** The clinical

toxicology service is a mandatory activity within the existing structure of the clinical pharmacology service. All toxicology patients require a call to the PC that initiates written patient specific advice, immediately via facsimile from a CSPI and within 24 hours via a written consult by a pharmacy practice resident reflecting PC advice. Cases requiring medical toxicology consultation receive additional advice and recommendations from the consultant. All cases are discussed daily during clinical pharmacology/toxicology rounds with medical students, pharmacy students, pharmacy, emergency medicine and medicine residents along with medical and clinical pharmacologists and toxicologists. **Results:** An 125% increase in call volume from the hospital to the PC occurred comparing the period of 1/1/00–4/15/00 (n = 162) to 1/1/99–4/15/99 (n = 72). Recommendations were followed in all cases in which PC recommendations were charted in the patient record. **Conclusion:** A successful clinical toxicology service can be initiated using existing resources in clinical pharmacology to ensure knowledge of poison center advice. The impact of this service on receptivity to poison center advice and ultimately, patient outcome deserves consideration.

### 153 POISON CENTER ACCESS TO PATIENT MEDICAL RECORDS.

Starr P, Giffin S. *Maryland Poison Center, Baltimore, MD, Oregon Poison Center, Portland, OR*

**Background:** Poison Centers (PC) serve as a resource to health care providers (HCP) for information regarding the assessment and treatment of toxic exposures. Access to patient information acquired during the initial assessment and throughout the hospital stay is essential to the provision of appropriate medical advice. PCs have expressed difficulty retrieving patient information from hospital staff concerned with violating patient confidentiality. A survey was conducted to determine the scope of the problem and resources PCs use to facilitate access to patient information. **Methods:** A E-mail survey was sent to managers of certified regional PCs. Survey questions addressed concerns regarding difficulty obtaining patient information and methods used to increase the likelihood of obtaining patient information. Responders were asked to submit a copy of any document used facilitate access to information. **Results:** 33 PCs responded. 3 centers claimed access to patient information was not a problem, 3 indicated it was rarely a problem, while 27 indicated ongoing problems trying to access information. 3 centers indicated an agreement with the department of health, 3 have obtained legal opinions regarding the right to access information and 2 indicated state regulations allowed access. Many centers utilize a letter describing their service and reasons for the information request. **Conclusions:** PC access to patient information is a widespread problem. Approaches currently in use to access information include judicial (legal opinion), legislative (state regulations) and cooperative interagency agreements. Laws and regulations regarding access to patient medical records vary among states. No universal mechanism is being used by PCs to assure access to patient medical records. Further evaluation is needed to determine if Federal regulations exist which could support PCs access to patient medical records.

### 154 A COMPARISON OF THE RECOMMENDATIONS FOR MANAGEMENT OF BREASTFEEDING IN THE FACE OF MATERNAL ANTIHYPERTENSIVES AND ANTIDEPRESSANTS IN SIX WELL-KNOWN REFERENCES.

Lawrence RA, McCooley AJM, Friedman LR. *Department of Pediatrics, University of Rochester School of Medicine, Rochester, NY*

**Background:** Poison Centers need reliable sources to refer to when confronted with breastfeeding questions. **Methods:** Common medications prescribed for hypertension and for depression and mental illness were investigated in six well-known references, Drugdex (Micromedex), Briggs, Freeman & Yaffe, American Academy of Pediatrics Drug List, Hale, the PDR and the Lactation Study Center database. The ratings were summarized from each reference as follows: A = Compatible; B = Modify breastfeeding/pump and discard; C = Caution needed; D = Discontinue; E = Unknown; F = Not Listed. **Results:** Drugdex listed 32 of 60 antihypertensives as unknown and 11 of 60 as controversial. 23 of 45 antidepressants were rated unknown and 12 of 45 were rated controversial. Briggs *et al.* rated most antihypertensives as unknown or did not list them. They approved 17 antihypertensives based on AAP listing. Of the antidepressants, 21 were not listed and 17 were not known. The AAP list only included 16 of the 60 antihypertensives, approving 14. Hale listed 7 of 60 as unknown, and 12 requiring discontinuation. The PDR rated antihypertensives as requiring caution in 23 of 60 and discontinuation of breastfeeding in 37. The antidepressants were rated 9 of 45 requiring caution and 23 discontinuation. The Lactation Center rated 44 of the 60 antihypertensives and all of the antidepressants. **Conclusion:** No drug was rated the same by all six well-known references. If the AAP rated the compound greater agreement was achieved. The PDR reported none on the list to be compatible with breastfeeding.

**155 AN INTERACTIVE INSTRUCTIONAL INTRANET SITE FOR MEDICAL STUDENTS IN A TOXICOLOGY CLERKSHIP.**

Sands T, Pali M. *California Poison Control System—Sacramento Division, Sacramento, CA*

**Background:** The California Poison Control System—Sacramento Division serves as one of the teaching sites for the University of California, Davis, School of Medicine. Fourth year medical students in their emergency medicine clerkship are required to participate in a 4-hour introduction to toxicology by spending this time in the poison control center. Up to 90 students a year rotate through the poison control center to fulfill this toxicology clerkship requirement. Students are familiarized with the Micromedex<sup>R</sup> databases, specifically PoisIndex<sup>R</sup>, by researching answers to standardized questions. They are also exposed to a variety of poison calls through on-site call monitoring. However, depending on the type and volume of calls coming in on that shift, students obtain clinical experiences that vary in value. In addition, fitting a structured academic teaching program into the 4-hour period is difficult. **Methods:** An evolving toxicology intranet site using FrontPage 2000<sup>R</sup> was created. The program has four modules: 1) Introduction to the California Poison Control System, 2) Learning to use PoisIndex<sup>R</sup>, 3) Basic principles of toxicology, and 4) Self-paced case presentations with interactive questions. In this module, toxicology case histories are presented in which the student selects treatment choices and receives feedback. More information on the case is then revealed, allowing the student to be involved in further treatment recommendations. **Conclusion:** Along with on-site call monitoring, this toxicology intranet modular system provides all medical students rotating through the poison control center with a uniform academic toxicology experience that does not depend on variations in call type and call volume.

**156 IS MEDICAL TOXICOLOGY BECOMING A PASTIME: GRADUATES OF MEDICAL TOXICOLOGY FELLOWSHIP PROGRAMS WILL NOT PRACTICE THIS SPECIALTY.**

Hoffman RJ, Kwok MY, Nelson LS, Hoffman RS. *New York City Poison Control Center, New York, NY*

**Background:** Anecdotal evidence leads us to hypothesize that physicians completing fellowship training in medical toxicology are unlikely to practice toxicology primarily. **Methods:** Using a telephone survey, we contacted all physician fellows in toxicology fellowships in North America to determine if they will practice toxicology after graduation. Fellows in their final year of training were asked what their medical practice would be after completion of their fellowship, and were asked if they would primarily practice toxicology. **Results:** Seventeen graduating toxicology fellows from eighteen programs were surveyed. No graduating toxicology fellow remaining in North America after graduation ( $n = 12$ ) intends to practice toxicology primarily. Most indicated that they would practice their primary specialty, usually emergency medicine ( $n = 9$ ), and would practice toxicology to a limited extent or part-time ( $n = 9$ ). Several had no plan to practice toxicology to any extent ( $N = 3$ ). These latter such fellows reported preferring to practice toxicology, but stated that they had no employment opportunity to do so. All foreign nationals returning to their home country ( $n = 5$ ) indicated that they would practice toxicology, 4 working primarily as toxicologists and 1 practicing pediatrics primarily and toxicology part-time. **Conclusion:** No graduating toxicology fellows remaining in North America intend to practice toxicology primarily. Several may not practice toxicology at all. Foreign nationals returning to their home country will all practice medical toxicology, most as a primary practice. Efforts may be needed to develop employment opportunities for medical toxicologists in North America.

**157 KNOWLEDGE ABOUT ACETAMINOPHEN TOXICITY AMONG ED VISITORS.**

Chen L, Schneider S, Wax P. *University of Rochester, School of Medicine, Rochester, NY*

**Introduction:** Chronic and unintentional overdose of acetaminophen (APAP) is an increasingly common cause of acute liver failure. This study examines knowledge and perception about APAP therapeutic usage and toxicity in a convenience sampling of ED visitors. **Method:** During a 3-month period a questionnaire was distributed to adult visitors found in a ED waiting room. The subjects were asked about demographic data, maximum daily therapeutic APAP dose and dosing intervals, and self-reporting practices regarding APAP use. **Results:** 103 of 142 (79%) ED visitors approached completed the questionnaire. The respondents were 66% Caucasians, and 31% of African Americans. 17.5% of the subjects believed maximum daily APAP dose is  $\geq 5g$  (5.8% believed that it is  $\geq 10g$  per day). When asked to identify APAP-containing products among 10 commonly used pain medications, only 12.6% chose Percocet<sup>®</sup>, and 5.8% Vicodin<sup>®</sup> respectively. Motrin<sup>®</sup> was the medication respondents most frequently believed to contain APAP(sic). 51.5% did not know that APAP toxicity causes liver damage. Readers (55.5%) of APAP package labeling had comparable knowledge of APAP toxicity to nonreaders (44.5%). No statistically significant difference existed with regard to sex,

race and age in responding to these questions. However, more female subjects routinely inform doctors about their APAP use compared to males (64.1% vs 29.7%,  $p < 0.005$ ). Conclusion: Some study subjects appear to have very limited knowledge regarding therapeutic dosing of APAP and its toxicity. To reduce the occurrence of chronic and unintentional APAP toxicity from the improper use of this omnipresent medication, optimal education of the public is essential. Regarding self-reporting practice, special attention should be focused on male patients who are less likely to volunteer APAP use history.

### 158 CYTOKINE RESPONSES FOLLOWING ACUTE ACETAMINOPHEN OVERDOSE.

James L, Farrar H, Sullivan J, Givens T, Kearns G, Wasserman G, Simpson P, Hinson J. *University of Arkansas for Medical Sciences, Little Rock, AR; University of Louisville, Louisville, KY; University of Missouri, Kansas City, Kansas City, MO*

Background: Cytokine liberation has been reported in animal models of acetaminophen (APAP) hepatotoxicity. Interleukin 8 (IL-8), a pro-inflammatory cytokine, has been found to be elevated in patients with toxin associated hepatic disease. Cytokines have not previously been measured in humans with APAP overdose. Methods: Concentrations of IL-6, IL-8, IL-10 were measured by ELISA in banked serum samples from patients with acute APAP overdose. Serum samples were obtained at the time of routine blood sampling for the evaluation and management of APAP overdose. Peak cytokine concentrations were compared to biochemical evidence of hepatocellular injury, nomogram risk assessment and prothrombin time (PT). Results: Serum samples from 35 children and adolescents (ages 0.8–18 years; 27 females) were evaluated for cytokines. Four patients had AST or ALT  $>100$  IU/L; five had AST or ALT  $>1000$  IU/L and the remaining patients had no hepatotoxicity. Peak IL-8, but not IL-6 or IL-10 levels, correlated with hepatotoxicity (Mann-Whitney Exact Test;  $p < 0.001$ ). Peak IL-8 was greater in patients at “high risk” by the nomogram and those presenting  $>15$  hours, as compared to other patients (Mann Whitney U;  $p = 0.009$ ). A temporal, parallel relationship for IL-8 and AST/ALT was found for one patient with severe hepatotoxicity. Peak IL-8 concentrations of  $>20$  pg/mL were also associated with peak PT values (Mann-Whitney Exact Test;  $p = 0.006$ ). Conclusions: IL-8 is significantly elevated in patients with acute APAP hepatotoxicity and corresponds with other common clinical measures predictive and/or reflective of hepatocellular injury. Further study is warranted to evaluate the relationship between inflammatory biomarkers and APAP hepatotoxicity in children and adults.

### 159 PERSISTENTLY ELEVATED ACETAMINOPHEN CONCENTRATIONS FOR TWO DAYS AFTER INITIAL FOUR HOUR NONTOXIC CONCENTRATION.

Spiller HA, Ross MP. *Kentucky Regional Poison Center Louisville, KY*

Background: The decision to treat patients with acute acetaminophen (APAP) overdose with *n*-acetylcysteine is routinely made with a single APAP concentration drawn four or more hours postingestion. However, in cases where there are co-ingestants that may delay gastric emptying, there have been recommendations for additional concentrations to determine the peak APAP concentration. Case Report: A 57-year-old female presented to the ED with a history of a multiple drug ingestion one hour prior to presentation. The patient was drowsy with the following vitals signs: HR 84, BP 150/85, resp 20, and  $O_2$  of 97%. Empty pill bottles indicated a possible ingestion of klonopin, butalbital, APAP, APAP with propoxyphene and APAP with hydrocodone. An APAP concentration drawn at patient arrival was 72 mcg/mL. The patient was lavaged and then administered activated charcoal with magnesium citrate. A 4-hour postingestion APAP concentration was 83 mcg/mL. All laboratory studies were normal at this time. Due to the patient's continued lethargy repeat APAP concentrations were drawn and were 88 mcg/mL (8 hours), 116 mcg/mL (19 hours), 75 mcg/mL (26 hours), 52 mcg/mL (30 hours) and 27 mcg/mL (40 hours), with an elimination half-life of approximately 10 hours. The patient was not treated with NAC, due to a decision by the patient's treating physician. Laboratory studies, including transaminases, BUN, creatinine, bilirubin and prothrombin time remained normal throughout the 60 hours they were measured. Conclusion: A patient developed delayed and persistent elevation of serum APAP after an initial concentration in the no treatment range. This may suggest delayed tablet dissolution with concomitant drug-induced decreased bowel tone. No evidence of hepatic injury occurred despite lack of antidotal therapy.

### 160 SURVIVAL AFTER MASSIVE INGESTION OF ACETAMINOPHEN PRESENTING AS COMA AND METABOLIC ACIDOSIS.

Rusyniak D, Dribben W, Furbee B, Kirk M. *Indiana Poison Center, Indiana University School of Medicine, Clarian Health Partners, Indianapolis, IN*

**Objective:** We present an unusual clinical scenario associated with massive acetaminophen overdose that through aggressive supportive care resulted in a good outcome despite a complicated clinical course. **Case Report:** A previously healthy 26-year-old female presented 12 hours after ingesting approximately 125 grams of Extra-Strength Tylenol® comatose with a GCS of 3. Vital signs included temperature 35.6°C, SBP 60 mmHg, and HR 130/min. She was intubated, resuscitated with IV fluids and started on pressors. Initial laboratory data revealed marked metabolic acidosis (pH 6.7, bicarbonate 5 mmol/L), renal insufficiency (creatinine 1.8 mg/dL), mild hepatotoxicity (AST 121 U/L, total bilirubin 0.7 mg/dL), and mild coagulopathy (INR 1.38, platelets 80,000/mm<sup>3</sup>). A 12-hour acetaminophen level was 1,148 mcg/mL followed by an 18 hour level of 1328 mcg/mL. Workup for other causes of metabolic acidosis (salicylates, iron, toxic alcohols) was negative. Despite treatment with IV NAC, the patient developed fulminant hepatic failure and underwent a 12 week hospital course including: 3 weeks of ventilatory support, prolonged hypotension (10 days of norepinephrine, max 68 mcg/kg/min), CVVH for renal failure, episodes of complete heart block, pancreatitis with pseudocyst, sepsis and pneumonia, ARDS, upper GI bleed, tracheo-esophageal fistula, pleural hematoma, pancytopenia (treated with 27 units of PRBCs and 17 units of platelets), and coagulopathy requiring 20 units of FFP. She eventually recovered and was discharged home with a normal neurological outcome and normal hepatic function. **Conclusions:** Massive ingestions of acetaminophen can present as metabolic acidosis and coma before the onset of hepatic failure. Despite fulminant hepatic failure and criteria suggesting poor prognosis, patients can survive with aggressive supportive care and without liver transplantation.

### 161 SEVERE ACETAMINOPHEN HEPATIC AND RENAL TOXICITY FOLLOWING POSTOPERATIVE THERAPEUTIC DOSES.

Burkhart KK, Donovan JW. *The Pennsylvania State University, Hershey, PA*

**Background:** Acetaminophen (APAP) is used to help control pain postop. We describe a patient who had multiple APAP orders with the potential to receive excessive in-hospital APAP. Our patient received  $\leq 3.9$  g/d (total 15.6 g) and developed severe hepatic and renal toxicity. **Case Report:** A 47-year-old male presented with CHF. A MI was ruled out, but there was hepatic injury, ALT 94 U/L, and LDH 1611 U/L. SH included 6–8 beers/d and smoking. On day 4, CABPG was performed. The patient was not fed, but was started on iron sulfate 325 mg TID. On postop day 5 hypotension and disorientation developed. ALT/AST were 2613 and 4838 U/L. Lactic acidosis, hypoglycemia, pancreatitis, renal insufficiency, and thrombocytopenia followed. Postop APAP orders included propoxyphene 100 mg/APAP 650 mg, APAP 325 mg/oxycodone 5mg, and APAP 325 mg/codeine 30 mg for pain, and APAP 650 mg prn fever. Daily postop APAP was 2.6 g, 3.9 g, 3.9 g, 3.9 g, and 1.3 g on postop day 5. An APAP level 8 hours after the last dose was 15 mcg/mL. All cultures returned negative, while a liver biopsy showed centrilobular necrosis. *N*-acetylcysteine was given for 17 doses. With aggressive supportive care this patient recovered. **Conclusions:** This case is a rare report where therapeutic APAP doses produced severe toxicity. This patient had risk factors, preceding hepatic injury, postop wound healing and fasting, heavy alcohol consumer, and the iron. Hospitals must develop protocols that prevent patients from receiving  $\geq 4$  g/d of APAP. Our pharmacy instituted the following changes. Warning flags are in the computer to alert pharmacists to check doses. No more than 3 prn doses are sent to patient floors. Finally, labels have been placed on all APAP products from the automated dispensing equipment that warn nurses to check the patient's total APAP dosing.

### 162 HEMOLYSIS FOLLOWING ACETAMINOPHEN OVERDOSE IN A PATIENT WITH GLUCOSE-6-PHOSPHATE DEHYDROGENASE DEFICIENCY.

Ruha AM, Selden B, Brooks D. *Good Samaritan Regional Medical Center, Phoenix, AZ*

**Background:** Patients with glucose-6-phosphate dehydrogenase (G6PD) deficiency hemolyze when oxidant stress depletes reduced glutathione in erythrocytes. Therapeutic doses of many drugs precipitate hemolytic episodes in such patients, however, acetaminophen (APAP) is not considered one of them. We describe acute hemolysis following a large ingestion of (APAP) in a patient with unrecognized G6PD deficiency. **Case Report:** A 16-year-old African-American teenager, with previously undiagnosed G6PD deficiency, ingested an unknown amount of APAP, fluvoxamine, and clomipramine in a suicide attempt. A 6 hour. APAP level was 680 mg/L. He received intravenous *N*-acetylcysteine

(NAC) and supportive care; no serious hepatotoxicity ensued. Two days postingestion he developed scleral icterus and an elevated unconjugated bilirubin. Hemoglobin and hematocrit had fallen from initial levels of 13.7 g/dL and 40.0% to 9.7 g/dL and 27.9%, respectively, on day 3, reaching a nadir of 7.6 g/dL and 22.1%, respectively on day 8. Plasma free hemoglobin was 148 mg/dL (normal <7 mg/dL). A qualitative G6PD assay obtained during the crisis revealed deficiency of erythrocytic G6PD activity, suggesting a severe variant of this enzyme deficiency. Hemolysis following APAP overdose has been reported only once previously. The coingestants have not been reported to produce hemolysis. **Conclusion:** A combination of a severe variant of G6PD deficiency and massive APAP overdose probably produced hemolysis in our patient. Evidence of G6PD deficiency should be sought when hemolysis follows APAP overdose.

### 163 ACUTE PANCREATITIS ASSOCIATED WITH ACETAMINOPHEN HEPATOTOXICITY.

Yoo H, Witmer R, Wax P. *University of Rochester Medical Center, Rochester, NY*

**Objective:** Acute pancreatitis in the setting of acetaminophen hepatotoxicity has been reported in isolated case reports, but there are no series investigating the actual incidence. Our goal was to better define the incidence of acute pancreatitis in this setting. **Methods:** A retrospective review of medical records of 50 consecutive patients consulted on by the medical toxicology service at a tertiary care hospital with a diagnosis of acetaminophen toxicity over a 6-year period was performed. This hospital also serves as a liver transplant center. Acetaminophen hepatotoxicity was defined as AST >1000 IU/L in the setting of excessive acetaminophen use or overdose. Patients who had a serum lipase level three times above the upper limit of normal (upper limit of normal 208 IU/L) were considered to fulfill a commonly used laboratory diagnosis for acute pancreatitis. **Results:** 28 of 50 (56%) patients had peak AST >1000 IU/L. Serum amylase and lipase data were available for 16 of these 28 patients. Seven of 16 (44%) patients had hyperamylasemia (>130 IU/L), and 13 of 16 (81%) had hyperlipasemia (>208 IU/L). Nine of 16 (56%) had serum lipase >624 IU/L (3 × upper limit of normal), meeting the criteria for acute pancreatitis. **Conclusion:** Although 12 of 28 patients with AST >1000 IU/L in this study did not have serum lipase determinations, our data suggests that at least 9 of 28 (32%) of our patients with acetaminophen hepatotoxicity had fulfilled the criteria for acute pancreatitis. Acute pancreatitis may be more common in acetaminophen ingestion than previously described. Lipase assay is indicated in patients with significant acetaminophen ingestion for early and accurate diagnosis of pancreatitis, as a number of patients with pancreatitis in our study had serum amylase within the normal range.

### 164 COMPARISON OF APAP OUTCOMES AND MANAGEMENT GUIDELINES BETWEEN TWO REGIONAL POISON CENTERS.

Anderson BD, Crouch BI. *Maryland Poison Center, University of Maryland School of Pharmacy, Baltimore, MD; Utah Poison Center, University of Utah School of Pharmacy, Salt Lake City, UT*

**Objective:** To compare outcomes and referral patterns for pediatric acetaminophen (APAP) cases from two poison centers that used different treatment guidelines. **Methods:** All cases of APAP exposures in children less than age 6 years from two regional poison centers were compared. Data collected included total human exposures; total pediatric exposures; total pediatric APAP exposures; and referral patterns, hospitalization patterns, and outcomes for pediatric APAP exposures from 1993 through 1997. During the study period, Center A referred pediatric acute APAP exposures to an ED at  $\geq 140$  mg/kg; Center B referred patients to an ED at  $\geq 200$  mg/kg. **Results:** Summary data are presented in the table below. Center A managed an average of 33,939 exposure cases per year with 63.4% occurring in children <age 6 years. Center B managed an average of 38,043 cases per year with 54.0% occurring in children.

Center	Total APAP Exposures < Age 6	% Referred to HCF	% Outcome of No or Minor Effect
A	4,538	17.6	97.4
B	3,415	4.7	98.1

**Conclusions:** Differences in treatment guidelines resulted in more cases being referred to a health care facility with no apparent improvement in outcomes. Assuming a cost of \$250 for each subtoxic pediatric APAP exposure managed in

an ED, center A could save an average of \$29,186 per year by revising their referral amount for pediatric APAP exposures to  $\geq 200$  mg/kg.

### 165 TREATMENT OF ACETAMINOPHEN TOXICITY IN A 9-DAY-OLD INFANT.

Flaman Z, Cawley J. *Ontario Regional Poison Information Centre, The Hospital for Sick Children, Toronto, Ontario, Canada*

**Background:** Acetaminophen, a widely used over-the-counter analgesic and antipyretic, is recommended in doses of 10 to 15 mg/kg for infants and children. Since acetaminophen is not routinely administered to neonates, toxicity with this medication for this age group is unusual. **Case Report:** A 9-day-old male infant, weighing 4 kg, was mistakenly given 3 doses of acetaminophen (220 mg or 55 mg/kg) for a total of 660 mg (182 mg/kg) over a 12-hour period. At a local emergency department, blood was drawn to test the infant's acetaminophen and liver enzyme levels. The initial acetaminophen level was 224  $\mu\text{mol/L}$  (14 hours after the last dose); alkaline phosphates (AK) 164  $\mu\text{mol/L}$ ; aspartate aminotransferase (AST) 31  $\mu\text{mol/L}$ ; alanine aminotransferase (ALT) 32  $\mu\text{mol/L}$ ; and indirect bilirubin 61  $\mu\text{mol/L}$ . The infant was discharged from the hospital without treatment, and the parents were asked to return with him the next morning. On his return, the infant was admitted for another set of tests. The second acetaminophen level, taken 22 hours after ingestion, was 172  $\mu\text{mol/L}$ ; AK 172  $\mu\text{mol/L}$ ; AST 58  $\mu\text{mol/L}$ ; and ALT 10  $\mu\text{mol/L}$ . The infant was treated successfully with oral *N*-acetylcysteine (NAC) at an initial dose of 140 mg/kg, followed by 4 doses of NAC at 70 mg/kg over a period of 16 hours. He tolerated oral NAC well. His acetaminophen level decreased to less than 26  $\mu\text{mol/L}$ . AST was 24  $\mu\text{mol/L}$ ; ALT 27  $\mu\text{mol/L}$ ; and bilirubin 32  $\mu\text{mol/L}$ . The infant was discharged 72 hours after admission. **Conclusion:** NAC is a potentially effective treatment for acetaminophen toxicity in infants as young as 9 days.

### 166 THE DILEMMA OF NAC THERAPY IN A PREMATURE INFANT.

Sharma AN, Howland MA, Hoffman RS. *New York City Poison Control Center, St. John's University, New York, NY*

**Background:** Fetal acetaminophen (APAP) toxicity is presumed to increase with gestational age and fetal production of NAPQI. Oral feeding of premature infants is associated with necrotizing enterocolitis (NEC), a life-threatening illness. We report a case that deals with presumed APAP toxicity in a premature infant and the complex dilemma associated with NAC therapy. **Case:** A 27-year-old female, 30 weeks pregnant, reportedly ingested 100  $\times$  325 mg tablets of APAP over a two-day period for a toothache, four days prior to admission. She reported no APAP use for the two days immediately prior to admission, but did report cocaine use. Initial laboratory abnormalities included: INR 8; AST 11,316 U/L; ALT 3440 U/L; APAP 10  $\mu\text{g/mL}$  and Cr 2.1 mg/dL. At presentation she was in active labor and was taken for an immediate Cesarean section. The neonate, born at 30 weeks gestation, 1.5 kg, was admitted to the NICU. His initial cord blood analysis revealed: APAP 7  $\mu\text{g/mL}$ ; AST 229 U/L and Cr 2.4 mg/dL. The decision was made to treat with NAC, but the ideal route of administration was debated. The neonate was maintained NPO due to the risk of NEC and his 24-hour IV fluid intake was limited to 80 mL. Both the British and US IV protocols were considered, but fluid restrictions made IV NAC difficult to administer. Ultimately, it was decided to treat the child with oral NAC using the standard US protocol. The neonate tolerated all 18 doses without any evidence of NEC. Mother and child recovered without further complication. **Conclusion:** Oral NAC was administered without complication in this premature infant with presumed acetaminophen toxicity.

### 167 ORAL OVERDOSE OF N-ACETYL-CYSTEINE IN AN INFANT.

Grover J, Caravati EM. *Utah Poison Control Center, College of Pharmacy, University of Utah, Salt Lake City, UT*

**Background:** Reports of *N*-acetylcysteine (NAC) overdose are scarce and mainly involve intravenous (IV) administration. We report an  $>8$ -fold overdose of orally administered NAC that resulted in minor effects. Because NAC is only FDA approved for oral administration, this is a more likely scenario for overdose in the United States. **Case Report:** A healthy 8 kg, 9-month-old female was found sucking on acetaminophen (APAP) gel capsules (500 mg) at home. The maximum amount of APAP ingested was estimated to be 7.5 g (937 mg/kg). The patient did not receive GI decontamination and her 4-hour APAP concentration was 350 mcg/mL. A dose of charcoal was given at this time, as well as a loading dose of NAC (1120 mg). The patient was admitted and the maintenance dose was miscalculated by ten-fold. The patient received 28 cc of undiluted 20% NAC instead of 2.8 cc for a total of eight doses via a nasogastric tube. The total dose given over a 36-hour period was 45,920 mg. The recommended total dose for that time period would be 5600 mg. The patient experienced clear emesis 1–2 hours after several of the doses on the second day of

treatment. Serum potassium was 2.7 mEq/L 24 hours after admission. The NAC treatment was discontinued after the eighth maintenance dose. The patient was discharged approximately 50 hours after the exposure and was lost to follow up. Upon discharge, liver enzymes were normal, prothrombin time was elevated at 14.3 sec and electrolytes were normal. **Conclusion:** This infant received an >8-fold overdose of oral NAC over 36 hours that was associated with minor GI effects and hypokalemia. The minor effects from this overdose are consistent with a large therapeutic index for this antidote.

### 168 DIPHENHYDRAMINE-INDUCED WIDE-COMPLEX TACHYCARDIA RESPONDS TO BICARBONATE.

Sharma AN, Hexdall AH, Nelson L, Hoffman RS. *New York City Poison Control Center, New York, NY*

**Background:** Given its wide availability, diphenhydramine (DPH) is commonly taken in intentional overdose. Its ability to block sodium channels may result in QRS widening that should respond to NaHCO<sub>3</sub>, similar to that occurring with TCAs. To date, only one case of DPH-induced wide-complex tachycardia has been reported to respond to NaHCO<sub>3</sub>. We report three additional DPH overdoses with bicarbonate responsive wide-complex tachycardia. **Case Reports:** On three separate occasions patients presented to the emergency department following massive DPH overdoses. All patients had tachycardia, an altered mental status and other symptoms of anticholinergic toxicity. In addition, each patient had an initial QRS complex duration greater than 100 msec. IV NaHCO<sub>3</sub> at 1 mEq/kg was administered to each patient with QRS complex width narrowing. Serum assays for antihistamines and TCAs revealed only elevated DPH levels and no detectable co-ingestants.

Patient	Initial QRS Width	QRS Width Post NaHCO <sub>3</sub>	Diphenhydramine Level
1	128 msec	89 msec	1600 ng/mL
2	161 msec	155 msec	Qualitative positive, no quant.
3	102 msec	86 msec	380 ng/mL

**Conclusion:** In our case series, diphenhydramine overdose with resultant wide-complex tachycardia was amenable to sodium bicarbonate therapy. Although the clinical benefit of this therapy is unknown, we recommend NaHCO<sub>3</sub> for patients presenting with diphenhydramine-induced cardiotoxicity.

### 169 SODIUM BICARBONATE FOR TAXUS-INDUCED DYSRHYTHMIA.

Miller MB, Eng J, Curry SC. *Ingham Regional Medical Center, Dewitt, MI.*

**Background:** Suicidal ingestions of yew (*Taxus*) have been reported to cause fatal arrhythmia. The literature describes both tachyarrhythmias and bradyarrhythmias, but most reports confirm the presence of a prolonged ventricular complex. Animal studies of the toxic component, taxine, show it acts as a Class I antiarrhythmic agent, by blocking sodium channels. This case illustrates the use of sodium bicarbonate in treatment of a nearfatal ingestion of *Taxus*. **Case Report:** A 32-year-old male stated he combined unknown quantities of dehydrated yew, paroxetine, brodifacoum, and shellac, which he then baked. After eating one dozen "cookies" he experienced repeated vomiting. He was taken the hospital approximately three hours postingestion. Upon arrival he became unresponsive and suffered sudden cardiac arrest. ACLS measures were initiated. ABG at the time of arrest showed a pH of 7.34. Bicarbonate was given as the original ECG demonstrated a prolonged QRS complex. Circulation was reestablished and wide complex rhythm remained. Bicarbonate therapy was therefore continued. Original epinephrine and dopamine drips were converted to norepinephrine for persistent hypotension. Serial ABGs reveal that as the serum pH became greater than 7.5, the QRS complex narrowed. Urine analyzed by GCMS revealed only the presence of phenylpropranolamine. Detection of taxine would not be expected due to the technique used. The presence of taxine in the "dough" however, was confirmed by GCMS. The patient eventually recovered and was discharged to a psychiatric facility. **Conclusions:** Sodium bicarbonate is an accepted standard of care in treatment of wide complex arrhythmias associated with sodium channel blocking agents. This case describes the first use of sodium bicarbonate in the treatment of yew toxicity.



an ED, center A could save an average of \$29,186 per year by revising their referral amount for pediatric APAP exposures to  $\geq 200$  mg/kg.

### 165 TREATMENT OF ACETAMINOPHEN TOXICITY IN A 9-DAY-OLD INFANT.

Flaman Z, Cawley J. *Ontario Regional Poison Information Centre, The Hospital for Sick Children, Toronto, Ontario, Canada*

**Background:** Acetaminophen, a widely used over-the-counter analgesic and antipyretic, is recommended in doses of 10 to 15 mg/kg for infants and children. Since acetaminophen is not routinely administered to neonates, toxicity with this medication for this age group is unusual. **Case Report:** A 9-day-old male infant, weighing 4 kg, was mistakenly given 3 doses of acetaminophen (220 mg or 55 mg/kg) for a total of 660 mg (182 mg/kg) over a 12-hour period. At a local emergency department, blood was drawn to test the infant's acetaminophen and liver enzyme levels. The initial acetaminophen level was 224  $\mu\text{mol/L}$  (14 hours after the last dose); alkaline phosphates (AK) 164  $\mu\text{mol/L}$ ; aspartate aminotransferase (AST) 31  $\mu\text{mol/L}$ ; alanine aminotransferase (ALT) 32  $\mu\text{mol/L}$ ; and indirect bilirubin 61  $\mu\text{mol/L}$ . The infant was discharged from the hospital without treatment, and the parents were asked to return with him the next morning. On his return, the infant was admitted for another set of tests. The second acetaminophen level, taken 22 hours after ingestion, was 172  $\mu\text{mol/L}$ ; AK 172  $\mu\text{mol/L}$ ; AST 58  $\mu\text{mol/L}$ ; and ALT 10  $\mu\text{mol/L}$ . The infant was treated successfully with oral *N*-acetylcysteine (NAC) at an initial dose of 140 mg/kg, followed by 4 doses of NAC at 70 mg/kg over a period of 16 hours. He tolerated oral NAC well. His acetaminophen level decreased to less than 26  $\mu\text{mol/L}$ . AST was 24  $\mu\text{mol/L}$ ; ALT 27  $\mu\text{mol/L}$ ; and bilirubin 32  $\mu\text{mol/L}$ . The infant was discharged 72 hours after admission. **Conclusion:** NAC is a potentially effective treatment for acetaminophen toxicity in infants as young as 9 days.

### 166 THE DILEMMA OF NAC THERAPY IN A PREMATURE INFANT.

Sharma AN, Howland MA, Hoffman RS. *New York City Poison Control Center, St. John's University, New York, NY*

**Background:** Fetal acetaminophen (APAP) toxicity is presumed to increase with gestational age and fetal production of NAPQI. Oral feeding of premature infants is associated with necrotizing enterocolitis (NEC), a life-threatening illness. We report a case that deals with presumed APAP toxicity in a premature infant and the complex dilemma associated with NAC therapy. **Case:** A 27-year-old female, 30 weeks pregnant, reportedly ingested 100  $\times$  325 mg tablets of APAP over a two-day period for a toothache, four days prior to admission. She reported no APAP use for the two days immediately prior to admission, but did report cocaine use. Initial laboratory abnormalities included: INR 8; AST 11,316 U/L; ALT 3440 U/L; APAP 10  $\mu\text{g/mL}$  and Cr 2.1 mg/dL. At presentation she was in active labor and was taken for an immediate Cesarean section. The neonate, born at 30 weeks gestation, 1.5 kg, was admitted to the NICU. His initial cord blood analysis revealed: APAP 7  $\mu\text{g/mL}$ ; AST 229 U/L and Cr 2.4 mg/dL. The decision was made to treat with NAC, but the ideal route of administration was debated. The neonate was maintained NPO due to the risk of NEC and his 24-hour IV fluid intake was limited to 80 mL. Both the British and US IV protocols were considered, but fluid restrictions made IV NAC difficult to administer. Ultimately, it was decided to treat the child with oral NAC using the standard US protocol. The neonate tolerated all 18 doses without any evidence of NEC. Mother and child recovered without further complication. **Conclusion:** Oral NAC was administered without complication in this premature infant with presumed acetaminophen toxicity.

### 167 ORAL OVERDOSE OF N-ACETYLCYSTEINE IN AN INFANT.

Grover J, Caravati EM. *Utah Poison Control Center, College of Pharmacy, University of Utah, Salt Lake City, UT*

**Background:** Reports of *N*-acetylcysteine (NAC) overdose are scarce and mainly involve intravenous (IV) administration. We report an  $>8$ -fold overdose of orally administered NAC that resulted in minor effects. Because NAC is only FDA approved for oral administration, this is a more likely scenario for overdose in the United States. **Case Report:** A healthy 8 kg, 9-month-old female was found sucking on acetaminophen (APAP) gel capsules (500 mg) at home. The maximum amount of APAP ingested was estimated to be 7.5 g (937 mg/kg). The patient did not receive GI decontamination and her 4-hour APAP concentration was 350 mcg/mL. A dose of charcoal was given at this time, as well as a loading dose of NAC (1120 mg). The patient was admitted and the maintenance dose was miscalculated by ten-fold. The patient received 28 cc of undiluted 20% NAC instead of 2.8 cc for a total of eight doses via a nasogastric tube. The total dose given over a 36-hour period was 45,920 mg. The recommended total dose for that time period would be 5600 mg. The patient experienced clear emesis 1-2 hours after several of the doses on the second day of

treatment. Serum potassium was 2.7 mEq/L 24 hours after admission. The NAC treatment was discontinued after the eighth maintenance dose. The patient was discharged approximately 50 hours after the exposure and was lost to follow up. Upon discharge, liver enzymes were normal, prothrombin time was elevated at 14.3 sec and electrolytes were normal. **Conclusion:** This infant received an >8-fold overdose of oral NAC over 36 hours that was associated with minor GI effects and hypokalemia. The minor effects from this overdose are consistent with a large therapeutic index for this antidote.

### 168 DIPHENHYDRAMINE-INDUCED WIDE-COMPLEX TACHYCARDIA RESPONDS TO BICARBONATE.

Sharma AN, Hexdall AH, Nelson L, Hoffman RS. *New York City Poison Control Center, New York, NY*

**Background:** Given its wide availability, diphenhydramine (DPH) is commonly taken in intentional overdose. Its ability to block sodium channels may result in QRS widening that should respond to NaHCO<sub>3</sub>, similar to that occurring with TCAs. To date, only one case of DPH-induced wide-complex tachycardia has been reported to respond to NaHCO<sub>3</sub>. We report three additional DPH overdoses with bicarbonate responsive wide-complex tachycardia. **Case Reports:** On three separate occasions patients presented to the emergency department following massive DPH overdoses. All patients had tachycardia, an altered mental status and other symptoms of anticholinergic toxicity. In addition, each patient had an initial QRS complex duration greater than 100 msec. IV NaHCO<sub>3</sub> at 1 mEq/kg was administered to each patient with QRS complex width narrowing. Serum assays for antihistamines and TCAs revealed only elevated DPH levels and no detectable co-ingestants.

Patient	Initial QRS Width	QRS Width Post NaHCO <sub>3</sub>	Diphenhydramine Level
1	128 msec	89 msec	1600 ng/mL
2	161 msec	155 msec	Qualitative positive, no quant.
3	102 msec	86 msec	380 ng/mL

**Conclusion:** In our case series, diphenhydramine overdose with resultant wide-complex tachycardia was amenable to sodium bicarbonate therapy. Although the clinical benefit of this therapy is unknown, we recommend NaHCO<sub>3</sub> for patients presenting with diphenhydramine-induced cardiotoxicity.

### 169 SODIUM BICARBONATE FOR TAXUS-INDUCED DYSRHYTHMIA.

Miller MB, Eng J, Curry SC. *Ingham Regional Medical Center, Dewitt, MI.*

**Background:** Suicidal ingestions of yew (*Taxus*) have been reported to cause fatal arrhythmia. The literature describes both tachyarrhythmias and bradyarrhythmias, but most reports confirm the presence of a prolonged ventricular complex. Animal studies of the toxic component, taxine, show it acts as a Class I antiarrhythmic agent, by blocking sodium channels. This case illustrates the use of sodium bicarbonate in treatment of a nearfatal ingestion of *Taxus*. **Case Report:** A 32-year-old male stated he combined unknown quantities of dehydrated yew, paroxetine, brodifacoum, and shellac, which he then baked. After eating one dozen "cookies" he experienced repeated vomiting. He was taken to the hospital approximately three hours post-ingestion. Upon arrival he became unresponsive and suffered sudden cardiac arrest. ACLS measures were initiated. ABG at the time of arrest showed a pH of 7.34. Bicarbonate was given as the original ECG demonstrated a prolonged QRS complex. Circulation was reestablished and wide complex rhythm remained. Bicarbonate therapy was therefore continued. Original epinephrine and dopamine drips were converted to norepinephrine for persistent hypotension. Serial ABGs reveal that as the serum pH became greater than 7.5, the QRS complex narrowed. Urine analyzed by GCMS revealed only the presence of phenylpropranolamine. Detection of taxine would not be expected due to the technique used. The presence of taxine in the "dough" however, was confirmed by GCMS. The patient eventually recovered and was discharged to a psychiatric facility. **Conclusions:** Sodium bicarbonate is an accepted standard of care in treatment of wide complex arrhythmias associated with sodium channel blocking agents. This case describes the first use of sodium bicarbonate in the treatment of yew toxicity.

**170 ADULT BUPROPRION EXPOSURES REPORTED IN TEXAS DURING 1998 AND 1999.**

Shepherd G, Velez LI, Keyes C, James DK. *North Texas Poison Center, Dallas, TX*

**Background:** Currently there is limited information describing the effects of adult ingestion of bupropion. The purpose of this retrospective case review is to describe the amounts ingested, therapy and clinical effects. **Methods:** 633 cases of bupropion exposures in adults that were reported to the Texas Poison Center Network during 1998–1999 were reviewed. Cases with poor follow-up ( $n = 193$ ) were excluded. Evaluation was based on TESS coding and review of the written case record. This review was performed cases that were coded as having seizures, a moderate, major or death outcome. It evaluated dose, dosage form, management site, clinical effects, therapy, outcomes and duration of observation. Another important point that this review addressed was the temporal relationship between ingestion, therapy, and clinical effects. **Results:** Reasons included intentional ingestion ( $n = 253$ ), therapeutic errors ( $n = 154$ ), and adverse drug reactions ( $n = 33$ ). The female to male ratios were 1.3:1 overall and 2:1 for adverse reactions. Deaths were reported in 2 cases that involved other substances. Seizures were reported in 33 cases, 2 of which developed status epilepticus. The time of onset of seizures ranged from 1 to 14 hours (mode = 2). They occurred most commonly in patients that ingested more than 2500 mg or were also using stimulants at the time of exposure. Seizures were not observed following accidental double doses. Other prominent clinical findings included: lethargy, tachycardia, hypertension, hallucinations, and tremor. Decontamination therapy was used in 50% of reviewed cases. **Conclusions:** Seizures are most likely occur within 4 hours, especially following large ingestions or when stimulants are co-administered. Adverse reactions were reported twice as frequently in women. Prospective studies are needed to better define therapy and risk factors for seizures.

**171 FLUVOXAMINE OVERDOSE PRODUCING STATUS EPILEPTICUS.**

Hahn I, Blancaflor G, Hoffman RS, Howland MA, Nelson LS. *New York City Poison Control Center, Bellevue Hospital Center, St. John's University, New York, NY*

**Background:** Fluvoxamine, an antidepressant that predominantly inhibits serotonin reuptake, is rarely associated with seizures in the absence of an underlying seizure disorder. Hypotension is unreported and fatalities are rare. **Case Report:** A 40-year-old male with depression presented to the ED one hour after reportedly ingesting 10 g of fluvoxamine. Prior to arrival, the patient had a self-limited generalized seizure witnessed by EMS. His initial vital signs in the ED were BP, 72/palp mmHg; P 124/min; R 28/min; T 101.4°F; and O<sub>2</sub> saturation 98% on room air. On physical examination the patient was lethargic and had mydriasis, peripheral cyanosis, and dry mucous membranes. An ECG revealed sinus tachycardia with a normal QRS duration and a normal T40 axis. His laboratory evaluation was unremarkable. He remained hypotensive despite 3 liters of intravenous saline but responded to intravenous pressors. Following endotracheal intubation he received orogastric lavage, which retrieved many pill fragments, and activated charcoal was administered. He suffered multiple self-limited seizures during the first hospital day for which he received several doses of lorazepam, phenytoin and empiric pyridoxine. A chest radiograph, which was initially normal, revealed bibasilar infiltrates on day 2. He developed progressive hypoxia and ultimately succumbed to necrotizing pneumonia believed to be secondary to aspiration. His presenting serum fluvoxamine concentration was 3500 ng/mL (therapeutic range 88–543 ng/mL). Screening for tricyclic antidepressants and drugs of abuse were negative. **Conclusion:** Fluvoxamine poisoning typically causes only CNS depression and death in isolated overdose remains unreported. This is the first reported case of status epilepticus and refractory hypotension following isolated overdose.

**172 LITHIUM GREASE INGESTION IN A 14-MONTH-OLD CHILD.**

Elko C, Burgess J. *Washington Poison Center, Seattle, WA*

**Background:** Lithium grease is formed from a blend of a lithium soap (lithium stearate or lithium hydroxysterate) and a hydrocarbon such as mineral oil. This formulation makes lithium grease both water-resistant and stable as a high temperature lubricant. Depending on the particular formulation, the lithium soaps may be present at less than 1% or greater than 80%. Although bovine fatalities from lithium grease ingestion have been reported, no cases of human ingestions have been described. **Case Report:** A call was placed to the Poison Center by a mother who found her 14-month-old son eating from a container of lithium grease. The container label described the contents as a lithium grease complex with petroleum hydrocarbon and organic zinc. The only symptoms noted were a rash on his neck, chest, stomach, and his eyes looking red. The mother had wiped out his mouth, had given him a bath, and was instructed to begin an eye flush for a possible eye exposure. At the same time, the on-call toxicologist was contacted and recommended

the child be evaluated at a hospital for possible lithium toxicity. The child was seen at a local hospital and a lithium level was drawn approximately 6.5 hours postingestion. The child was observed, remained asymptomatic, and had a lithium level of 0.14 mmol/L. Per our recommendation, the child was then discharged home to the care of his parents. **Conclusion:** This is the first reported case of a measurable lithium level following lithium grease ingestion in a human. Although the serum level was low, cases of lithium grease ingestion in humans should be regarded cautiously until further research or information is available.

### 173 SUCCESSFUL MANAGEMENT OF LIFE-THREATENING LITHIUM INTOXICATION WITH CONTINUOUS VENO-VENOUS HEMODIALYSIS.

Pali M, Albertson TE, Swenson E. *California Poison Control System—Sacramento Division, Sacramento, CA*

**Background:** Conventional dialysis has been used as a mainstay for severe lithium toxicity. Critically elevated lithium levels in patients who have acute or chronic renal failure present a clinical challenge in that conventional dialysis must be repeated several times or require longer sessions than the usual 4 hours. Continuous veno-venous hemodialysis/hemofiltration has been used in case reports with good efficacy. Houzard *et al.* described a lithium clearance rate of 28 mL/min using continuous veno-venous hemodiafiltration in a lithium intoxicated patient. **Case Report:** A 72-year-old comatose female with an acute intoxication presented with an initial lithium level of 35.7 mEq/L. The patient's initial creatinine was 3.7 mg/dL and she was anuric. Two 4-hour sessions of conventional dialysis resulted in rebound levels greater than >9.9 mEq/L. The patient remained comatose, flaccid and hypotensive. She was transferred to a tertiary care center where she was placed on continuous veno-venous hemodialysis (CVVHD) for 5 days. Her lithium levels while on CVVHD are shown in the table below. The patient was eventually discharged ambulatory.

Time on CVVHD	0 h	12 h	25 h	38 h	62 h	73 h	86 h
[Li] (mEq/L)	>9.9	7.6	5.8	4.34	2.56	1.73	1.61
Serum Cr	2.3	1.9	1.8	1.5	1.3	1.1	1.3

**Conclusion:** CVVHD use in a patient with a critically elevated lithium level and renal failure resulted in a good outcome.

### 174 UNUSUAL PRESENTATION OF IATROGENIC PHENYTOIN TOXICITY IN A NEWBORN.

Lowry JA, Vandover JC, DeGreeff JL, Scalzo AJ. *Cardinal Glennon Regional Poison Center, Saint Louis University School of Medicine, St. Louis, MO and Children's Mercy Hospital, Kansas City, MO*

**Background:** Medication errors may produce severe toxicity resulting in hospitalization. This can be compounded if the physician obtains the wrong concentration from a reference manual and a pharmacy miscalculates the conversion. We report a child presenting with ileus, hypothermia, and lethargy after receiving supratherapeutic dosing of phenytoin after a concentration miscalculation. **Case Report:** A 2-month-old infant presented to the ED with progressive worsening of abdominal distension, diminished activity, and 1 day Hx of difficulty feeding secondary to ↓ LOC. PMHx was significant for neonatal Group B Strep meningitis with seizures. Among the child's discharge medications was an Rx for phenytoin (30 mg/5 mL) 2.5 cc po tid. On ER exam, the child was hypothermic with pink mottled skin, poor responsiveness, prolonged capillary refill, abdominal distension with hypoactive bowel sounds, and dysconjugate gaze. Initial phenytoin level was 91.8 mcg/mL. She was admitted to the PICU and was started on ampicillin and cefotaxime for R/O sepsis. Phenytoin was withheld and subsequent levels revealed an extremely slow elimination (mcg/mL vs time pair coordinates were 78.2/13.3 h; 74.3/62.3 h; 43.7/109.6 h; 10.8/160.9 h) reflecting zero-order kinetics. Postdiscontinuing antibiotics, phenytoin levels ↓ at rates expected. She was discharged after resolution of symptoms. The MD who had written the phenytoin Rx had based it on the *Harriet Lane Handbook*, 2000 Ed. The 30 mg/5 mL formulation has been unavailable in the US for several years. A community pharmacy substituted the 125 mg/5 mL formulation, but miscalculated the dosage to be 1.6 cc (40 mg) tid. **Conclusions:** Abdominal distension and ileus may be presenting symptoms in children at toxic phenytoin levels. Ampicillin and cefotaxime may affect the elimination rate of phenytoin at such levels. We report one of the highest phenytoin levels recorded after therapeutic misadventure. Physicians must be aware of inaccuracies in reference manuals that may result in dosing errors.

**175 MULTICENTER CASE SERIES OF GABAPENTIN EXPOSURES.**

Hopkins U, Shepherd G, Klein-Schwartz W, Gorman S, Crouch B. *Maryland Poison Center, University of Maryland School of Pharmacy Baltimore, MD; North Texas Poison Center, Dallas, TX; Georgia Poison Center, Atlanta, GA; Utah Poison Control Center, Salt Lake City, UT*

**Background:** Gabapentin is a newer anticonvulsant that is being used for an increasing number of off label indications. There are no published case series of overdose. The purpose of this study is to document the clinical manifestations and outcomes of gabapentin overdoses. **Methods:** A multicenter prospective observational study of all exposures reported to three Poison Centers was conducted between 4/1/98 and 4/1/2000. **Results:** There were 72 cases of which 20 involved gabapentin alone. Nine of the exposures to gabapentin alone involved children and adolescents. Seven of the 20 gabapentin alone exposures were managed in the home with only observation. Four of these patients remained asymptomatic during the observation period. Effects reported in the 3 symptomatic patients were drowsiness (3) and ataxia (1). Thirteen of the gabapentin alone exposures were managed in a healthcare facility. Effects reported in the 9 symptomatic patients included drowsiness (6), dizziness (3), tachycardia (2), and hypotension (2). None of the patients exposed to gabapentin alone were admitted for medical treatment. No moderate effects, major effects, or deaths occurred. Fifty-two of the 72 cases collected involved the ingestion of gabapentin with another substance. Ten of these cases were patients under 19 years of age. Forty-nine were managed in a healthcare facility of which 14 were discharged directly from the emergency department, 17 were admitted to a critical care unit, 4 were admitted to a noncritical care unit, and 14 were admitted to psychiatry. No deaths occurred in this group. **Conclusion:** Gabapentin produces minimal toxicity when ingested alone. When gabapentin was ingested with another substance the effects seen were generally consistent with the coingestant.

**176 PHYSOSTIGMINE AWAKENS PATIENT FROM BACLOFEN SEIZURES.**

Dickinson E, Burkhart KK, Donovan JW. *York–Hershey Emergency Medicine Residency Program, Central Pennsylvania Poison Center, Pennsylvania State University, Hershey, PA*

**Background:** A few reports describe patients that have awakened from baclofen coma when given physostigmine. We describe the changes on electroencephalogram (EEG) of a patient who received physostigmine for baclofen toxicity. **Case Report:** A 27-year-old paraplegic female had been on a stable dose of 120 mg/d baclofen. After four days of Unasyn® the patient developed renal insufficiency (peak serum creatinine 3.3 mg/dL) and became baclofen toxic. Classic symptoms had evolved over two days including “tearing” abdominal pain and progressive lethargy into deep coma. During the following two days, the patient would intermittently have eye opening to deep pain, but otherwise remained flaccid. Periodic facial twitching was noted. An EEG documented subclinical seizure activity. Physostigmine, two mg, halted these seizures. The patient’s EEG became normal, as the patient awakened and followed commands. A physostigmine infusion was used to keep the patient sufficiently awake to protect her airway. Hemodialysis was performed for four hours. One hour into the hemodialysis the patient became fully awake and the physostigmine was discontinued. The baclofen level before hemodialysis was 15 mg/L (therapeutic 0.2–1.2 mg/L). After hemodialysis it became undetectable. **Conclusions:** Baclofen coma may include seizure activity that is reversible with physostigmine. Physostigmine infusions may maintain airway patency. Baclofen is excreted unchanged in the urine. Coma may be prolonged when renal insufficiency develops. Hemodialysis therefore may enhance recovery.

**177 SEVERE TOXICITY DUE TO 4-AMINOPYRIDINE INGESTION IN A PEDIATRIC PATIENT.**

Velez LI, Shirazi F, Goto CS, Shepherd JG, Roth B. *The University of Texas Southwestern Medical Center and the North Texas Poison Center, Dallas, TX*

**Background:** Four aminopyridine (4-AP) is a potassium channel blocker used to improve motor strength in demyelinating diseases such as multiple sclerosis. We describe a case of a single tablet ingestion in an 8-month-old child that resulted in severe but transient symptoms. **Case Report:** An 8-month-old male was found with greenish saliva. His grandmother has multiple sclerosis, and one of her green 4-AP tablets was missing. Upon arrival to an outlying hospital, he was jittery, tachycardic, and tachypneic. He was decontaminated with activated charcoal and cathartic (1 g/kg by NGT) and given midazolam (0.5 mg/kg IV). He was then transported to our Emergency Department, where he was jittery, tachycardic, tachypneic, with upward gaze deviation, and hyperreflexia. A wide pulse pressure was treated with 0.9NS at 20 cc/kg IV. He then progressed to opisthotonic posturing with tongue fasciculations. This was treated with two doses of lorazepam (0.05 mg/kg IV). After transfer to the intensive care unit, an EEG was performed; it was negative for seizure activity. The patient was completely asymptomatic 20 hours after the ingestion. **Conclusion:** This case is

the first documented pediatric 4-AP ingestion. Our patient had severe but transient symptoms, which were managed with supportive care. One of the well-documented toxicities of 4-AP is convulsions. It is interesting that the EEG performed while the patient was symptomatic was negative for seizures. Other adverse effects include extrapyramidal reactions and tremors. Our patient responded well to benzodiazepines, which have been utilized previously in the management of 4-AP poisoning.

### **178 PROLONGED OCTREOTIDE INFUSION TO TREAT GLYBURIDE-INDUCED HYPOGLYCEMIA.**

Bui L, Adler D, Keller KH. *California Poison Control System, San Francisco, CA*

**Background:** Octreotide, a synthetic somatostatin analogue that inhibits insulin secretion from pancreatic beta cells, has demonstrated efficacy in sustaining blood glucose in experimental and case reports of sulfonylurea overdoses. We report a case of glyburide overdose requiring six days of octreotide infusion, the longest reported use of constant octreotide infusion in the treatment of sulfonylurea overdose. **Case Report:** A 23-year-old diabetic female ingested fifty 5 mg tablets of glyburide. On paramedic evaluation 30 minutes after ingestion, she had normal mentation with a D-stick of 73 mg/dL. She was given 25 g of intravenous D50W. In the ED, her glucose level was 87 mg/dL. A second dose of D50W was given and a D5W IV drip was started at 150 cc/h. Oral gastric lavage was performed, returning pill fragments, and 50 g of activated charcoal was given. Over the next two hours, despite increasing the dextrose infusion to a D10W drip at 200 cc/h, she developed symptomatic hypoglycemia with blood glucose levels in the 30s requiring an additional 50 gm of D50W. Three hours after the ingestion, intravenous octreotide was started at 50 mcg/h. At 8 hours, the octreotide drip was increased to 100 mcg/h to maintain the blood glucose near 100 mg/dL. Once octreotide was instituted, no additional D50W boluses were required. The octreotide infusion was reduced on Day 5 and discontinued on Day 7, and the patient was discharged on Hospital Day 8. **Conclusion:** Octreotide, as an adjunct to IV glucose, helped maintain a stable blood glucose level. Although an optimal dose regimen has yet to be established, octreotide should be considered in the therapy of sulfonylurea overdose where refractory hypoglycemia is encountered.

### **179 MASSIVE STEROID OVERDOSE PRECIPITATING DIABETIC KETOACIDOSIS IN A NONDIABETIC ADULT.**

Lee DC, Rudolph GR. *North Shore Hospital, Manhasset, NY*

**Background:** Life-threatening sequelae to an acute steroid overdose in a healthy adult is extremely rare. Although diabetic ketoacidosis (DKA) has been reported as an adverse effect with chronic administration in patients with diabetes and/or pregnancy, this has not been reported in an acute setting in a relatively healthy patient. We present a case report of massive iatrogenic methylprednisolone overdose inducing DKA nondiabetic patient. **Case Report:** A previously healthy 44-year-old male without significant medical history presented with abdominal and back pain, and difficulty walking after a fall. On examination, the patient had a Glasgow Coma Scale of 15 with normal vital signs. The patient was treated for a presumed spinal cord injury with high dose methylprednisolone. However 50.4 mg/kg (30 grams) was administered intravenously rather than the correct dose of 5.4 mg/kg. Within 24 hours, the patient became lethargic and developed DKA requiring continuous insulin and bicarbonate infusions. Within 24 hours, significant lab results included: Na<sup>+</sup> 141 mEq/L, K<sup>+</sup> 3.1 mEq/L, Cl<sup>-</sup> 105 mEq/L, HCO<sub>3</sub><sup>-</sup> 10 mEq/L, BUN 21 mg/dL, Cr 1.6 mg/dL, Glu 650 mg/dL, ABG (room air) pH 7.16, pCO<sub>2</sub> 22, pO<sub>2</sub> 117, urinalysis was positive for ketones. The patient's trauma work-up included normal CT scans of the head, spine, and abdomen. MRI of the spine revealed multiple disc herniations and paraspinal muscle swelling. There was no evidence of abdominal injury and no surgical intervention was required. The patient was discharged in 8 days in good condition but was lost to further follow-up. **Conclusion:** An acute massive steroid overdose can cause life-threatening reactions (DKA) in a healthy adult.

### **180 HYPERINSULIN THERAPY IN THE TREATMENT OF VERAPAMIL OVERDOSE.**

Place R, Carlson A, Leiken J, Hanashiro P. *Rush-Presbyterian-St. Luke's Medical Center, Toxikon, Chicago, IL*

**Background:** Hyperinsulinemia-euglycemia (HIE) has been demonstrated to be useful in the treatment of calcium antagonist (CA) overdose. We present a case of CA toxicity in which hemodynamic data was obtained, and in which the

patient responded to a higher dose of insulin than previously reported. **Case Report:** A 49-year-old male (100 kg) ingested 80 tablets of 240 mg Verapamil SR. At presentation he was alert but hypotensive, requiring dopamine. At hour 18 he developed 3° AV block, which required transvenous pacing. Serum verapamil level was 4620 ng/mL (norverapamil 2180 ng/mL). Cardiogenic shock worsened despite therapy with calcium chloride (4 g), glucagon (15 mg), and norepinephrine (20 µg/kg/min). HIE therapy with 1 U/kg of insulin and continuous glucose was planned. Inadvertently, 1000 U of insulin was administered. Rapid hemodynamic improvement ensued: Hemodynamic data (Swan-Ganz)

	Pretreatment	15 min	30 min	45 min	60 min	9 h
CO	3.75	5.73	6.42	7.01	7.40	6.44
SV	42.1	64.4	72.1	78.8	83.1	72.4
SVR	789	558	598	536	519	819
MAP	58	61	69	69	71	86

Within 5 hours after the insulin bolus, all vasoactive medications were withdrawn. The patient never developed hypoglycemia. **Conclusion:** HIE therapy rapidly improves hemodynamic parameters in severe CA toxicity. Insulin doses approaching 10 U/kg appear to be both safe and efficacious.

### 181 INSULIN "EUGLYCEMIA" THERAPY FOR ACCIDENTAL NIFEDIPINE OVERDOSE.

Morris-Kukoski CL, Biswas AK, Parra M, Smith C. *Naval Medical Center Portsmouth, Portsmouth, VA*

**Background:** Hypodynamic shock is a common manifestation of calcium channel blocker toxicity. Reported treatment regimens have involved the use of IV fluids, calcium, glucagon, catecholamines, atropine/pacing, and phosphodiesterase inhibitors. Insulin "euglycemia" therapy has been documented and reported as an effective adjunct to conventional treatment. **Case Report:** A 5-month-old (5.5 kg), with pulmonary HTN s/p surgical closure PDA, was started on 1 mg nifedipine per ng tube for afterload reduction. Inadvertently, 2 nifedipine 10 mg capsules were given. Twenty minutes later, the patient became profoundly hypotensive, required hand bagged ventilatory assistance with sedation, and vasopressors and inotropes. Treatment over the next several hours consisted of Ca chloride (stopped when ionized Ca 2.91 mmol/L), glucagon, dopamine, epinephrine, phenylephrine, and milrinone. Despite these therapies at or beyond maximum doses, systolic BP was in the 50s (arterial pH 7.05). High dose insulin therapy (max 1 U/kg/h) with D50 was started (initial glucose 246 mg/dL). Subsequently, systolic BPs sustained in the 80s. Insulin therapy allowed cessation of glucagon within ½ hour and phenylephrine within 2 hours (glucose 296 mg/dL, arterial pH 7.24). Prolonged hypotension led to anuric renal failure. As systolic BP rose, epinephrine, dopamine, and milrinone were tapered off within 72 hours, 90 hours, and 90 hours respectively. Insulin infusion was tapered off within 96 hours (mean glucose 126 mg/dL, arterial pH 7.36). Complications (renal and respiratory failure) from nifedipine-induced hypodynamic shock resolved within 30 days. **Conclusion:** Insulin "euglycemia" therapy in this patient helped restore and maintain efficient myocardial metabolism and performance as evidence by resolution of hypotension, acidosis, and renal and respiratory failure, with return to native euglycemic state.

### 182 BETA BLOCKER INGESTIONS IN CHILDREN.

Belson MG, Geller RJ. *Georgia Poison Center and Emory University, Department of Pediatrics, Atlanta, GA*

**Background:** Limited data exists regarding toxicity of beta blockers (BB) in children. We sought to determine the range of toxicity in accidental pediatric BB ingestions. **Case Series:** A 7-year retrospective review of acute BB ingestions in children under 7 years of age at one regional poison center, excluding patients with coingestants with recognized cardiovascular or CNS effects. 378/411 patients met criteria; mean age 27 months, 56% male. Metoprolol (28%), atenolol (27%), and propranolol (25%) were most commonly ingested. 80% of ingestions involved ≤1 pill. Symptoms occurred in 2%: 2/378 with lethargy and 6/378 with bradycardia and/or ↓BP. 7/8 symptomatic patients involved regular-release (reg.) preparations (median time to onset of symptoms = 3 hours, range 1-3.5 hours). The range of toxicity in 299/378 (79%) patients in whom a specific amount of drug was reportedly ingested is summarized in the following table:

Beta Blocker	N	Smallest Toxic Dose	Largest Nontoxic Dose	Median Nontoxic Dose
Propranolol reg	60	5.0 mg/kg	15.4 mg/kg	2.1 mg/kg
Propranolol SR	16	12.0 mg/kg	13.0 mg/kg	3.6 mg/kg
Metoprolol reg	61	no symptoms	11.5 mg/kg	3.1 mg/kg
Metoprolol SR	22	no symptoms	18.0 mg/kg	4.1 mg/kg
Atenolol	76	5.3 mg/kg	16.7 mg/kg	3.6 mg/kg
Other BBs	64	—	—	—

41% of patients were managed at home, 43% were evaluated in an ED then discharged. The median observation time in a hospital was 4 (reg.) and 12 hours (SR). Outcome: 72% of patients had no clinical effect, 2% minor effect, and 0.5% moderate effect. Conclusions: Beta blockers appear to have a wide margin of safety in young children, as the majority of these exposures are asymptomatic. These data suggest potential triage values for acute pediatric BB ingestions.

### 183 SYSTEMIC ALPHA-2 POISONING WITH THERAPEUTIC USE OF ALPHAGAN® EYE DROPS IN AN INFANT.

Rangan C, Ingels M, Clark RF. *California Poison Control System, University of California at San Diego, San Diego, CA*

Background: Brimonidine eye drops are imidazoline derivatives chemically related to clonidine and oxymetazoline. We report the first case of systemic alpha-2 poisoning from therapeutic use of these eye drops. Case Report: A 2½-month-old male infant with Goldenhar's Syndrome and a surgical history of corneal transplants was seen in the Ophthalmology Clinic for a routine visit. He was feeding and growing normally with a normal echocardiogram, brain CT, and brain MRI. In the clinic he was administered his first-ever dose of Alphagan 0.2% (brimonidine, Allergan Inc.), one drop into each eye. Soon thereafter he became acutely pale and lethargic, with shallow, infrequent respirations. He was administered 100% oxygen by facemask and transferred to the Emergency Department. Vital signs revealed temperature 96.3°F, pulse 126, blood pressure 100/41, and respiratory rate 8. He was somnolent with shallow respirations when left alone, but continually regained a normal mental status and respirations >25 after tactile stimulation. The rest of his physical exam was unremarkable. Serum electrolytes, CBC, and urine toxicology screen were within normal limits. He was admitted to a critical care unit, where he continued to have intermittent lethargy with shallow respirations that were easily reversed by tactile stimulation. After 6 hours he regained his baseline mental status with a normal respiratory pattern. He was discharged in stable condition. Conclusion: This case is similar to previous reports of pediatric ingestion of alpha-2 agonists like clonidine. Alpha-2 eye drops have not been tested in children, and physicians should be aware that therapeutic use could lead to severe systemic intoxication.

### 184 SEIZURES, RESPIRATORY ARREST FOLLOWING TOPICAL APPLICATION OF EMLA CREAM.

Hiotis M, Doyon S. *Maryland Poison Center, University of Maryland at Baltimore, Baltimore, MD*

Background: Emla cream (a eutectic mixture of lidocaine and prilocaine) is a local anesthetic that is considered safe and effective. We report the first case of EMLA-induced seizures, coma, and respiratory arrest following topical application. Case Report: A 29-year-old female applied EMLA Cream on her abdomen, buttocks and thighs in preparation of a cosmetic dermatological procedure. Within 1 hour she developed generalized tonic clonic seizures and respiratory arrest. Seizures were treated with benzodiazepines and phenobarbital and she required endotracheal intubation and mechanical ventilation. Her lidocaine level was 8 mcg/mL 4 hours after the application of the cream. Her skin was reported to be erythematous and peeling as a result of Retin-A cream that she used daily for 4 weeks prior to application of EMLA. Conclusion: Local anesthetics are potentially toxic when used inappropriately. The size of the surface area, the integrity of the skin, the use of occlusive dressings, and hepatic function are some of the factors that should be assessed before they are used.



**Platform Session 5****Monday, September 18  
Abstracts #185–#190****10:30 am–12:00 pm****185 HEPATOCELLULAR APOPTOSIS IN A PATIENT EXPOSED TO *LEPIOTA SP.***

Brouxhon S, Ryan C, Wax P, Schneider S. *Department of Emergency Medicine, University of Rochester School of Medicine & Dentistry, Rochester, NY*

**Background:** Apoptosis represents a genetically programmed and highly selective mechanism of cell death that eliminates redundant or excessively damaged cells in response to a wide array of stimuli (viruses, radiation, toxins, etc.). Previous data have shown that amatoxins induce structural changes in liver and kidney cell nuclei by binding RNA polymerase II, thereby inhibiting DNA transcription and subsequent protein synthesis. The purpose of the present study was to investigate whether apoptosis played a role in the acute fulminant hepatic failure that developed in a patient following amatoxin (*Lepiota sp.*) ingestion. **Methods:** Surgically excised liver was formalin-fixed, paraffin-embedded, cut into 4  $\mu\text{m}$  thick sections and mounted onto glass slides. Furthermore, formalin-fixed, paraffin-embedded liver and thymus sections from healthy patients were included in the study and served as a control. Nucleosomal DNA fragmentation was assessed with the Hoescht stain and the fragmented DNA of apoptotic cells was measured by using the terminal transferase deoxynucleotide end labeling (TUNEL) assay. **Results:** Our results demonstrated a dramatically increased number of cells positive for both the Hoechst stain and the TUNEL assay ( $p < 0.001$ ) in the amatoxin exposed liver relative to control as assessed by the unpaired student *t*-test, indicating that a large number of amatoxin exposed cells underwent apoptosis. **Conclusion:** The data presented here suggest that amatoxin induced hepatotoxicity is largely mediated by apoptosis. The exact apoptotic pathway mediating this hepatotoxicity remains to be elucidated, however if it involves caspase activation, caspase-based therapeutic strategies may be useful for the management of these patients in the acute setting.

**186 ACUTE ACONITE POISONINGS: A REVIEW OF 219 CASES.**

Birtanov Y, Toibaeva G, Birtanov A. *Republican Toxicology Center (RTC), City Emergency Medicine Hospital, Almaty, Kazakstan*

**Background:** Aconite (common name for *Aconitum napellus*) is one of the well-known toxic plants since antiquity. There are 14 species of aconite growing in Kazakstan where it is still popular as a part of traditional herbal medicine. Therefore it often becomes a reason for severe accidental and suicidal poisonings. During the last 5 years (1995–1999) 278 patients were hospitalized with acute aconite poisoning and 36 of them died (12.9%). The objective of our study was to characterize the clinical presentation of acute aconite poisonings in our region. **Methods:** A retrospective case review of 219 hospitalized patients including 8 fatalities was performed. **Results:** There were 53 (24.2%) suicidal and 166 (75.8%) accidental poisonings. In 101 (46.1%) cases symptoms appeared within first 15–20 minutes after ingestion, in 39 (17.8%) cases within 20–60 minutes, in 29 (13.2%) cases after 1 hour. Major symptoms of poisoning included: nausea (80.8%), vomiting (62.%), sensory disturbances (84.0%), agitation (51.1%), coma (14.1%), respiratory arrest (16.9%), cardiac disturbances (85.6%). Cardiac disturbances included: bradycardia (6.8%), tachycardia (11.9%), extrasystole (73.1%), fibrillation (7.8%) and arterial hypotension (51.1%). In 199 (90.9%) cases aconite alkaloids were detected in patients urine. Treatment included gastric decontamination, forced diuresis and supportive treatment. Lidocaine was used routinely to reverse cardiac arrhythmias. In 26 (11.9%) cases cardiopulmonary resuscitation was performed. **Conclusion:** Acute aconite poisoning has potentially dangerous clinical features. Further investigations are necessary to define an effective treatment protocols including a search for specific antidotes for major aconite alkaloids.

**187 COMPLICATIONS AND PROGNOSIS OF FACTORS OF ACETIC ACID POISONINGS.**

Sarmanaev SKh. *Toxicological Center, Hospital, Ufa, Bashkortostan, Russia*

**Objective:** Acute Poisoning by Acetic Acid (APAA) by adults is a frequently emerging problem in Russian toxicological practice. The aim of the investigation is to disclose the character of consequences of APAA, leading to fatal outcome so as to enable prognosis of such cases. **Methods:** Out of 9448 medical cards of hospitalized patients at the toxicological

center in Ufa, we collected data from all consecutive cases of APAA between 1995–1999 for a retrospective analysis. This data base was analyzed with regard to initial symptoms and consequent complications resulting in fatal outcomes. **Results:** There were 268 cases of APAA (2.8% of the hospitalized patients) included in the study, 26 of these being *exitus letalis* (10.1% of all fatal cases). The following table summarizes the parameters that were investigated:

	Successful Outcome	Deaths
Mean age $\pm$ SD	45.09 $\pm$ 2.04 years	56.69 $\pm$ 3.18 years (p < 0.01)
Mean dose $\pm$ SD	45.21 $\pm$ 1.20 mL	96.15 $\pm$ 12.63 mL (p < 0.01)
Mean time lag $\pm$ SD	65.21 $\pm$ 0.81 min	93.40 $\pm$ 15.15 min (p < 0.05)
Intentional	42.8%	88.5%
Gastrointestinal hemorrhage	3.3%	84.61%
Acute kidney failure (AKF)	15.70%	84.61%
Leukocytes (number/ $\mu$ L)	11,360 $\pm$ 480	18,700 $\pm$ 1,970 (p < 0.05)
Sample size	242	26

**Conclusions:** Such factors as age, dose, time lag, intentional poisoning, presence of gastrointestinal hemorrhage, AKF, and leukocytosis can be used as prognostic criteria of determining outcome in cases of APAA.

#### 188 MALIGNANT HYPERTHERMIA: EXPERIENCE WITH HOTLINE CASES.

Caliva M, Stork CM, Sopchak C, Cantor R. *Central New York City Regional Poison Center, Department of Emergency Medicine, University Hospital, Upstate Medical University, Syracuse, NY*

**Background:** Malignant Hyperthermia (MH) is a rare hypermetabolic disorder that occurs in genetically predisposed individuals receiving anesthetic triggers. We examined the incidence of common MH clinical findings to determine if any correlated with a diagnosis of MH. **Methods:** This was a retrospective review of 617 MH cases reported to a regional poison center from 1/1/97 to 12/31/99. Only patient care cases were included. The consulting MH expert diagnosis of MH or not MH along with the incidence of the following findings were extracted from the case record; muscle rigidity, masseter rigidity (MR), hyperthermia, tachycardia, increased end tidal CO<sub>2</sub>, metabolic acidosis, creatinine kinase (CK) elevations and myoglobinuria. The incidence of these findings among MH and not MH cases were compared using Chi-square analysis. **Results:** 617 calls meet inclusion criteria. 133 calls were MH and 484 not MH. The following findings were found in MH: not MH calls respectively; Muscle rigidity: 23%/9% (p < .35), MR: 8%/7% (p < .47), Hyperthermia: 68%/82% (p < .0006), Tachycardia: 44%/49% (p < .35), Increased end tidal CO<sub>2</sub>: 54%/35% (p < .00005), metabolic acidosis: 44%/30% (p < .0035), CK elevation: 14%/17% (p < .43), myoglobinuria: 8%/9% (p < .82). History of a trigger was 73% versus 12% of MH and not MH cases. (p < .00015) There was no difference in the number of presenting symptoms between the MH and not MH group (2.9%: 3%) **Conclusion:** Findings of hyperthermia, increased end tidal CO<sub>2</sub>, acidosis, and history of a trigger are significantly more often found in patients diagnosed with MH. The number of presenting symptoms and other historical MH symptoms did not influence the diagnosis of MH. A prospective trial with diagnostic accuracy is warranted to confirm these findings.

#### 189 REPEATED SUPRATHERAPEUTIC DOSING OF ACETAMINOPHEN (APAP): CAN SERUM TRANSAMINASE LEVELS PREDICT THE RISK OF HEPATOTOXICITY?

Daly FFS, Dart RC, Bogdan GM, Jolliff HA, Waksman JC. *Rocky Mountain Poison & Drug Center—Denver Health, University of Colorado Health Sciences Center, Denver, CO*

**Objective:** Repeated suprathreshold dosing accounts for 9% of APAP-related deaths and has not been studied systematically. We hypothesize that AST/ALT at presentation identifies patients at risk for APAP-induced hepatotoxicity follow-

ing repeated supratherapeutic dosing. **Methods:** A prospective cohort study of poison center patients presenting with repeated supratherapeutic APAP dosing (ingestion of >4 g/24 h; pediatric >90 mg/kg) was performed using structured data collection. Patients were managed by standing poison center protocol (presentation with abnormal AST/ALT or APAP level >10 mcg/mL prompted treatment with *N*-acetylcysteine-NAC) and followed for 48 hours, or until resolution of symptoms and laboratory abnormalities. **Results:** 44,689 calls were screened; 2,335 were APAP exposures. 114 cases (5%) were repeated supratherapeutic ingestions and 92 consented to enrollment.

AST/ALT at Presentation	n (%)	Median APAP (g/d)	Hepatotoxicity (AST > 1000 IU/L)	Lost to Follow Up
Normal AST/ALT	41 (44.6)	10	0 (15 treated with NAC)	7
AST/ALT >50 IU/L	40 (43.5)	12	13 (4 deaths, 1 transplant)	2
No laboratory data	11 (11.9)	7.5	0 (3 well at follow-up)	8

The likelihood ratio for patients with normal AST/ALT at presentation developing hepatotoxicity was zero. This study is limited by small numbers, administration of NAC to patients with normal AST, and 18% lost to follow-up rate. **Conclusion:** Patients who present after repeated supratherapeutic APAP ingestions with normal AST/ALT may not need treatment with NAC. The role of an APAP level in directing treatment and predicting outcome is not clear.

#### 190 OBSERVATION UNIT EVALUATION OF LOW-RISK DRUG INDUCED CHEST PAIN.

Tijunelis M, Leikin J, Hanashiro P, Timmons J, Kissane K. *Rush-Presbyterian-St. Luke's Medical Center, Toxikon, Chicago, IL*

**Introduction:** Drug related chest pain (DRCP) is one of the most common presentations of drug abuse to an ED. We present our experience of evaluation of DRCP to the ED based observation unit (ED-OBS) in patients with chest pain under the age of 41 years. **Methods:** 18 month (10/1/98 to 3/30/00) retrospective chart audit of all low risk chest pain patients admitted to ED-OBS was performed. Data collection focused upon risk factors, drug screens, acute history of drug abuse, and cardiac event. All patients received serial EKG, cardiac enzymes, and ECHO; when these tests were negative, a provocative stress test was performed. **Results:** Of the 670 total patients admitted into the ED-OBS with chest pain, 122 (18%) were under 41 years old with 17 of these patients admitted as inpatients (14% admission rate). 32 patients (26%) were DRCP; of these 6 patients (19%) were admitted but none had an acute cardiac ischemic event. Three of the eleven patients who were admitted but did not exhibit DRCP had an acute cardiac ischemic event. 21 of the 32 DRCP patients were related to cocaine abuse, 9 utilized marijuana alone, 1 heroin, 1 anabolic steroids. **Conclusion:** An ED-OBS unit can distinguish a majority of DRCP patients without requiring inpatient admission. No acute cardiac events were documented in DRCP patients. It appears that DRCP patients have a low likelihood of the need for hospital inpatient admission. Young DRCP patients who do not exhibit objective evidence of acute cardiac ischemia on E.D. presentation may represent a lower cardiac risk than previously recognized.

#### 191 USE OF 4-METHYLPYRAZOLE (4-MP) AS AN ANTIDOTE FOR 1,4-BUTANEDIOL (BD) TOXICITY IN CD-1 MICE.\*

Quang L, Maher T, Shannon M, Woolf A. *Children's Hospital, Massachusetts/Rhode Island Poison Control System, Harvard Medical School, Massachusetts College of Pharmacy and Allied Health Sciences, Boston, MA*

**Objective:** BD is a prodrug that is biotransformed by alcohol dehydrogenase to gamma-hydroxybutyrate (GHB). This study examined if 4-MP can be used as an antidote for BD toxicity. **Methods:** Male CD-1 mice were given i.p. injections of BD at 600 mg/kg (TD<sub>50</sub>) and divided into 3 groups. Group 1 received i.p. 4-MP at 25 mg/kg or deionized, distilled water (controls) 1 minute after BD. Group 2 received the same interventions 5 minutes after BD. Group 3 received the same interventions at the time they failed the roto-rod (RoRo) test after BD. Toxicity was assessed by the righting reflex and roto-rod test at 10-minute intervals up to 3 hours. **Results:**

	Time After 1,4-Butanediol I.P. Injection		
	Group 1 (1 min)	Group 2 (5 min)	Group 3 (failed RoRo)
Righting Reflex			
4-MP Group	10/10	10/10	10/10
Control Group	8/10	8/10	7/10
p-value	NS	NS	NS
Roto-rod Test			
4-MP Group	10/10	10/10	9/10
Control Group	1/10	1/10	1/10
p-value	<.001	<.001	<.001

**Conclusion:** 4-MP confers an antidotal effect on CD-1 mice for the roto-rod test when given at 1 and 5 minutes postBD, and at onset of BD toxicity, presumably by blocking biotransformation of BD to GHB.

**\*Winner of the American Academy of Clinical Toxicology 1999 Research Award.**